the racehorse
a veterinary manual

second edition

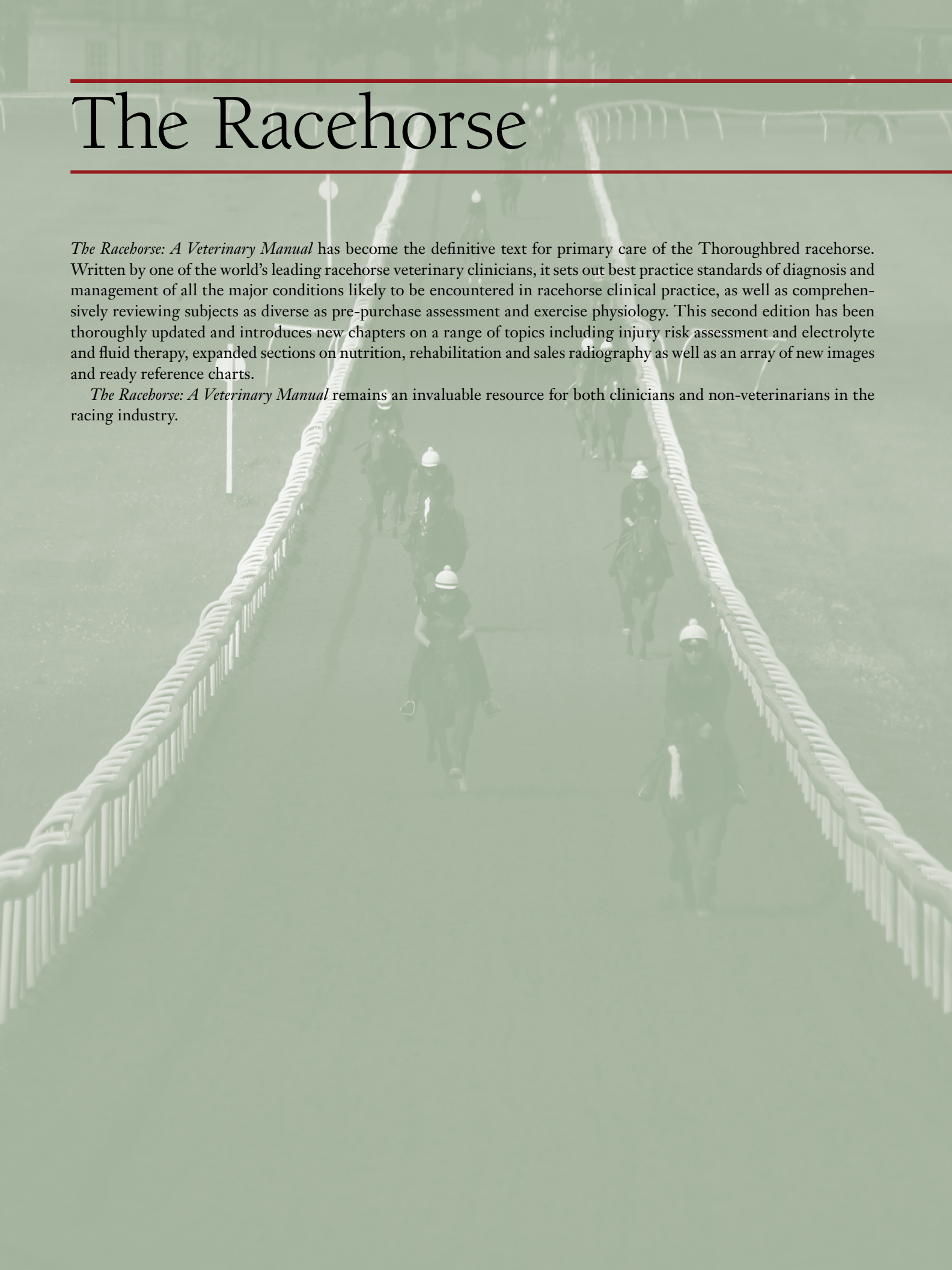
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The Racehorse

The Racehorse: A Veterinary Manual has become the definitive text for primary care of the Thoroughbred racehorse. Written by one of the world's leading racehorse veterinary clinicians, it sets out best practice standards of diagnosis and management of all the major conditions likely to be encountered in racehorse clinical practice, as well as comprehensively reviewing subjects as diverse as pre-purchase assessment and exercise physiology. This second edition has been thoroughly updated and introduces new chapters on a range of topics including injury risk assessment and electrolyte and fluid therapy, expanded sections on nutrition, rehabilitation and sales radiography as well as an array of new images and ready reference charts.

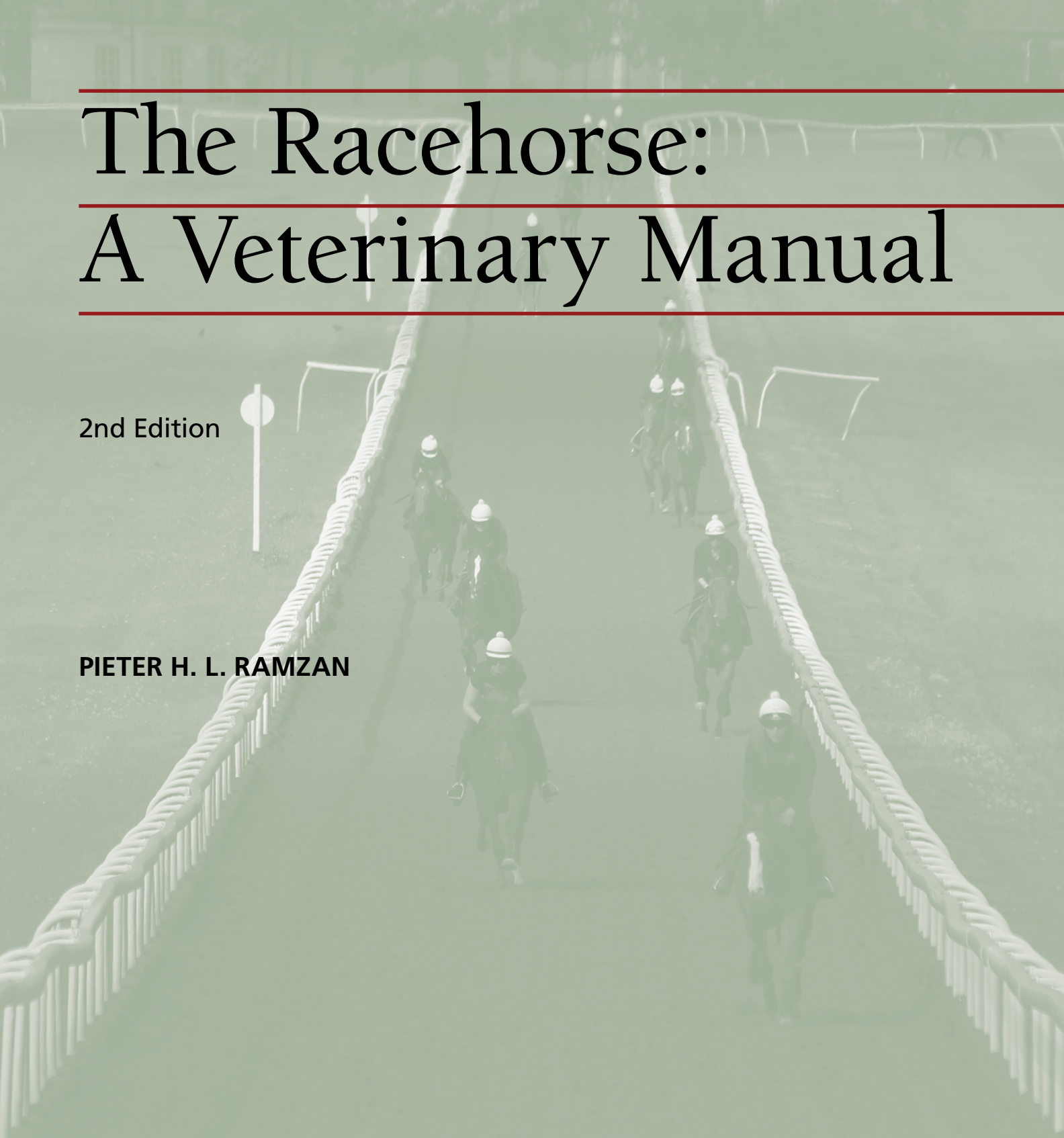
The Racehorse: A Veterinary Manual remains an invaluable resource for both clinicians and non-veterinarians in the racing industry.



"*The Racehorse: A Veterinary Manual* is a must-have book for anyone involved with a racehorse. I would encourage all new graduates as well as veteran practitioners to read this book cover to cover. After nearly two decades as a racetrack veterinarian, I continue to reference it frequently. The additions in this second edition elevate an already outstanding resource."

Ryan Carpenter, DVM, MS, DACVS, Equine Medical Center, USA

The Racehorse: A Veterinary Manual



2nd Edition

PIETER H. L. RAMZAN

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PREFACE

In the world of textbook publishing I expect that it is a relatively common phenomenon for authors, from the moment a manuscript is submitted, to simultaneously vow never to take on such a project again, and start planning how to do things better next time around. *The Racehorse* is no exception, and I hope that in presenting this second edition I have succeeded in both updating and improving it while maintaining the central goal of producing a work that distils the specialism of racehorse sports medicine in a manner that is useful to both veterinary clinicians and the wider racing community.

In compiling this edition I have once again drawn upon the deep knowledge and experience of colleagues from both within Rossdales and amongst the wider racing and sales vet community in the UK, Ireland and further abroad; I feel lucky to share this professional road with Tom O'Keeffe, Ian Cameron, Mike Shepherd, John Hanly, Patrick Sells, Jamie O'Gorman and Peter Hynes among many others. In particular I must acknowledge Mathieu Spriet, Ryan Carpenter and David Beylin, whose seminal work with standing PET is producing the biggest leap forward in our understanding of fetlock pathologies since the advent of standing MRI, with tangible effects on race day safety. Mathieu has provided PET images for this publication for which I am grateful, and I also acknowledge the contributions of Tim Barnett, Sarah Powell, Celia Marr, Emily Floyd, Mariana Castro Martins, Vanessa Peter, Claire Wylie, Tim Parkin, Fil De Oliveira, Matthew Briggs for his outstanding illustrations, and Alice Oven for editorial support. On a personal level I wish to thank Alberto Baragiola, Colin Planas, Sam Goldsmith, Gary Smith, Conor Norris, Rob Moore, Mary Black, Lorraine Palmer and Alvise Pasinato; and above all my wife Sarah and son Floris for their unwavering support and tolerance.

Since the publication of the first edition my clinical work has intersected with a long-term passion, a rarity in busy professional life. In transitioning from follower of the Palio of Siena to working within it I owe a great debt of gratitude in particular to Fabio Miraldi, Bernardo Bandini and Giuseppe Incastrone. There can be no other city in the world in which the horse and racing are so integral (and beneficial) to the fabric of society, and to be even a small part of this rich tapestry has been an enormous privilege and the high point of my career. I thank Emiliano Cioni, Tommaso Giuntini and Riccardo Vegni for cherished moments shared in the stalla, as well as Gian Piero Cervellera, Paolo Marucelli, Filippo Rossi, Jacopo Gotti, Francesco Mugnaini, Francesco Gerardi and the people of Camporegio. Also I am grateful to those in the wider community of Siena who have shown my family and me such hospitality and generosity over the years, particularly Susi Meiattini, Viola Carignani and the late Andrea Mari and his family.

Finally, I acknowledge the passing of Dr Peter Rossdale since the first edition was published. The ethos that Peter instilled in his colleagues of practice-based research continues to this day; indeed in reviewing the wider literature for this book the overwhelming sense is of how little of the science is settled and how much scope there is for enhancing our veterinary management of the racehorse. Whether it is poulticing feet or arthroscopic surgery, much of what we do and advise as equine clinicians has received very little rigorous scientific critique, and the published evidence base is embarrassingly limited when compared with human medicine. As Thoroughbred clinicians we frequently (particularly in the context of pre-purchase examinations) make judgements that can have far-reaching impacts not just for horses under our care but for the wide network of people whose livelihoods depend on them. It is incumbent on all of us to strive to base those decisions on the best available evidence, to have sufficient self-awareness to recognise the limitations of current scientific understanding, and to remain open-minded to better ways of doing things in future. If one can draw any conclusion from this publication, it is that despite the wealth of accumulated veterinary knowledge concerning the racehorse that exists there are limitless opportunities for grassroots research to challenge those same received wisdoms and push the science ever forward.

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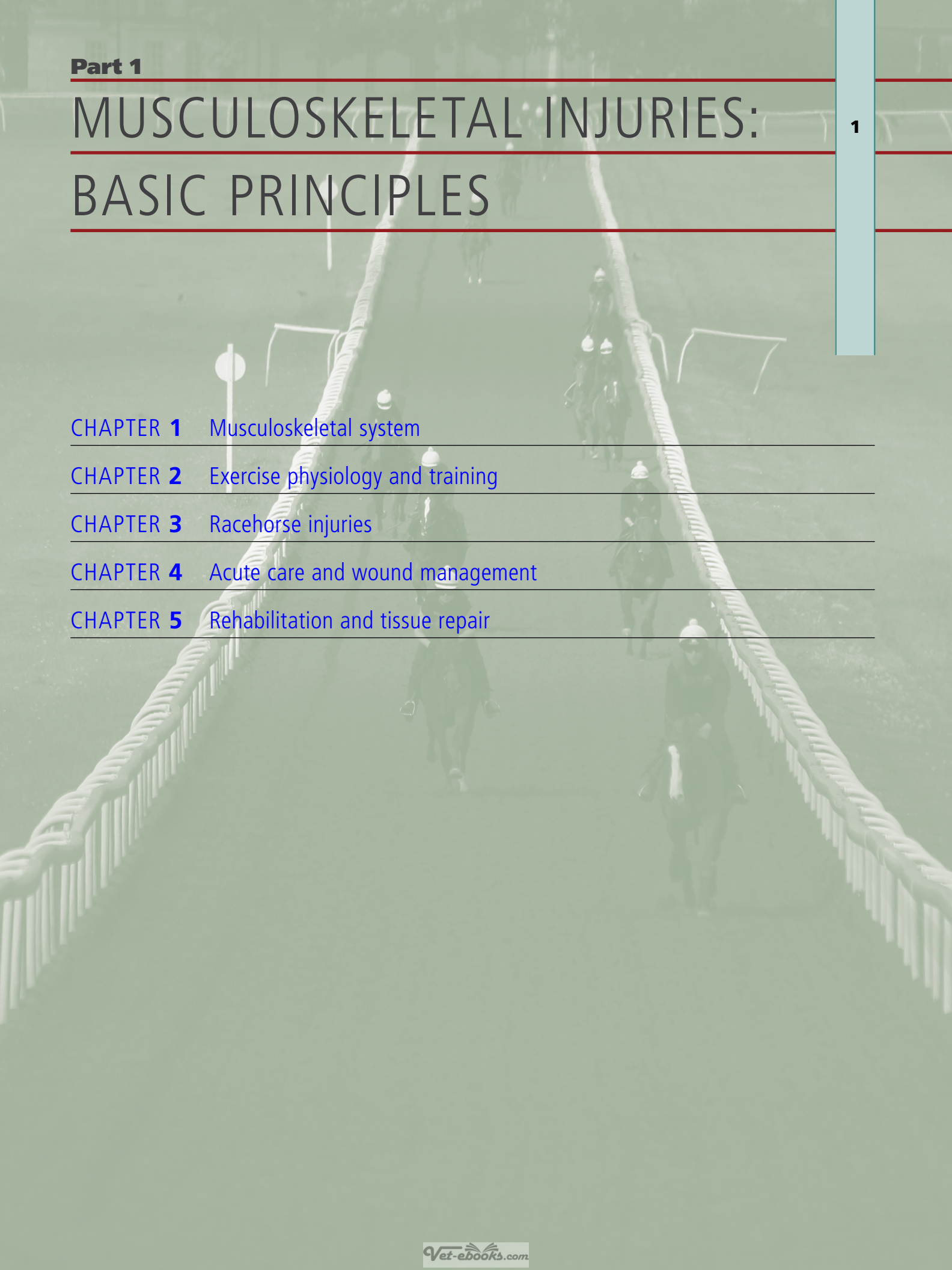
AUTHOR BIOGRAPHY

Piet Ramzan is one of the leading veterinary clinicians of his generation working in Thoroughbred practice. An upbringing riding cutting horses in the Hunter Valley led to graduation from the University of Sydney Veterinary School in 1994, and following a season working as a stockman in the Kimberley region of Northern Australia he moved to the UK and entered veterinary practice. In 1998 he joined Rossdale and Partners, Europe's largest equine practice, and commenced a distinguished career that encompasses first-opinion work in Newmarket racing yards and pre-purchase consultancy across the Northern and Southern hemispheres. He has a strong interest in advancing the science and practice of racehorse sports medicine and has drawn on busy clinical practice to inform academic research and drive innovation in many fields, publishing a large number of peer-reviewed papers and regularly contributing to scientific symposia and industry advisory boards. He is a previous recipient of the BEVA Richard Hartley Clinical Award and in 2018 was awarded Fellowship of the Royal College of Veterinary Surgeons (RCVS) for Meritorious Contributions to Clinical Practice.

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Part 1

MUSCULOSKELETAL INJURIES: BASIC PRINCIPLES



CHAPTER 1 Musculoskeletal system

CHAPTER 2 Exercise physiology and training

CHAPTER 3 Racehorse injuries

CHAPTER 4 Acute care and wound management

CHAPTER 5 Rehabilitation and tissue repair

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CHAPTER 1

MUSCULOSKELETAL SYSTEM

By definition, training for any athletic pursuit involves progressive overloading of the musculoskeletal system to stimulate adaptations to prepare for competition during which limits of fitness and strength are tested. Prevention, early recognition and enhanced rehabilitation of injuries is probably the most important aspect of racehorse sports medicine for equine clinicians, with direct impact on both horse welfare and the health of the wider sport. To optimise the well-being and career opportunities of the equine athlete, it is necessary to have a basic understanding of the behaviour of musculoskeletal tissues in health and disease as well as the specific physiological demands faced by the Thoroughbred racehorse in training.

MUSCULOSKELETAL TISSUES

Musculoskeletal injuries can involve bone, joints and soft-tissue structures such as tendons, ligaments, muscles and cartilage. Injuries may arise from a single acute overloading event, but far more commonly they are the result of cumulative damage built up over a period of weeks or months. This applies particularly to bone injuries (including fracture) with the great majority of serious fractures in racehorses associated with pre-existing disease, but it equally accounts for important injuries involving tendons and ligaments. Musculoskeletal tissues have (to a variable degree) an innate ability to repair microdamage that arises from athletic training and competition; however, the equilibrium between damage and repair can be upset by excessive training loads or inadequate recovery time and subsequent mechanical failure of the tissues may result in clinical injury. Key features of the main musculoskeletal tissues and the mechanisms that lead to injury are summarised next.

Bone

Structure

Bone is a mineralised connective tissue that serves several important roles in the body including structural support, facilitating movement (through attachment of musculature) and protection of vital organs, as well as accommodating bone marrow and acting as the body's store of calcium and phosphorus. Despite its apparent inertness bone is actually a dynamic, living tissue that is continually renewing itself and adapting to the forces it experiences, and this continues throughout life. Architecturally, bone is classified as either cortical ('compact') or cancellous ('spongy'). Cortical bone is essentially 'solid' bone and makes up the majority of bone mass in the body; it is the stiff outer 'shell' of most bones and the main component of the long bones of the limb. Cancellous bone is a three-dimensional matrix of trabecular 'plates', which are oriented in relation to the direction of mechanical strains running through that particular part of the bone. Although less strong than cortical bone, cancellous bone is much more dynamic and responsive to loads imposed on it; therefore, it plays a great role in the adaptation of the skeleton to compressive forces in particular.

At a cellular level, bone turnover occurs through the action of osteoclasts (which resorb existing bone) and osteoblasts (which lay down new bone). The latter produce bone matrix, which becomes mineralised: the mineral component is primarily hydroxyapatite crystals composed of calcium and phosphorus. Osteocytes (the most common type of bone cell) maintain communication with each other through an extensive network of canals; they have a mechanosensor function and are well placed to regulate the activity of osteoclasts and osteoblasts. The whole process of bone turnover is complex and finely orchestrated by local/cellular and systemic (hormonal) influences; however, essentially there is a threshold of loading/strain above which bone cells are stimulated to produce bone matrix, and if loading drops substantially below that threshold, then bone removal occurs.

Bone *remodelling* is the process by which 'old' or damaged bone is removed and replaced (at the same site) by new bone, and this occurs throughout life. Osteoclasts remove bone tissue through demineralisation, and osteoblasts follow to deposit the organic part of the matrix, which is subsequently mineralised to become new bone. This process is tightly coupled such that at the end of the process (providing there is no injury or disease at play), there is no net gain or loss of bone tissue at the site. Bone *modelling*, by contrast, occurs

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when osteoclast and osteoblast actions are not coupled (or occur on different surfaces), leading to a net change in bone shape. This is a means for the body to adapt structure to load, or to grow: obvious examples are 'splint' enlargements, or bone 'spurs' at articular margins. Bone modelling occurs on bone surfaces, whereas remodelling may occur both at the surfaces (outer: periosteal; inner: endocortical) and within the substance of cortical and cancellous bone.

Mechanism of injury

Athletic training, regardless of species, involves readying the musculoskeletal system for competition by progressively increasing exercise loads (volume and speed). Bone is remarkably adaptable; however, the health of the skeleton relies on the maintenance of an equilibrium between bone formation and resorption. In response to loading, new bone may be laid down on the external (periosteal) and/or internal (endosteal) surfaces of the site under strain in an effort to buttress the bone against these forces. In subchondral regions, buttressing cannot occur on the articular surface because of the presence of overlying cartilage, and consequently the response is characterised by thickening (sclerosis) of the subchondral bone plate and underlying trabecular bone. When training volumes are excessive or do not allow for recovery, this bone response and ongoing repair may not keep pace with deterioration; it is also known that at least in some key sites (such as the lower cannon of the fetlock) bone remodelling rates are inhibited by high training loads. The changes in bone microarchitecture (whether excessive thickening and resulting stiffness, or excessive resorption and therefore porosity) can lead to accumulation of microdamage and ultimately the development of stress injuries. Bone weakened in this manner is more susceptible to failure when high strains are imposed on it during high-intensity (fast gallop/racing) exercise or when muscular fatigue forces the affected bone to take a greater share of the impact load. Such stress injuries/fractures may be encountered across a spectrum of severity from subclinical to catastrophic. Determining the point at which the level of adaptive response (or accumulated microdamage) tips over from 'normal' to 'pathologic' levels is difficult; similarly, the point at which clinical injury may be manifested is influenced by many factors and therefore highly variable between individuals.

Tendons and ligaments

Structure

Tendons and ligaments are collagenous structures that serve important roles in stabilising joints and assisting locomotion in the horse. By definition tendons connect muscle to bone while ligaments connect bone to bone; however, both types of structures show great variety depending on their anatomical location and function, and the difference between them can sometimes be purely technical. Tendons in particular can vary greatly in their physical characteristics: 'positional' tendons (such as the extensor tendons over the front of the forelimb) are much weaker and less elastic structures than the 'energy storing' flexor tendons that are important to musculoskeletal health in the racehorse. While tendons often have an active role in locomotion, ligaments are generally considered to passively support joints and assist proprioception (the unconscious awareness of joint and limb position).

Tendons are composed of parallel collagen fibrils that band together to form fibres, with groups of fibres forming larger subunits called fascicles. Fascicles have the ability to move independently of each other and are bound up within the outer lining (peritenon) of the tendon, which permits it to glide against adjacent structures. Tendons are relatively sparsely populated with tendon cells (tenocytes), which are responsible for producing and maintaining tendon matrix; in a mature horse the level of turnover and production of tendon tissue are low. Ligaments share many of these characteristics (low cellularity and tissue turnover, and poor blood supply) but may differ from some tendons by having a less organised collagen structure. Ligaments may be intra-articular (localised within a joint), capsular or extra-capsular, with location having an influence on healing potential due to particular characteristics pertaining to blood supply. Extra-articular ligaments have a very thin cell- and blood-vessel-enriched outer lining (epiligament) that serves an important role in maintenance and healing. Ligaments attach to bone either directly (with a transition zone of fibrocartilage and specialised collagen fibres that enter bone) or indirectly by merging with the lining of the bone (periosteum).

Mechanism of injury

As with many other kinds of musculoskeletal pathologies, the majority of tendon and ligament injuries encountered in racehorse practice are generally considered to be the result of cumulative microdamage from repetitive loading cycles rather than a single overloading event, although the latter can certainly occur. Ligaments in particular, in their role as passive joint stabilisers, may be injured by acute overloading or asymmetric loading of a joint or limb beyond its usual range of motion: this can cause stretching or rupture of inelastic collagen fibrils or avulsion of bony attachments. The low cellularity of tendon/ligament tissue limits its capacity for ongoing repair and maintenance, and when structural damage occurs above a threshold level irreversible damage becomes inevitable. Other factors

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may contribute to the development of injury: changes in mechanical properties of tendons/ligaments occur with age that make them stiffer and less resistant to fatigue, and intra-tendinous heating associated with exercise can also lead to cell damage and predispose to injury. In an animal as specialised for athleticism as the racehorse it is understandable that the most important tendons (and ligaments) probably function within a very narrow mechanical safety margin, and the contribution of intrinsic (such as age, conformation and bodyweight) and extrinsic risk factors (training loads, track characteristics and jockey weight) can tip a horse over into clinical injury.

Skeletal muscle

Structure

Skeletal muscles are complex organs consisting of a variety of integrated tissues including muscle fibres, connective tissue, blood vessels and nerves. Muscle cells are highly specialised and protein dense. They are generally referred to as fibres because of their elongated, cylindrical shape. Bundles of fibres form fascicles, and the connective tissue that surrounds them and the entire muscle belly serves to compartmentalise the muscle and give it structure. Muscle contraction creates tension that is transferred to attached structures (typically bone; but also fascia-sheets of connective tissue) to create skeletal movement.

Mechanism of injury

Muscles may be damaged by direct trauma (lacerations and bruises are common, due to their superficial location) or indirect insults such as strains. Strains can occur when a muscle is actively stretched (eccentric motion) against concurrent muscle contraction (concentric motion), resulting in torn muscle fibrils. Some of the most common muscle injuries involve torn muscle bellies (+/– overlying fascia) in the thigh resulting from a slip over/backwards, with associated haematoma formation and sometimes avulsion injuries of bone attachments (such as tuber ischium of pelvis, or third trochanter of femur). Other common injuries arise from kicks (to chest or upper limb). Strains involving the belly or musculotendinous junction of the forearm muscles are rare but significant injuries.

Joints

Structure

Joints facilitate the transfer of load between bones. In most cases this involves repetitive movement in one or more planes; in order to minimize wear and distribute load evenly a near frictionless environment is required. Excessive or abnormal movements are limited passively by the geometric 'fit' between adjoining bones as well as the support structures such as the joint capsule, collateral ligaments and surrounding tendons. Active stability is provided by muscular activity and mediated by proprioceptive nerve endings. In a healthy joint the intrasynovial pressure is usually subatmospheric (influenced by synovial fluid volume and degree of extension/flexion), which also assists stability.

The joint capsule is a dense fibrous structure lined with synovial membrane that, in addition to stabilizing the joint, acts as a seal for joint fluid. The capsule forms a sleeve around the joint and is anchored on adjoining bones through specialized fibrocartilaginous attachments. Thickness varies with location and function, with most joint capsules incorporating some stabilizing ligaments.

Articular (hyaline) cartilage lines the articulating surfaces of opposing bones and along with lubricating synovial fluid serves to provide near-frictionless movement. Articular cartilage varies in structure both between different joints and different weight-bearing locations within individual joints, but it is essentially a high-water content matrix of collagen fibrils and proteoglycans: its composition makes it resilient and able to rebound to normal dimensions after deformation under load. In key load-bearing regions, cartilage is thicker and mechanically strongest. Cartilage is avascular and the cartilage cells (chondrocytes) that produce matrix receive their nutrition by diffusion from synovial fluid within the joint.

The synovial membrane is the secretory lining on the inner surface of the joint capsule; it covers all internal joint surfaces apart from articular cartilage. The membrane produces hyaluronic acid and other components of synovial fluid, and mediates nutrient exchange between blood and joint. Synovial fluid acts both as a lubricant to facilitate frictionless joint movement and as a transport medium for nutrition of articular cartilage. It is a filtrate of plasma and derives its viscosity from hyaluronic acid; volume and viscosity vary between joints.

It is now recognised that a fundamental part of the joint complex is the bone that directly underlies the articular cartilage – the subchondral bone. The subchondral bone connects to the articular cartilage through its deepest layer – the articular calcified cartilage – from which it is only distinguishable microscopically. The architecture of the subchondral bone varies by region, from the compact subchondral bone 'plate' immediately adjacent to the calcified cartilage to the trabecular bone further away from the articular surface. Subchondral bone is deformable and serves an important role by dispersing axial loads across the joint, thereby preserving the articular cartilage which overlies it. Subchondral bone also has a great capacity to adapt rapidly to training stresses by strengthening its microarchitecture (through trabecular thickening).

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Articular cartilage contains no nerves; however, pain perception from the joint comes from several sources. The joint capsule is richly innervated both superficially and deeply with low-threshold mechanoreceptor nerve endings; mechanical stimuli and increased intra-articular pressure (from joint distension) can therefore contribute to the development of lameness. Higher threshold mechanoreceptors are found near the bony insertions of articular ligaments. Synovial and capsular pain receptors (nociceptors) have a high threshold for pain perception in normal joints, but the chemical stimuli associated with osteoarthritis (OA) and synovitis can reduce this tolerance. Pain in many equine orthopaedic conditions arises from the highly innervated subchondral bone rather than the joint itself: loading of the joint leads to raised intraosseous fluid pressure (and therefore pain) at sites of osteochondral damage.

Mechanism of injury

Damage to articular cartilage can arise from a single traumatic event; however, OA in the racehorse is more typically the result of chronic repetitive overloading and an imbalance between damage and repair. Articular cartilage has little capacity to heal itself following injury: chondrocytes have only a very limited ability to replicate, and repair of cartilage matrix and collagen is slow. Accumulation of subclinical cartilage lesions such as surface roughening and wear lines occurs commonly with training but does not necessarily lead directly to arthritis; the factors that determine why pathology progresses in certain individuals are not completely understood. When it does, the cascade of events involves degradation of matrix resulting in swelling and softening of cartilage and loss of normal mechanical function, leading to fibrillation/erosion. This is sometimes accompanied by an inflammatory response with excessive joint fluid of reduced viscosity; secondary interruption to normal functioning of the synovial lining of the joint can lead to chronic joint effusion. OA is also a disease of the subchondral bone, which is an integral part of the osteochondral unit. Thickening ('sclerosis') of the subchondral bone in response to loading, whilst an important part of the adaptation of the joint to the demands of training, can reduce the mechanical support the bone normally provides to the overlying articular cartilage, reducing its resilience and creating stresses and clefts in the calcified cartilage layer. Subchondral bone disease is also recognised as a repetitive stress injury in its own right, in which the pace of bone microdamage exceeds its repair: such pathology can exist even when the overlying articular cartilage is intact.

BIOMECHANICS

Gaits

Locomotion comes about from the generation of force by muscular activity resulting in the movement of limbs, and therefore has an energy cost. Any animal, if permitted free choice, can generally be expected to move at the gait (and the speed within that gait) that is the most biomechanically and energetically efficient for the activity being undertaken. A 'gait' is defined as a specific pattern of limb coordination that is repeated each stride, with a 'stride' being a single full cycle of motion.

Horses can utilise and continually transition between many gaits, with the most identifiable of these being the walk, trot, canter and gallop. Within each of these primary gaits there are many variations aligned with speed and relative collection or extension. Each of these gaits has a characteristic footfall sequence, summarised in Figure 1.1: walking is a 4-beat gait, trotting a 2-beat gait (in which diagonal limb pairs hit the ground simultaneously), canter a 3-beat gait and gallop a 4-beat gait. Symmetrical gaits

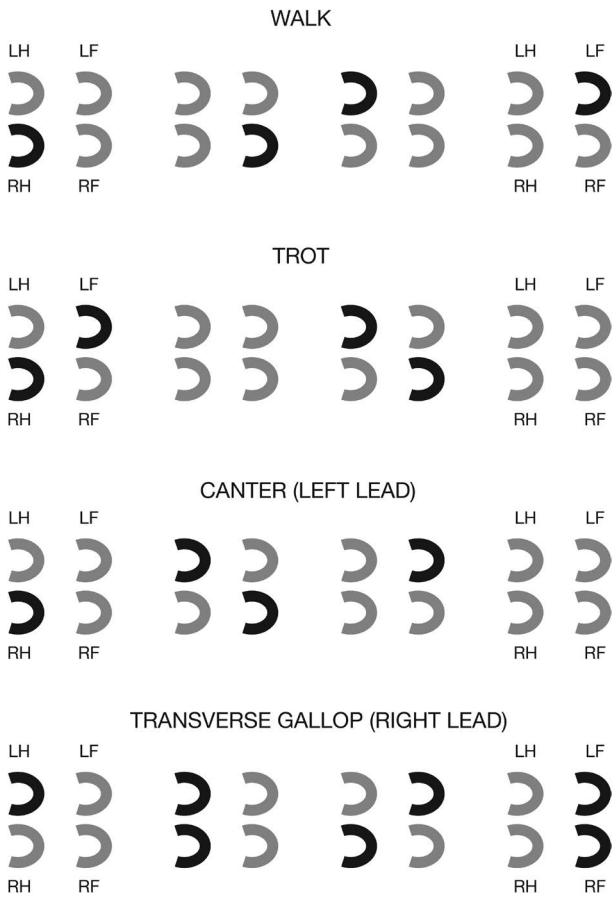


Fig. 1.1 Footfall sequence of primary gaits. LF, left fore; RF, right fore; LH, left hind; RH, right hind.

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(walk, trot) are those in which the limb coordination pattern of one side exactly repeats that of the opposite side, whereas asymmetric gaits (canter, gallop) do not.

Irrespective of gait, each of the four limbs undergoes a cycle of movement comprising a stance phase (when the hoof is in contact with the ground) and a swing phase when it moves through a non-weightbearing arc to its next ground contact site. Additionally, for some gaits the stride as a whole may be divided into phases in which limb(s) are in contact with the ground (stance phase) or all concurrently in the air (suspension phase).

To further define the motion of limbs within the pattern of a gait, the terms 'lead' and 'non-lead' legs are used for the canter and gallop. The 'lead' legs are those that reach forward to a greater degree than their opposites: the lead leg can be identified as the last of a contralateral limb pair to leave the ground during a stride cycle. Non-lead legs are subject to greater loading forces during the stance phase of the stride than their lead leg counterparts.

Stride length is the distance between successive ground placements of the same limb, stride frequency is the number of strides per unit time, and stride duration is the length of time taken to complete one stride. When analysing gait a gallop stride is considered to start when the non-lead hindlimb contacts the ground.

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CHAPTER 2

EXERCISE PHYSIOLOGY AND TRAINING

GLOSSARY

ATP:	adenosine triphosphate
BCAA:	branched-chain amino acids
bpm:	beats per minute
BWT:	bodyweight
CO:	cardiac output
CO_2:	carbon dioxide
ECG:	electrocardiogram
EPO:	erythropoietin
HCT:	haematocrit
HR:	heart rate
HR_{max}:	highest heart rate achieved during peak exertion
HRV:	heart rate variability
NO:	nitric oxide
PCV:	packed cell volume
SV:	stroke volume
VHR_{max}:	velocity at maximal heart rate
$VO_{2\ max}$:	velocity that elicits maximal rate of oxygen consumption

EXERCISE PHYSIOLOGY

The racehorse as athlete

Evolution as a fight-or-flight prey animal has resulted in the modern horse being skeletally mature at a young age and capable of tasks requiring speed or stamina. Further selective breeding has given the racehorse several intrinsic anatomical and physiological characteristics that make it a superior and economical athlete at high speed:

- Large muscle mass relative to BWT, with muscles primarily positioned in the upper limbs/trunk allowing for great efficiency of motion.
- Large cardiovascular capacity to supply this muscle mass with oxygenated blood: the main feature is the ability to greatly increase the HR in response to workload.
- Large reserve of red blood cells stored in the spleen, which is released at onset of exercise to dramatically increase oxygen carrying capacity of blood.
- Efficient cellular-level utilization of oxygen in skeletal muscle through high activity of enzymes involved in energy pathways.

Cardiovascular system

The cardiovascular system of the racehorse is well adapted to support the high metabolic demands and oxygen consumption associated with high-speed exercise. The large heart (weighing on average 4–5 kg) pumps blood around the body and maintains blood pressure to the tissues; arterial blood pressure is a function of both CO and resistance to flow within the smallest vessels (arterioles). CO is the volume of blood pumped by the heart per minute, and is determined by HR and SV (the volume of blood ejected from the left ventricle per beat). At the onset of exertion (or excitement), the HR rises dramatically: within approximately 20 seconds it is elevated from a resting level of 28–36 bpm to racing levels of >200–240 bpm. Despite high HRs reducing the time available for the cardiac chambers to fill, the horse succeeds in also raising the amount of blood pumped with each beat (SV), partly through increased venous return assisted by muscular contraction. SV at rest is in the region of 800–900 mL, and it approximately doubles in the fit racehorse during peak exertion. In this way up to 400 L/min of blood can be circulated at peak exercise. Athletic capacity is also aided by increased CO and by blood flow preferentially directed towards tissues utilised during exertion (locomotory and respiratory muscles) and away from organs less essential for the immediate demands of movement (such as the gastrointestinal tract and kidneys).

In addition to a highly adaptable cardiac response to exercise demands, the racehorse has, through its splenic reserve of blood, the ability to greatly boost circulating blood volume and red blood cell numbers and thereby the blood's oxygen carrying capacity. Total circulating blood volume in the resting racehorse is about 45 L (9% of BWT); splenic blood volume at rest is estimated at between 6 and 12 L, with a higher red cell concentration (HCT 70–80%) than circulating blood. Within the first 30–60 seconds of onset of exertion (or excitement)

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splenic contraction can effectively double the circulating red blood cell concentration (PCV/HCT) from resting levels of 0.35 to 0.65 L/L.

Cardiac parameters rapidly return to resting levels following the cessation of exercise, with HR dropping dramatically in the first minutes. Recovery of resting blood parameters takes longer, with red blood cell figures (PCV) not returning to resting levels for at least 1–2 hours and white blood cell figures taking up to 6 hours.

Respiratory system

The lung is the organ of gas exchange between blood and inspired air. Within the millions of tiny air sacs (alveoli) at the terminal portion of the pulmonary 'tree', oxygen passively passes across the thin alveolar-capillary membrane from airway to blood, while CO_2 passes in the opposite direction and is removed from the body in the exhaled air. The vascular side of this exchange, a rich plexus of pulmonary capillaries, is fed by the pulmonary artery, which delivers deoxygenated blood from the right side of the heart. Although pulmonary arterial pressures rise dramatically during high-speed exercise, dilation of small vessels and fuller utilisation of the vascular plexus results in a reduction in pulmonary vascular resistance.

At both rest and during exercise the work of respiration is primarily done by the diaphragmatic muscle; the ribcage does not expand greatly, even during periods of great respiratory effort. The amount of air that moves in or out of the lungs with a single breath is the tidal volume, which in the resting racehorse is approximately 6 L, rising to 12–15 L at peak exercise. 'Minute volume' is the amount of air expired over a minute, and thereby determined by respiration rate: resting respiration rate is typically 8–12 breaths/min. During exercise, respiration rate increases in line with speed; however, there is a natural upper limit to this because at faster speeds respiration and stride are linked. This locomotor-respiratory coupling permits breathing to benefit from a range of biomechanical efficiencies, including the gut contents pushing forward on the diaphragm (and thereby acting as a 'piston' on the lungs) and recoil inhalation aided by the expansion of the ribcage that comes with forelimb protraction. As peak respiration rate at exercise approaches 130–150 breaths/min, minute volume can be as high as 1800–2000 L. Due to gravity and regional differences in pressure on the ribcage, alveolar ventilation throughout the lung is not uniform, with intrapleural pressure (and therefore ventilation) lower in the dorsal portions.

Despite this, the ability of the respiratory system to respond to exercise demands is more modest than the cardiovascular system and it is likely to be the principal factor that limits athletic capacity. Although ventilatory volumes increase during exercise, there are both anatomical (airway resistance) and biomechanical (breathing pattern in relation to stride) constraints to the amount of air that can reach the alveoli. Due to the anatomical particularities of its larynx and pharynx, the horse can breathe only through its nostrils: regardless of respiratory demand, breathing through the mouth is not possible. The upper airway plays a significant role in humidifying inspired air, as well as filtering particles and defending the lower airway against potential pathogens, but it is also the largest source of resistance to airflow. Air passes first through the dilatable nostrils. Then it is exposed to the highly vascular and rigid mucosal surfaces of the nasal turbinates before passing through the nasopharynx and larynx into the long semi-rigid cartilaginous tube of the trachea. Airway resistance is a direct function of the cross-sectional area of the airways that inspired air has to traverse; in simple terms, any reduction in the diameter of the 'tube' has an exponential effect on the resistance to airflow (and therefore the workload needed to overcome it) as a result of both friction and turbulence.

Muscles

Skeletal muscle mass in the racehorse accounts for a large proportion (53–57%) of total BWT. Specialisations both in terms of locomotor muscle location and architecture (orientation of muscle fibres in relation to their tendons and the direction of muscular action) and their fibre type composition contribute to the racehorse's effectiveness as an athlete. Muscle fibres are broadly classified by their metabolic and morphological properties as being 'slow-twitch', 'fast-twitch' or 'hybrid/transitional'. Slow-twitch fibres have an endurance function and are capable of long periods of work without fatiguing, whereas fast-twitch fibres are capable of rapid and large power production but fatigue easily. Muscle bellies are not homogenous structures. They are comprised of a mix of fibre types, with their individual composition dependent on muscle function and location; muscles with a predominantly postural role stabilising the limb or body have a greater proportion of slow-twitch fibres than muscles that are heavily involved in propulsion. Even within the same muscle there will be variation in the proportion of fibre types depending on location, with superficial parts often predominantly fast-twitch glycolytic and deeper parts slow-twitch. Throughout the body the relative proportions of these fibre types are largely genetically predetermined, although some modification of fibre subtypes can occur through training.

Slow-twitch (or type I) fibres have a relatively small cross-sectional area, and therefore less capacity to store energy substrates within the cell. As a result they rely

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predominantly on aerobic metabolism of fuels delivered by the bloodstream through a rich capillary network; these are considered to be the 'slow oxidative' type. In contrast, fast-twitch (type IIA) fibres have a large cross-sectional area and greater capacity to store energy substrates (glycogen), but also primarily use aerobic metabolism to produce energy. Type IIA fibres make up the greatest proportion of skeletal muscle cell types in the racehorse. Type IIX (or IIB) is a fast-twitch fibre type that primarily uses anaerobic metabolism for energy production.

Genetics

The athletic potential of a racehorse is determined by the complex interplay of many factors: environmental variables such as early-life nutrition and conditioning, training programme and genetic and epigenetic (non-hereditary gene expression) factors. Muscle mass and composition (ratio of fast-twitch/slow-twitch fibre types), conformation, precocity and growth potential are all traits with a strong underlying genetic basis, but the expression of these genes can be promoted or inhibited by a large array of non-genetic influences. The gene for myostatin (a muscle-specific hormone) in particular has been found to be one of the most important determinants of athletic potential: it is directly involved in the regulation of muscle development, and variations at the myostatin gene locus have a strong association with a horse's optimal racing distance ('distance aptitude'). As a result race distance aptitude is highly heritable: horses with a CC genotype are more likely to perform best over shorter race distances (<1600 m), those with a TT genotype are more likely to have greater stamina and perform best over staying distances and those with the heterozygous genotype CT are likely to perform best over middle distances.

Despite the importance of these factors it is notable that the closed Thoroughbred gene pool and intense selection for performance has resulted in a population that genetically is relatively uniform. Because of this the heritable component of key measures of athletic success in the racehorse is likely to be significantly lower than for comparable aspects of human athletic performance. When considering the many influences on racehorse performance it is useful to view the genetic material that a horse inherits as determining its 'absolute potential', but whether (or to what extent) that potential is realised is largely the result of how the horse is reared, trained and campaigned.

Speed and stride

Locomotion in the horse is driven by power generation from the strong hindquarter musculature. The peak speed of the racing Thoroughbred racehorse exceeds 40 mph (65 km/h, 18 m/s). The best performers can run at speeds averaging between 15 and 16 m/s over race distances of 1000–2000 m.

There is an intimate relationship between speed and stride parameters (stride length, duration and frequency). When at non-maximal canter/gallop pace, the horse is able to alter stride parameters to suit exercise conditions; however, the galloping horse can only achieve its maximal speed by maximising its stride length and minimising stride duration. When a horse fatigues during maximal exercise, stride frequency, stride length and extension of the body begins to diminish. This is likely to be due to reduced power generation by the major locomotory muscle groups. Stride length and duration can be influenced by many factors, including horse-level variables such as age, sex, fitness and weight carried and external factors such as type (and condition) of track surface, gradient and radius of turns. Stride length (and jumping trajectory) can also be influenced by girth tension and use of breastplates. Average stride length of male racehorses is greater than females, and that of older horses is greater than 2 YOs. In general, faster horses have a longer stride length than slower horses, and sprinters have a greater stride rate than stayers; however, many variations between individuals exist in terms of interaction between stride length and rate at different stages of a race. To some extent the stride characteristics particular to a horse may determine its success under different race conditions (track geometry, race distance and pace): a horse with faster stride rate or greater acceleration may be a more effective athlete over shorter distances or tighter tracks than a horse with greater stride length. Stride characteristics are integral to loading of limbs; therefore, they contribute to an individual's risk of developing both acute and chronic injury.

Energy metabolism

Metabolic pathways

All cells in the body need chemical energy to perform their tasks, which range from cross-membrane transport (of ions and small molecules) to muscular contraction. ATP is the basic molecular unit involved in the energy transfer essential to these cellular functions. Energy is stored in the bonds between its phosphate groups, and breaking of these bonds and transfer of phosphates generates energy; during biological processes ATP is constantly broken down and reformed.

The ability to perform and sustain athletic effort is determined by energy production in skeletal muscle. The amount of ATP stored within muscle cells is small, therefore, it must be continually synthesised for muscle activity

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to take place. Fuels used by muscle to create ATP include glycogen (a carbohydrate stored within muscle and liver cells) and to a lesser extent amino acids, acetate and fatty acids. These fuels are metabolized through either aerobic or anaerobic pathways, with exercise intensity and duration determining the relative contribution of each pathway. Prolonged, low-intensity exercise utilises both fats (lipids) and carbohydrates, whereas high-intensity exercise relies primarily on carbohydrates (glycogen). The generation of energy by aerobic means is most efficient, but it is limited by the many factors that make up the supply chain of oxygen to muscle, chiefly cardiorespiratory function and blood oxygen carrying capacity. Anaerobic metabolism (glycolysis) generates force rapidly but is inefficient and can only be sustained for short periods; it also leads to the production of lactate as a by-product. Although lactate can accumulate and ultimately contributes to the onset of fatigue, it is also utilised by oxidative (type IIA) fibres as an important secondary energy source.

Equine muscle contains very large stores of glycogen; at the high exercise intensities typical of horseracing oxidation of glycogen is the dominant pathway for generating energy, although anaerobic metabolism also plays a part. Slow paces (trot and slow canter) are purely aerobic; however, all performances at racing speeds derive energy from both pathways, regardless of distance. Energy production during races over staying distances (>3200 m/2 miles) is estimated to be around 80–90% aerobic, and even in those run over sprint distances (<1200 m/6 furlongs) energy production is predominantly (70%) aerobic. Short periods of acceleration during a race (whether at the start or in the closing stages) are anaerobic efforts and consequently cannot be sustained for long (typically <600 m/3 furlongs).

The metabolic processes involved in athletic effort result in large shifts in fluid (between extracellular and intracellular fluid compartments) and electrolyte balance, which in turn affect acid-base status. These interactions are complex and are primarily influenced by intensity and duration of exercise. High-speed, intense exercise causes a metabolic acidosis, whereas submaximal/endurance exercise can result in a metabolic alkalosis. These alterations are highly dynamic and typically corrected within 60–90 minutes following exercise.

Fatigue

Fatigue is a reduction in muscular power production. The mechanisms that lead to muscular fatigue are complex and not fully understood, but they may be generalised as impairment of cellular functions arising from accumulation of 'by-product' metabolites and to a much lesser extent depleted energy stores. When glycogen is broken down by the anaerobic pathway, not only are muscle stores of this important carbohydrate depleted, but lactate is produced. Although lactate also becomes an energy substrate itself, its accumulation within the cell causes acidosis, which inhibits glycolysis and other essential processes. Capacity to resist the onset of fatigue varies with muscle cell type, and is greatest for slow-twitch fibres. Equine muscle in general is considered to have high innate capacity to 'buffer' against acidosis; this comes about through the action of membrane transporters (that correct acid-base imbalances) and high intracellular levels of the peptide carnosine.

Recovery

The immediate post-race period is characterised by a profound metabolic acidosis. The locomotor-respiratory coupling, which has constrained oxygenation during high-speed locomotion, ceases and the horse transitions through a phase of hyperventilation in which excess CO_2 is exhaled and acid-base status restored.

Depletion of muscle glycogen stores resulting from high-speed training workouts is estimated at between 20% and 25%, and that resulting from flat racing around 25–30%. Restoration of these glycogen stores to pre-exercise levels occurs naturally and without intervention but is slow in the horse compared with humans, taking 2–3 days. Whether a horse exercises at walk or light canter during these recovery days does not appear to influence (negatively or positively) the rate of muscle glycogen repletion. It is unlikely that glycogen depletion is a significant factor in performances over flat racing distances, and the need for enhanced repletion is therefore doubtful. If faster recovery is desired, however (e.g., due to proximity of next race), it is important to provide water, electrolytes and high-energy diet and avoid low-energy feed (hay) in the post-race period; intragastric or intravenous fluid therapy can also be considered.

TRAINING

Tissue response to training

The goal of training is to stimulate adaptations of key body systems (skeletal, muscular and cardiovascular) to the demands of racing. Conditioning strengthens the musculoskeletal system to withstand the forces experienced during peak exercise and maximises the efficiency of energy generation and utilization: the result is a racehorse that can both attain higher speeds and maintain athletic effort for longer before the onset of fatigue. Racetrack performance of the average racehorse improves considerably between 2 and 4–5 YO. Much of this development is training related; however, some development is also a natural consequence of maturation. The horse is a

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precocious animal: in human-comparative terms the racehorse completes the 'adolescent growth spurt' between 12 and 18 months of age and onset of skeletal maturity occurs at approximately 24 months. Physical development and athletic capacity continue to develop through a horse's career, with running speeds peaking at around 4–5 YO.

Although the overall effect of a training programme is to increase the aerobic capacity of the racehorse, the changes that contribute to this differ between body systems. Additionally, the threshold of training loads required to stimulate adaptations varies with tissue type and is determined by both duration and intensity of exercise. Beyond the foundation phase of training, periodic overload is introduced such that the horse 'overreaches': if calibrated correctly and followed by a recovery phase, such 'functional overreaching' produces tissue remodelling that results in positive athletic development.

Cardiovascular adaptations

An early and universal consequence of training is enlargement and strengthening of cardiac muscle: with training loads and maturation the heart of the young Thoroughbred develops thicker muscular walls and becomes heavier. Hypertrophy of the left ventricular muscle in particular permits greater propulsion of blood to the exercising body. Modest increases in CO and SV (latter increasing by around 10%) contribute to greater aerobic capacity. Training does not, however, result in any significant change in either the HR_{max} (the highest HR achieved during peak exertion) or resting HR (unlike in human athletes).

Training induces little in the way of measurable alterations in blood parameters. Although increases in red blood cell numbers and plasma volume may occur (and assist in improved capacity of the circulation to carry oxygen), the magnitude of these changes is modest and difficult to detect (requires sampling during a treadmill test). Parameters measurable at rest fall well within the range of variation caused by many other factors such as feeding/hydration status and excitement. Most other biochemical, haematological and electrolyte variables remain unchanged.

Respiratory adaptations

As with other tissues the adaptations of the respiratory system to training depend on many factors such as current and previous training loads. Although one of the principal effects of training is an increase in the amount of oxygen that the horse can utilise during maximal exercise ($VO_{2\ max}$), this comes about through muscular/cardiovascular adaptation rather than any significant training-related increase in ventilatory capacity. The key measures of ventilatory capacity, respiration rate and tidal (or minute) volume, are generally considered to not be significantly altered by training. Although lung and airway function do not adapt to training loads, there is some evidence that the main respiratory muscle (diaphragm) strengthens during the foundational phase of training and potentially through use of some training interventions, and this adaptation may play a role in fatigue resistance.

Muscular adaptations

Of all the tissues and organs involved in athletic effort, the most responsive to training is muscle. A range of training adaptations occur, and while some are specific to the type of training load (sprint or endurance) as well as the type and location of muscle (postural or locomotory), the overarching effect is an expanded capacity for power generation and greater resistance to fatigue. This comes about through an increase in muscle mass and increased capillarization of muscle fibres, as well as cellular changes such as increased mitochondrial density and greater activity of enzymes involved in aerobic metabolism (resulting in greater efficiency in use of energy substrates). The majority of these changes occur early (within the first 2 months) in the training programme.

Both aerobic and anaerobic exercise stimulate overall growth of muscle bulk, but at a cellular level (and dependent on fibre type) the cross-sectional area of muscle fibres typically diminishes. It is thought that this facilitates easier transport to and diffusion into the cell of oxygen and fuels required for muscle contraction. Another key adaptation is that training induces a shift in proportion of fibre subtypes, such that overall oxidative capacity of muscle improves. Although it is not thought that slow-twitch fibres can to turn into fast-twitch (or vice versa) fibres, a transformation of fast-twitch muscle mass from high glycolytic (subtype IIX/IIB) to high oxidative (subtype IIA) is a consistent outcome of training. The domination of IIA fibres means that not only can trained muscle generate greater power but that muscular effort can be sustained for longer.

In addition to 'plasticity' of muscle architecture and makeup, training results in upregulation of several metabolic mechanisms that allow muscle cells to improve their oxidative capacity and counteract the onset and effects of lactate accumulation.

Bone adaptations

Bone is a dynamic tissue that remodels continually throughout life in response to the loads placed upon it. These loads are influenced by speed, conformation, track surface and jockey weight, among other factors. Bone is known to be most responsive to loading stimuli before the age of 2 YO and well before full skeletal 'maturity' occurs. Architectural adaptations involving bone shape and size, and trabecular

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orientation of cancellous bone appear to be more important to acquired strength than increases in bone density or mass. These adaptations (along with those of other musculoskeletal tissues) appear to have clinically significant specificity to training track surface and geometry, such that horses may be predisposed to injury if trained at high intensity on surfaces to which they are not accustomed.

Tendon and ligament adaptations

Adaptation of the flexor tendons to conditioning exercise occurs early in life. From the age of 2 years, no strengthening of tendon occurs, although some increase in tendon size is often noted (as a result of greater water content of tendon matrix rather than tendon hypertrophy). The suspensory ligament undergoes some strengthening into early training but is then largely unresponsive to training loads (as for flexor tendons).

Cartilage adaptations

Although training has little or no adaptive effect on articular cartilage, free exercise as a foal is important to future health of the joint. Restricted exercise early in life has the potential to hinder normal articular cartilage development, making it less able to withstand the rigours of training.

GROWTH PLATES AND MATURITY

- Radiographic survey of growth plate above knee/carpus (distal radial epiphysis) to assess maturity and 'readiness to train' has been practiced for decades.
- The growth plate is a zone of hyaline cartilage that generates long bone growth and appears on radiographs as a radiolucent line (Figure 2.1a). Radiological closure of the growth plate occurs when the growth plate has ossified and the radiolucent line is no longer visible (Figure 2.1b).
- The term 'open knees' is poorly defined and refers to physical (not radiological) appearance: it is influenced by relative prominence of lower radius and carpal joints/bones and does not correlate well to 'open growth plates'.
- In the racehorse, growth of the radius ceases before radiological closure of the radial growth plate occurs.
- The Thoroughbred is a precocious animal and there is ample evidence that commencing training before full skeletal maturity is more likely to be beneficial than harmful.
- The limited scientific evidence available supports no correlation between radial growth plate closure and measures of both injury risk and racetrack performance.
- Radiographic classification of closure of the radial growth plate is a poor measure of skeletal maturity of the racehorse and when used as a guide to training and management is highly likely to be misleading.



Fig. 2.1 Radiological appearance of 'open' (a) and 'closed' (b) distal radial growth plate (DPa radiographs).

Training programmes

Training usually commences before the age of 2 years and produces rapid effects: between the end of its yearling year and the first spring of its 2 YO season the racehorse will already have greatly increased its aerobic capacity and maximal running speed. Training strategies vary widely both geographically and between individual trainers and in large part are shaped by tradition, available tracks and

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local climate/weather conditions. Although there is no universal template for conditioning a racehorse, most programmes share elements of the following:

- Foundation low-intensity phase (typically lasting 6–12 weeks): low-speed, predominantly aerobic exercise (trot and slow canter). HR <160 bpm and no increase in blood lactate. Goal is to improve aerobic capacity and limb strength as well as educate the horse. Daily distance cantered typically commences at 1000–1600 m.
- Gradual increase in training load (cantering speed +/or distance) every 2–3 weeks ('overload principle') to improve aerobic and anaerobic fitness as well as musculoskeletal strength. Exercise intensity is more important than duration.
- Combined aerobic and anaerobic phase: periodic high-intensity exercise improves muscular strength and stamina. Causes adaptation/recruitment of the fast-twitch (type IIX) muscle fibres that assist in developing anaerobic metabolism. Typically done by closing submaximal (70–80% of race speed; 14–16 s/furlong) workouts with a short distance (1–3 furlongs) of top speed (95–100% of maximal speed).
- Once the conditioning programme is complete and the horse has commenced racing, training loads (particularly high-intensity exercise sessions) can be reduced depending on racing intervals.

The capacity for exercise (mental and physical) varies between horses. Training programmes are individualised to account for this as well as stage of maturity and career goals. Training not only targets physical fitness but mental development (stalls and race training). Rest days after fast exercise or racing days are important for recovery, and frequency of high-intensity bouts (typically one to two per week) is determined on an individual basis. Overreaching (in this context: short-term/single-session excessive training load relative to fitness) may diminish performance and cause loss of appetite. Fitness is maintained well in the short term and in general it is preferable to apply short intense training loads on an intermittent basis, with allowance for rest/light exercise periods, than to maintain constant high-volume training stimulus.

The goal of achieving muscular and cardiovascular fitness must be balanced with conditioning of the musculoskeletal system. Incremental increase in workload (through manipulation of intensity, duration or frequency of exercise) stimulates conditioning; however, unless sufficient time is allowed for adaptation, stress-related injuries may result.

Training speeds

Terminology to describe canter speeds differs worldwide. Frequently, there is also considerable variation between trainer/jockey perception of paces and measured speeds. Factors such as stage of season, time of week, track surface and flat or hill work have been demonstrated to have an impact on actual (as opposed to described) speeds. To determine work intensity accurately, objective measurement using GPS equipment is required. Typical training and racing speeds are shown in Table 2.1.

Training the young horse

The long-standing notion that subjecting horses to high-speed training and racing before they have reached skeletal maturity is detrimental to well-being and athletic longevity has been comprehensively refuted by a large and diverse body of scientific research. In fact, it is now known that the best opportunity that the main musculoskeletal tissues have to adapt to future athletic challenges is if they are stimulated by some training loads well before

Table 2.1 **Typical training speeds**

	TROT	CANTER (SLOW)	CANTER (1/2 PACE)	CANTER (3/4 PACE)	FAST GALLOP/BREEZE
km/h	10–16	18–29	36–47	48–54	>54
mph	6.5–10	11–18	22–29	30–34	>34
m/s	3–4.5	5–8	10–13	13–15	>15
s/furlong		>25	20–15	15–13	<13

km/h, kilometres/hour; mph, miles/hour; m/s, metres/second; s/furlong, seconds/furlong.

1 furlong = 220 yards = 201.2 m.

1 mile = 8 furlongs = 1609.3 m.

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skeletal maturity occurs (<2 years of age). For cartilage, tendons and ligaments the window of adaptive opportunity is in the first year of life and there is some indication that judicious application of conditioning exercise to the juvenile horse in addition to spontaneous paddock activity might be advantageous. Bone strength is highly responsive to early training, and the better overall career longevity of horses trained and raced as 2 YOs probably reflects greater potential for the musculoskeletal system to adapt during this period of relative immaturity.

Overtraining

'Overtraining' is a poorly defined syndrome characterized by long-term underperformance or reduced exercise capacity, with or without other physical or behavioural signs (see p. 432). There is limited understanding of the underlying cause(s) and why certain individuals are affected, although it is considered to represent a mismatch between training load and recovery and oxidative stress may be implicated. Clinical features include loss of condition/BWT (with or without a drop in feed intake) and irritability and unwillingness to train. Diagnosis is difficult and frequently inconclusive due to the current absence of any single effective diagnostic test, although HRV (see p. 18) is considered useful in human athletes.

Detraining

Removal of the stimulus of full training has a range of effects on different musculoskeletal tissues and body systems. Short periods of restricted exercise (<1–2 weeks) do not result in great loss of fitness.

There is some evidence that cardiovascular fitness ($VO_{2\ max}$) then declines rapidly (within 2–3 weeks) with inactivity; however, most objective measures of muscular fitness are relatively slow (>5–6 weeks) to diminish after the cessation of training. Loss of cardiovascular fitness can be significantly offset if the horse is allowed some daily walking/paddock turnout rather than being confined to the stable; however, evidence-based guidelines regarding the threshold levels of exercise that might be beneficial have not yet been established. Effects of detraining on the musculoskeletal system can be variable and tissue dependent; some degree of limb loading and motion is required to keep articular cartilage healthy.

Training aids

Hill work

- Inclines of 5–10% are typical.
- Exercising uphill comes at greater metabolic cost and permits application of a similar training load at slower speeds than would be needed if exercised on the flat (maximal exercise intensity occurs at 10–12 m/s on 6% hill, but 14–16 m/s on flat).
- Permits increase in volume of training, which stimulates anaerobic pathways without causing fatigue and overtraining.
- Training at lower speeds reduces risk of certain musculoskeletal injuries, although greater loading of hindlimbs can lead to higher prevalence of some hindlimb problems.

Treadmills

- Adjustable for speed (up to gallop) and incline (typically 0–10%).
- Majority of horses readily become accustomed to treadmill exercise after short introduction period (one to two sessions).
- Allows great control over training loads and individual tailoring of exercise programmes.
- Useful when lighter exercise is desirable without the need to train under a rider.
- Also for horses that are unwilling/difficult to train under a rider, horses recovering from musculoskeletal injury or those with sore backs preventing use of tack.
- Limited information available on treadmill-specific injury patterns; however, risk of serious/catastrophic injury during use is very low.

Water treadmills

- Adjustable water height: encourages change in stride length/character during use (see p. 62).
- Maximum speed typically around 6 m/s.
- Increased workload relative to regular horse-walker.
- Unless utilized heavily, has little demonstrable conditioning effect on cardiorespiratory or muscle function.

Swimming

- Generally considered a non-essential cross-training aid, as stimulates adaptations specific to swimming rather than running.
- Has a role in rehabilitation and some training programmes.
- Exercise intensity similar to trotting/slow cantering (HR 140–180 bpm): submaximal aerobic exercise.
- Tethered (tail) swimming with short periods of encouragement may achieve efforts with HR >200 bpm.
- Markedly altered respiration pattern compared with ridden exercise: brief inspiration, breath holding and prolonged, explosive expiration.

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- Greater pressure of water on chest and abdomen may necessitate more forceful breathing: possible 'training' of respiratory muscles.

GPS/ECG/stride monitors

- GPS tracking can allow accurate monitoring of daily workload (speed and distance).
- Primary benefit is objective analysis of fast exercise performance (Figure 2.2a,b). Speed and pace data are used to interpret training gallop outcomes and plan optimum racing strategy for individual.
- Encourages improvement in pace consistency of work riders.
- HR, speed and stride data may be used longitudinally over a period of time to document fitness/athletic potential of an individual as well as possible role in detection of developing injury; many variables may influence data and it requires caution in interpretation of patterns.

Warm-up

- Has little effect on injury risk, but can enhance performance.
- 'Primes' the muscles: improved oxygen and fuel availability/utilization such that during a subsequent race more energy is provided by aerobic sources and onset of fatigue is delayed.
- Splenic contraction increases circulating red cell fraction (and oxygen carrying capacity) of blood; may also occur through anticipation/excitement.
- Likely that high-intensity (canter) warm-up is more beneficial than low-intensity (walk/trot) warm-up.
- Cantering down to start of race probably beneficial to subsequent race performance.

Cool-down

- 'Active' cool-down (low- to moderate-intensity exercise in the immediate period post-race/gallop) does not appear to have any impact on injuries or delayed-onset muscle soreness, but it may have beneficial effects on cardiovascular and metabolic recovery including accelerated recovery of blood lactate to pre-exercise levels.
- Most efficient cool-down exercise for optimizing clearance of blood lactate is a 10-minute fast trot (at 65–70% of maximal HR) rather than passive recovery or walking.
- Overall effect on athletic performance(s) in subsequent days likely to be minimal.

Other recovery aids

- Glycogen repletion (see p. 452, p. 457).
- A variety of other strategies/aids are used in human/equine sports in attempts to assist recovery from



(a)

Split Description	Split (secs)	Elapsed (-secs)	Min Speed (mph)	Max Speed (mph)	Ave Speed (mph)	HR Min (bpm)	Peak HR (bpm)	HR Avg (bpm)	Stride Count	Stride Length (m)
Start->Furlong 1	**14.8**	89.90	21.2	31.2	27.6	175.0	200.0	189.0	29.94	6.72
Furlong 1->2	**13.5**	75.10	31.4	35.1	33.4	201.0	209.0	206.0	29.36	6.85
Furlong 2->3	**12.7**	61.60	34.1	35.8	35.0	208.0	212.0	210.0	28.20	7.13
Furlong 3->4	**12.1**	48.90	36.0	39.5	37.3	212.0	214.0	213.0	27.55	7.30
Furlong 4->5	**12.3**	36.80	35.8	38.7	37.3	213.0	215.0	214.0	28.15	7.15
Furlong 5->6	**12.1**	**24.50**	35.9	38.1	36.9	215.0	216.0	215.0	28.13	7.15
Furlong 6->7	**12.4**	**12.40**	34.9	38.2	36.5	216.0	218.0	217.0	28.27	7.12
Summary	89.9	0.00	21.2	39.5	34.6	175.0	218.0	208.0	28.51	7.06

(b)

Fig. 2.2 (a) Speed/heart rate graph from training gallop. (b) Associated breakdown of sectional speed, stride count and derived stride length data.

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strenuous exercise. These include cold-water immersion, static stretching and compression blankets. There is little current evidence that these interventions substantially accelerate recovery or reduce post-exercise muscle soreness.
- Use of non-steroidal anti-inflammatory drugs (NSAIDs) and anti-oxidants in the recovery phase is common practice; however, there is substantial evidence that these interventions may inhibit adaptive responses to training (through interference with biochemical signalling pathways) and could have a negative long-term effect on performance.

Measuring fitness and ability

Assessment of fitness (readiness to run) and individual athletic ability is the central role of the trainer. This is typically judged through appraisal of body condition and observation of an individual's performance during fast exercise or trial gallops relative to that of other horses in the work group (accounting for factors such as jockey weight and instructions). The ultimate measure of fitness is racetrack performance, where these estimations can be tested against a broader group of animals. This process is lengthy and inexact, and more objective methods of determining fitness are desirable. Measurement of aerobic capacity (maximal oxygen uptake) is possible with treadmill studies incorporating ventilation capture, but this is a specialist procedure that is rarely practical. Some other measures that are sometimes applied are discussed below.

Blood analysis (haematology and biochemistry)

- Not useful for assessing fitness.
- Changes in oxygen carrying capacity of blood occur early in training but are an unreliable guide to fitness later in preparation.
- Sampling circulating blood does not necessarily permit assessment of total red blood cell volume, due to large splenic reserve of blood cells; excitement, exercise and recent meals can all influence commonly used red cell variables such as PCV/HCT, red blood cell count and haemoglobin concentration.
- No correlation between PCV/HCT and racing performance.
- Little correlation between resting or post-exercise red or white blood cell values (or blood enzymes) and fitness.

HR

- Unreliable measure of fitness in flat racehorses.
- Maximal and resting HR vary between horses; each horse has its own HR_{max}.
- Training does not alter resting HR (unlike human athletes).
- Training does not alter maximum HR at exercise; rather, with increasing fitness a horse will achieve higher speeds at this HR_{max}, and this speed is known as VHR_{max}.
- No correlation between maximal HR and racetrack performance.
- VHR_{max} can be used as a measure of fitness but requires standardized conditions and GPS/ECG monitoring.
- HR recovery after standardized exercise test difficult to utilize for fitness assessment, as HR naturally drops rapidly at end of exercise.

HRV

- Although normal heart rhythm (in the absence of arrhythmias) is regular and repetitive, small variations can occur in the intervals between beats.
- These fluctuations are linked to the state of relaxation of the nervous system and specifically activity of the parasympathetic nervous system, and are considered to reflect total stress influences on the individual (i.e., exercise, travel, nutrition and sleep).
- HRV is measured at rest over short periods (approximately 1 hour); interpretation depends on establishing a baseline set of measurements for each individual and sampling at same time of day/under same conditions (in relation to training times) is important.
- In human athletes HRV is correlated to performance and may be used as an aid to monitor recovery from training/competition. In general, high HRV is desirable and linked to fitness, and uncharacteristic dips in HRV may indicate the need for recovery from training.
- Research in racehorses is limited at present.

Respiration rate (recovery)

- Commonly used for subjective appraisal of fitness by training staff but is of little value as an objective, measurable fitness parameter.
- Poor/prolonged return to normal respiration rate after fast exercise is an indicator of overexertion/unfitness/illness.

Body condition

- Highly subjective and requires close familiarity with individual horse and knowledge of workload and appetite.
- Considerable variation in ideal condition and BWT at peak fitness between individuals.
- BWT increases as a horse matures, peaking at 4–5 YO; average gain between 2 and 5 YO is 20–25 kg.

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- Average mature BWT of colts and geldings is 480–490 kg, and mares 465 kg.
- BWT can be used as an indirect measure of race readiness for the individual; it is of most use when information on previous weights in relation to performance is available.

Speed

- VHR_{max} broadly correlates to ability and is a good measure of fitness.
- However, interpretation of VHR_{max} can be confounded by influence of many variables (e.g., stress/illness/rider/track).
- Measurement of HR at submaximal speeds is an unreliable measure of fitness.

Blood lactate (lactic acid)

- Buildup of lactate in muscle and blood occurs when intensity of exercise exceeds aerobic capacity; this typically occurs during medium/fast cantering.
- 'Anaerobic threshold' is considered to be the speed at which a horse has a blood lactate concentration of 4 mmol/L (V_{LA4}).
- Increasing fitness allows work at faster speeds without lactate accumulation (rise in threshold/V_{LA4}).
- Blood lactate can rise from resting levels of <2 mmol/L to >20 mmol/L with intense exercise; it returns to normal levels within 3 hours post exercise.
- Measurement of blood lactate immediately after (or during) exercise is a good test of aerobic (but not sprint) fitness; it can also be used as a guide to ability.
- Portable handheld, whole-blood lactate analysers permit accurate measurement.
- More suited for treadmill testing: exercise variables must be tightly controlled and samples obtained within 3 minutes of cessation of cantering (or trotting).
- Treadmill field test: blood lactate obtained immediately after strenuous but submaximal exercise; speed of test can vary but typically medium-fast canter. Variables such as warm-up/distance should be kept constant.

TRAINING AND RACING: FURTHER ASPECTS

Track types

A variety of track types are used in training and racing worldwide and in many training centres a choice of track surfaces is available for slow and fast exercise. It is recognised that musculoskeletal adaptation can be specific to track type, and ensuring tracks of the same type to the expected racing surface are utilised during any conditioning programme is an important part of injury risk mitigation. The main classes of track are discussed next.

Turf

Turf is the preferred surface for racing and training in many jurisdictions. While many turf racetracks are purposefully built (with layered construction and drainage), many turf training areas have developed with no planned construction over generations of use. Turf racetracks typically consist of a topsoil/root zone layer that is comparatively shallow (10–15 cm), above sand (25–50 cm depth) and deeper gravel layers that serve to drain water away. Shear strength of the topsoil layer is determined not only by grass root structure but by its soil content, with clay being relatively more cohesive than sand, particularly in the presence of moisture. The incorporation of polypropylene fibre, mesh or strands in the upper sand layer is common in high-rainfall climates and fortifies the shear strength of the root zone (smaller divot size). Suitability to local growing conditions determines optimum grass type, with root growth, strength and resilience to wear more important than sward above the ground. Ideal soil temperatures for root growth differ between grass varieties: common cool-season grasses are Kentucky bluegrass and ryegrass, whereas kikuyu, bermudagrass and couch predominate in warmer climates. It is common for racetracks to be composed of a mix of grass varieties to cope with local weather conditions. The length of sward is typically maintained at 10–15 cm; however, while this may provide some slight cushioning effect for footfall, the interaction between horse and ground is primarily a function of the topsoil/root zone.

Maintenance of grass tracks is labour intensive, requiring specialized management of soil, grass and water resources. Ease of management and uniformity of surface under different weather conditions varies with track design and drainage as well as local factors such as soil type and water table depth. Regular upkeep includes replacement of divots, rolling and thatch removal; heavy use or use in wet conditions can result in serious deterioration of the track. In many jurisdictions turf racetracks are maintained to higher standards than turf training grounds despite the latter carrying the majority of traffic.

Dirt

Dirt racing tracks typically consist of varying proportions of medium, fine and coarse sand with a smaller amount (approximately 5%) of clay that acts to bind the sand and therefore contributes to shear strength. The main track designs in use include those with a shallow (9–12 cm)

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dirt cushion lying directly on a hard foundation base, and deeper dirt tracks in which selective management of different depths creates a shallow cushion over either a 'false base' hardpan layer or a partially compacted pad. The level of maintenance required depends on design and rainfall, but in general dirt tracks weather well and can support heavy use.

Sand

Sand tracks are in wide use in training centres as a relatively cheap all-weather surface. They can support heavy use and are used for both foundation and fast work. Sand tracks vary widely in construction and material; training surfaces are typically deep, while those used for racing may have a shallow (7–8 cm) cushion layer over a hard substrate level. Incorporating a non-porous base enables some retention of moisture, thereby assisting with cohesion of sand particles. Maintenance requirements (regular harrowing/rolling, occasional top-ups) are generally lower than many other surfaces. Shear strength diminishes considerably when saturated.

Synthetics

Synthetic tracks were initially developed to provide consistent footing and a lower maintenance surface in the face of weather challenges and high-volume traffic. Synthetic surfaces vary in their composition between manufacturers, but generally consist of mixtures of sand, synthetic/natural fibres and non-compressible fillers (rubber/foam) coated with waxes, oils or polymers. Fibres provide shear resistance and act much in the same way as root structure does in turf tracks, while the waxes or oils bind the constituents together and repel water. Synthetic tracks are typically laid as a layer 15–25 cm thick (with a loose top 1/3) on a multilayer porous foundation of aggregate rock that serves to drain water away from the track, with central perforated drainage pipes beneath. Synthetic surfaces are generally softer than dirt and generate lower peak ground reaction, acceleration and hoof vibration forces than dirt and turf tracks to the extent that they have been proposed to reduce injury rates. Track characteristics (speed) can vary markedly with ambient temperature. Maintenance is crucial to provision of a safe and consistent surface, particularly with respect to minimising compaction at sites that experience high usage. Maintenance protocols vary with different products and geographical location/season but typically involve regular (daily/multiple daily) rolling/grooming and occasional top-up or refurbishment of degraded tracks. The working depth of the top surface can be altered with the depth of harrowing.

Woodchip

Woodchip surfaces are in wide use as all-weather training tracks. The construction characteristics of the foundation layers vary between sites. They are considered to provide safe, even footing if maintained (harrowed, rolled) regularly (daily/multiple daily) but replacement/resurfacing is required on a regular basis.

Track design

Tracks vary widely in configuration and design. Local topography and historical factors account for much of this variation. In addition to track surface, crossfall and other drainage factors contribute to a track's performance under different conditions and determine whether and how track bias may develop with weather, traffic and maintenance. The design of new tracks increasingly has to incorporate features that contribute to horse and jockey safety, that minimize disruption to race meetings from weather events and that take account of local water availability and quality. Design and banking of curves, and incline of straights affects speed and gait (lead changes, and stride length and frequency) and can have a direct influence on injury rates. More gradual transition between straight and curved portions of a track (increased radius of curvature), and banked rather than flat curves reduce incidence of injury.

Track condition and maintenance

The provision of a surface that is consistent in character during the course of a training session or race meet, or in the face of weather and climatic variables, is challenging. Hardness distribution may vary with location on the track, with propensity for greater localised compaction at sites such as just before certain curves. In general terms, the greatest influence on the mechanical characteristics of dirt and turf tracks is moisture, and on synthetic tracks it is temperature. The maintenance regimen required to provide a consistent surface therefore depends on geographical location and local weather variability.

Turf tracks require complex maintenance year-round that includes watering, mowing, soil aeration and thatch removal. Wear of the track can be managed by moving the inner rail to provide fresh ground. Dirt and synthetic tracks require regular light harrowing and grooming during use to prevent compaction from increasing the hardness of the track in areas of high use; dirt tracks are more susceptible to this compaction. Deeper tilling and rolling on a periodic basis is also important to freshen the track. Such maintenance has minimal impact on shear strength, which is determined by moisture (and clay) content in dirt

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tracks and fibre density of synthetic tracks. The mechanical characteristics (and thereby speed) of synthetic tracks change with ambient temperature due to changes in wax viscosity; peak temperatures in the top layer usually occur mid-afternoon. They can also change in composition over time, with degradation of fibres, loss of wax/oil and contamination with organic material; the need for top-ups and re-waxes is guided by regular depth checks and quality control.

Accurate determination of racetrack firmness is essential for trainers, ground staff and the betting public. Official pre-confirmation/declaration and race day reports of turf track conditions (ranging from 'heavy/soft/yielding' to 'firm/hard') have traditionally relied on subjective assessment, but instrumentation is now used routinely to derive the 'going' from aggregated measurements of cushion depth, penetration, shear strength or impact deceleration. Such measurements are interpreted in the light of local track characteristics and do not necessarily permit direct comparisons between tracks; they do, however, allow detection of variability in the track and can guide maintenance and watering efforts.

Racing strategies

The combination of innate athletic ability and fitness are clearly major determinants of racetrack performance. While many factors that occur during a race can affect outcome, the metabolic principles relating to energy production and expenditure remain fundamental to both the peak speed that a racehorse can attain, and the duration of time that it can sustain peak effort before the onset of fatigue. These metabolic constraints help to shape optimal racing strategy.

Aerobic fitness is the main contributor to athletic performance in races of all types, from sprint to staying distances; the longer a horse can derive muscular energy from aerobic pathways the better its performance is likely to be. However, the complex interplay between aerobic and anaerobic pathways is equally important as energy expenditure (and utilisation of available energy substrates) will vary depending on how, when and at what rate a horse accelerates during a race. The importance of pacing strategies is determined by race distance: over sprinting distances rapid acceleration to peak speed and subsequent maintenance of peak speed throughout the race is a feature, while for longer distances pacing (and conservation of energy) is important as it is not possible to maintain maximum athletic effort for the entire race over these trips. Additionally, drafting ('slipstreaming') is a rational strategy to minimise energy expenditure through reduction of drag, and can be used to advantage in horseracing just as in many other sports.

ERGOGENIC AIDS

An ergogenic aid is any external factor beyond normal training that can enhance physical performance (speed, strength or endurance). It can refer not just to pharmaceutical agents but to training aids and nutritional manipulations. Scientific support for the most commonly used ergogenic aids (human and equine) is sparse due to difficulties associated with measuring performance; as a result many are considered theoretically advantageous but unproven.

Ergogenic aids should always be viewed in the context that physical, training and genetic factors account for the major part of any athletic performance. In certain circumstances, however, there may be a rationale for the use of some products or methods. Specifically these should be scientifically valid, not harmful and comply with regulatory requirements. Many ergogenic aids used in people are not necessarily applicable to equine use because of metabolic and physiological differences between the species. The main groups of ergogenic aids that have been applied to human and equine pursuits are discussed here, with particular emphasis on their validity for use in the racehorse; these can be split into two broad categories: nutritional and non-nutritional aids.

Nutritional aids

Nutritional ergogenic aids act through modification of a nutritional metabolic pathway to delay the onset of fatigue. Dietary manipulation or the provision of a key nutrient can lead to greater availability of stored energy or greater efficiency of energy production. There may be multiple potential pathways to usefulness for some nutrients. When evaluating the merits of a nutritional supplement, considerations include not just potential efficacy of active ingredient(s), but interactions with diet (possible toxicity through over supplementation of certain nutrients) and potential for contamination with prohibited substances. Popularity should not be taken as evidence of efficacy.

Carbohydrate (glycogen) 'loading'

- Common practice in human endurance disciplines where duration of exercise is >90 minutes. High-carbohydrate diet combined with exercise taper 1–4 days out from competition can boost muscle glycogen stores and delay onset of fatigue during event.

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- Little rationale for use in racehorses: they have naturally high muscle glycogen content, and only small (around 10%) increases are possible with dietary manipulation; also, high- starch diet is associated with significant risks (colic, diarrhoea, laminitis).
- Horses: glycogen replenishment as an aid to recovery after intense exercise may be more useful (but only in limited circumstances); it takes 2–3 days on a normal diet to correct the 20–30% depletion of muscle glycogen stores resulting from high-intensity exercise.

Amino acids

- Important role in many metabolic processes such as 'building blocks' of protein and regulators of peptide synthesis.
- Amino acids with proposed ergogenic effects include glutamine, glycine, tryptophan and the essential amino acids (leucine/isoleucine/valine) known as BCAA that are found in significant quantities in skeletal muscle. Relative quantity of BCAA to aromatic amino acids in diet may be of importance.
- Amino acid supplementation ('protein shakes') is widespread in human sports: it is regarded as a potentially useful nutritional supplement to support muscle development (and reduced protein breakdown) in training. BCAA is thought to have a potential role in delaying the onset of central fatigue.
- While no significant ergogenic effect has been demonstrated in people or horses, there is some evidence that BCAA may act as an alternative energy source during exercise.
- Likely to have beneficial effects on recovery following intense exercise.

L-arginine

- Amino acid that is oxidised to NO.
- Proposed mechanism: increased NO production in skeletal muscle, causing vasodilation/greater blood flow and improved tolerance to aerobic and anaerobic exercise.
- Little evidence to support use in fit athletes (or horses).

Beta-alanine

- Naturally occurring amino acid.
- Substrate for synthesis of muscle carnosine, an important intracellular buffer in type II skeletal muscle.
- Oral supplementation of β-alanine increases carnosine content of skeletal muscle in humans and horses.
- Ergogenic effect in human athletes: delayed fatigue and increased capacity for exercise (for exercise lasting >60 seconds).
- Horses: good bioavailability; biological rationale for use but no current scientific validation of clinical effect.

Creatine

- Organic acid stored in muscle and derived from amino acids. Has a role in energy production within cell.
- Increasing muscle creatine content has an ergogenic effect (increased work capacity and peak strength) in human athletes.
- Horses: poor oral bioavailability (not a natural part of equine diet) limits use.

L-carnitine

- Enzyme essential to cellular energy production through transport of long-chain fatty acids across inner mitochondrial membrane.
- Increased muscle concentration theoretically advantageous.
- Horses: supplementation (oral or intravenous) has little effect on muscle carnitine content and therefore not considered to confer ergogenic benefits.

Antioxidants

- High levels of reactive oxygen species are produced in skeletal muscle during intense exercise.
- Oral antioxidant (e.g., vitamins C, E) supplementation is proposed to reduce oxidative stress and thereby improve muscle function.
- Most antioxidants have consistently been shown to have no effect either on performance or on prevention of muscle damage.
- Coenzyme Q10 is an antioxidant that also has a role in mitochondrial aerobic metabolism; it may have potential impact on efficiency of energy production. However, scientific understanding of possible benefits is limited at present. Supplementation has demonstrable effects on plasma and muscle levels.

Flavonoids

- Broad category of bioactive compounds present at low levels in many plants, seeds and fruits.
- Many different flavonoids have received attention in human sports for their potential ergogenic properties: quercetin, cocoa derivatives, green tea extract, anthocyanins (including black currants, grape juice) and mango leaf extract.
- Potential effects thought to be related primarily to antioxidant activity, although some modifications relating to energy metabolism have been documented.
- Diversity of compounds and mixed research results in humans mean that conclusions cannot be drawn

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at present regarding potential usefulness in equine performance.

B vitamins

- Water-soluble vitamins essential for proper cell function.
- Thiamine/riboflavin/niacin/pantothenic acid have roles in muscle energy pathways.
- Folate and B_{12} have roles in cell synthesis and repair.
- Exercise increases requirements for some B vitamins.
- Thiamine (B_1) supplementation has been shown to delay fatigue (increases the anaerobic threshold) in some human athletic pursuits.
- Horses: little research is available; however, injectable supplementation has demonstrable positive short-term effect on plasma levels of B_{12}.

Beetroot juice

- Rich source of nitrate.
- Metabolization to NO: possible effects on increased blood flow, gas exchange and strength of muscle contraction.
- In some human athletic disciplines an ergogenic effect on endurance performance has been demonstrated following chronic daily supplementation, although the magnitude of effect is small. Possible positive effects on post-exercise recovery and muscle soreness are also recognised.
- Available evidence from racehorses demonstrates no significant systemic uptake following oral supplementation; horses may lack transport proteins/enzymes necessary for digestion.
- No current evidence to support use in athletic horses.

Other nutrient supplementation

- Supplementation of nutrients over and above a well-balanced diet generally has no ergogenic effect (in both people and horses).
- Little support for megadose supplementation; high doses of some vitamins can be harmful.
- Plant and synthetic 'adaptogens' are a diverse group of pharmacologically active compounds from a variety of sources; long history of use in human sports to 'support' body's ability to cope with training (e.g., ginseng) but good research evidence of efficacy is largely lacking.

Sodium bicarbonate

- 0.3–1 g/kg BWT administered via intragastric route (nasogastric intubation) 2.5–5 hours pre-exercise ('milkshaking') reduces blood lactate acidosis and is widely believed to delay fatigue over middle-distance trips.
- Generally considered a performance-enhancing substance; administration on race day is prohibited and enforced through established threshold for blood CO_2 concentration.
- Variation in blood levels of CO_2 can occur with exercise, furosemide administration and between individuals, but the established threshold at >1 hour post-race used by regulatory bodies has been adequately validated.
- Diets containing sufficient quantities of bicarbonate or alkalinizing agents have the potential to result in a positive test.
- Current evidence does not appear to support claims that administration of alkalinizing agents improves performance significantly over flat racing distances.

Non-nutritional aids

Real/simulated high-altitude training (hypoxic/hypobaric aids)

- Low oxygen tension has the potential to stimulate production of red blood cells, conferring a potential advantage for later performance at sea level or as an aid to acclimatization.
- Achieved by true altitude (>3500 m) or hypoxic/hypobaric conditions.
- Two main strategies: 'live low-train high' and 'live high-train low'.
- Live low-train high: several weeks of daily treadmill cantering in hypoxic (typically <15% O_2, compared with normal level of 21%) conditions can improve athletic performance. Reduced oxygen supply to skeletal muscle during exercise can stimulate adaptations in muscle that favour glycolytic metabolism, thereby increasing anaerobic capacity.
- Live high-train low: may be the most beneficial ergogenic strategy but is best achieved by spending >16 hours/day (preferably 20–22 hours/day) in hypoxic conditions.

Inspiratory muscle training

- During high-speed exercise the metabolic cost of breathing can be high due to upper airway resistance and obligate nasal breathing. Anything which reduces the work of breathing or delays fatigue of muscles involved in respiration has the potential to aid athletic performance.

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- Resistance training through use of a valved mask results in increased strength of inspiratory muscles (upper airway and diaphragm) in racehorses, and has the potential to improve race performance (as it does in humans).
- Training involves short (few minutes, several times daily) sessions of breathing against the resistance created by adjustable valves; training takes place at rest.
- Measurable effects after 8 weeks of training.
- Protocol guidelines for optimal effect have not been established.

Autologous blood transfusions ('blood doping')

- Boosting of 'race day' red blood cell numbers by reinfusion of autologous blood obtained (and stored) 8–10 weeks prior to competition.
- Detection in humans relies on indirect methods such as longitudinal monitoring of biomarkers ('biological passport').
- As with other 'blood-boosting' aids, effect is likely to be less important in horses than in humans due to naturally large splenic reserve of red cells.

EPO

- Glycoprotein hormone originating from kidney cells that regulates the body's production of red blood cells.
- Produced in response to hypoxia or anaemia, and stimulates bone marrow to upregulate red cell production.
- Causes increase in circulating red cell numbers and haemoglobin, with resulting improvement in aerobic capacity.
- Potential for adverse side-effects including severe anaemia and/or thrombotic/embolic disease.
- Recombinant and biosimilar EPOs banned by sporting regulatory bodies.
- Detection capability for emerging analogues is a constantly evolving field.
- Horses: limited available information on efficacy; however, as with other blood-boosting aids, effect is likely to be of less importance than in humans due to naturally large splenic reserve of red cells.

Cobalt chloride

- Cobalt is an essential trace element that is essential for the synthesis of vitamin B_{12}.
- Present at low levels in dietary components and naturally present in urine and faeces.
- Also a component of commonly used injectable B vitamin supplements.
- Supraphysiological dosing can increase expression of EPO and thereby increase circulating red cell numbers (with potential to enhance aerobic performance, as mentioned earlier).
- Potential for adverse side-effects including cardiac arrhythmias and hypertension.
- Supraphysiological administration banned by most regulatory bodies and enforced through application of threshold.

Anabolic steroids

- Natural or synthetic derivatives of testosterone.
- Multiple anabolic effects including increased protein synthesis, increased lean body mass and reduced fat mass (partitioning), and enhanced recovery from injury.
- Use banned by most regulatory bodies.

Clenbuterol

- β_2-adrenergic agonist commonly used for treatment of equine respiratory disease.
- Demonstrable anabolic/partitioning effect in several species, although limited information available in horses.
- Any anabolic effect is likely to require prolonged administration.

Growth hormone

- Endogenous growth hormone is produced in the anterior pituitary gland.
- Multiple anabolic effects including increased protein synthesis, increased lean body mass and reduced fat mass (partitioning), increased power:mass ratio, enhanced cardiovascular function and recovery from injury.
- Range of synthetic substances (performance-enhancing peptides and non-peptides) may stimulate release of growth hormone ('secretagogues') and thereby stimulate anabolic effects.
- Perceived benefits from recombinant growth hormone doping in human sports exceed those determined scientifically.
- Use of exogenous growth hormone and growth hormone secretagogues banned by most regulatory bodies.

Gene doping

- Non-therapeutic manipulation of genes or expression of genes to improve athletic performance.

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- Genes involved in muscle metabolism and key energy pathways (e.g., myostatin, EPO, insulin-like growth factor-1, endorphin) are of greatest interest; these can potentially be manipulated either directly or through stimulation/inhibition of their expression (e.g., affecting the proteins that they produce) resulting in performance-enhancing effects.
- Potential for serious negative effects on health and therefore welfare implications.
- Delivery of 'gene therapy/doping' agent typically through use of a modified viral carrier ('vector').
- Use banned by most regulatory bodies; detection is difficult because rather than pharmaceutical molecules or metabolites the target agent is typically indistinguishable from naturally occurring substances (proteins). Constantly evolving field and development of reliable blood tests (e.g., immunological profiling to identify exposure to viral vector) and use of biological passports likely to enhance detection in future.

CAN RACEHORSES GET ANY FASTER?

- The Thoroughbred population is highly inbred as a result of selective breeding and a closed gene pool.
- This has led to speculation that athletic potential of the breed has 'peaked' and that further improvements in speed should not be expected.
- Race times of the average racehorse have improved over the past 150+ years; however, these improvements have not been linear.
- Analysis is complicated by many non-genetic factors such as training methods, nutrition and jockey technique.
- Current evidence suggests that racehorses have reached/nearly reached their absolute athletic potential over middle and staying distances, but that sprinting performances are continuing to improve.
- There remains potential for further athletic improvement in the breed with the application of new training (and other environmental) factors in combination with evidence-based genetic selection.
- Genetic variation remains an important factor in what makes a successful racehorse.

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CHAPTER 3

RACEHORSE INJURIES

INTRODUCTION

Horses hold a unique place in the animal world for the great variety and global distribution of athletic pursuits in which they participate. As with all athletes, racehorses are exposed to training loads that may result in musculoskeletal injury and, in common with all sports, experience a range of discipline-specific injuries. These reflect both the anatomical adaptations of the racehorse and the biomechanical demands arising from the high-magnitude limb loading particular to racing, and occur at locations and with configurations that are highly predictable. Although injury patterns may vary with training style or local conditions, the genetic and physical uniformity of the Thoroughbred population ensures a similar spectrum of pathology regardless of geographical location. In racehorse practice a small number of musculoskeletal conditions are encountered with great regularity, with the variety of clinical presentation and outcomes coming from individual expression of similar pathologies.

Injuries (specifically fractures) of sufficient severity that they merit euthanasia on humane or practical grounds are rare events; however, as they predominantly occur on the racetrack in view of a public audience they have understandably become the yardstick by which the welfare of racing's equine participants is judged. The overall prevalence of musculoskeletal injury in horseracing actually compares favourably to that seen in some human athletic populations (e.g., military recruits and track and field athletes); however, the consequences of injury at high speed can be much more serious for horses. The ratio of non-fatal to fatal fractures sustained on the racetrack is reported to be >3:1, and <10% of all fractures sustained during racing and training result in fatality. The majority of catastrophic injuries predominantly involve the fetlock or its support structures, and most (70–80%) occur in the forelimb. Risk of all-causes fatality is considerably greater (typically by a multiple of >5) in jump racing than flat racing and reflects the increased likelihood of falling, as well as longer distances raced. Not all race day fatalities are the result of fracture, with up to 20% caused by cardiovascular or respiratory incidents.

It has long been recognised that the majority of musculoskeletal pathologies encountered in racehorse practice are repetitive stress injuries and a consequence of fatigue microdamage that accumulates over sometimes lengthy time frames, rather than of acute insult. This is substantiated by extensive postmortem research programmes as well as a range of empirical evidence that can be simplified to the basic premise that training loads applied to excess or for excessive periods lead to biomechanical failure of musculoskeletal tissue. That catastrophic fractures are generally preceded by prodromal pathology provides opportunities for reducing fatality rates through early detection of injury, and it follows that a key responsibility of racehorse clinicians in the modern era is risk management to safeguard the musculoskeletal health of horses under their care.

Fracture terminology

When describing fractures it is important to include location of injury, relationship to joints (whether intra- or extra-articular) and growth plates, orientation (transverse, oblique, spiral) and degree of displacement. Standardised terminology relating to fractures is shown in Table 3.1.

WASTAGE

'Wastage' is a term transposed from human labour resource analysis and in the context of the racing industry is used to describe numerical attrition from the 'pool' of racehorses over time. It is generally used in the context of an annual cohort of animals, and encompasses losses (from all reasons, not only injury) representing all stages of development, from failed pregnancies through to end-of-career injuries. In the past, research into the levels and types of wastage has focused on the economic impact of losses; however, latterly the interest of the public and of racing regulators has been the welfare and ethical implications of how and why racehorses exit the industry, and where they end up after racing.

While reasons for and rates of horses leaving racing can vary considerably between geographical locations and over time (for instance, in response to economic cycles), previous research provides some information that is broadly applicable to most jurisdictions. Features of

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Table 3.1 **Fracture terminology**

TERM	DESCRIPTION
Incomplete	Fracture that does not completely traverse the bone.
Complete	Fracture that completely traverses the bone, with total separation into two or more fragments.
Non-displaced	Fragments remain in normal anatomic alignment.
Displaced	Fragments are out of normal anatomic alignment; may be separated or overriding.
Comminuted	Fracture configuration that divides the bone into more than two pieces.
Closed (simple)	Skin overlying the fracture is intact.
Open (compound)	Skin overlying the fracture is broken.
Slab	Biarticular fracture that extends completely through the bone to involve both the proximal and distal articular surfaces.
Avulsion	Fragment distracted from parent bone due to tension placed upon it by a ligamentous/tendinous attachment.

career progression and attrition from the flat racehorse cohort from yearling stage onwards are as follows:

- Approximately 40–50% of yearlings will race by the end of their 2 YO season.
- Time to first start is not influenced by whether horse was born early, mid or late foaling season.
- Approximately 20% of yearlings will not have started a race by the end of their 3 YO season.
- Median age at retirement is 5 years.
- Females are more likely to remain unraced than males.
- Females that do race have shorter average careers and lower average number of race starts than males.
- Likely that 15–20% of the racehorse population retires each year.
- Retirement is predominantly voluntary and based on factors such as performance or owners' requests.
- Injuries represent a minority proportion of reasons for retirement.
- Less than 5% of musculoskeletal injuries (and <10% of fractures) are fatal.
- More than 75% of horses sustaining non-fatal fractures will race again.

EPIDEMIOLOGY OF RACEHORSE INJURIES

Overview

Most research and public interest to date regarding racehorse injury rates has centred on race day fatalities or serious injuries, as these are both high profile and the easiest data to record and analyse. The majority (approximately 80%) of racehorse injuries, however, occur during training rather than racing, although these are predominantly non-fatal. Few studies of training injuries exist, but from those that do it is clear that while a high proportion (>25%) of horses experience an injury setback at some point during their racing career these for the most part resolve without complication and simply result in temporary interruption to training rather than in curtailment of career. Clinical syndromes relating to skeletal adaptation to training loads, such as dorsal metacarpal disease ('sore shins') or 'splints', bone stress injuries at predictable predilection sites, osteochondral injuries of the middle carpal and fetlock joints and a small range of tendon and ligament strains make up the majority of conditions encountered in the training population. Conversely, while injuries sustained during racing or high-speed training exercise are less frequent, they are more likely to be serious or catastrophic because of the extreme loading conditions the limb is under at the time of tissue failure; in consequence the majority (>60%) of racehorse fatalities arising from injury occurs on the racetrack. The best data that permit an overall view to be taken of risk of developing clinical injury (catastrophic or otherwise) in a racehorse population come from jurisdictions where horses are trained under close regulatory supervision (principally Hong Kong and Japan). From these, it appears that fractures account for approximately 40–50% of total injuries, with soft tissue injuries (involving the flexor tendon or suspensory ligament) accounting for 30–40% of the total.

Defining and measuring injuries

Routine collection of data concerning serious and catastrophic race day musculoskeletal injuries occurs in many racing jurisdictions. Definition of what constitutes such an injury (particularly the time frame beyond race day that qualifies a case for inclusion) and the way it is diagnosed

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(clinical, postmortem examination or aided by imaging) varies widely however, such that direct comparison of injury rates between jurisdictions is problematic. By way of example the Equine Injury Database in the USA records data on horses that die or are euthanized within 3 days of injuries sustained during racing, the British Horseracing Authority within 2 days of racing and the Hong Kong Jockey Club records fatalities up to 5 days post-race. Data obtained without the benefit of postmortem examination is inherently flawed, and in the case of jump racing there is the additional confounding factor that horses that fall (or die) for other reasons (such as cardiovascular failure) may present at postmortem with fractures of the upper limb or spinal column sustained secondary to (and not causative of) the fatal incident.

Furthermore, horses that have sustained injuries that are potentially treatable but are subsequently euthanized electively at the request of connections may not be captured in 'fatality' or 'catastrophic injury' data at all, depending on jurisdiction. Additionally, it is known that race day injuries and fatalities constitute only a proportion of those experienced by the racehorse population as a whole, yet accurate and reliable reporting of injuries sustained during training is a rarity in the scientific literature. As a result it is currently very difficult to compare relative rates of different injury types (and severity) between racehorse populations, whether those populations are at a local (different training yards) or international level. It is also noteworthy that discussion of fracture and race day injury rates is completely separate and distinct from current understanding of some of the fatigue-related pathologies affecting subchondral bone (particularly in fetlock and carpus): the latter are common and are certainly classified as injuries, but relationships between clinical and postmortem findings are undefined at present.

Risk of injury and injury rates are two distinct ways of measuring occurrence of injury in an athletic population. Race day injuries are typically expressed in terms of incidence. *Incidence* in sports medicine epidemiology refers to the number of new injuries during a specified time period and is calculated by dividing the number of new injuries by the number of exposures to risk: in horseracing, incidence is generally reported as number of injuries (or fatalities) sustained per 1000 race starts. It is best suited to describing acute events such as catastrophic fracture and used in this way it is a direct measure of average injury risk (i.e., the risk of any single horse sustaining a serious/fatal injury if it starts a race). *Prevalence* by contrast is the proportion of individuals within a population who have an existing injury at a given point in time, and is therefore best suited to describing some of the fatigue/overuse/chronic musculoskeletal pathologies seen in the racehorse.

Regional variations and trends in injury patterns

Rates and types of serious race day musculoskeletal injury differ between racing jurisdictions, and reflect a range of influences such as training strategies and track surfaces. Globally, the average incidence of catastrophic injury sustained during racing is approximately 1/1000 starts, with individual figures for racing jurisdictions for which information is available noted in Table 3.2. As noted previously, methodology for race day injury surveillance programmes

Table 3.2 **Catastrophic musculoskeletal injuries (CMI) during flat racing**

COUNTRY	CMI (PER 1000 STARTS)	DATA PERIOD	PREDOMINANT FRACTURE (WHERE DATA AVAILABLE)
USA	Overall: 1.40 Dirt: 1.50 Turf: 1.26 Synthetic: 0.88	2020–2021	PSB
Great Britain	Overall (turf + synthetic): 0.7	2000–2013	Metacarpal condyle
Australia (NSW)	Turf: 0.52	2009–2014	PSB
Hong Kong	Turf: 0.6	2004–2011	PSB
Japan	Turf: 0.82	2017–2021	
South Africa	Turf: 0.56	1998–2012	PSB
New Zealand	Turf: 0.41	2005–2011	

PSB, proximal sesamoid bone.

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varies between jurisdictions and comparison of some injury metrics can be problematic. Recording of dominant injury type associated with race day fatality/euthanasia is, however, generally more consistent. In all countries the forelimb fetlock is the predominant site of race day fracture or breakdown, with either the proximal sesamoid bones/suspensory apparatus (USA, AUS, HK, SAF) or distal cannon (UK) being the most common injury.

Regardless of jurisdiction, the broad trend of recent decades has been of steadily diminishing rates of catastrophic race day injuries. This has come about through greater awareness of the underlying causes of many pathologies and the implementation of measures that modify some contributory factors, such as safer track surfaces/design and tighter medication controls. It is highly likely that with the sustained interest of stakeholders this direction of travel will continue such that greater alignment (or even parity) of overall injury rates between the major horseracing nations might be expected in future.

COMPARING INJURY RATES

- Comparing injury rates between racing jurisdictions, training centres/tracks, trainers and time periods is fundamental to understanding risk factors and trends and determining success or otherwise of injury prevention strategies.
- Patterns of both serious/catastrophic racetrack injuries and non-fatal training injuries are of interest.
- Little uniformity in injury definitions and methods used for data capture (past and current) both by racing regulators and in research publications.
- Consequently, it is very difficult to make meaningful comparisons in injury rates between countries/training centres/yards (an example of estimated incidence of different types of musculoskeletal injuries in an average Newmarket, UK, flat racing yard is found in Table 21.1, p. 439).

CAUSES OF INJURY

The development of injury in the racehorse is complex and multifactorial, with many interactions between contributory risk factors. Broadly, various biological factors (e.g., genetic, sex, conformation, previous injury) may predispose a horse to development of a particular pathology, added to which exposure to external risk factors (such as training strategies/loads and medications) creates a horse 'at risk' of injury. The final mechanism that leads to clinical injury is often an inciting event, such as the extreme loading of the affected limb that occurs during high-speed exercise. Fractures during racing most commonly involve the leading forelimb. The horse-, exercise-, race- and management-level risk factors about which most is known are summarised in Table 3.3, and some of the key variables are discussed briefly later.

Horse-level factors

Genetic and heritable predispositions to injury are known to exist but associations are still being investigated. Prior to the availability of molecular genomic studies, limb conformation and to a lesser extent familial histories of racing soundness were the main indicators used in racehorse husbandry to assess risk of future injury, although statistical investigations of even these factors have been limited. As the strength of musculoskeletal tissues and their ability to repair/remodel are determined by cellular activities, it is understandable that some aspects of injury susceptibility have an underlying genetic basis: while fracture risk is complex with multiple genomic regions contributing, it is likely that useful genetic markers will be identified in future that assist risk profiling of individuals.

Despite long-held assumptions that the training of 'immature' horses is a potential welfare problem in racing, there is abundant evidence that exposure of young (≤2 YO) horses to high-speed exercise results in equine athletes that are more robust, less prone to injury and fatality over the course of a career and more likely to have longer, more successful careers (more starts, more wins, more years in training) than horses that start training later in life. Adaptation of the musculoskeletal tissues to training loads must happen in all racehorses before they can reach the racetrack; however, the innate adaptive processes that permit this are far more efficient in the yearling/2 YO than in the older horse. Although the incidence of injuries during the 2-YO season is greater than for other age-groups, these injuries are generally of the minor/adaptive type: risk of fatality is consistently lower than that of older horses. Horses that start their first race later in life have a higher risk of suffering fatal injury.

While high-speed exercise is necessary for successful skeletal adaptation, it is also clear that above a certain threshold it can become damaging. The slow accumulation of bone (and soft tissue) microdamage over time, particularly at important sites in the fetlock, is thought to explain the increased risk of distal limb catastrophic injury that occurs with increasing age.

Exercise-level factors

Cumulative exposure to high-speed exercise (defined as training/racing at speeds ≥48 km/h/13.3 m/s/15 s/furlong) beyond that which is necessary for healthy skeletal

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Table 3.3 **Factors contributing to risk of catastrophic musculoskeletal injury in flat racing**

	RISK FACTOR		EFFECT	LIKELY REASON
Horse-level	Age		Risk increases with age.	Accumulation of microdamage.
	Sex		Entire males (colts/stallions) at >50% greater risk of injury than females/geldings.	Reasons undetermined at present but likely to be multifactorial – both horse level (weight, biomechanics) and managerial level (fillies more likely to retire early; therefore less accumulated microdamage).
	Genetics		Possible genetic basis for susceptibility to fracture risk.	Associations have not been defined at present. Possible that heritable traits relating to collagen cross-linking, conformation and speed will prove to have influence on injury risk.
	Exercise history	Age at first start	Increased risk of injury for horses that start first race later in life (≥3 YO).	Musculoskeletal adaptation to training loads much more efficient at young age (≤2 YO) than when already skeletally mature.
		First year of racing	Increased risk of injury during first year of training (irrespective of age).	Horses have to go through a cycle of training to adapt to high-speed exercise and are at elevated risk until this adaptation occurs.
		Cumulative high-speed exercise*	Risk increases with total distance exercised/raced at high speed over career.	Beyond initial conditioning (during which high-speed exercise is necessary/protective), high-intensity loads cause accumulation of microdamage and inhibit some innate regenerative processes.
		Average high-speed exercise/day	Risk increases with greater average distance trained at high speed per day.	Accumulation of microdamage.
		Performance	Better-performing horses (or horses expected to perform, as judged by starting race odds) at greater risk of injury.	Reasons undetermined at present.
Race-level	Track surface		Turf: in most jurisdictions is the safest surface relative to synthetics and dirt. Dirt associated with >30% greater risk of fatal injury compared to synthetics. Aside from racing, some evidence that greater training exposure to dirt is an important risk factor for proximal sesamoid bone fracture.	Loading impact on limb, resulting fetlock hyperextension, and limb slide greater for dirt than synthetics (synthetics: more cohesive surface and greater shoe-surface grip interaction). May contribute to both acute and fatigue injuries.
	Track condition		Turf: higher risk of both fatal and non-fatal injury with faster/firmer (drier) tracks (risk of serious injury >3 times that of heavy track). Dirt: higher risk with high water content (muddy/sloppy), poorly cohesive tracks. Synthetics: variations in injury rate less notable and primarily related to track maintenance (e.g., time elapsed since surface refurbishment/change)	Turf: fast/firm tracks = higher speeds, greater loading forces on limbs, greater fetlock extension, less cushioning effect, rapid deceleration, increased impact. Dirt: lower cohesion with sloppy tracks limits support during propulsion phase.
	Track geometry		Injuries are more likely to occur on a bend than on a straight segment of track. Uphill training tracks are associated with higher incidence of hindlimb injuries.	Altered loading of limbs on bends: greater peak impact forces on outside limb (providing in correct lead). Greater strains with smaller turn radius; greater angle of inclination of cannon bones on flat compared with banked curves. Uphill tracks: greater peak forces in hindlimb and increased stride frequency (more loading cycles).

(Continued)

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Table 3.3 **Factors contributing to risk of catastrophic musculoskeletal injury in flat racing *(Continued)***

	RISK FACTOR		EFFECT	LIKELY REASON
	Seasonality		Increased risk coinciding with racing season.	Greater intensity of workload (training and racing) leads to accumulation of fatigue pathology.
	Race distance		Mixed evidence: risk of injury in most jurisdictions increases with longer race distances; data from USA show risk is greatest over middle (6–8 f) and sprint (<6 f) distances.	Possible role of fatigue (with effects on limb loading) +/or speed.
	Race type		Greater risk of injury with both low-value claiming/selling races, and elite (Group/Grade 1) races.	Claiming/selling races may be utilised by trainers to sell horses with pre-existing orthopaedic conditions; this may predispose them to injury during the race. Increased risk experienced by horses running in elite races is of uncertain origin but may relate to faster speeds/greater competitiveness at this level.
	Jockey experience		Less successful, and non-professional jockeys, more likely to ride a horse sustaining fatal injury than successful jockeys.	Reasons undetermined at present but proposed that experienced jockeys may recognise injury/injury risk earlier and pull horse up before catastrophic injury ensues.
Management	Medication		Complex relationship between risk of injury and previous/recent administration of intra-articular medication (primarily corticosteroids). Increased risk with horses that have previously received multiple (>2) medication sessions. Increased risk of fatal injury following pre-race administration of phenylbutazone (where permitted).	Medications (both systemic and intra-articular) that eliminate/diminish early clinical indicators (lameness) of impending injury can lead to horses being trained/raced when they are at risk of serious injury; also possible effect on limb loading patterns.
	Previous injury		Currently unknown.	Relationship between previous injury and risk of future serious injury is complex and has not been fully investigated. Likely that some injuries (MC3/MT3 condylar fracture, pelvic-iliac fracture) may be associated with increased risk of future fracture.
	Trainer		Injury (fatal and non-fatal) rates differ between trainers. Additionally, some mixed evidence that risk of injury may be greater for horses trained by successful trainers relative to those trained by poorer performing trainers.	Many variables likely to influence differing injury rates between yards including training strategies, quality of horses and riders. Possible that better average racetrack success may arise from more intense training strategies but that consequence is increased risk of injury.

* High-speed exercise defined as training/racing at speeds ≥48 km/h (13.3 m/s; 15 s/furlong).

adaptation/maintenance is known to be a risk factor for development of injury. While research has produced some numerical outcomes for the increase in odds of incurring injury with average daily and total cumulative distances trained at high speeds, robust practical advice is limited. In general terms low and high (or high, without rest periods) volumes of fast work are considered undesirable, but the point at which the influence of high-speed exercise changes from being protective against injury to being potentially harmful in any given individual cannot currently be predicted. The optimal levels of fast work required for health, when it is best introduced into a training programme and the influence of training practices and facilities all need further investigation.

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Race-level factors

Most serious/catastrophic race day injuries involve the forelimb fetlock. The fetlock of the racehorse is put under extreme loads during high-speed exercise (Figure 6.27, p. 99), and probably functions at close to its biomechanical limits. Factors that either increase or alter the direction of loading forces through this crucial articulation, or that weaken or inhibit the capacity of the articulation to bear these loads (such as subchondral microdamage or ligamentous pathology), can therefore lead to serious injury. Track surface type, condition and geometry all affect the way the foot lands and decelerates, thereby affecting the magnitude and direction of loads imposed upon the limbs of the galloping horse. While there is some variability between jurisdictions, sufficient injury data exist to state in general terms that, all else being equal, average risk of race day serious injury is lower on synthetic surfaces than on dirt by more than 30%. Depending on jurisdiction (and presumably related to weather conditions, maintenance and track geometry) turf is generally regarded as either the safest racing surface or one that holds an intermediate position between dirt and synthetics in terms of overall risk of serious injury. Risk of proximal sesamoid bone fracture is greater on synthetics than on grass. Hoof-track surface interactions that influence injury risk are complex, however, and the mechanical properties (vertical firmness and horizontal shear strength) of a surface arising from preparation, maintenance and moisture may in many circumstances be more important to generation of injury than the type of surface material. Racecourse operators strive to maintain racing surfaces that are consistent and safe; however, it is inevitable that variables such as weather, maintenance strategies, sites of heavy use and in the case of synthetics, degradation and refurbishment can affect the condition of a track (or part of a track) on any given day. For both dirt and turf, variation in injury rates is most often associated with moisture content of the track: higher risk on dirt occurs with sloppy, poorly cohesive surfaces while higher risk on turf and synthetics occurs with dry or fast tracks.

Management factors

Medication

Medication use (specifically that used for managing musculoskeletal pathology, such as corticosteroids and non-steroidal anti-inflammatory drugs) and its association with race day injuries is a controversial topic and one which has not been comprehensively resolved statistically. That there is conflicting research about whether anti-inflammatory medications increase the risk of serious injury may be explained by differences in prevalence of medication use in the population of interest, and the way that it is applied. Where medication use is commonplace, it is difficult to investigate any influence on injury risk because both injured and uninjured cohorts may have received treatments. Conversely, in populations in which medication use is less common, differences in injury rates can be expected between animals that have undergone thorough diagnostic investigations prior to administration, and those that have received medication without any appropriate risk assessment.

Trainer

Rates of both race day and in-training injuries can vary significantly between trainers, even when they are using the same training facilities. These differences have received little research attention to date; however, factors such as training strategies, value/pedigree/origin of horses, quality of staff/riders, management of lame horses, experience and veterinary input are likely to be major contributors to this variation.

MANIFESTATIONS OF INJURY

Lameness

Most clinicians, trainers and riders would consider they understand the concept of lameness (and soundness) enough to recognise it in horses under their care, given that it is a routine aspect of racehorse husbandry. Describing lameness objectively and determining the role that pain or musculoskeletal dysfunction plays in observed gait abnormalities is, however, an area of considerable scientific uncertainty. Additionally, consensus on the best ways to measure and grade lameness remains elusive. Some important aspects of lameness and its place in management of the racehorse follow next.

What is lameness?

Lameness can best be defined as a clinical sign of pain, functional or structural disorder manifested as an alteration of normal gait.

While lameness frequently results in an asymmetric gait, this is not always the case; asymmetric gaits can also exist in the absence of lameness, and hence the terms 'asymmetry' and 'lameness' should not be taken to mean the same thing.

The term lameness has welfare connotations for the public; however, whether (or to what degree) lameness actually represents 'experienced pain' is difficult to determine and varies among individuals. Pain thresholds differ between horses and as in people chronic pain (in which sensory pathways are often altered) may be experienced very differently from acute pain.

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What is 'soundness'?

'Soundness' is one of the most commonly used terms in relation to the equine athlete and is generally taken to mean the absence of lameness, although it can also be interpreted in a looser fashion as indicating musculoskeletal health or 'fitness for training/racing'. Given that lameness itself can be difficult to define and detect and in practice is largely a subjective concept, it follows that soundness is also a subjective rather than an absolute determination.

When trotted in straight lines, it is generally considered that a horse without lameness should display a high degree of symmetry in the movement and loading of the left and right diagonal limb pairs, as well as left-right symmetry in the motion of the head, neck and pelvis in relation to footfall. Exceptions to this rule would include horses that have an asymmetric gait not considered to be arising from lameness. Reasons for asymmetric gait in the absence of lameness include physical asymmetry (such as conformational faults and differences in foot shape/size/balance, muscle atrophy or previous injury such as pelvic fracture) as well as laterality or 'handedness' (the favouring of one limb or side of the body over the other, as in people).

Observing lameness

As trotting is a symmetric gait, it is the standard one used to assess lameness. In racing practice it is most common to conduct lameness assessments with the horse being trotted in hand in straight lines while observing from both in front/behind and from the side. Observations are also frequently made of the horse being ridden (typically in an oval-shaped warm-up trotting ring or arena) from the side. Side observations permit better assessment of foot flight arc height and degree of extension (and thereby of loading) of the fetlocks. Surface used, and trotting speed can significantly affect observed lameness, and should therefore be used with consistency during a lameness examination in order not to confound interpretation. Ability to visually detect subtle lameness is known to diminish with faster trotting speeds, and evaluation at slower speeds is recommended. Sedative agents may affect (predominantly hindlimb) coordination and limb placement and can therefore interfere with assessment of mild lameness; however, they may also be useful in maintaining consistency of the trot-up in horses that are fresh or unruly.

It is important to note that lameness or gait abnormality observed at trot does not always correlate to gait abnormalities at other paces, and conversely that apparent freedom from lameness when trotted in straight lines does not necessarily indicate an absence of musculoskeletal pain or pathology.

Patterns of lameness

Limb(s) of origin of lameness may be associated with characteristic observable patterns of movement as follows:

Forelimb lameness: typically observed as an asymmetric head nod, with the head/neck/wither dipping down when the non-lame limb strikes the ground (and rising when the lame limb is in stance phase).

Hindlimb lameness: greater individual variability and complexity in movement patterns exists for hindlimb lameness than for forelimb lameness. When viewed from behind, the vertical movement of the bony landmarks of the pelvis (principally the tuber coxae) can be used to assess hindlimb lameness. Vertical displacement ('hip hike') just before foot-ground contact is greatest on the lame side; pain during push off (impulsion) may inhibit the degree to which the pelvis rises and these factors are usually manifested as a greater 'dip' of the pelvis on the lame side. The pelvis may also rotate towards the lame limb, increasing the complexity of assessment as it can affect the detection of tuber coxae movement. Depending on severity, hindlimb lameness can also induce a head nod, which may mislead the observer into considering the presence of forelimb lameness. Some hindlimb lameness is also associated with reduced flexion or protraction of the lame limb, result in consistent or intermittent catching of the toe on the ground.

Bilateral lameness: Because observer perception of lameness typically relies on distinguishing asymmetric features in a horse's gait, the presence of bilateral lameness can inhibit detection of musculoskeletal pain. Features of bilateral lameness that may assist the observer are primarily related to limb placement: in the forelimb this is most commonly manifested as wider placement of the limb(s), while in the hindlimb for many bilateral pathologies the limbs contact the ground closer to midline (and may even cross over during protraction: 'plaiting'). A bilateral (or quadrilateral) lameness at trot is typically associated with a shorter suspension phase between the diagonal stance phases, which gives the impression of a short-stepping 'choppy' gait. At canter pace a bilateral hindlimb lameness may cause poor impulsion from the hindlimbs or alteration in timing of footfall and manifest as a 'bunny hopping' gait.

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'Tracking off': in a horse with a symmetric gait at trot the hindlimbs should normally follow the 'track' of the forelimb on the same side, resulting in a 'two-track' gait. Lameness, or musculoskeletal pain arising from the topline, trunk or pelvis may cause a horse to drift off to one side (thereby effectively moving on three 'tracks'): most typically, direction of 'drift' is away from the lame/lamest side.

How is lameness best detected?

Lameness assessment has traditionally relied upon visual observation of a horse's gait under a variety of conditions. This is inherently a qualitative and very subjective process, and studies have consistently shown high variability between observers in detection and grading of lameness. However, humans are known to be highly effective at pattern recognition, and there is ample empirical evidence in equine sport that regular observation of horses trotting under standardised conditions can lead to acquisition of great expertise in appraisal of equine gait and considerable alignment in opinion between experienced observers. In horseracing such expertise is frequently found among both non-veterinary and veterinary personnel.

While recognition of lameness patterns by the brain may be excellent, limitations exist in terms of spatial and temporal resolution. Bias may also significantly impair true assessment, as knowledge of or assumptions about a horse's history or recent interventions may strongly affect whether (or by how much) a horse is judged to be lame. To counteract these influences, objective gait analysis is increasingly used to assist clinical decision-making. Quantitative analysis of the complex movements of limbs and body can be achieved through the use of horse-mounted inertial measurement units or camera-based data acquisition. These analyses can be undertaken at all paces, and it is expected that documentation of gait in a variety of situations from lameness investigations to pre-purchase examinations will increasingly benefit from utilisation of technology. As asymmetries (or particular features) of gait do not strictly equate to diagnosis of lameness, such objective gait analysis should be used to aid rather than supplant observer interpretation within the context of the clinical concern. Given the wide biological variation in clinical presentation and outcome of equine lameness, it is likely that gait analysis, as with subjective assessment, will prove to be most beneficial when used longitudinally to build up an information profile relating to the individual, rather than in comparisons between horses or against arbitrary threshold values.

Localising the source of lameness in clinical practice often involves utilising local anaesthetic agents to desensitise regions of the affected limb and thereby rule them in (or out) of the investigation. Local anaesthetics may be instilled around peripheral nerves with the object of blocking sensory transmission from particular anatomical regions (and generally applied progressively higher in the limb), or deposited within synovial structures such as joints or tendon sheaths. Both nerve blocks and intra-articular/thecal analgesia can affect regions remote to the intended structure as well as occasionally fail to abolish lameness from the structure injected, and awareness of this lack of specificity is important when conducting a lameness investigation. It is generally considered that substantial improvement in baseline lameness following local analgesia may be interpreted as a positive response.

Grading lameness severity

There is currently no universally accepted grading system for lameness that both allows for the wide range of clinical presentations encountered in racing practice and that can be applied with consistency by different observers. Such a system would necessitate precise and reproducible definition of gait features, which is not possible to achieve based on subjective evaluation. Additionally, grading systems typically rely on interpreting severity of gait asymmetry, and therefore cannot permit grading of bilateral or quadrilateral lameness. Aside from numerical scales, lameness can be classified as 'mild', 'moderate' or 'severe/marked'; however, these are also subjective and undefined categorizations.

Despite their inherent flaws, it is commonplace for clinicians to utilise numerical grading systems to record observations about lameness. These include the 0–5 grade AAEP grading (Table 3.4) that predominates in the USA and more widely in the scientific literature, and a 0–10 grade scale more common in the UK. One of the chief problems associated with the AAEP system is its insensitivity for lameness severity: the large majority of racehorse lameness must be given either the grade 2 or grade 3 assignation, which prevents adequate differentiation of widely differing clinical presentations. The ideal grading system would be one with sufficient categories to accommodate this differentiation, that could be applied independently to walk and trot and to which some narrative description of gait characteristics (such as 'toe catch', 'reduced protraction' and 'intermittent') could be added.

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Table 3.4 **AAEP lameness scale**

GRADE	DESCRIPTION
0	Lameness not perceptible under any circumstances.
1	Lameness is difficult to observe and is not consistently apparent, regardless of circumstances (e.g., under saddle, circling, inclines, hard surface, etc.)
2	Lameness is difficult to observe at a walk or when trotting in a straight line but consistently apparent under certain circumstances (e.g., weight-carrying, circling, inclines, hard surface, etc.)
3	Lameness is consistently observable at a trot under all circumstances.
4	Lameness is obvious at a walk.
5	Lameness produces minimal weight-bearing in motion and/or at rest or a complete inability to move.

Variation

Musculoskeletal pathologies experienced by racehorses are associated with a wide range of lameness and gait patterns. The relationship between type and stage of pathology and demonstrable lameness is highly variable between both conditions and individuals, and may vary over short and long time frames and under different conditions (e.g., ridden/in-hand, track surface, concurrent medications). Associations between pathologies and practical manifestations such as whether a horse might 'warm up' during an exercise session, or worsen (or improve) after high-speed exercise have received almost no research attention and while patterns are recognised and relied upon by experienced clinicians the evidence base is currently solely empirical. In general terms the correct management of chronic/long-term pathologies or lameness in the racehorse is greatly assisted by regular and ongoing observations of gait under different conditions and in response to different intensities of workload. Individual horses with chronic pathologies tend to have their own characteristic and repeatable gait/lameness patterns, and it is deviation from what is 'normal' for each horse that tends to be more useful in injury risk management than the presence of lameness per se.

Relationship with performance

Whether (or to what degree) the presence of lameness may affect performance (particularly race performance) is a matter of considerable interest given that many veterinary interventions in racing practice are aimed at abolishing or managing it. It is reasonable to expect that at high speeds, when the horse is utilising an asymmetric gait (gallop), pain arising from a limb(s) may introduce some inefficiencies into locomotion through alteration of stride, with consequences for energy utilisation and thereby onset of fatigue. This relationship, however, remains theoretical and is balanced by a large amount of empirical evidence that lameness (as judged by assessment at trot) does not necessarily translate to impairment of performance: many elite racehorses 'fail' pre-purchase examinations on the basis of detected lameness. As with many other aspects of lameness in the equine athlete, it is best to base clinical interpretations on knowledge of individual circumstances rather than generalisations.

LAMENESS RULES OF THUMB

- Most clinically significant observable racehorse lameness can be localised to joints or bones (rarely muscular).
- Severity of lameness should not be assumed to correlate with seriousness of pathology.
- Common pathologies often present with consistent patterns; history and clinical features are strong indicator of likely origin of problem.
- How lameness changes over time and in response to training loads can guide whether/when diagnostic investigation is warranted.
- Lameness arising from stress or fatigue fracture may be temporarily abolished with a few days of light exercise; care should be taken when resuming faster paces.
- Ruling out the fetlock early in any investigation through blocking/imaging should be considered a priority.
- Many perineural and intra-synovial analgesic blocks have an effect distant to their expected region of influence; it is important to be aware of the potential for blocking patterns to mislead.

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CHAPTER 4

ACUTE CARE AND WOUND MANAGEMENT

INJURY MANAGEMENT ON THE TRACK

The clinician attending an injured horse on the track usually finds themselves in charge of managing the immediate situation rather than just the horse, including directing the actions of horse handlers and ground staff. If not already aware, ground staff should be notified to implement closure or rerouting of the track, and horse recovery transport mobilized. At a race meeting, evaluation and treatment should be conducted behind screens or vehicles. The goals of emergency care on the track are to relieve the horse's pain and anxiety, assess the severity and likely diagnosis of the injury, and make appropriate decisions about transport (how, and to where), further management or euthanasia. While the welfare considerations and acute care of the patient is paramount there is also an imperative to ensure the safety of the rider, other personnel, the public and other horses.

Establishing a definitive diagnosis is frequently not possible (without imaging); however, judging whether a horse can/should be moved safely to where further assessment can take place is usually an uncomplicated matter. The clinician should act calmly and decisively, with awareness of ethical responsibilities. Despite these being emergency situations, good use can be made of available time whilst the horse is sedated, restrained and prepared for transport to obtain a full clinical history and observe the horse's demeanour and consequently decision-making need not be rushed.

Restraint

The injured horse is often excitable and may be unaware of its injury, and immediate administration of a sedative will facilitate safe assessment/stabilization. Alpha-2 adrenergic agonists are preferred: high doses (up to/greater than twice 'normal' dose rate) are usually required to settle horses in the immediate post-exercise phase. Concurrent administration of acepromazine is useful to relieve anxiety and prolong action of other sedative(s), although use merits caution in exhausted horses as may cause hypotension.

Once sedated and if available, a headcollar with lead rope/lunge line should be placed over the bridle to accomplish better restraint. The saddle should be removed.

Initial assessment: Life or death?

Even while initially approaching the injured horse it is often possible to judge the severity of the injury from the posture the animal takes and how it is carrying the affected leg as well as overt evidence of bony failure/instability. Determining whether an injury is (or is likely to be) catastrophic or potentially treatable is the initial goal and generally straightforward (Appendix 5, pp. 477–478). The clinician's primary responsibility is the welfare of the horse, and if assessment determines the presence of an injury that has a hopeless prognosis for life, is causing excessive pain and for which transport to a treatment facility cannot be justified on welfare grounds, immediate euthanasia is warranted. Complete, open fractures of the cannon or fetlock are the most commonly encountered examples of this. Informed consent for euthanasia should be obtained from the trainer/owner but unnecessary delay avoided. In the case of a catastrophic injury an insured horse should be positively identified and an independent postmortem examination may be required.

If uncertainty exists over the severity of or potential to treat an injury, a second opinion should be obtained or the horse stabilized and removed to appropriate premises such as a treatment area or referral hospital for further assessment.

Elective euthanasia may be conducted at the request of the owner regardless of injury status; however, connections should be advised that to do so without meeting recognized criteria for immediate humane destruction might invalidate any potential insurance claim.

Stabilization

Fracture management takes priority over dressing of skin wounds. If the site of injury is known or suspected (usually an easy clinical determination in the lower limb), it should be stabilized (using the guidelines shown in Table 4.1) to permit the horse to be safely transported. The objective of

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Table 4.1 **Guidelines for initial stabilization of fractures for transport or to prevent further injury**

SITE OF INJURY	PREFERRED OPTIONS FOR STABILIZATION
MC3/MT3 condylar fracture	Immobilizing (Robert Jones) bandage (+/– medial and lateral splints) Compression boot Bandage cast (Kimzey Leg Saver)*
P1 fracture	Immobilizing (Robert Jones) bandage (+/– medial and lateral splints) Compression boot Bandage cast (Kimzey Leg Saver)*
Proximal sesamoid bone fracture	Kimzey Leg Saver Compression boot Dorsal splint + heel wedge (or plantar board/splint for hindlimb)
Mid-forelimb fracture (mid-cannon to lower forearm)	Robert Jones bandage with lateral and caudal splints. Splints extend from ground to top of elbow (lateral splint to above shoulder for injuries of forearm)
Mid-hindlimb fracture (cannon and hock)	Robert Jones bandage with lateral and caudal splints (to top of hock)
Elbow	Robert Jones bandage with caudal splint from ground to elbow. Stabilizes carpus and allows weight-bearing
Tibia (only if complete/unstable fracture)	Robert Jones bandage with lateral splint from ground to hip
Fractures above shoulder or stifle	Splinting is contraindicated in most cases

* Satisfactory for short-distance transport.

stabilization is to nullify as best as possible the likely forces of distraction acting on the fracture site, thereby minimising further injury, preserving vascular and soft tissue integrity, limiting swelling and ensuring the best possible chance of repair and recovery. Injured horses typically continue to move even when restrained and despite the injured limb being non-weightbearing the act of swinging it can result in further damage. Immobilization of the fracture site (along with sedation) greatly assists relief of any anxiety that the horse may exhibit in the immediate post-injury phase. Stabilization usually involves immobilisation of the joints above and below the fracture site, with choice of device or bandage determined on an individual basis (Figure 4.1).

The type of splint or bandage used should be determined by the location of injury and ideally diagnosis/configuration of fracture. If the chief goal of immobilization is to prevent further displacement or movement at the fracture site, the forces of distraction through the site should be taken into consideration: supporting a limb in a dorsopalmar direction for instance is of little benefit to a fracture that has mediolateral instability. The problem faced on the track is that diagnosis remains presumptive in the absence of imaging, and it is therefore acceptable to temporarily fit a proprietary aid to facilitate movement to a treatment facility. Hinged compression boots (preferred) and dorsal splints (Kimzey Leg Saver) have the great advantage of being rapidly applied to fetlock injuries in the field in a matter of seconds. Although the latter type of splint provides dorsopalmar/plantar rather than mediolateral stabilization (therefore indicated for proximal sesamoid bone, and not condylar/P1 fractures), in practice no apparent difference is noted in outcomes for short-distance transport and in these circumstances

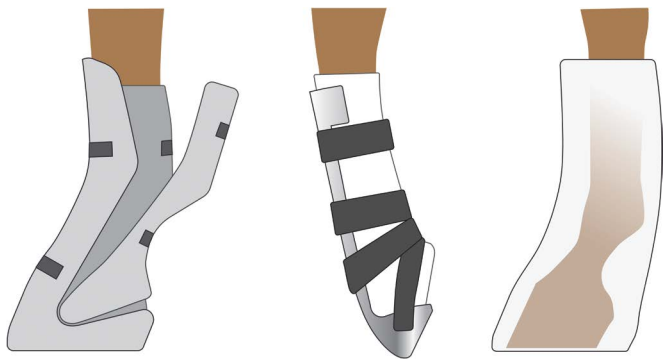


Fig. 4.1 Main classes of aids for short-term stabilization of fetlock fractures. From left: Hinged compression boot, dorsal splint (Kimzey Leg Saver), and Robert Jones bandage/bandage cast.

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they are satisfactory (and recommended) for use with all fetlock injuries. Immobilizing bandages (Robert Jones bandage) +/– incorporated splint(s), and bandage casts provide excellent stabilization and are more readily applied satisfactorily if the horse is in a treatment area. Due to the reciprocal apparatus of the hindlimb, alignment of the dorsal cortices of the cannon and pastern is not possible and fitting proprietary dorsal splints or compression boots satisfactorily is more difficult than for the forelimb.

APPLYING A DISTAL LIMB ROBERT JONES BANDAGE (RJB)

- Goal is to immobilize/splint the limb and appropriate joints through use of a bulky, multi-layer conforming bandage. Bandage should extend from ground level to level of carpometacarpal or tarsometatarsal joint.
- Approximate quantities of materials needed: 1–2 rolls orthopaedic padding +/– length of Gamgee, 3 rolls cotton wool, 8–12 rolls wide open weave (gauze/crepe) conforming bandage, 2–4 rolls elastic adhesive bandage.
- If horse is reluctant to weight-bear, it may be necessary to extend leg forward so that the foot is flat to ground.
- First layer: orthopaedic padding +/– single length of Gamgee; ensure no creases/unevenness.
- Subsequent thin/single layers of cotton wool, each held in place/tightened by conforming open weave (gauze/crepe) bandage. Apply all layers in an overlapping spiral fashion to avoid development of creases in bandage.
- Apply inner layers of cotton wool to slightly greater thickness in pastern and upper cannon (or where needed) to account for limb contour and achieve parallel column shape to bandage.
- Progressively greater compressive force used with each successive layer of conforming gauze: very light conforming pressure used for initial (limb contact) layer building up to maximum tightness by outer layer.
- Outer layer of elastic adhesive bandage; running adhesive bandage +/– duct tape under foot assists longevity and secures RJB in place.
- End result should be a parallel-sided column at least three times the diameter of the leg.

Transport

Providing the injured site has been adequately immobilized/supported the risks posed by travel are a relatively minor consideration, although long-distance travel should be avoided if possible. Suspected/confirmed unstable pelvic fractures at risk of catastrophic internal haemorrhage are poor candidates for long-distance transport and consideration should be given to local stabling.

Care should be taken to ensure loading and unloading are conducted smoothly and without undue strain being placed on the injured limb; the transport vehicle should be brought as close to the horse as possible to limit distance walked. As the horse may be reluctant to protract/place the injured limb some manual assistance may be required to assist movement onto and up the ramp: two handlers can readily push a horse's hindquarters forward under these circumstances.

Horse ambulances should be low-loading to minimize ramp incline; hydraulic-assisted vehicles are ideal as the ramp can be lowered to near-flat. Facility to unload from front and rear so that the horse can be walked forward off the ambulance on arrival at destination is advantageous. Adjustable side partitions and front and rear bars are used to support the horse during transit. While the direction that the horse faces may have some potential advantages in terms of limb loading (facing forward for hindlimb fractures, backward for forelimb fractures), in practical terms orientation for travel is much less important than fracture stabilization and ease of loading/unloading.

THE RECUMBENT HORSE

Management of the recumbent horse can be challenging and while persistent recumbency may ultimately justify euthanasia, arriving at a definitive diagnosis that qualifies that decision may not be possible in all cases premortem. Several diverse conditions can cause a horse to fail to rise, and complete examination is often impaired by limited diagnostic resources in the field. Detailed history, methodical clinical examination and observation of the horse's demeanour and attempts to rise are important factors that may lead to provisional diagnosis. Likely causes and approach to management of recumbency differ between horses that collapse/fall at or after exercise, and those that are found recumbent in the stable or yard setting.

Recumbency during/after exercise

Recumbency may occur due to musculoskeletal injury (incurred during exercise or as a result of a fall or collision), trauma involving the central nervous system (head, neck or back), cardiovascular event or exhaustion/heat stress. Circumstances leading to collapse/fall, where and when during the race/exercise session it has taken place and demeanour of horse and subsequent attempts to rise are valuable details that can help direct subsequent investigation.

- A horse brought down (or falling) during a flat race that subsequently fails to rise is likely to have sustained trauma, and involvement of a fractured limb

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or pelvis (or less commonly of spinal column) should be suspected in the first instance.
- Recumbency in jump racing after a fall is relatively common and when in the latter stages of a race is often simply caused by horse being exhausted or 'winded'. Such horses lie still with strong/laboured breathing and generally rise uneventfully and without assistance within 20 minutes.
- Failure to rise when encouraged to do so (when breathing has normalized) or further collapse indicates a condition more complicated than simple hypoxia/exhaustion, and possible musculoskeletal injury should be investigated.
- Somersault falls or those in which the neck undergoes extreme flexion may cause fracture, dislocation or contusion of the spinal column of the neck.
- Cardiovascular events (cardiac arrhythmia or failure, ruptured blood vessel or aneurysm) are usually distinguished by a brief period (sometimes lasting only seconds) of staggering prior to collapse.

Recumbency not associated with exercise

When a recumbent horse is encountered in a stable, yard setting or field, background information including duration of recumbency may be limited. The possibility of illness as well as injury should be considered.

- One of the most common causes of recumbency or acute severe hindlimb lameness apparently sustained in the stable is fracture of the ventral pelvis (+/– acetabular involvement) (p. 247). This is usually caused by a slip or fall, and is most commonly seen in fillies.
- Recumbency may also occur in horses being rehabilitated for serious pelvic or proximal limb stress fracture. In some cases this recumbency results from serious/catastrophic deterioration, and in others it may be simply that a cross-tied horse is fatigued, chooses to lie down and has difficulty rising again.
- Evidence of current/recent distress (sweating) or exhaustion can be associated with a range of conditions, including musculoskeletal injury, colic or prolonged attempts to rise having become cast.
- Trauma to the head may result from a horse rearing over backwards: fracture of the base of the cranium may be associated with acute neurological signs, severe loss of coordination, unilateral or bilateral blindness and blood from nostrils or ears.

General approach to the recumbent horse

Initial examination

- Obtain detailed history: Seen to fall? Nature of fall? If found in stable, was anything heard/when was horse last seen standing? Any attempts to rise observed, and if so did horse regain feet/weight-bear on hindlimb(s)?
- Demeanour/consciousness (pupillary and withdrawal reflexes)/mucous membrane colour/pulse and respiration rate/demonstrable pain give some indication of severity and whether primary problem is musculoskeletal, cardiovascular or neurological.
- Poor mucous membrane colour/rapid pulse and deteriorating vital signs may indicate catastrophic internal bleeding.
- Signs of exhaustion/sweating may indicate a period of struggling to rise.

Musculoskeletal examination

- Assessment of musculoskeletal integrity, particularly that of pelvis and back, can be difficult in a recumbent horse.
- Care should be taken to assess the horse from the dorsal aspect to avoid danger posed by limbs.
- Position of recumbent horse in the stable may create access challenges/dangers and personal protective equipment should ideally be worn by anybody in proximity of horse. Use of sedatives may be warranted.
- Assess neck, back, uppermost side of pelvis and all four distal limbs (without moving horse) for obvious fracture or instability.
- Tightness of gluteal/caudal thigh muscles may indicate exertional rhabdomyolysis.
- Rectal examination of pelvis is usually safe to undertake while horse is recumbent and may permit detection of obvious pelvic/acetabular instability or internal haematoma indicating fracture.

Neurological examination

- Neurological reflexes (withdrawal reflexes, tail and anal tone) tested to identify site of any neurological dysfunction.
- Reflexes typically subdued in a stressed/recently exercised horse; full assessment may be hindered during initial 30 minutes to 2 hours following incident.
- If fully conscious but unable to move hindlimbs +/– forelimbs: vertebral fracture with spinal involvement likely.
- Spinal injury in cranial neck: only able to raise head; exaggerated withdrawal reflex all four limbs.

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- Spinal injury C6–T2: only able to raise head and neck; absence of forelimb withdrawal reflex.
- Spinal injury caudal to T2: can dog-sit; loss of skin sensation over trunk (if T2–L4: exaggerated hindlimb withdrawal reflex).

Actions

- Winded horse: allow to rest +/– administration of oxygen (nasal insufflation): should rise unassisted within 20 min.
- For horse with suspected musculoskeletal injury: if limbs/pelvis appear intact and no apparent injury is detected, horse may be encouraged to rise. This can either be achieved by propping horse into sternal position with front legs extended and encouraging/assisting horse to stand (with tail and head support); or placing ropes/lunge lines under lower side forelimb and hindlimb, running ropes over body and rolling horse onto opposite side (concurrently restraining head). Rolling over allows assessment of previously inaccessible side of pelvis/limbs and frequently also stimulates attempts to rise. When rolling a horse care should be taken by handlers to stay out of kicking range.
- Horses recumbent in a stable may need to be repositioned (by rolling, or by pulling head/neck +/or tail around) before assessment or attempts to rise are possible; available space may also limit attempts at the latter. If horse appears exhausted but is comfortable and not struggling it may be preferable to position favourably and leave horse to rise itself (rather than assisted recovery).
- In exceptional circumstances and dictated by available facilities, equipment and structural features of stable/yard, it may be possible to attempt hoisting/slinging of horse to assist further diagnostic and recovery efforts.
- For horses with catastrophic limb, pelvic or vertebral fracture: immediate euthanasia.
- For conscious cases in which catastrophic musculoskeletal injury is suspected but cannot be confirmed premortem, it is generally acceptable to consider euthanasia if no successful attempts to rise have been made after >1 hour has elapsed. This time frame should be extended if horse has received sedatives during the examination process.
- Comatose/semi-comatose: may recover (if not deteriorating) and prolonged assessment/recovery period may be warranted. Persistently fixed/dilated pupils associated with poor prognosis and warrants euthanasia.
- When the reason for recumbency remains uncertain following preliminary investigation and condition is not deteriorating, anti-inflammatory drugs (corticosteroids and non-steroidal anti-inflammatory drugs) should be administered and removal to quieter location (drag mat and transport) may be recommended.
- Neurological cases: if still recumbent and para-or tetraplegic after 1–2 hours euthanasia is warranted.

HEAT STRESS/EXHAUSTION

- A large amount of metabolic heat is produced by high intensity/prolonged exercise; if this is not dissipated effectively then a rise in core body temperature (hyperthermia) can result. Exacerbated by large fluid/electrolyte losses in sweat.
- Dissipation of heat occurs at skin-air interface; if ambient conditions are hot + humid evaporation of sweat can be inhibited.
- Typical presentation is initially unsettled behaviour/discomfort and high respiration and heart rate, altered mentation/depression/disinterest in surroundings, absence of thirst/appetite, tacky/dry gums and prolonged capillary refill time. Kicking out is common; also colic secondary to gastrointestinal stasis (ileus) may occur. May progress to staggering/incoordination and recumbency.
- Horse should be cooled off aggressively by repeatedly pouring copious volumes of cold/iced water over body (shoulders/back/hindquarters). Scraping off excess water between applications is not necessary but may be beneficial if humidity is high and evaporation impeded. Shade and use of fans if available. Monitor effect: target is reduction in skin temperature to approximately 30°C.
- Preferable to keep walking if possible (rather than permit recumbency).
- Sedation may be indicated to control fractious/agitated behaviour: detomidine is recommended.
- +/– single dose of flunixin (to counter inflammatory/endotoxic aspects of heat stress).
- If severe: large volume intravenous fluid administration. Can tolerate administration rate of 10–20mL/kg bodyweight (BWT)/h (5–10L/h); preferably non-lactated polyionic isotonic fluids; however, re-establishing perfusion is more important than choice of fluid.
- Intra-gastric administration of fluids (4–8 L, repeated hourly) also useful providing no gastric reflux is present.

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- Referral to hospital facility recommended if horse fails to urinate (or improve clinically) after approximately 2–3 hours (around 40 L) of intravenous fluid therapy.

SKIN WOUNDS

The approach to treatment of skin wounds is determined primarily by anatomical location, severity and involvement of adjacent/underlying structures. Choice of wound repair and bandaging is often geared towards minimizing interruption to training.

Assessment of wounds

- Determine proximity to important underlying structures (joints/tendon sheaths/tendons).
- Injury to deeper structures may not always be visible through skin wound when limb is in weight-bearing position: wound should also be explored with limb flexed.
- Suspected synovial involvement warrants synoviocentesis (or referral to surgical facility).
- Exploration of wound +/– ultrasonography/radiography to ensure no foreign body present.

Management

- Primary wound closure through suturing/stapling results in faster healing and better cosmetic outcome, but may not always be necessary or desirable.
- 'Clean' wounds: 'golden' period for primary wound closure is approximately 4 hours (>4 hours: significant bacterial contamination and greater likelihood of eventual wound breakdown).
- Small (<1 cm) lacerations/abrasions/flaps on lower leg are often best left unrepaired, as wound closure frequently results in abscessation and wound breakdown. Manage by cleansing + wet poultice for initial 12–24 hours + continued exercise; antibiotic therapy frequently necessary for wounds on lateral/medial aspect of cannon due to propensity to develop secondary cellulitis.
- Larger lacerations: irrigate/debride and full or partial primary closure (staples/sutures).
- Falling/stumbling/road injuries: partial- or full-thickness abrasions over front of fetlock, carpal or stifle regions, also point of elbow (Figure 4.2a,b). Skin loss usually prevents primary closure and the most important consideration is to rule out synovial involvement. Fetlock wounds: initial wet poultice then bandage for exercise. Carpal and proximal limb wounds may be left unbandaged. May require antibiotic therapy.
- Wound cleansing/irrigation: may use tap water, tap water saline, dilute (0.1–0.2%) povidone–iodine or chlorhexidine (0.05%). Good irrigation pressures achieved using 18- or 19-gauge needle/syringe combination.
- Sutures are preferred for areas under high tension (greater strength).
- Staples can be applied quickly and often without need for administering drugs; are preferred for low-tension wounds.
- Puncture wounds may be troublesome as infection is seeded in deeper tissues and little opportunity for drainage.
- Major wounds sometimes best treated under general anaesthesia (GA): permits better debridement, irrigation and repair.

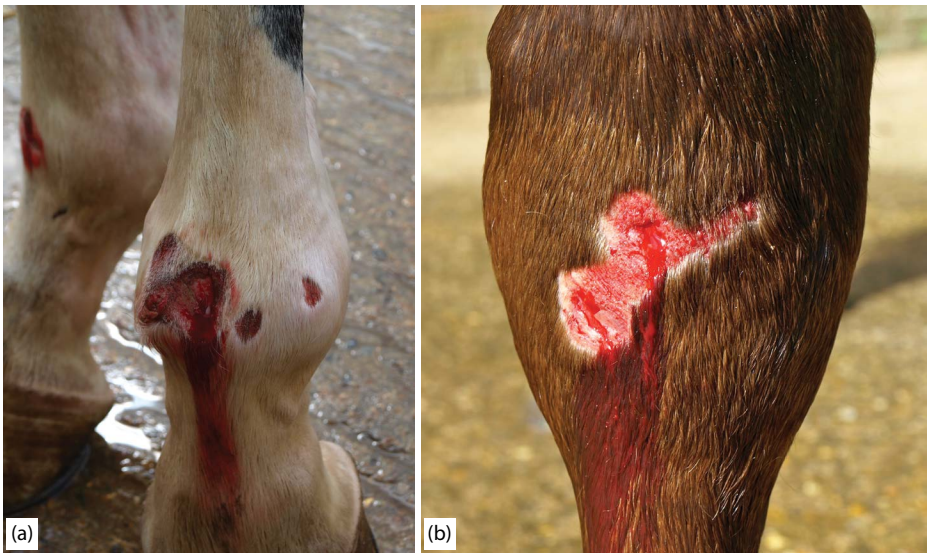


Fig. 4.2 **Typical wounds to front of hind fetlock (a) and front of knee (b) following fall/slip.**

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Wound dressings and exercise

- Bandaging assists healing by minimizing wound movement and contamination and providing conditions (warm, moist) conducive to repair.
- Acute phase: antiseptic contact dressings can reduce surface contamination and assist healing.
- Frequency of dressing change dependent on amount of exudate.
- Once healthy granulation tissue bed has formed, little need for antibiotic therapy.
- Wounds with significant 'dead space' cavity may benefit from packing/dressing with medical-grade manuka honey to encourage rapid granulation/infilling of defect.
- Exercise during healing phase: dependent on severity of initial injury, type of repair and desire to avoid wound breakdown.
- Horses with wounds at 'high stretch' sites may require removal from ridden exercise until sutures/staples removed.
- Horses with many other repaired wounds, including those on the upper limb, can remain in work with little risk of wound breakdown.

Proud flesh

- Infilling of wound cavity with granulation tissue is normal part of repair process.
- Infection/wound movement inhibits wound edge contraction and stimulates granulation tissue production (Figure 4.3).



Fig. 4.3 'Proud' flesh: Exuberant granulation tissue raised above level of surrounding skin.

- Wounds on lower limb more prone to development of proud flesh due to greater contamination and relative lack of supportive tissue underlying the skin.
- Non-healing wounds should be investigated for possible presence of foreign body, bone sequestrum or infection.
- Treatment: topical corticosteroid cream plus bandaging +/– antibiotic therapy until granulation tissue back to level of surrounding wound edge.
- Topical astringents such as zinc sulphate useful for limiting granulation tissue proliferation (used in preference to caustic agents such copper sulphate/silver nitrate, which can harm wound edges and delay healing).
- Initial debulking by surgical trimming useful in some circumstances (granulation tissue is free of sensory innervation and can be excised without analgesia).

Bone sequestration

- Thin outer lining of bone (periosteum) supplies blood to outer cortex and serves as an osteogenic organ to assist healing, particularly following fracture.
- Damage to periosteum (direct cortical trauma/local infection) can cause disruption to blood supply of underlying bone, resulting in separation of an avascular bone sequestrum from the underlying parent bone.
- Occurs most commonly in cannon (metacarpal/metatarsal) region (poor protective covering of soft tissue).
- Causes non-healing wound +/– draining tract +/– lameness.
- Diagnosis is by radiography: signs typically delayed >2 weeks following initial injury (Figure 4.4a–c).
- Treatment: antibiotic therapy (typically 2–4+ weeks duration) resolves most cases.
- Surgical debridement often advocated as treatment of choice, but rarely necessary and usually reserved for large sequestra or those non-responsive to antibiotic therapy (Figure 4.4d).

WOUNDS INVOLVING TENDON/LIGAMENT

Assessment

- Involvement of tendon(s) +/– synovial structures in any open wound can be career/life-threatening.
- Most commonly involve the hindlimb and are sustained during high-speed exercise or racing (Figure 4.5a).
- Deep wounds overlying tendons/ligaments should be assessed clinically/ultrasonographically to determine extent of injury.

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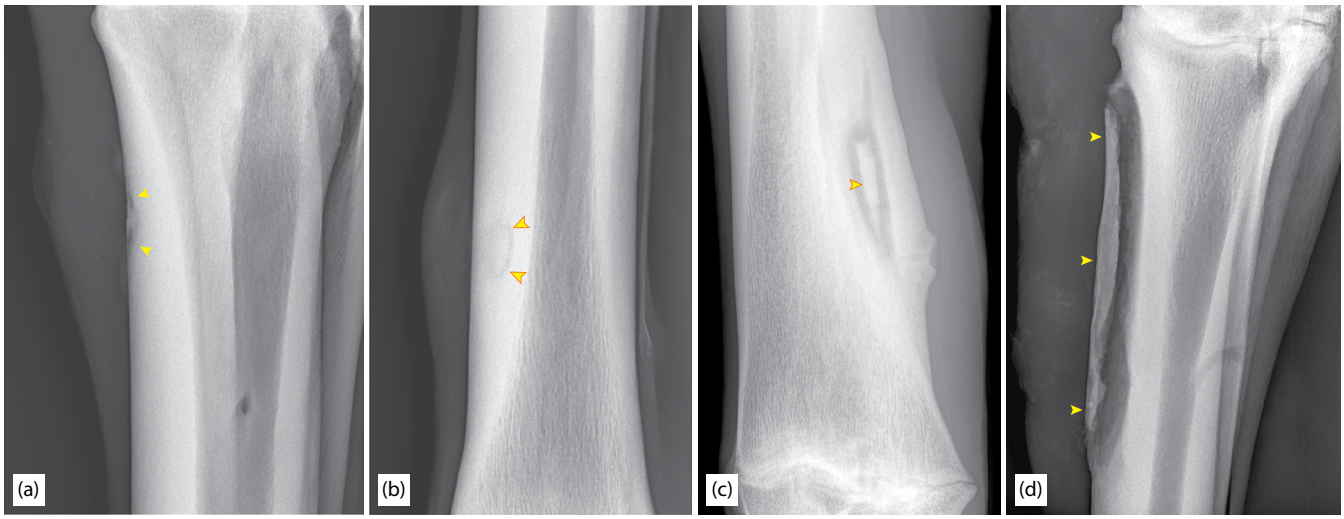


Fig. 4.4 **Examples of bone sequestration. (a) Small saucer-shaped lytic defect (arrowheads) on dorsal aspect of cannon underlying skin wound, with indistinct periosteal reaction proximal and distal to it consistent with early lesion. (b) Circular lytic defect consistent with separation of devitalised fragment from surrounding cannon. (c) Clearly defined sequestered fragment on medial aspect of radius. (d) Large fragment on dorsal aspect of hindlimb cannon necessitating surgical removal.**

- Injury to tendon may not be immediately apparent at level of wound until limb is raised/flexed.
- Concurrent involvement of digital tendon sheath possible with injuries in fetlock region (Figure 4.5b).

Management

- Suspected tendon laceration: leg should be dressed and splinted with fetlock in slight flexion for transport to surgical facility.

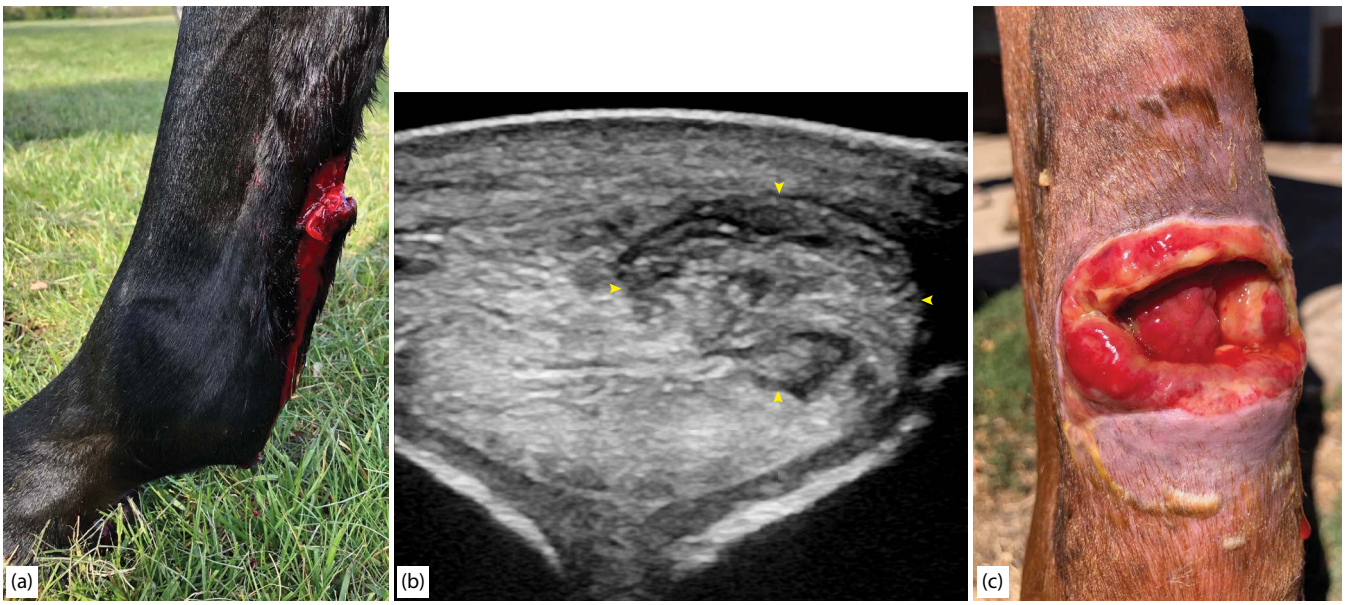


Fig. 4.5 **(a) Serious hindlimb wound with tendon involvement caused by interference from another horse during high-speed exercise. (b) Traumatic injury to SDFT within digital tendon sheath (at level of fetlock) with large zone of disrupted tendon tissue noted. (c) Typical appearance of interference injury to back of cannon during rehabilitation, with proud tissue present at both the skin wound edge and within the wound (from paratenon).**

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- Tendons heal at a slow rate and repair never results in recreation of normal tendon tissue or function.
- Healing of wounds involving paratenon/tendon often complicated by independent movement of tendon through granulation tissue (Figure 4.5c); immobilization (casting) may be beneficial.
- Partially lacerated tendon: limb immobilization for up to 6 weeks.
- Lacerated tendon: suture repair plus limb immobilization possible. Total/near-total lacerations +/– synovial bursa involvement/contamination are challenging to return to full athletic use and may merit euthanasia.
- Superficial traumatic injuries to tendons/ligaments carry a better prognosis (and generally require less recuperation time) than comparable 'overstrain' tendon injury.
- Rehabilitation guided by ultrasonographic reassessment.
- Prognosis for return to full athletic function is considerably better for hindlimb than for forelimb: complete laceration of hindlimb flexor tendon does not preclude return to training.

INTERFERENCE INJURIES

Traumatic skin/soft-tissue injuries sustained at exercise have characteristic features depending on the type of interference; individual management is determined principally by the severity/depth of the injury. Sporadic/accidental injuries may be attributed to deep track surface, freshness of horse, inattentive rider or use of sedatives, whereas recurrent injuries may arise from conformation faults, poor shoeing or lameness. Limbs/feet responsible for interference may not be easy to determine retrospectively as limb interactions during high-speed exercise are complex and affected by variables such as curvature of track or which limb lead the horse is in at moment of contact.

Overreach

- Occurs when back of forelimb is struck by toe of the back foot. 'Forging' is an overreach that strikes the shoe/sole of front foot (with associated characteristic sound of contact) rather than soft tissues above the foot.
- Usually incurred at slow paces and/or on deep tracks.
- Typically involves a single heel (injuries to back of fetlock/pastern or cannon uncommon).
- Varies in depth/severity: superficial grazes to deep flap wounds.
- Most commonly: superficial flap of skin/hoof at heel bulb coronet (Figure 4.6a,b). Often deeply embedded with dirt and may be (or become) lame if infected or when subsequently exercised on deep track.
- Management: initial wet poultice to clean site, thereafter keep open. Flap may be excised if pocketing dirt (Figure 4.6c). Antibiotic therapy if local infection develops.
- Prevention (if recurrent problem): overreach boots +/– altered shoeing.
- Shoeing: flight proximity of fore-and hindlimb feet can be altered by improving forelimb breakover (e.g., using rolled toe shoe for front feet) +/or shoeing front feet with aluminium plates and hind feet with steel shoes; hind shoes can be set back +/– quarter clips.



Fig. 4.6 Typical overreach injury to heel (a), with full-thickness flap of soft hoof tissue pulled down (b) and following flap excision (c).

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Brushing

- Cuts/abrasions/knocks on inside of pastern/fetlock caused by interference with foot of opposite limb (Figure 4.7a,b). Usually sustained at faster paces.
- When on forelimb: usually with base narrow/toe-out conformation.
- When on hindlimb: often result of a close/'plaiting' hindlimb action or bilateral hindlimb lameness.
- Prevention (forelimb): exercise bandages/change of shoeing: either inside of shoe set tight/rounded edge, or redirect breakover laterally.
- Prevention (hindlimb): exercise bandages or boots/change of shoeing: ¾ shoe (less shoe to catch) or lateral trailer (pivots toe laterally). Three-quarter shoes best used as a temporary measure otherwise distortion/imbalance of hoof may result long-term. If brushing is arising from hindlimb lameness/change of hindlimb action, underlying problem should be investigated/treated.
- Hindlimb interference frequently diminishes over time with strengthening through training/maturity.

Medial fetlock interference injury

- Single/repetitive blunt injury to inside of forelimb fetlock may cause acute inflammation and lead to persistent subcutaneous thickening/acquired bursa over the medial sesamoid bone.
- +/– skin wound with secondary infection.
- Usually caused by contact with foot of opposite forelimb.
- Subsequent prominence of site may predispose to further recurrent trauma (Figure 4.7c).
- Varies in severity: may initially be intensely painful to palpate but lameness generally mild.
- Exposed position of palmar nerve over sesamoid bone may expose it to trauma: focal neuritis.
- Usually occurs with toe-out/base narrow conformation.
- Can usually be differentiated from more serious injuries on clinical grounds and response to treatment: swelling/palpable pain associated with injuries that occur at same site (suspensory ligament branch desmopathy, sesamoid bone fracture) typically more focal to injured structure.
- Management: systemic/topical anti-inflammatory +/– antibiotic medication. Slow resolution of swelling is typical (days/weeks depending on initial severity).
- Exercise bandages may prevent recurrence of trauma in some cases.
- Long-standing subcutaneous seromas can be treated with intrabursal corticosteroid administration, but rarely necessary.

'Speedy' cuts

- Short lacerations on inside of one or both hocks/hind cannons (Figure 4.7d,e). Less commonly, can involve inside of knee/lower radius (Figure 4.7f).
- Usually incurred during high-speed exercise.
- Limb interactions are complex and vary with speed, limb lead and whether travelling in a straight line or rounding a bend. Injuries involving the hindlimbs may be caused by contact from either the outer margin of the shoe of the same side front foot or inner margin of opposite side front foot. When involving the knee, they are likely to be caused by inner margin of opposite forelimb foot.
- Generally require only topical wound management and do not interfere with training, although more severe/recurrent injuries may develop proud tissue and require wound management.
- Prevention: ensure front shoes are set tight/rounded on the aspects of concern relating to particular injury. Neoprene boots on upper hind cannons may be protective.

Scalping

- Injury to front of hind pastern (Figure 4.7g) caused by the toe of the front foot on same side.
- Prevention: subtle alteration of breakover of front feet or hind feet often sufficient to prevent limbs meeting during exercise. Change in forelimb breakover achieved by either using rolled toe shoe or shoeing front feet with aluminium plates and hind feet with steel shoes.

Tendon knock/bandage 'bow' or 'bind'

- Subcutaneous swelling over back of forelimb tendon bundle noted after exercise or when bandages removed.
- May result from blunt trauma or tight/slipped bandage.
- Clinically may resemble superficial digital flexor tendon (SDFT) tendinitis in acute phase and ultrasonography may be warranted to assess integrity of tendon (typically reveals thickened subcutaneous/peritendinous tissues) (Figure 4.8a).
- Focal damage to tendon may occur but is rare: if present, generally confined to margin(s); however, more profound damage such as longitudinal split of tendon (Figure 4.8b) may occur. Tendon damage necessitates rehabilitation, but typically carries a better prognosis (and usually shorter rehabilitation time) than typical 'overstrain' tendon injury.

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Fig. 4.7 **Interference injuries: Brushing (a, b); trauma to medial fetlock (c) with prominent thickening of soft tissues over medial proximal sesamoid bone (PSB); 'speedy' cuts on the medial aspect of hock (d), hind cannon (e) and knee (f); chronic 'scalping' wound to hindlimb with scarring on front of lower pastern (g).**

- If no SDFT involvement, injury is self-resolving, although systemic and topical anti-inflammatory measures may assist return to normal appearance; rest is not usually required.
- In very rare cases of severe bandage 'bow' (prolonged or overtight bandage application), avascular necrosis of tendon +/– overlying skin may occur: guarded to poor prognosis for return to full athletic function.

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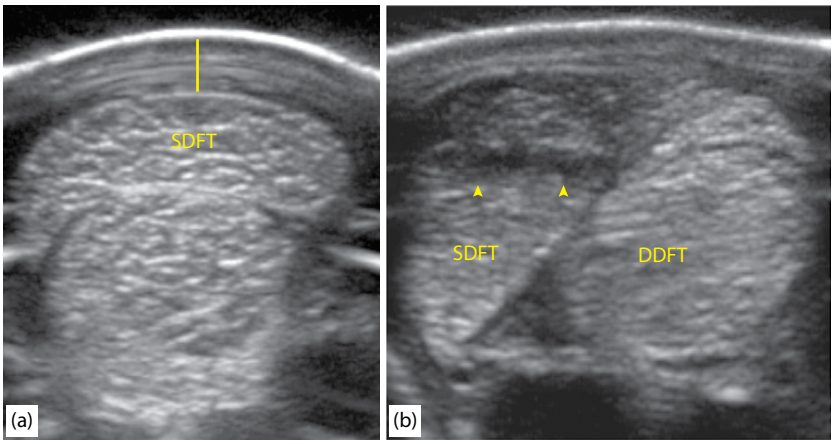


Fig. 4.8 **(a) Bandage 'bind' with 'bowed' profile to tendon bundle caused by thickening of subcutaneous tissue but without underlying tendon damage. (b) 'Split' SDFT arising from blunt trauma to tendon bundle at exercise.**

KICK WOUNDS

Assessment

- Kick wounds sustained at exercise most commonly involve the forearm, point of shoulder, chest or upper hindlimb (Figure 4.9a,b).
- Many occur during warm-up exercise; if the horse has subsequently exercised the likely presence of significant orthopaedic injury can be assessed from degree of lameness.
- Most are benign and of little concern.
- Skin wounds usually small (<5 cm) and associated with local bruising/bleeding.
- Synovial involvement is rare.
- Wounds directly overlying bone may require radiographic assessment depending on presence of lameness.

Management

- Wound closure generally not required due to small size and location in area of high mobility.
- Antibiotic +/– anti-inflammatory therapy if local infection develops.
- Horses with uncomplicated wounds can generally continue training without interruption.
- Injuries at sites with little muscle protection may result in local bone sequestration or (rarely) cortical fracture: inner aspect of radius/tibia most susceptible.
- Possibility of fracture should be investigated if degree (or persistence) of lameness is inconsistent with local infection.
- Radiological evidence of fracture propagation may not be apparent until 1–2 weeks post injury.
- Suspected fracture: stable rest (+/–walking exercise to manage any concurrent cellulitis) until radiological confirmation.



Fig. 4.9 **Kick wounds to elbow (a) and lower neck/shoulder (b).**

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SYNOVIAL INFECTION

Infection within a synovial space (joint or synovial bursa) is potentially a life-threatening condition. Unless recognized and treated promptly and aggressively, persistent infection of the synovial space or irreversible destruction of articular tissue may occur, leading to lameness that may be incompatible with an athletic or non-athletic future. Wounds in the region of synovial cavities or lameness that might be attributable to synovial sepsis are emergencies and should be investigated without delay. Infections resulting from direct wound contamination usually involve mixed bacterial species, whereas iatrogenic infections (following intra-articular injection) most commonly involve *Staphylococcus aureus*.

Assessment

- Acute-onset, moderate-severe lameness.
- Not invariably associated with obvious wound or history of trauma.
- When iatrogenic (e.g., following intra-articular injection) may have a more insidious/ambiguous presentation if intra-articular corticosteroids have been administered (due to dampening of inflammatory response).
- Marked effusion of affected joint +/– localized cellulitis usually noted.
- Infection of anatomically well-protected joints/bursae (e.g., navicular bursa) may be associated with little palpable abnormality.
- Synoviocentesis: collection of synovial fluid sample for cytological analysis +/– through-and-through needle lavage to assess wound communication.
- Synovial sample: total white blood cell (WBC) count >10 x 10^9/L (10,000/μL), total protein >50 g/L (5 g/dL) and toxic/degenerative cytological change strongly indicative of infection.
- Cytological findings not always definitive, particularly for cases attended promptly. Clinical suspicion, multidisciplinary assessment and relative risks of immediate/delayed treatment should inform decision-making.

Management

- Hospitalization and prompt treatment improves outcome. Systemic antibiotic therapy should commence as early as possible.
- Large-volume lavage of synovial cavity (frequently under general anaesthetic) plus antibiotic therapy (systemic/regional perfusion) to clear bacterial contamination and debris.
- With appropriate management, prognosis for survival to discharge is good (>80%) and return to athletic use fair to good (>50%).
- Poorer prognosis with concurrent bone/tendon/ligament involvement.

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CHAPTER 5

REHABILITATION AND TISSUE REPAIR

OVERVIEW

Rehabilitation is the process by which recovery from injury is assisted by interventions and structured exercise intended to optimise return to athletic function. Rehabilitation also encompasses methods used to manage, minimise or slow the progression of chronic musculoskeletal pathologies. A clear understanding of the mechanisms of injury and repair and the way these may be modified is fundamental for clinicians to devise treatment protocols and provide valid recommendations to clients. The extent to which these methods are applied to any given individual case will depend on many factors such as stage of career, economic considerations and available facilities.

Whether an injury arises due to fatigue damage that accumulates over long periods in the face of inadequate tissue repair processes (the most common cause of racehorse musculoskeletal injury) or from an acute overloading event, the final act in the process is typically a physical disruption to the structure in question. Regardless of the tissue involved, the natural healing response that follows generally goes through a short initial inflammatory phase, then a proliferative phase in which new tissue is laid down, followed by a final (and prolonged) maturation/remodelling phase in which the repair tissue adapts and strengthens in response to the loads imposed upon it. When stress or overuse injuries are detected (and allowed to repair) before 'macroscopic' disruption to the structure occurs, there is no well-defined traumatic event and healing generally occurs through tissue remodelling alone. Rehabilitation involves ensuring that these phases of repair occur smoothly and without impediment, while minimising unnecessary wastage of training time and resources. The intrinsic properties of the injured tissue define the rate and quality of healing that is possible, and along with severity and staging (acute, subacute, chronic) of the injury determine the broad parameters within which a return to athletic use can be expected. It is important that veterinary recommendations are based on best current knowledge of tissue healing time frames as well as being tailored to individual circumstances: many examples exist of racehorse careers being curtailed more by inappropriate rehabilitation advice than by the actual injury.

EXPECTATIONS AND PROGNOSIS

Prognosis is defined as the prediction of the likely outcome of an injury and is derived from both general knowledge of that injury from the larger population and specific clinical details of the individual case being considered. Prognosis is dynamic and may be influenced by factors arising throughout treatment or rehabilitation: it is frequently the case that prognosis for some cases improves as potential complications are eliminated through the immediate post-injury phase.

Despite the inherent flaws associated with attempting to predict individual outcome based on population averages, it is useful to broadly categorize prognosis so that trainers/owners can make informed management decisions. The evidence base available to racehorse clinicians to formulate strong guidelines on likelihood of favourable outcome with any particular course of action is very limited; most interventional studies involve small numbers of horses and do not utilize controls, and do not differentiate the reasons for retirement (which may include pragmatic/breeding decisions rather than injury outcome). The prognosis recommendations in Chapters 6 and 7 therefore draw on relevant published work and clinical experience and, where available, more specific outcome figures are included in the text. These should be considered guidelines only and subject to the circumstances of the individual case as determined by the treating clinician: there are exceptions to almost any rule and most trainers/vets can recall multiple cases of horses defying predictions of poor outcomes by resuming successful careers. Return to full use is defined as full, unrestricted participation in training/racing. The likelihood of expected return to full use (if so desired) is categorized in Table 5.1.

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Table 5.1 **Categorization of the likelihood of expected return to full use following injury**

PROGNOSIS	DESCRIPTION	EXPECTED RETURN TO FULL USE (%)
Excellent	Full recovery to normal function can be expected	>80
Good	Expect improvement; most recover to full function	70–80
Fair	Expect improvement; significant chance of insufficient recovery/recurrence/ongoing problems	60–70
Guarded	Not possible to predict with certainty whether full or partial recovery to full use will occur; recurrence or need for ongoing attention may be expected	40–60
Poor	Unlikely to recover fully regardless of management; may deteriorate	<40

TREATMENT MODALITIES

Effectiveness

Whether a rehabilitation recommendation or therapy will be effective or not, and the extent to which it might influence outcome in a particular individual, are the most important considerations for trainers/owners when offered veterinary advice on the management of injury. Although rarely validated by controlled studies, the prescription of rest and exercise appropriate for effective rehabilitation of the majority of racehorse injuries is based on a solid foundation of empirical knowledge accumulated over many veterinary generations. By contrast, most intervention therapies applied to racehorses are underpinned by little or no good quality evidence. While this should not necessarily preclude their use, it is important that expectations around treatment outcomes and communication with trainers/owners are informed by facts as far as they are available.

The general trajectory of scientific validation and development of treatments in both pharmaceutical research and human sports medicine follows a staged progression from basic tissue studies through to large 'real-world' trials assessing effectiveness and cost-benefit determinations in target populations. Early proof-of-concept testing in a small number of patients is used to support the proposed theory by providing evidence that the treatment can or may (rather than does) work; this is followed by larger studies assessing treatment effects in a population with a more representational range of symptoms. Once treatment efficacy for the injury/disease has been demonstrated, further trials assessing 'effectiveness' (the impact of the treatment in routine clinical practice) allow conclusions to be drawn about which patient groups are likely to experience substantial practical benefits from the treatment, balanced against risks and costs.

Almost without exception this template of translational research does not occur in equine sports medicine. Scientific trials are costly and with the relatively small size of the equine market it is understandable that therapies may be adopted directly into clinical practice from other fields without any preparatory research. That they become established adjunctive therapies in the veterinary arsenal without significant critique is of concern given that for many interventions there is substantial evidence (from human sports medicine) that they are unlikely to have a positive clinical impact, or at the very least that there is little good evidence to support their use. Furthermore, without research foundations, the effectiveness in different patient or injury categories, cost-benefit determinations and understanding about optimal dosing (dose, timing, frequency) of the intervention for equine injuries must remain speculative. Given that scientifically validated return to racing success rates and time periods (with 'normal' rehabilitation protocols) do not exist for most racehorse injuries, it follows that claims (either general or specific) of improved outcomes following application of any particular intervention (whether medical, surgical or physical) are generally unsubstantiated. Clinicians should remain conscious of these scientific inadequacies in equine sports medicine and strive to remain objective and avoid strong recommendations for the use of therapies for which there is little or no substantial evidence of efficacy.

Exercise prescription

Exercise prescription is the most important component of racehorse rehabilitation. Guidelines for the type and duration of rest from active training for most musculoskeletal injuries are well established in clinical practice and are supported by a large amount of empirical evidence. In its simplest form – that of recommending defined periods of stable confinement, walking and trotting exercise – most

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injuries can be assisted successfully through the phases of tissue repair that lead to functional recovery. Refinement of exercise therapy, including the use of training aids such as water and land treadmills, has the further potential to minimise lost training time and improve quality of injury repair. While many of the strengthening and non-weight-bearing exercises available to the human athlete cannot be replicated in the horse, the fundamentals of controlled and graduated loading and conditioning of the damaged tissues remain the same and protocols can be adapted to individual circumstances depending on diagnosis and clinical/imaging progress throughout rehabilitation.

Pharmaceutical management of pain and inflammation

In the immediate post-injury phase it may be necessary to administer anti-inflammatory medications, particularly when lameness and/or localised swelling is profound. Injury type, site and severity determine whether and how analgesic and anti-inflammatory measures should be implemented; for example, it may be best in cases of proximal limb or pelvic injuries at risk of destabilisation to limit analgesia so that voluntary mobilisation by the horse of the injured structure is minimised. Systemic anti-inflammatory medications in common equine use fall into two main categories: non-steroidal anti-inflammatory drugs (NSAIDs) or corticosteroid drugs. Mechanism of anti-inflammatory action differs between these drug classes, with NSAIDs having additional analgesic effects.

While previously a key component of acute injury treatment, NSAIDs are now known to have a potentially negative effect on tissue (especially soft tissue) healing. The prostaglandin-induced recruitment of inflammatory cells at the injury site that occurs early after injury is important for the initiation of the healing process, and inhibiting this phase can have long-term consequences for the speed and quality of any subsequent tissue repair. Through their analgesic function NSAIDs also have the ability to mask lameness or inflammation such that ongoing or recurring injury may go undetected. In view of this, routine prescription of NSAIDs for chronic soft tissue injuries is generally not recommended, and for acute injuries application should be judicious and for the shortest period of time possible, if at all.

Regenerative medicine

Many of the injuries (both acute and chronic) encountered in equine sports medicine undergo repair processes that leave the injured structure functionally inferior to pre-injury tissue. This is most typically the case for soft tissue injuries involving tendon, ligament and cartilage. Regenerative medicine involves the use of therapeutic agents to assist the regeneration or replacement of damaged tissues and restore them to normal function. The mechanisms of the inflammatory response and subsequent injury repair are complex and tightly regulated, involving interaction of a wide range of growth factors and cellular mediators. While this offers multiple potential pathways to directing repair processes to favour regeneration rather than scar tissue in cases of clinical injury, knowledge in this field is evolving rapidly and it is likely that the promise of many novel treatments will be balanced by the disappointments of others for the immediate future. There is considerable evidence that regenerative therapies have potential anti-inflammatory and disease-modifying effects; however, 'real-world' therapeutic efficacy in common racehorse injuries is largely undetermined and even in human sports medicine the science behind these interventions is far from settled.

Regenerative medicine treatments may be cell based or cell free. They have previously been primarily derived from tissues or blood harvested from the injured animal itself; however, allogeneic therapies (from donor tissue) also exist. Several therapies have entered clinical use in horses and these are described in the following sections.

Mesenchymal stem cells

Mesenchymal stem cells are cells that have the ability to differentiate into specific tissue-generating cells such as those that synthesise bone (osteocytes), cartilage (chondrocytes) and fat (adipocytes). They may be harvested from a range of tissues including bone marrow, fat and umbilical cord or amniotic membranes and both their tissue of origin and the horse's age at time of sampling may affect their properties and inherent value as therapeutic agents. It was originally considered that the main benefits derived from administration of mesenchymal stem cells to an injury site might come from their differentiation into tissue-specific cells helpful for regeneration; however, it is now known that survival time of the stem cells after transplantation is typically very short. It is likely that any therapeutic effect therefore comes from their secretion of 'bioactive' substances (the 'secretome') that help to modulate the healing process in the surrounding microenvironment. Latterly, cell-free therapies based upon these bioactive substances (rather than stem cells themselves) have shown considerable promise.

Autologous blood products

Autologous blood products are plasma- or serum-based medical therapies derived from minor manipulation of

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blood drawn from the injured horse. The blood products used most widely in both horses and people are autologous conditioned serum (ACS) and platelet-rich plasma (PRP). ACS is produced by incubating the blood with borosilicate beads to stimulate release of growth factors and inflammatory mediators by blood cells and platelets; subsequent centrifugation results in a cell-free product that may interfere with the destructive cycle of inflammation when injected intra-articularly. PRP is a simpler product produced from anticoagulated blood, being by definition plasma with a higher concentration of platelets than whole blood; in common with ACS, it contains a variety of growth factors and inflammatory mediators that may be beneficial to the healing process.

Gene therapy

Gene therapy is the transfer of specific genes to cells in the target tissue to allow for the enhanced or sustained production of therapeutic or regenerative products (proteins) by those cells. The goal is to alter the function of the target tissue so that it 'heals' itself, or at least continues to produce beneficial bioactive products that combat ongoing damage.

Physical therapies

Physical therapies may be used to limit inflammation and further tissue damage during the acute phase following injury, to modulate the healing response to optimize the return to full normal function and to manage acute and chronic pain. Many physical therapies used in human sports rehabilitation (Table 5.2) have been adopted into equine sports medicine; however, despite widespread application the scientific basis to support their use for most conditions is lacking. Often, therapies are adopted based on their popularity in the human field, or on some plausible scientific rationale derived from human or laboratory research that may be translatable to racehorses. It is important to recognise that even in human rehabilitation the majority of evidence used to support the use of many of these physical therapies is of low or very low quality: research studies are frequently compromised by high levels of bias, inconsistency and small sample sizes. National health boards, which review all available evidence and give judgements on both efficacy and cost-benefit determinations, frequently recommend against the use of such therapies in all but a few well-defined circumstances. Given the absence of equine research for most modalities, and inconclusive findings for many modalities in the human field, it is best in general to view physical therapies as adjuncts and co-interventions to rest/exercise prescription rather than as vital components of rehabilitation. They should in the first instance do no harm, and given that in most cases contribution to overall outcome (if any) is likely to be small they should not be recommended to clients as having efficacy beyond their evidence base.

Cold therapy

By reducing blood flow (through vasoconstriction) and thereby tissue metabolism, cooling can reduce the local swelling (oedema/haematoma) that occurs in the acute phase following injury, as well as assisting analgesia. It is most effective when utilized within the first 24–48 hours and should be applied for 15- to 30-minute periods 2–4 times/day. 'Rebound' vasodilation occurs in cyclical fashion in response to application of cold therapy. Regular post-exercise cold therapy is also useful as a management aid for horses in fast work to minimize joint/soft-tissue inflammation.

Effective application of cold therapy to the lower limb is easily accomplished. Ice baths, purpose-designed cooling units and cold-water spas can be very effective at deep cooling of the lower limb. Reusable ice boots/gel wraps can also be efficient cooling aids; however, it is important that they conform to the leg. Cold water hosing is relatively inefficient.

Heat therapy

Heat increases local circulation (and thereby metabolic rate in affected tissues) through vasodilation. For this reason it is usually avoided in the acute phase following injury, but may be beneficial during the repair phase that follows to increase metabolism and speed healing. Relief of muscle spasm is another application. Heat can be applied through direct conduction by using heat packs/bags (40–45°C, for 15–30 minutes); care should be taken to avoid burns when doing so. Depth of penetration of heat with direct conductive methods is limited to approximately 1 cm. Infrared lamps are also used for radiant heating. For heating of the deeper tissues (up to 10 cm), therapeutic ultrasound is required; heating is greatest at tissue interfaces and in tissues with a high collagen or protein content.

Manual therapy

Various forms of manual therapy ranging from massage to manipulative techniques are commonly used in racehorses. These are applied both as rehabilitation treatments and as 'maintenance' therapies (ostensibly to 'optimise' functional capacity). Practitioners of manual therapies may be skilled, but degree of training can be highly variable and trainers/owners generally have only a poor understanding of (or interest in) qualifications held

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Table 5.2 **Physical therapies used in equine sports rehabilitation**

THERAPY	DESCRIPTION	USE AND CLINICAL EFFECT	RESEARCH SUPPORT OF EFFICACY
Massage	Increases range of movement of joints and may have a role in restoring flexibility and function after injury	Limited evidence that it may reduce muscle soreness after fatiguing exercise; optimal timing for assisting recovery unknown	Uncertain
Manipulative therapy/ chiropractic	Proposed to improve vertebral symmetry and restore normal joint motion and back/ pelvic kinematics	Little equine research. Some support for treatment of human back pain; however, clinical effect (reduced pain/return to normal function) is limited	Weak
Extracorporeal shockwave therapy (ESWT)	Pressure wave generated by mechanical concussion, with release of kinetic energy at interfaces of different tissue densities. Proposed to upregulate healing particularly at ligament/bone interfaces	No strong evidence that ESWT improves outcomes in most equine/human orthopaedic conditions. Considered most useful in management of insertional tendinopathies; may induce neovascularisation and repair. Short-term analgesic effect reported. Denervation through long-term use is a possible explanation for some reports of clinical efficacy Can damage normal tendon/ligament tissue	Uncertain
Electrotherapy (TENS: transcutaneous electrical nerve stimulation; NMES: neuromuscular electrical stimulation)	Direct application of electrical current to skin to stimulate nerves or neuromuscular units	Used in human medicine primarily as adjunct to drug administration for management of acute and chronic pain. Current evidence is insufficient to support use for pain control Some evidence of efficacy in managing/ limiting muscle atrophy (following surgical or neurological trauma)	None/weak (for pain management).
Therapeutic ultrasound	Ultrasound generates acoustic energy that is absorbed in tissues through which sound waves pass. Can generate heat; also non-thermal effects such as cavitation and thought to have positive effects on blood flow and tissue metabolism. Depth of penetration up to 10 cm for 1 MHz	Little evidence of efficacy in management of musculoskeletal pain and soft tissue injury. Low-intensity pulsed ultrasound (daily) shortens healing time of acute, non-surgical fractures in people. Use in stress fractures and following surgical repair is not considered beneficial	None/weak
Acupuncture	Proposed to achieve physiological effects through stimulation of specific points by variety of means (needles/ pressure/ electricity/laser)	Large placebo effect in human trials. Positive effects (such as analgesia) generally of small magnitude; however, it is harmless and use for management of chronic pain in humans is therefore not deterred	Uncertain
Low-level laser therapy (LLLT)	Proposed to induce photochemical effects (poorly defined) on tissue metabolism	Some evidence in human medicine that use as adjunctive therapy can reduce pain/ stiffness for some chronic musculoskeletal conditions. Most laser radiation is absorbed/ scattered in skin: skin colour, thickness and preparation all affect penetration	Uncertain
High-intensity laser therapy (HILT)	Multidirectional/scattered energy applied to deep tissues proposed to induce photothermal and photochemical effects: possible effects on cell metabolism, vascular permeability, sensory conduction	Some evidence in human medicine that use as adjunctive therapy can improve pain/ function for some chronic musculoskeletal conditions. Some improved healing of experimental ligamentous injury reported in horses. Tissue effects potentially influenced by wavelength/power/pulse variables	Uncertain
Pulsed magnetic field therapy	Proposed to enhance blood flow and tissue metabolism, with resulting analgesic effect and reduction in healing times	Mixed evidence in human medicine of some efficacy in improving pain, stiffness and physical function for a variety of conditions	Uncertain

(Continued)

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Table 5.2 **Physical therapies used in equine sports rehabilitation** ***(Continued)***

THERAPY	DESCRIPTION	USE AND CLINICAL EFFECT	RESEARCH SUPPORT OF EFFICACY
Vibration therapy	Mechanical vibration platforms proposed to have effect on bone metabolism and theoretical positive effect on bone strength, fracture healing and proprioceptive repair	Current evidence is insufficient to support use in human/equine athletes	None/weak
Hyperbaric oxygen therapy (HBOT)	Increased oxygenation of tissues proposed to have physiological effects that may lead to enhanced healing	Some evidence that healing of certain conditions (arterial ulcers, osteomyelitis) is improved with HBOT. Current evidence is insufficient to support its use for musculoskeletal injury	None/weak

Research support definitions: **strong**, studies generally support effectiveness; **uncertain**, some positive findings are available, but confirming research needed; **none/weak**, little or no positive data available.

or what different types of therapies involve. Practitioners often become successful through word of mouth rather than any objective measures of competence. Titles of some disciplines (physiotherapist, chiropractic, osteopath) may be protected by legislation, and in human medicine these titles provide some assurance of minimum levels of training, regulation and professional indemnity insurance; however, these protections frequently do not extend to veterinary applications.

Manipulative therapies may incorporate passive stretching and range-of-motion exercises for the back, neck and limbs; tissue mobilisation through massage; myofascial release and joint mobilisation through passive and active manipulations. Lunging and ridden aids can be used to strengthen the musculature of the topline and improve balance. While validation of specific treatment/maintenance protocols (including type and frequency of treatments) is absent in the scientific literature, there is some evidence to support the use of physiotherapy techniques to reduce back (epaxial muscle) spasm and pain and improve range of movement of the spine in the equine athlete. Evidence of efficacy related to many of the other (unregulated) manual therapies used in racehorses is weak or absent.

Acupuncture

Acupuncture, and allied therapies (dry-needling and electro-acupuncture) are widely used in human medicine. Each therapy has its own philosophy and treatment points, but in short they involve stimulation of acupuncture or trigger points in soft tissue in an attempt to restore normal function or 'balance'. A large body of scientific research exists in the human field regarding its use in a multitude of conditions ranging from chronic musculoskeletal pain to migraines and labour pain; however, the quality of much of the research evidence (both supporting and opposing) of efficacy is poor. As it is considered to be a harmless intervention with some evidence of benefits in alleviating/management of chronic pain its use is supported for some conditions. It follows that it may have a role in equine sports medicine; however, it is reasonable to assume the magnitude of any potential benefit to be low.

Extracorporeal shockwave therapy (ESWT)

Extracorporeal shockwave therapy (ESWT) utilises pressure waves travelling through soft tissues to transmit energy and stimulate biological effects proposed to assist in healing of injuries. Kinetic energy is released primarily at interfaces between tissues of different densities, and effects are considered to be greatest where soft tissue meets bone such as at ligament insertions. The shockwaves can be either focused or unfocused (latter are 'radial' pulses, generated pneumatically).

Energy transmitted per unit pulse is the 'energy flux density' (EFD). Treatment protocols vary in terms of energy density and frequency of the shockwaves. In general the biological effects of ESWT are dose dependent: low EFD is broadly associated with stimulatory effects (largely considered beneficial, such as growth of blood vessels), and higher EFD is broadly associated with dose-related levels of cellular damage. Additionally, ESWT is also considered to have potential short-term analgesic effects (through unknown mechanisms) and because of this its use is regulated in the pre-race period in many jurisdictions.

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ESWT in human sports medicine has been widely researched and evidence of its efficacy in assisting rehabilitation or pain management of the main pathologies that are comparable to equine applications is inconsistent at best and frequently poor/lacking. Sufficient evidence to justify its use as a routine rehabilitation therapy in equine sports medicine is currently lacking.

Therapeutic ultrasound

Ultrasound therapy involves the delivery of soundwaves to soft and hard tissues: the resulting low-level micromechanical forces that are induced are thought to stimulate biological effects, with enhanced healing times for fractures being one of the proposed uses of the modality. While it is safe to use, there is little current evidence of efficacy, and as a monotherapy it is unlikely to result in quantifiable functional improvement in most equine orthopaedic conditions.

Therapeutic laser

As with other electrotherapeutic therapies, therapeutic laser therapy ('photobiomodulation therapy') is proposed to induce biological effects in the tissues to which it is applied that may be beneficial to rehabilitation and pain management. Specific mechanisms are poorly defined but include effects on cell metabolism, blood supply and mediation/conduction of pain. With low-level light therapy (LLLT; power <500 mW) the majority of energy applied is absorbed/scattered in the superficial layers of equine skin and the likelihood of substantial disease-modifying clinical effect resulting from use is low. High-intensity laser therapy (HILT; power >500 mW) has deeper penetration and ability to stimulate a biological effect for equine conditions is more plausible. Evidence to support use for both LLLT and HILT to assist pain management and return to function for a variety of chronic musculoskeletal conditions exists in human medicine but is of low/moderate quality; efficacy is largely untested in equine sports medicine.

Pulsed magnetic field therapy

Pulsed magnetic field therapy is used in a range of human rehabilitation applications with proposed effects on blood flow and tissue metabolism. Some evidence that low-intensity/low-frequency pulsed magnetic field therapy may assist reduction of pain and stiffness and improve physical function in a variety of conditions, although prescription of therapy remains controversial due to conflicting evidence base. It should be differentiated from static magnetic therapy (commonly applied to horses in the form of 'magnetic blankets'): no evidence for efficacy of the latter for any orthopaedic/rehabilitation use and indeed good evidence that static magnetic therapy does not induce any beneficial effects on blood flow, muscle tension, pain thresholds or skin temperature.

Vibration therapy

Whole body vibration therapy is claimed to assist fracture healing and minimise bone deconditioning during rest periods; it is applied through the use of large rubber-topped platforms or installed stable floors that oscillate at variable frequency. Mechanism of action considered to be that vibrations lead to reflex stabilising muscle contractions with resulting forces on skeleton stimulating bone formation/suppressing bone resorption. Evidence from non-equine research: mixed and inconsistent results for effects on bone metabolism, fracture healing and bone callus strength/quality, and highly dependent on frequency of applied vibrations (with possibility of negative effects as well). No overall effect on bone mineral density. Caution is advised in clinical applications, as even small changes in vibration settings could be associated with disruption of fracture healing; general application to horses rehabilitating from injury is considered overly simplistic and of questionable benefit.

Topical therapy

A wide variety of topical liniments, cooling gels, osmotic sweats and clays are used on the lower limb to manage inflammation. Use of these products is supported by empirical evidence only, but many appear to have a positive effect in reducing heat or swelling. Protocols for application vary between products but can be classified as open or bandaged. Common ingredients of many anti-inflammatory preparations include menthol, witch hazel distillate *(Hamamelis virginiana)*, peppermint oil, eucalyptus oil, salicylate and aloe vera. Menthol is an active ingredient that elicits a strong perception of cooling (mediated by stimulation of thermosensitive nerves) and through inhibition of nerve conduction also acts as an analgesic. Witch hazel has anti-inflammatory effects but is less potent than corticosteroids. Dimethyl sulfoxide (DMSO) is frequently used as a skin penetration enhancer in compounded preparations for delivery of anti-inflammatory medication (usually corticosteroids) into deeper tissues. Other ingredients have little or no support for efficacy but are generally considered benign. Use of some products (including kaolin clay) should be avoided on broken or infected skin.

Capsaicin is a naturally occurring irritant compound that can be found in low- and high-concentration topical products. It appears to act through initial irritation/

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hypersensitivity, which is followed by a longer period of desensitization, particularly when used over a long period. Efficacy is likely to be restricted to the skin.

Counter-irritation

The principle underlying the use of counter-irritation is that provocation of an inflammatory response may assist or revitalize healing processes in the limb. Counter-irritation may take the form of topical 'blisters', cryotherapy (freeze firing) or thermocautery (pin or bar firing). Blistering is most commonly used for dorsal metacarpal disease, splints and curbs and involves the use of an irritant or caustic skin dressing/paint (iodine/cantharidin/mercuric iodide-based) that is applied to effect depending on the product. The result is usually a superficial dermatitis or cellulitis, with thickening of the limb that may persist for weeks. Cryotherapy (freeze firing) is the application of a liquid nitrogen-cooled probe to the skin; local hair depigmentation (or loss) and temporary desensitization of the skin result after a mild inflammatory reaction. Pin or bar firing involves the invasive cautery of skin +/– underlying tendon or bone. Another counter-irritation technique is direct (needle) scarification of the cannon, typically used for the treatment of dorsal metacarpal disease (+/– infiltration of corticosteroid).

Aside from needle scarification for dorsal metacarpal disease, there is no evidence base for the efficacy of any counter-irritation method in assisting the quality or speed of healing. In particular, support for the use of firing as a preferred treatment for superficial digital flexor tendinitis has diminished with evidence that healing may be compromised. Any observed benefits of blistering/firing can usually be attributed to the enforced rest that accompanies these interventions. There is limited anecdotal support for the use of thermocautery in selected cases of persistently aggravated splints or curbs where prolonged rest and other therapies have been ineffectual.

Sclerotherapy

A feature of some chronic ligament and tendon pathologies is the growth of new blood vessels and nerves into and around the site of the lesion. There is some evidence from human sports medicine that this may be implicated in ongoing pain arising from certain injuries, although very little is known about comparable findings in equine injuries. Identification of vasculature using power Doppler ultrasound, and injection of sclerosing agents (polidocanol) under ultrasound guidance, can be very effective in shutting these blood vessels down, and is considered to be safe. However, evidence of efficacy is conflicting and there is currently no solid scientific foundation to support use as a routine treatment in equine conditions. It is known that nerve growth often accompanies neovascularization in these conditions, and it is possible that some of the empirical evidence of positive outcomes (for pain relief rather than healing of injury) may be related to desensitization (rather than ablation of blood vessels).

APPROACHES TO REHABILITATION

Introduction

While healing of any injury is primarily defined by the diagnosis, severity and musculoskeletal tissue involved, approach to rehabilitation is defined by individual circumstances pertaining to the horse, trainer/owner and veterinary advice, and may differ considerably between horses with similar injuries. Career priorities for the injured horse, time of year/season and training and rehabilitation facilities available are all important factors that may influence recommendations for rest, exercise and utilisation of adjunctive therapies. Compliance with these recommendations may vary depending on trainer/management-side factors unrelated to veterinary considerations. Furthermore, realised return to training times may be influenced by clinical progress (or setbacks) throughout the rehabilitation programme.

Individualised rehabilitation

The goal of any rehabilitation programme is not solely the satisfactory repair of the injured structure, but also to minimise the time out of training needed to achieve return to full function. Many flat racehorse careers are relatively short and in most jurisdictions the financial rewards arising from prize money and resale opportunities heavily favour horses that can achieve success during their 2- and 3-YO seasons. Setbacks arising from musculoskeletal problems are common during the first year in training, with the timing of any injury often being the crucial factor in whether career goals (ranging from racing targets to presentation for sale at a breeze-up/ready-to-run auctions) are achieved. Even minor adjustments to 'time off' may be career-changing in some circumstances, and intelligent application of individualised rehabilitation advice can realise significant economic benefits to trainers/owners.

Many factors involved in the repair of musculoskeletal injury vary between individuals: these include pathological

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severity and stage of training at time of diagnosis, intrinsic factors relating to conformation, body type and genetics and extrinsic factors such as track surface and training style.

It follows that modification (to a greater or lesser degree) of rehabilitation advice to individual circumstances is almost always possible. Whether or not individualised rehabilitation is necessary or desirable, however, may be influenced by time of year/season and horse value and pedigree, as well as variables as diverse as physiological propensity to lose/gain body condition or temperamental capacity of an individual to cope with different types of restricted exercise.

Rehabilitation recommendations in Chapters 6 and 7 should be regarded as guidelines only: they are derived largely from empirical evidence and benchmark the approximate periods and type of rest required for respective injuries to heal and return to full training. For every injury and rehabilitation programme there will always be horses that are statistical 'outliers' in terms of speed and success of recovery. However, it is also important to recognise that most established 'healing' time frames broadly correlate to those used for comparable human sports injuries and that expectations of fast-tracked return to full training derived from tailored rehabilitation should not be unrealistic: benefits are usually in the order of a few weeks at most.

When to individualise rehabilitation

Understanding when individualisation of rehabilitation might be possible, and when it might not, is an important component of veterinary oversight of a racing stable. Rate and quality of healing is primarily determined by the intrinsic properties of the particular tissue and anatomical structure involved, and recognition of the broad time frames required for structural repair of these tissues is fundamental. More severe or high-risk injuries require longer initial immobilisation periods than mild/low-risk injuries and this will necessarily be the greatest determinant of overall rehabilitation time. For some minor injuries that arise during a racing season there may be a desire to tailor rehabilitation to minimise loss of fitness, through use of training aids (such as treadmills) that involve some cardiovascular stimulus. However, significant cardiac and muscular deconditioning occurs after approximately 4 weeks out of active training, and as most bone stress and ligament/tendon injuries in racehorses necessitate at least 6–8 weeks out of cantering exercise, the focus of rehabilitation in most situations should be on repair rather than retention of fitness.

How to individualise rehabilitation

Individualising rehabilitation relies on accurate diagnosis as well as clearly defined staging of the tissue injury as acute, subacute or chronic. Consideration should be given to the presence of other pathologies (musculoskeletal or otherwise) that the horse may have that might benefit/suffer from application of exercise protocols or interventional therapies. As the key component of rehabilitation advice relates to prescription of rest/exercise, goals for progressing (or regressing) in the programme should be set and reappraisals scheduled at appropriate times. Progression through each stage of training should be based on objective criteria as far as possible, with the contribution of clinical and imaging information determined by the nature of the injury. At its simplest, individualisation takes the form of brief clinical reappraisals to assess the presence/resolution of lameness or palpable signs of inflammation; this can be a very effective method for ensuring that the conditioning of repairing tissues progresses at the correct pace as well as reducing unnecessary time spent confined or in low-loading exercise. By way of example, practical application in the case of bone stress injuries would involve checking that a horse is free from lameness in order to progress to the next exercise stage, then re-examining a few days/week into the next phase to ensure that no clinical setback has ensued.

Risks

Early introduction of loading exercise or shortening of total rehabilitation time can prove beneficial in terms of minimizing lost training time. However, there are also potential negative consequences of conditioning repairing tissues close to their physiological limits and these should be understood and communicated to the trainer/owner. If intensity or duration of loading at any stage is excessive then healing may become stalled or incomplete, and in some circumstances reinjury may result. Providing there is adequate clinical/imaging monitoring throughout rehabilitation, however, the greatest risk of any individualised rehabilitation that aims to minimise wasted training time is generally that of setback to repair of the healing tissues, rather than serious injury. When during the rehabilitation programme any setback becomes apparent depends on many factors but is typically not before a horse has resumed cantering. For some injuries (e.g., low-risk bone stress injuries) the potential benefits of a faster return to racing may outweigh the risks of recurrence of lameness (and further rehabilitation), whereas for injuries in which the consequences of reinjury may be career-ending (e.g., tendon strains, or cortical fractures at high-loading sites)

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a few weeks potentially saved in return to training may not be sufficiently attractive to warrant modifying rehabilitation recommendations.

Rest and exercise

Rehabilitation typically involves prescription of an initial rest period of variable length to minimise further injury and allow repair processes to occur, followed by a gradually increasing level of loading exercise to encourage damaged tissue to remodel/strengthen back to optimal function. This tissue remodelling phase is characterised by incremental rises in exercise intensity and duration. In human sports medicine guidelines for these incremental rises generally refer to a 10% increase in distance or intensity of exercise per week. By the nature of the equine gait, exercise phases are necessarily divided into walking, trotting, cantering and galloping, and smoothly graduated increases in training loads are rarely possible. Further modification is possible through time spent in each phase, method of application (horse-walker, ridden or treadmill) and speed/duration of exercise bouts. Alternate-day training is also sometimes utilised. How loading (and therefore healing) of specific anatomical sites is affected by choice of gait or details pertaining to application of exercise has received only limited research attention, such that there is not even a basic understanding of whether (for example) ridden trotting is preferable to treadmill cantering for a particular injury.

The need for an initial period of inactivity is determined by injury type/severity and should be prescribed on an individual basis. Horses with mild lameness or stress injuries detected early in the pathological process can often be removed from cantering/fast exercise and safely walked or trotted from the outset, while other injuries may necessitate an initial period of confinement. In the later stages of rehabilitation, the emphasis shifts from conditioning of the repairing tissue to restoration of aerobic fitness and musculoskeletal strength, and there should be an awareness that incremental progression through the exercise programme is necessary to minimise the risk of other injuries arising from overloading of deconditioned tissues.

Stable rest

Stable rest/confinement is widely used for the initial post-injury period, although it is not always necessary or ideal for healing of many injuries. Approach to confinement is generally influenced by the need to stabilise the injury site, and if injuries are considered to be stable or of low seriousness then complete stable rest is generally not necessary. This, however, is an aspect of equine sports medicine that has received almost no research attention and the relative benefits of stable rest with or without daily walking exercise (or indeed the amount of loading/movement that occurs even during periods of complete stable confinement) for rehabilitation of any given injury are unknown. From a management perspective, feed energy intake should be reduced and precautions taken to minimize the risk of colonic impaction (Chapter 12, p. 336). It is preferable to feed haylage and avoid straw bedding.

Cross-tying/tying up

For some fractures at high risk of catastrophic displacement it is sometimes advisable to tie the injured horse up to discourage lying down/rising and limit the strains that this imposes on the hindlimbs. The duration that this is attempted is determined by location and severity of injury; however, at least 3–4 weeks is required for the formation of bridging callus. Some unstable fractures of the pelvis require 6–12 weeks tied up (if tolerated). The question of whether to tie a horse up or not should be considered a choice of the 'least bad option', as there are significant risks inherent to both courses of action. While it is generally preferable for a horse with a high-risk fracture involving a hindlimb (typically the pelvis or tibia) to not lie down, some individuals left loose successfully protect themselves when doing so and have a good outcome, whereas others that are tied up may still suffer catastrophic deterioration. It is not uncommon for horses to experience fatigue after days/weeks tied up, which sometimes results in attempts to lie while restrained, a perilous situation particularly if the tether remains attached. In these circumstances it is often preferable to let the horse down fully rather than risk it getting down (frequently in the night, whilst unobserved) and struggling to rise.

When horses are tied up for orthopaedic injury they should be permitted to feed with their heads down to assist the clearance of lower respiratory tract secretions and minimise bacterial colonisation of the lungs, thereby lowering the risk of pleuropneumonia (Chapter 9, p. 309). Regular monitoring of temperature (or blood sampling to monitor inflammatory markers if patients are receiving anti-inflammatory/analgesic medication that might mask pyrexia) can permit early detection of illness.

In exceptional circumstances it might be desirable/possible to support the injured horse in a sling for the initial weeks following fracture. This is a specialised and intensive undertaking necessitating substantial nursing support (the major challenge being prevention of pressure sores when redistributing load away from the limbs) and is therefore rarely utilised outside of a hospital setting.

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CROSS-TYING HORSES

- Choosing a rubber-floored stable in quiet part of yard, and tying up to front of stable (to allow horse to face main areas of visual interest) is desirable.
- Tie-up should be long enough to allow horse to lower head/neck but not long enough to permit horse to lie down easily. Attachment to headcollar should be twine or similar to allow tether to break under tension.
- Haynet and water bucket should be placed at an appropriate height within easy reach of horse.
- Allowing horse to eat from the floor (i.e., let down) whilst under observation once/twice daily may reduce risk of development of pleuropneumonia.
- Twice daily monitoring of rectal temperature, +/– periodic blood sampling, for early detection of pleuropneumonia.
- Common for hindlimbs to 'fill' with lymphoedema and recommended that stable bandages are applied to minimise this.
- The goal is to discourage the horse from lying down; however, it is not possible (or desirable) to completely prevent a horse from attempting to lie down if it wants to, unless using a sling.
- Decision to tie up should be kept under constant review and horses managed on an individual basis: some horses tolerate long-term cross-tying (weeks/months) very well with few attempts to lie down, while others fatigue easily and break loose or lie down repeatedly.
- If a horse is displaying repeated (successful/unsuccessful) attempts to lie down, it may be judged safer to let the horse down fully than risk it becoming recumbent and struggling to rise whilst tied up.
- If horse tolerates cross-tying, determining when to cease is determined by clinical and imaging progress.

Walking

Some daily light-loading exercise is considered to have advantages over complete inactivity for the repair of many musculoskeletal injuries. Choice of in-hand walking or horse-walker exercise is dependent on the type of injury and the horse's temperament. Low-dose sedative medication (acepromazine) may be used to ensure overactivity is avoided.

Paddock turnout

For most injuries controlled exercise is preferable to unrestricted turnout, as even in small pens horses are capable of abrupt motion that can overload the repairing limb. If turnout is utilized during rehabilitation it should generally not commence until the period when trotting exercise would have resumed. However, when the primary objective is removal from training loads rather than rehabilitation (such as for carpal immaturity), paddock turnout at an earlier stage of the rehabilitation programme is permissible.

Treadmills

Treadmill exercise is useful for the rehabilitation of many injuries. Exercise duration and speed can be tightly controlled and the intensity of workload increased according to the planned programme without the variability and risks inherent to training on the track. The uniform treadmill belt surface and ability to train to faster paces without the weight of a rider avoids unnecessary and irregular loading of the limb and has the potential to improve outcomes of some injuries.

Surfaces

Range of motion of joints varies between ground surfaces of different types, with potential effects on rehabilitation of injuries. Of particular interest is degree of joint extension when considering rehabilitation of some key overextension/overloading pathologies (such as those associated with the fetlock, Chapter 6). Some surfaces have been commonly utilised (or avoided) in prevention or rehabilitation programmes for certain injuries by trainers: an example is ridden 'roadwork' to assist training preparation or recovery from tendon injuries. It is likely that surfaces and their interaction with loading of particular tissues will receive greater attention as an important component of the rehabilitation process; however, at present information is largely anecdotal. While the amount of flexion/extension of joints beneficial or detrimental to the rehabilitation of injury is unknown, the following points may be relevant:

- Stance phase (and therefore limb loading) during walking exercise is shorter on soft ground compared with hard ground and land treadmill.
- Greatest degree of extension of the fetlock during walking exercise occurs on the land treadmill.
- Greatest degree of extension of both carpus and tarsus occurs on hard ground.

Swimming

Swimming is a cross-training aid that permits low-level aerobic exercise without limb loading, and is commonly used as a partial substitute for trackwork for horses with limb injuries or soundness issues. Horses are not natural swimmers and when in the water use their hindlimbs for propulsion and forelimbs for balance. While swimming

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permits activity and some retention of fitness, it is not appropriate in all circumstances and its effect on the healing of many conditions is undetermined. Swimming involves extreme ranges of movement through the hip, stifle and hock joints, and the 'lordotic' posture it induces through the topline (extension of the neck, thoracolumbar spine and pelvis) may be detrimental to the rehabilitation of some conditions at these sites.

Water walkers/treadmills

Water walkers and treadmills are commonly used both for rehabilitation and as a training aid, and are considered to offer advantages over land horse-walkers and treadmills by providing low-impact, high-resistance work. Additionally, immersion of the limbs in cold water has potentially anti-inflammatory effects: hydrostatic pressure reduces oedema and increases lymphatic return, while low temperatures reduce blood flow and modulate pain. Increased mobilisation of joints assists rehabilitation of some injuries in human sports and could add to the beneficial effects of water walking for some conditions in horses, although effects remain undetermined. Depth of water used markedly influences both gait parameters (stride frequency, stride length, duration of stance and swing phases and protraction and retraction of limbs) and limb loading (through bodyweight reduction), and these factors should be considered carefully when incorporating its use into a rehabilitation programme.

- Water depth: buoyancy is minimal (and therefore any advantages for injury site of reduced loading negligible) when only the distal limbs are immersed. Bodyweight reduction with water depth at elbow level is approximately 10–15%; when the trunk is immersed buoyancy is substantial (at chest level bodyweight reduction is 60%; at pelvic level 75%).
- When water depth is at or below the level of the carpus, horses 'step over' the water to conserve energy. This results in increased rotation and flexion of the pelvis and back. Once water levels approach the upper limb, this is reversed and lateral bending and rotation of the topline is minimal.
- Effect of water depth on stride parameters differs between forelimbs and hindlimbs. As water depth increases, protraction of forelimbs reduces while that of hindlimbs may increase.
- Range of motion of specific joints is affected by water depth. Fetlock extension (and therefore limb loading) is greatest with fetlock-depth water, whereas fetlock flexion is greatest with hock-depth water. Hock-depth water also induces the greatest range of movement of the carpus, whereas stifle-depth water induces the greatest hock movement.

Although rehabilitation protocols incorporating the use of water walkers/treadmills have not been validated, given the current knowledge about the effect of water depth on gait, range of joint movement and limb loading some comments are applicable to specific injury sites:

- Joint injuries: not recommended during acute phase due to increased range of movement of joints; however, the same effects on joint flexion/extension (and reduced limb loading) may be beneficial for chronic joint injuries. Potential beneficial effects also include strengthening of the limb musculature supporting the affected joint, return of functional symmetry with the other limb(s) and improved joint stabilisation.
- Tendon (superficial digital flexor tendon [SDFT]) and suspensory ligament injuries (forelimb): not recommended during acute phase of injury because of changes in gait resulting from greater activation of the muscles that swing the leg forward. Following the acute phase of injury, the benefits of water walker/treadmill use come primarily from reduced limb loading: as the greatest strain in the tendon/ligament occurs during the stance phase, there is a rationale for starting rehabilitation exercise with deep water (for buoyancy) and progressively reducing water depth over time. Water walker/treadmill exercise does not appear to be strenuous enough to stimulate clinically relevant levels of strengthening/fitness adaptation in the superficial digital flexor muscle.
- Hindlimb proximal suspensory ligament injuries: treadmill (both dry and water) use causes increased retraction (caudal phase) in the gait of the hindlimb, and could potentially be detrimental to rehabilitation.

Other aspects of rehabilitation

Nutrition

Providing the rehabilitating horse is fed a balanced diet with sufficient trace elements and an appropriate calcium–phosphorus ratio, there is little nutritional advantage to be gained from any form of supplementation in respect to bone healing. Although nutritional products (such as essential and branch-chain amino acids, omega-3 fatty acids, antioxidants, vitamin K) aimed at assisting recovery from injury are widely marketed, there is no substantial

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evidence that supplementation of any nutrient beyond a normal diet is efficacious. Carbohydrate (starch) intake should be reduced and free-choice forage made available. Excessive bodyweight gain should be avoided as this may increase loading on the limb (and the need for conditioning) on return to exercise.

Rehabilitation: Fractures

Overview of bone healing

Bone repairs itself with functionally normal tissue and in doing so is the only tissue not to heal with a 'scar'. Providing bony union is not impaired by instability, infection or inadequate blood supply at the fracture site the repaired bone is generally as strong as (and sometimes stronger) than the original structure.

Most racehorse injuries involving bone arise as a result of 'stress' pathology, and if detected (and rested) prior to overt cortical fracture have the opportunity to repair through a return to equilibrium of bone remodelling at the affected site. When cortical fracture occurs (either subsequent to pre-existing stress pathology, or following application of a direct insult to the bone such as a kick or single supraphysiological loading event), healing must pass through several distinct and coordinated phases. The first is an inflammatory response at the fracture site, in which a blood clot forms and inflammatory cells migrate to the area. Injured and dead tissue is removed, and blood vessels grow into the site. The 'soft' callus that forms is comprised of chondrocytes (cartilage cells) and undifferentiated bone cells, and provides a scaffold for subsequent bone formation, which transforms it to 'hard' callus. Finally, bone remodelling over a longer period replaces the immature (and structurally weak) 'woven' bone with mature lamellar bone, and also ultimately returns the bone to approximate pre-injury dimensions. The process is regulated by complex interactions between cells; however, at a functional level factors such as too much instability, or insufficient blood supply at the fracture site can lead to an impairment of conversion of soft to hard callus, thereby resulting in a fracture non-union. Similarly, if immature healing tissue is subjected to strains that exceed its mechanical strength then reinjury and setback in ultimate healing time can result.

Classifying injuries

Stress fractures may become clinically apparent at any point on the spectrum of severity from early microfailure to complete cortical disruption, and rehabilitation times broadly correlate with the position on this spectrum of pathology at which diagnosis occurs. The severity of the injury also determines the need for surgical fixation or limb immobilization. An important part of planning rehabilitation therefore is accurate classification of injury.

Classification of stress fractures is based on two components: categorisation of injury type/location as 'low risk' (those with reliable healing patterns, and a low probability of propagation or of delayed/non-union) or 'high risk' (those with a greater likelihood of propagation to complete fracture, or of delayed/non-union), and grading of severity. Severity is broadly categorised as low grade for microfractures or injuries without a visible fracture line on (advanced) imaging, and high grade for injuries with a visible fracture line. To comprehensively categorise an injury therefore relies on both diagnosis and use of imaging techniques appropriate to assess extent of injury. Radiography (including CT) is considered an inferior technique for classifying stress injuries, as bone changes may either be too subtle for radiographic detection or lag behind the onset of clinical signs by some weeks; however, good refinement of injury classification for selected sites (e.g., tibia, fetlock) is possible with excellent technique and proficiency. Bone scan is a technique with good sensitivity for the detection of injury, but is also unsatisfactory for grading severity or staging progression. Magnetic resonance imaging (MRI) is the 'gold standard' modality for injury classification; however, it is currently only feasible to perform standing MRI of the lower limb (fetlock/cannon/lower carpus). Positron emission tomography (PET) has emerged as a useful modality to classify injury severity and activity and has many advantages over bone scan but remains to be validated against MRI for key injuries.

The diagnosis and classification of many racehorse injuries, particularly those higher in the limb, therefore relies on clinical findings aided by combinations of radiography, ultrasonography or bone scan. This does not generally allow the precise staging of pathology that is possible in human athletes; additionally, humans can communicate the presence and quality of pain experienced during recuperation, permitting appropriate adjustment of exercise intensity. Racehorse clinicians are restricted by the insensitivity of available imaging modalities, and recuperation programmes for many injuries are empirical. As a general rule, however, severity of initial lameness and the rapidity with which it diminishes (with rest) correlate well with the length of rehabilitation required. A broad classification that may be used as a guide to injury classification is shown in Table 5.3.

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Table 5.3 **Classification of stress injury severity**

GRADE	LAMENESS*	RADIOGRAPHY	BONE SCAN
1	None/subtle	Normal	Mild IRU
2	Mild-moderate	Normal	Moderate IRU
3	Moderate-marked	Periosteal/endosteal reactive change	Moderate-marked IRU
4	Moderate-marked	Periosteal/endosteal reactive change +/– cortical fracture line	IRU involving more than one cortex
5	Severe	Complete fracture of one/both cortices	Not required

IRU, increased radiopharmaceutical uptake.

* Unilateral lameness may not always be a feature (due to bilateral pathology).

DELAYED RADIOGRAPHIC EXAMINATION

- When fracture is suspected and initial radiographic survey proves unremarkable, it may sometimes be useful to re-xray 1–2 weeks later to assist diagnosis.
- Rationale is that bone loss/production resulting from innate repair processes will cause injury to become more apparent on delayed imaging.
- This presupposes significant bone activity in intervening period.
- In practice, re-xray is only useful with lameness of moderate or greater severity with relatively acute onset: chronic, low-grade lameness typically is not associated with rapid changes in bone metabolism.
- If the clinical picture hasn't changed substantially recently then don't expect radiographs to.

Rehabilitation

Recommendations are influenced by grade and location of injury. For fractures at low-risk sites, more rapid return to full use (up to 1/3 less rehabilitation time) is usually possible for 'low-grade' (i.e., no visible fracture line on advanced imaging) injuries than for 'high-grade' injuries. Stress fractures at anatomical sites that are considered high risk for either catastrophic deterioration or recurrence of injury must be treated more conservatively than injuries at 'low-risk' sites. Some stress fractures (e.g., humeral injuries) may propagate at low levels of exercise due to poorly-defined anatomical predispositions (such as forces of tension at fracture site). The relative proportion of time spent in stable confinement or walking should therefore be determined on an individual basis.

General recommendations are:

- Mild stress injuries (grades 1 and 2):
  - Stable confinement until sound at trot.
  - Return to walking as soon as possible.
  - Monitor soundness at each 'step-up'.
  - Total time out of cantering exercise: 6–8 weeks.
- Moderate stress fractures (grades 2 and 3):
  - 3–4 weeks stable confinement.
  - 3–4 weeks walking.
  - Monitor soundness at each 'step-up'.
  - Total time out of cantering exercise: 9–12 weeks.
- High-risk fractures (graded severe +/– potential for catastrophic propagation):
  - Fetlock: consider surgical fixation or bandage immobilization depending on fracture type.
  - Following surgical fixation: 4–8 weeks stable rest/4–6 weeks walking is typical.
  - Pelvis/tibia: consider tying up for initial 4+ weeks to minimize risk of fracture propagation when moving or rising in the stable. Further 4–6 weeks stable rest/6–8 weeks walking.

Monitoring healing

Monitoring of healing to ensure that it is safe to step a horse back into faster work is desirable particularly if recuperation times have been minimized. Unfortunately, accurate monitoring of bone strength is not possible in the racehorse. Radiography is of little use for most stress injuries, although it has a role in ongoing assessment of cortical fractures at sites such as the third carpal and tarsal bones, or fetlock. Bone scan is also of little use for determining when training loads may be increased, as scintigraphic activity usually remains elevated long after effective healing has taken place: the presence of increased radiopharmaceutical uptake (IRU) at the injury site does not preclude a return to full training. MRI and PET may be useful to determine reduction of activity in the case of fetlock injuries, but are not widely applied in practice. Therefore, the most practical way to monitor progress is regular clinical evaluation to ensure that lameness does

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not recur when exercise intensity increases. In human sports medicine it is widely accepted that during the rehabilitation of bone stress injuries an increase in training loads should not cause pain, and that the presence of pain during weight-bearing indicates that tissues are not ready for greater loading. While the relationship between pain and lameness/gait abnormalities in the racehorse may sometimes be uncertain, it is reasonable to use the initial lameness at time of injury diagnosis and its persistence or absence as a clinical yardstick to assess progress and guide exercise prescription.

During the early rehabilitation period ultrasonographic or radiographic imaging may assist managerial decisions such as appropriate timing to let a cross-tied horse down or resumption of walking or ridden exercise. It is only useful when obvious bone callus or a cortical fracture is present and progression (smoothing) of periosteal modelling or disappearance of visible fracture line can permit estimation of injury repair.

REHABILITATION RULES OF THUMB

- Initial clinical severity (of stress injuries) is frequently a good guide to likely length of rehabilitation needed, regardless of imaging findings.
- Walking exercise is preferred to complete inactivity (stable rest) unless the latter is necessitated by nature/severity of injury.
- Individualisation of rehabilitation is possible for majority of injury types and can be successfully achieved with minimal effort.
- Increased training load should not cause pain/lameness: monitor progression at each stage of rehabilitation and remain alert to signs that injury is still active.
- Most adjunctive physical or medical therapies used during rehabilitation are of uncertain efficacy and do not permit shortening of rehabilitation time recommendations.

Rehabilitation: Tendons and ligaments

Overview of tendon/ligament healing

Tendons and ligaments have generally poor innate regenerative capacity. They are slow to heal (typically many months) and do not fully regain their pre-injury physical characteristics. As with other tissues, repair of tendon/ligament injuries involves an initial inflammatory phase lasting a few days (with arrival of inflammatory cells and removal of debris) followed by a proliferative phase in which the laying down of an early matrix serves as a scaffold for later repair processes. Over several weeks more collagen is deposited, which then undergoes maturation over a longer 'remodelling' phase lasting months to years. Unlike with bone tissue (in which the repair process is a pathway to full return of 'normal' tissue), tendons and ligaments repair with disorganised scar tissue. The repair tissue that replaces the torn collagen type I fibrils of a strained tendon has poorer elastic strength and capacity for repetitive loading under high strains: the result is typically a structure that is mechanically inferior to undamaged tendon/ligament, even after the remodelling phase is complete. The resulting step change in stiffness between the site of previous injury and healthy tissue means that this interface is predisposed to recurrent injury upon a return to training.

In the acute phase of severe tendon/ligament injuries (or those with associated joint/limb instability) immobilisation may be justified to limit further damage. The nature of this immobilisation will be determined by the location and type of injury. It is important to recognise that tendon/ligament tissue requires loading to stimulate/direct the important remodelling phase of the repair process, and that restricting loading beyond what is necessary to control further damage can itself lead to much poorer healing due to reduced collagen production and haphazard arrangement of collagen fibrils. Early, controlled resumption of exercise is generally considered to have beneficial effects on healing and enable more rapid/successful return to maximum function. Rehabilitation strategies utilised to assist tendon/ligament healing in people such as exercises to promote range of motion and improved neuromuscular function and proprioception of joints have limited applicability in horses.

Successful return to racing following a tendon strain is generally considered to be a direct function of how closely the mechanical properties of the recovered tendon approach that of 'normal' tendon and this is determined by the extent of original injury as well as the quality of its repair. There appears to be an opportunity during the repair process for tendon tissue to remodel to some degree such that disorganised collagen fibrils partially regain parallel alignment (and therefore better overall mechanical function). Optimising the collagenous architecture of the injured zone of the tendon remains the key objective of most rehabilitation therapies in the horse; however, there is compelling evidence in human sports medicine that rehabilitation strategies that sustain/improve the uninjured part of the tendon are potentially more important for athletic outcome than those concentrating on the injured portion.

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Classifying SDFT injuries

Objective assessment of lesion severity is of use in determining a rehabilitation programme and prognosis. The full extent of the injury is best assessed after the initial swelling subsides (1–3 weeks after the initial signs). The entire tendon should be scanned using B-mode or ultrasound tissue characterisation (UTC) ultrasonography and the cross-sectional area of tendon and lesion measured, along with grading of tendon echogenicity and fibre alignment. Location of injury (both proximodistal, and within or on margin(s) of the tendon) should be determined and recorded. Basic classifications of injury using B-mode ultrasonography are shown in Table 5.4.

Management of SDFT injury: Acute phase

It has previously been considered that aggressive anti-inflammatory measures (both through drug administration and topical cold therapy) in the initial days following injury are useful interventions to limit the extent of injury. There is now much greater understanding that the initial inflammatory phase that occurs after 'acute' tendon injury plays an important role in setting a foundation for the repair process, and that inhibiting the complex cellular events taking place during this phase may be detrimental to overall outcome. Therefore any use of anti-inflammatory measures should be judicious and sparing and only to address excessive pain and acute swelling.

Exercise limitation during the acute phase is important, with stable confinement +/– limited hand walking only for the initial weeks. Supportive bandaging of the limb is only required for severe injuries with total/near total loss of tendon integrity (+/– sinking of fetlock): in these cases an immobilizing (Robert Jones) bandage may be required during the initial weeks.

Table 5.4 **Basic classifications of injury using B-mode ultrasonography**

Mild injury	0–15% tendon volume	<10% lesion size at maximum injury zone
Moderate injury	16–25% tendon volume	10–40% lesion size at maximum injury zone
Severe injury	>25% tendon volume	>40% lesion size at maximum injury zone

From Smith RKW, McIlwraith CW (2012). Consensus on equine tendon disease: building on the 2007 Havemeyer symposium. *Equine Vet J* 44(1):2–6.

Management of SDFT injury: Rehabilitation phase

Gradual reintroduction to controlled exercise is preferable. If turnout is desired, this should only follow on from the walking/trotting phase and should initially be in a small paddock. Treatment protocols are empirical, but the key points are:

- Severity of injury determines recuperation time, but in general at least 6 months out of cantering required. Strong empirical evidence that lengthier rehabilitation periods (>12 months) are more likely to result in successful long-term outcome.
- Monitoring of healing by serial B-mode or UTC ultrasound examinations (every 3 months or as required).
- Quality of fibre pattern alignment is an important prognostic indicator for likelihood of reinjury on return to fast work.
- Decrease or stability in size of tendon/size of lesion and increase in echogenicity are main determinants of progress.
- Enlargement of the tendon (by >10%) or further disorganisation of fibre pattern between scans may indicate excessive loading for the stage of healing and the exercise level should be reduced.
- Training programmes, aids and surfaces that minimize peak strains/high-impact loading in the injured tendon may improve likelihood of successful return to racing. Real-world effectiveness of these have been poorly researched; however, it is highly likely that future developments in successful management of SDFT injuries will arise in the field of biomechanics and training modifications. From what is currently known treadmill use (reduced load bearing) and inclined/uphill (lower speeds and SDFT strain) training may be beneficial. Further information on use of hydrotherapy can be found on page-62.

Additional interventions

Additional therapies (Table 5.5) aimed at reducing the likelihood of tendon reinjury are varied and frequently prescribed/recommended without full consideration of their efficacy. Good quality scientific evidence to support these interventions, particularly in respect to improved real-world outcomes is generally lacking.

Intra-lesional therapies are generally administered in the late acute phase (approximately 2 weeks post-injury). These therapies should, in any case, be considered as an optional adjunct (rather than an alternative) to a controlled exercise programme.

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Table 5.5 **Adjunctive therapies for tendon injury**

THERAPY	ACTION	EFFICACY
Intralesional injection: stem cells	Injection of mesenchymal stem, stromal or progenitor cells proposed to improve qualities of healed tendon	Evidence of enhanced quality of tendon repair and preliminary evidence of reduced reinjury rate; however, true clinical efficacy remains undetermined
Intralesional injection (other): Platelet-rich plasma, insulin-like growth factor-1, polysulphated GAG, hyaluronic acid	Various therapies proposed to optimize quality of healing or speed maturation of healing tissue	Currently no definitive evidence of effectiveness for most interventions Some evidence that intra-lesional platelet-rich plasma may enhance return to optimal fibre pattern through injured area although effect on reinjury rate remains unknown
Tendon splitting	Scalpel/needle lancing of core lesion during acute phase may limit physical/enzymatic damage to adjacent tendon tissue	Little/no good quality evidence of efficacy
Surgery: desmotomy of accessory ligament of the SDFT (AL-SDFT or 'superior check' ligament)	Sectioning of AL-SDFT reduces future load bearing in the SDFT	May reduce risk of reinjury; however, results in redistribution of load-bearing forces: increased risk of suspensory ligament injury upon return to training.
Thermocautery (bar/pin firing)	Intended to cause counter-irritation and stimulate inflammatory response +/– formation of supportive scar tissue	Not shown to reduce risk of reinjury and ethical concerns limit use
Pulsed magnetic field therapy	Proposed to modulate inflammation/stimulate cellular repair	Current evidence does not support use
Therapeutic ultrasound	Thermal (heating from sound wave absorption) and non-thermal (cavitation) effects	Conflicting evidence (human studies). No good quality evidence of efficacy in horses
Low-level laser therapy ('cold laser')	May reduce inflammatory response	Current evidence does not support use

AL-SDFT, accessory ligament of superficial digital flexor tendon; GAG, glycosaminoglycan; SDFT, superficial digital flexor tendon.

The following exercise programme (Table 5.6) can be used as a general guide, with progression of exercise determined on an individual basis by ultrasonographic re-examinations.

A shorter rehabilitation programme may be considered for subtle/mild injuries for which the risk of any reinjury with more rapid return to training is deemed acceptable. In these cases ultrasonographic monitoring throughout the rehabilitation period is the best guide to determining the readiness for increased loading on an individual basis. With satisfactory clinical and ultrasonographic progress, a return to trotting at 8–10 weeks, cantering at 16–20 weeks and fast work at approximately 6 months may be possible.

Rehabilitation: Skeletal muscle injuries

Overview of muscle healing

At a cellular level the repair of muscle shares some characteristics with repair of other tissues. There is an initial inflammatory phase (lasting 1–3 days) in which influx of inflammatory cells occurs and devitalised muscle tissue is removed; this is followed by a repair phase (of a few weeks) in which precursor (satellite) cells become activated, regeneration of muscle cells occurs and scar tissue forms. And finally there is a longer remodelling phase in which the regenerated muscle fibres mature and normal strength and function is regained.

Following minor injuries, skeletal muscle tissue can regenerate completely. By contrast, severe injuries, or those in which the healing process is compromised, can result in the formation of scar (fibrotic) tissue that is mechanically very different to the contractile muscle tissue around it and therefore impairs function. Scarring following muscular injury to the extent that athletic function is affected is a rare outcome in racehorses.

Management of muscle injury

Focal muscle injuries of clinical significance (i.e., causing obvious lameness or impaired function) are relatively uncommon in racehorses. Most heal satisfactorily without any intervention; therefore there is little incentive to manage muscle rehabilitation beyond adhering to some general principles of treatment. Initial clinical severity

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Table 5.6 **Guide exercise programme/SDFT injury**

WEEK	EXERCISE
0	stable confinement
1	stable confinement
2	stable confinement
3	10 min walk
4	15 min walk
5	20 min walk
6	20 min walk
7	25 min walk
8	25 min walk
9	30 min walk
10	30 min walk
11	35 min walk
12	35 min walk
13	40 min walk
14	40 min walk
15	40 min walk/5 min trot
16	40 min walk/5 min trot
17	40 min walk/5 min trot
18	40 min walk/5 min trot
19	35 min walk/10 min trot
20	35 min walk/10 min trot
21	35 min walk/10 min trot
22	35 min walk/10 min trot
23	30 min walk/15 min trot
24	30 min walk/15 min trot
25	30 min walk/15 min trot
26	30 min walk/15 min trot
27	25 min walk/20 min trot
28	25 min walk/20 min trot
29	25 min walk/20 min trot
30	25 min walk/20 min trot
31	20 min walk/25 min trot
32	20 min walk/25 min trot
33	15 min walk/30 min trot
34	15 min walk/30 min trot
35	Resume cantering
36	Build up cantering
37	
38	
39	
40	
41	
42	
43	
44	Resume fast work/galloping

Shading: ultrasonographic re-examination recommended.

and location of injury will determine whether consideration should be given to a 'managed' rehabilitation: most thigh haematomas, for instance, despite arising from a (presumably significant) muscle belly tear heal fully with no impairment of function.

In the initial days following injury the goal is to minimise further damage: the presence of lameness, gait restriction or local inflammation indicates that rest from full training is appropriate. Anti-inflammatory medication may be used to moderate pain and muscular spasm; however, excessive use within the first 2 days following injury may interfere with the important early phase of the repair process and is discouraged, along with prolonged use beyond the acute phase. Gradual increase in mobilisation can begin within a few days, with clinical progress guiding management at all stages. While mechanical conditioning of the regenerating muscle is important for optimal healing, detailed rehabilitation guidance is not usually required to achieve it.

In human sports medicine many interventions have been utilised for some of the important muscle injury types, in an attempt to maximise the quality of repair and speed of return to full use. These include injection of biological agents (e.g., PRP) and electrotherapies. There is little scientific support for efficacy of most of these interventions and the indications for considering their use in the equine athlete are extremely limited. Some anti-fibrotic agents have promise as therapies to limit the formation of scar tissue; however, as noted previously the fact that most racehorse muscle injuries heal uneventfully makes the routine use of any medical interventions difficult to justify.

Rehabilitation: Joint disease

Overview of joint healing

Articular cartilage has no direct blood supply and is sparsely populated by the cartilage cells (chondrocytes) essential for production of cartilage matrix, and therefore has a poor capacity for regeneration. Healing is incomplete and slow. Depth, severity and location of cartilage injury within the joint all influence overall functional outcome. Repair of partial-thickness ('chondral') injuries relies on the very limited capabilities of the few undamaged chondrocytes at the injury site, and in consequence is usually incomplete and consists of fibrous scar tissue. Full-thickness ('osteochondral') defects may penetrate the underlying subchondral bone and if so benefit from a direct blood supply, proximity of bone marrow and availability of mesenchymal stem cells leading to better overall functional repair. However, healing of even these deeper lesions does not return the joint surface to normality, as

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the fibrocartilage that fills the defect is mechanically inferior to articular cartilage.

The principal racehorse injuries that involve joints are rarely pathologies of articular cartilage alone. Chronic joint injury may result in proliferation of soft tissue and bone at joint margins (osteophytes/'bone spurs'), in part as a response to joint instability: fragmentation or chip fracture of these joint margins may result. Adaptive or pathologic change in the underlying subchondral bone may frequently contribute more to the overall clinical picture than the purely articular component of the osteoarthritic joint, and returning the subchondral bone to health is an important consideration for rehabilitation.

Subchondral bone injury and its repair have been extensively researched in the fetlock, the key site for the most important racehorse articular pathologies. It is likely that some of what is known from the fetlock also applies to other important injury sites such as the third carpal bone. Subchondral bone remodelling (replacement of old/damaged tissue with new bone) rates are suppressed during sustained high-intensity training; however, the switchover to intense repair activity can be dramatic as soon as rest is permitted. While there is large variation between horses in this response to rest, maximal bone repair typically occurs within the first 10 weeks. A feature of this 'rebound' remodelling can be greatly increased porosity of the subchondral bone, and if this weakened bone is subjected to excessive loads then collapse of the subchondral bone plate can ensue. Rehabilitation of horses with some subchondral bone pathologies therefore carries with it a significant risk of irreversible deterioration (sometimes necessitating retirement/euthanasia), and the potential benefits of rehabilitation should therefore be balanced against the necessity for any such rehabilitation based on clinical history. Risk factors for determining which horses are at enhanced risk of serious subchondral deterioration if removed from training, and factors that might mitigate that risk, have not been established. More broadly, validated recommendations on type and duration of rest required to rehabilitate subchondral bone pathologies do not exist and it is preferable to structure rehabilitation programmes on individual circumstances, taking into account the site of pathology, severity of disease and sequential clinical and imaging monitoring to minimise the risk of deterioration.

Management of joint disease

The diagnosis and severity of injury largely determine appropriate management. The approach to acute injuries (synovitis, osteochondral fragmentation) or those associated with risk of fracture may differ considerably from that taken with chronic/long-standing joint disease. Most articular conditions in the racehorse tend to affect only a small number of joints – sometimes only one. This favours intra-articular therapies, where the therapeutic agent can be targeted directly to the site of importance. Goals of treatment include returning affected joint(s) to normal function as rapidly as possible, optimizing the long-term health of joint(s) by limiting progression of disease, and managing pain/lameness to permit continuation of training.

Some of the rehabilitation strategies considered important for articular cartilage repair (particularly after surgical intervention) in people are not feasible in horses. Passive motion exercises, and joint-specific programmes structured around defined range-of-motion and load parameters do not translate well to equine rehabilitation.

Exercise modification

The diagnosis and chronicity of injury determine whether exercise modification is required, and the optimum rest/rehabilitation period. In many cases of low-grade or chronic joint pathology rest is neither curative nor desirable. Continued training in some circumstances may be aided by the use of treadmill or swimming exercise, or tracks with surface/geometry considered sympathetic to the particular injury. Cartilage requires some mechanical stimulus to optimise healing and so the reduced-weight-bearing exercise that is possible with water walkers/treadmills may be beneficial.

Physical therapy

Acute synovitis may benefit from cold therapy and topical anti-inflammatory measures. Physical therapy is generally of little use in chronic joint conditions; however, regular cold therapy is widely used and is possibly beneficial to recovery from fast exercise.

Surgery

While arthroscopy may be useful as a diagnostic procedure in some circumstances, the majority of racehorse joint disease is manageable non-surgically. Surgery is not the leading intervention for osteoarthritis (OA) but may be advocated in the treatment of conditions such as chronic proliferative synovitis and certain types of osteochondral fragmentation/fracture in which conservative therapy has proven unrewarding. In these cases debridement of osteochondral defects may improve the prognosis for future soundness. Surgical interventions beyond simple debridement include cartilage repair, grafting and subchondral

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bone stimulation: techniques are evolving and application should be guided by individual circumstances.

Intra-articular therapies

Joint disease is often best managed by delivering medication directly to the target site. While current intra-articular medications are not considered to 'heal' cartilage, they may be disease- or symptom-modifying; interference with the inflammatory cascade can break the cycle of cartilage degeneration (and therefore limit joint pain). The goal of intra-articular therapy is long-term preservation of athletic function through protection of cartilage; elimination of lameness is also important to normalize gait and limit overloading of other limbs. Although the relationship between observable lameness at a trot and action/performance at faster paces is not currently known, in the absence of evidence to the contrary it is presumed that managing such lameness is beneficial at some level to the athlete with joint pathology. While intra-articular drugs are rapidly removed from the joint space after administration, many of them (such as corticosteroids) exert an influence on cartilage metabolism that persists beyond the time that the product is actually present in the joint.

The objective risks associated with intra-articular medication include introduction of infection into the joint (synovial sepsis) and, in the case of corticosteroids, laminitis. These risks are negligible (combined incidence <0.04%) in the racing Thoroughbred with appropriate technique, and corticosteroids are no longer considered to cause increased statistical risk of laminitis. Some medications may be associated with occasional inflammatory joint 'flare' and justify combination therapy to prevent occurrence. Of greater importance is the potential to mask warning signs of impending fracture through elimination of lameness, and in many circumstances it is now recommended that medication is used only after undertaking a 'risk assessment' for potential fracture propagation (particularly with fetlock disease). Another significant risk is that of a regulatory adverse analytical finding at post-race sampling: while there is reasonable confidence around 'safe' withdrawal times in most jurisdictions for corticosteroids in the most commonly treated joints, significant individual variation in excretion times is well documented and may arise from factors such as joint disease status or inadvertent administration into synovial or ligamentous tissues (resulting in 'depot' effect with considerably lengthened detection times).

The main classes of intra-articular medication in common use are corticosteroids, sodium hyaluronate (hyaluronic acid/hyaluronan), biocompatible hydrogels and biological ('biologics'/'regenerative') products (Table 5.7). Clinical presentation, diagnosis, regulatory constraints and cost are key factors in the choice of appropriate therapy for the individual. Indications for treatment range from reduction of acute inflammation or assisting post-surgical rehabilitation, to controlling chronic lameness in full work. Treatment is often empirical and even in horses with similar pathology there may be considerable individual variation in response; post-medication exercise protocols and interval to re-medication should reflect this. A 24-hour rest (stable rest/walk) period following any intra-articular medication is recommended and may prolong the effects of medication.

There is no single intra-articular therapy that is most appropriate for all joint conditions, and the limited current research does not permit firm conclusions to be drawn about relative clinical efficacy of the main product categories for key racehorse applications. Corticosteroids (most commonly triamcinolone acetonide) have been the mainstay of intra-articular therapy for synovitis and OA and are generally considered to give excellent short- to medium-term control of inflammation/lameness while optimizing joint/cartilage health. It has been common practice to administer corticosteroids in combination with sodium hyaluronate; however, current evidence does not support routine use of the latter. It has also been common practice to combine intra-articular medications with antibiotics; however, there is no supporting evidence that this practice has any effect in diminishing the (already very low) risk of joint infection, and there is evidence that intra-articular amikacin may have detrimental effects on cartilage.

In the human field there is great interest in developing therapeutic agents (such as gene therapies and oligonucleotide therapeutics) that alter the course of joint disease by modifying cellular function: these hold considerable promise that future intra-articular treatments may return articular tissues to good health rather than just mitigate inflammation on a short-term basis as is currently the case.

Systemic medications

Systemic medications may be used as an adjunct to other OA therapies (Table 5.8). NSAIDs have an established role in managing lameness; however, long-term use or administration without risk assessment for further injury is discouraged. Other systemic medications are proposed to assist cartilage repair; however, strong evidence of efficacy is generally lacking and clinical effect likely to be small.

Nutraceuticals (oral joint supplements)

Nutraceuticals (Table 5.9) are dietary supplements with little primary nutritional value that are proposed to assist

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Table 5.7 **Intra-articular medications for management of joint disease in horses**

PRODUCT		ACTION	USE AND EFFICACY
Corticosteroids	Triamcinolone acetonide (TA), methylprednisolone acetate (MPA), betamethasone, dexamethasone	Strongest anti-inflammatory agents available for intra-articular use and have greatest potential to modify lameness. Anti-inflammatory effects include reduced vascular permeability (joint filling), reduced migration of inflammatory cells and inhibition of pro-inflammatory enzyme release. Relatively short half-life within joint space but can exert prolonged effect on synovial lining and cartilage. TA: may be chondroprotective; no detrimental effects with normal use. MPA: longest acting but can potentiate cartilage degeneration.	TA: 6–10 mg per joint. Duration of therapeutic effect typically 4–8 weeks. Betamethasone and dexamethasone: less potent and limited efficacy for lameness. MPA efficacious but used infrequently because of prolonged detection time
Hyaluronic acid (HA)	Non-sulphated glycosaminoglycan (GAG) found in synovial fluid and cartilage. Primary function is joint lubrication (determines viscoelasticity of synovial fluid)	Half-life within joint is short (few days in normal joint, few hours in arthritic joint). Primary effect on cartilage and synovial metabolism rather than as replacement for endogenous HA; anti-inflammatory effects and inhibits cartilage degeneration. Correlation between molecular weight/concentration/volume and clinical effect is uncertain; currently not possible to differentiate between products clinically. May cause joint flare if administered alone (without corticosteroid).	Typically used in combination with intra-articular corticosteroid (TA). Moderate level evidence that has no/little clinical effect; current evidence does not support routine use.
Polyacrylamide hydrogel (PAAG)	Synthetic, non-degradable hydrogel	Becomes incorporated into synovial lining in the weeks following injection, forming a subsynovial layer with long-term (years) persistence. Hypothesised to cause long-term augmentation of joint capsule and synovial lining, possibly increasing capsular elasticity and strength and reducing cartilage friction.	Most commonly applied as one-time injection. Validated efficacy for diminishing lameness. Full clinical effect may not be evident until 4–6 weeks. Evidence of superior relative efficacy to TA, HA. Duration of clinical effect may be >12 weeks.
Autologous or allogeneic biological products ('regenerative therapies')	Autologous conditioned serum/interleukin-1 receptor antagonist protein (ACS/IRAP): disease-modifying osteoarthritis (OA) product; harvested blood incubated with glass spheres, which stimulate leukocytes to produce anti-inflammatory cytokines and growth factors. Platelet-rich plasma (PRP): concentrated platelet (and growth factor)-rich portion of plasma. Mesenchymal stem cells (derived from range of tissues)	ACS/IRAP: disease-modifying protective and anti-inflammatory activity. PRP: growth factors proposed to enhance tissue repair. Mesenchymal stem cells: intrinsic properties and mechanism of action within joint not fully understood. Cells survive in joint only for short period but may exert effect by secreting bioactive factors that modulate immune response and endogenous regeneration.	ACS/IRAP: variable treatment frequency (course of 2–3 injections, or regular treatments). Limited analgesic effect but may reduce joint inflammation. PRP: variable treatment frequency. Efficacy undetermined. Both products have benefit of being free of prohibited substances (if peripheral blood also clear at time of harvesting). Scientific validation and standardization of protocols needed. Favourable clinical reports (equine and human) but high-quality evidence of practical efficacy lacking. Mesenchymal stem cells: most commonly applied as one-time injection. Validated efficacy for OA of mild-moderate severity.
Anabolic steroids	Stanozolol	Upregulates collagen synthesis, chondrocyte and osteoblast proliferation and has chondroprotective and anti-inflammatory effects *in vitro*. Use may be limited by regulatory controls.	Low-dose (5 mg; <2% of intramuscular 'anabolic' dose) medication at 1–2 weekly intervals for 3–6 treatments. Favourable clinical reports (equine) but high-quality evidence of efficacy lacking.
Gene therapy, immunologic and oligonucleotide therapeutics	Diverse range of potentially disease- and pain-modifying agents including nerve growth factor (NGF) antagonists.	Goal of these therapies is to alter the function of cells within the joint to combat/reverse joint inflammation and cartilage degeneration; in the case of NGF antagonists, to interfere with nociceptive signalling pathways and reduce pain sensitization.	Promising alternative therapeutic pathways and rapidly evolving field of sports medicine; however, limited clinical research evidence at present.

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Table 5.8 **Systemic medications used in the management of joint disease in horses**

PRODUCT		ACTION	USE AND EFFICACY
Non-steroidal anti-inflammatory drugs (NSAIDs)	Class of drugs with similar action that includes phenylbutazone, meloxicam and aspirin (acetylsalicylic acid)	Inhibit inflammatory pathways. Efficacy greater for pain than inflammation/filling. Aspirin: poor analgesic with short half-life; proposed to improve blood supply in sclerotic bone conditions (decreases platelet aggregation) but little/no supporting evidence for this.	Useful in acute synovitis; more effective in inflamed than normal tissues. Long-term use of NSAIDs discouraged as may mask signs of injury and accelerate cartilage degeneration.
Corticosteroids	Dexamethasone most commonly used for orthopaedic disease	Potent inhibitor of multiple inflammatory pathways	Single administration (IV/IM) during acute phase of injury; useful drug for reducing synovitis but poor analgesic.
Polysulphated glycosaminoglycans (GAGs)	Disease-modifying osteoarthritis (OA) medication Derived from bovine trachea	IM administration results in therapeutic concentrations in joint. No direct analgesic effect. Inhibits inflammatory cascade of OA; also chondroprotective (stimulates hyaluronic acid [HA] and collagen production).	Weekly administration (IM) after initial loading course. Reduces severity of OA signs in humans/horses. Clinical effect undetermined.
Pentosan polysulphate	Disease-modifying OA medication Derived from beechwood hemicellulose	IM administration results in therapeutic concentrations in joint. No direct analgesic effect. Anti-inflammatory and chondroprotective (stimulates HA and proteoglycan production) effects.	Weekly administration (IM). Currently no definitive evidence of clinical effectiveness.
HA (sodium hyaluronan)	Produced through biosynthesis	IV and oral forms. Possible anti-inflammatory effects in joint have been demonstrated after IV/oral administration.	Regular (weekly) IV administration or daily oral use. Clinical effect likely to be small.
Tiludronate Clodronate disodium	Bisphosphonate drugs	Systemic administration. Acts on bone turnover and has disease-modifying and analgesic effects in sclerotic and osteopenic bone conditions.	Single or periodic administration. Duration of effect on bone turnover likely to be months. Efficacy undetermined for most equine conditions.

Table 5.9 **Nutraceuticals used in the management of joint disease in horses**

PRODUCT		USE AND EFFICACY
Glucosamine	Precursor of chondrocyte glycosaminoglycan (GAG); proposed to have chondroprotective and anti-inflammatory effects. Derived from shellfish/animal/fungal sources	Conflicting evidence of efficacy in humans; some modification of osteoarthritis (OA) symptoms demonstrated. Biological rationale for use but currently little/no evidence of clinical efficacy in horses.
Chondroitin sulphate	'Building block' of cartilage. Derived from bovine trachea.	Conflicting evidence of efficacy in humans; less support for use than glucosamine. Any clinical effect likely to be small.
HA (sodium hyaluronan)	Produced through biosynthesis.	Limited evidence of efficacy in modifying synovitis/OA.
Avocado/soybean lipids (unsaponifiables)	Anti-inflammatory and possible regenerative effect on cartilage.	Some evidence of efficacy in modifying OA symptoms/disease in humans and horses but any clinical effect likely to be small.
Green-lipped mussel lipids	Contains omega-3 fatty-acids, vitamins and minerals.	Biological rationale for use but limited evidence of efficacy. Any clinical effect likely to be small.
Methylsulphonylmethane (MSM)	Proposed to be a source of biologically active sulphur.	Limited evidence of efficacy. Any clinical effect likely to be small.
Biota orientalis oil extract	Seed oil from coniferous tree. Proposed to have anti-inflammatory/anti-oxidant effects.	Some multi-species evidence of efficacy in modifying OA symptoms/disease.

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cartilage healing either through provision of 'building blocks' of cartilage metabolism or interference with the inflammatory cascade of OA. Long-term daily supplementation with products containing one or more of these nutrients is common. Considered safe to use; however, strong supporting evidence of efficacy (humans/equine) is lacking for all products, bioavailability of some products in horses is doubtful and any clinical effect is likely to be small. Use should be considered on cost-benefit grounds.

Topical anti-inflammatory medications

Topical products used to cool or tighten the lower leg are in common use. Topical anti-inflammatory drugs intended to achieve therapeutic levels within the joint are a more specific treatment and limited at present to a small number of drugs. Efficacy with daily/twice daily application appears to be similar to oral NSAID therapy. Topical therapy merits consideration as a management option for OA, but is not in widespread use at present. Prolonged clearance times can occur, with regulatory implications.

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Part 2

REGIONAL MUSCULOSKELETAL CONDITIONS

CHAPTER 6 Appendicular conditions

CHAPTER 7 Axial and miscellaneous conditions

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THE FOOT

Applied anatomy

The hoof capsule (comprising wall, sole and frog) is a specialized structure of keratinized horn that encases the distal extremity of the limb. Germinal cells at the coronary band ('coronet') continually produce hoof tissue that grows down the wall to be worn away and lost at the ground surface; wall growth is affected by many factors but typically occurs at a rate of 8–10 mm/month. The hoof wall is thickest at the toe and becomes progressively thinner towards the heels. On the sole, the 'white line' is the junction between the soft inner horn of the hoof wall and the solar horn. The frog is a wedge of more pliable horn that is bordered on each side by a deep groove (sulcus).

Suspended within the hoof capsule by a system of interlocking lamellae is the distal phalanx (P3 or pedal bone) (Figure 6.1). Enzyme-mediated remodelling of these lamellae permits the continuous 'ratcheting' downward of maturing keratin while maintaining the strong bond between P3 and hoof. At the back of the foot, between P3 and the frog and heels, lies the loosely collagenous digital cushion. When the foot strikes ground the thinner wall at the quarters allows expansion of the heels; the digital cushion, along with collateral cartilages, acts as a 'shock absorber' to dampen downward movement of the middle phalanx (P2).

The distal interphalangeal joint (DIPJ), or 'coffin' joint, is a hinge joint between P2 and P3 and the boat-shaped navicular bone, which is maintained behind them by a sling of ligaments. This joint is encased within the hoof capsule; however, the dorsal pouch extends above the coronet. The common digital extensor tendon passes down the front of the lower pastern over this synovial pouch to insert on the extensor process of P3. The DIPJ is capable of movement in several planes including rotation and sliding; however, its primary action is flexion/extension, being stabilized by articular congruity, collateral ligaments and the deep digital flexor tendon (DDFT). At the back of the foot, the DDFT passes around the palmar/plantar face of the navicular bone then fans out into its insertion on the underside of P3. The DDFT is separated from the navicular bone by the navicular synovial bursa.

Synovial communications

DIPJ and navicular bursa: there is good diffusion of treatment drugs (in therapeutic quantities) in normal horses.

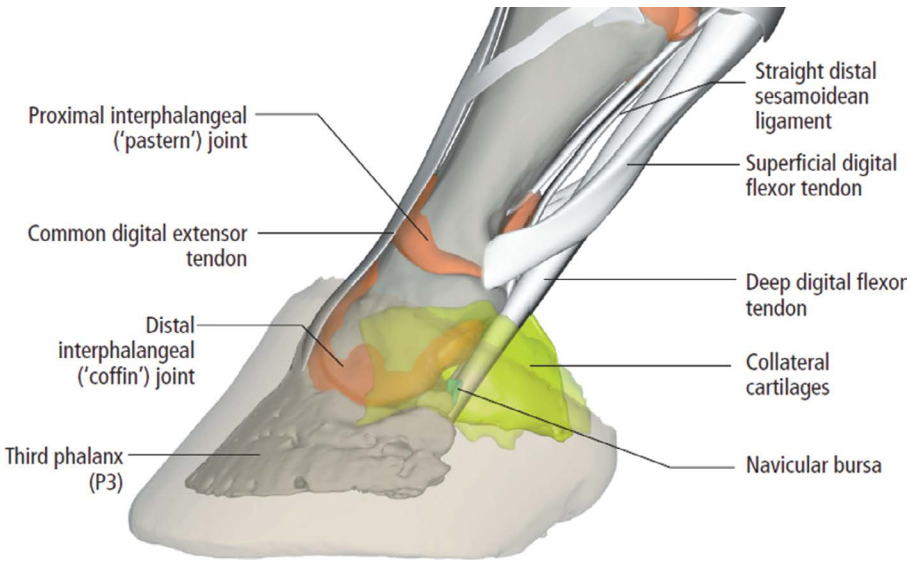


Fig. 6.1 **Major structures of the foot.**

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Examination

Feet should be observed both weight-bearing (from side and front/rear) and raised for symmetry of size and shape (with opposite foot) and dorsopalmar and mediolateral (ML) balance. Dynamic foot balance is assessed by observing how the feet strike the ground at walk and trot (should land level). Digital pulse and foot warmth relative to the opposite limb, and assessment of shoe fit, nail holes and hoof capsule defects, may assist diagnosis.

Palpation of the coronet and heels may elicit a focal pain response in cases of infection. DIPJ effusion is best noted by palpation of the dorsal pouch (above the coronet) with the limb weight-bearing, and should be compared with the opposite foot. Pain on deep palpation of the flexor tendon at its most distal extent between the heels may indicate navicular bursa pathology.

Lameness arising from the foot is often characterised by being exacerbated when the horse is turned tightly. Response to careful and systematic application of hoof testers to all regions of the sole and frog will usually localize pain arising from infection or bruising. Comparison with the opposite foot and determining repeatability is advisable before commencing to explore the foot invasively. Removal of the shoe is necessary for assessment of the white line, and the sole should be cleaned (wire brush) or lightly pared before exploration.

Approach to foot lameness in the racehorse

Lameness arising from the foot is common. The overwhelming majority of lameness is caused by either bruising or infection, is self-limiting and is inconvenient rather than serious. The challenge for the clinician and farrier is to promptly identify the nature of the problem and assist its resolution through targeted interventions to minimise lost training days. This can be of the utmost importance when lameness arises in proximity to a planned race or training gallop, and pressure to reshoe and resume exercise has to be balanced against the likelihood of recurrence if the primary problem has not settled sufficiently. It is important that clinician and farrier collaborate to achieve both the best short- and long-term outcome for foot lameness cases; daily re-examination until the horse resumes training permits agile decision-making and is a preferable approach to simply removing shoe and poulticing over a matter of days.

In most cases it is relatively straightforward to differentiate, through thorough clinical examination, whether foot lameness is the result of bruising or infection. While the two may be related to an extent (bruising/imbalance may predispose to infection), when faced with the acutely lame horse the important choice is generally whether to invasively explore the foot or not. Attempting to establish drainage at a focal area of concern with hoof knives is warranted for suspected infection, but if bruising is suspected thinning or opening the sole often risks exacerbating the lameness. Deciding which approach to take, how far exploration of the foot should proceed and when to reshoe in any given case (or at a particular stage of the lameness) is a matter of experience and clinical judgement. Choice of correct shoe to follow a resolving foot lameness is often a matter of trial and error with response being assessed on hard and soft surfaces and following ridden exercise.

When clinical presentation or progression of lameness is atypical, consideration of causes of lameness other than subsolar infection or bruising might be merited. Most of these (fractures, osseous cyst-like lesions [OCLs], soft tissue injuries) are rare but generally readily identified (or eliminated) through initial ambulatory imaging. Pathologies such as 'navicular syndrome' and DIPJ subchondral bone +/– ligament injuries that are encountered in other equine disciplines (and typically require advanced imaging for diagnosis) occur only very rarely in racehorses and are not covered in the following section.

Bruised foot ('corn')

Bruising of the heel angle ('seat of corn') is largely a problem of front feet and predominantly affects the medial side of the foot. The condition is manifested by increased sensitivity to pressure on the affected heel angle and typically associated with evidence of acute/chronic bruising or localized infection at the white line. Bruising of other parts of the sole is less common and when it occurs is usually the result of direct trauma ('stone bruise').

Cause

- Loading forces concentrated by shoe, particularly if shod close +/– collapsed heels.
- Uneven footfall (secondary to ML foot imbalance) may contribute.
- Toe-out conformation: lateral toe strikes ground first then medial heel, predisposing to shunted heel/bruising.
- Damage at white line frequently predisposes to secondary infection at site.

Risk factors

- Bruised foot is the most common cause of foot lameness in training.
- Risk factors include foot conformation (low/collapsed heels; ML imbalance) or inappropriate/inadequate trimming or shoeing (e.g., shod too close on inside bar).

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History
- Acute onset forelimb lameness.
- Most commonly arises when due for reshoeing (rather than when freshly shod).

Signs
- Mild-moderate lameness; usually better on grass/soft surface.
- Unilateral/bilateral.
- Focal pain response to hoof testers.
- +/– increased digital pulse (usually mild).
- Signs generally milder than with subsolar abscess (p. 83).

Diagnosis
- Clinical findings usually definitive.
- Appearance of affected heel angle: can range from normal to thin/flat-soled, with normal or discoloured/disrupted white line +/– evidence of fresh or prior haemorrhage (Figure 6.2a,b).
- Diagnostic blocking if required: lameness abolished with blocking of relevant (medial or lateral) palmar digital nerve.

Management
- If no infection, avoid unnecessary paring of foot otherwise thinning of sole/prolapse of sensitive solar corium may result, delaying recovery (Figure 6.3).
- Poultice if cannot distinguish from infection (typically not beyond initial 12–24 hours).
- Excessive softening of sole should be avoided (unless infection suspected); topical use of osmotic gels or hardening agents (e.g., iodine) under a foot dressing is preferred.
- Most cases managed satisfactorily by brief respite from shoe (24–48 hours) followed by reshoeing that aims to minimize pressure on affected site ('seating-out') +/– set full/wide at the quarters.
- Shoes that redistribute weight away from affected heel/solar angle (bar shoe, digital/frog support pad +/– impression material) or unload it altogether (¾ plate/¾ bar shoe) sometimes required for troublesome cases (Figure 6.4). Response to latter may be dramatic but considered an interim measure. Choice of shoe determined by individual foot/frog/heel shape and balance. Typically used only in the short term (single shoeing cycle) to facilitate return to/continuation of training.
- Horses with poor foot balance +/– propensity to recurrently develop bruised heels should be subjected to greater routine attention by farrier. Allowing collapsed heels to 'run forward' can exacerbate the problem, and shorter shoeing cycles, appropriate dressing back of heels +/– use of digital/frog support (+/– wedged pad) may be necessary in the medium term to improve overall heel shape. 'Remedial' shoes generally replaced by regular racing plates for racing engagements.

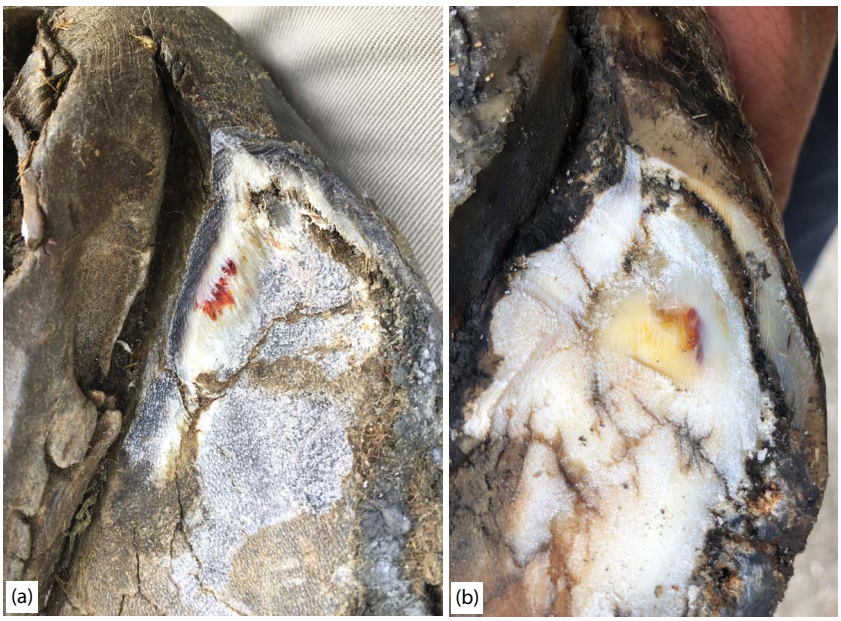


Fig. 6.2 **Bruised foot: (a) haemorrhage at the white line of the medial bar; (b) subsolar bruising at medial 'seat of corn'.**



Fig. 6.3 **Prolapse of solar corium (arrowhead).**

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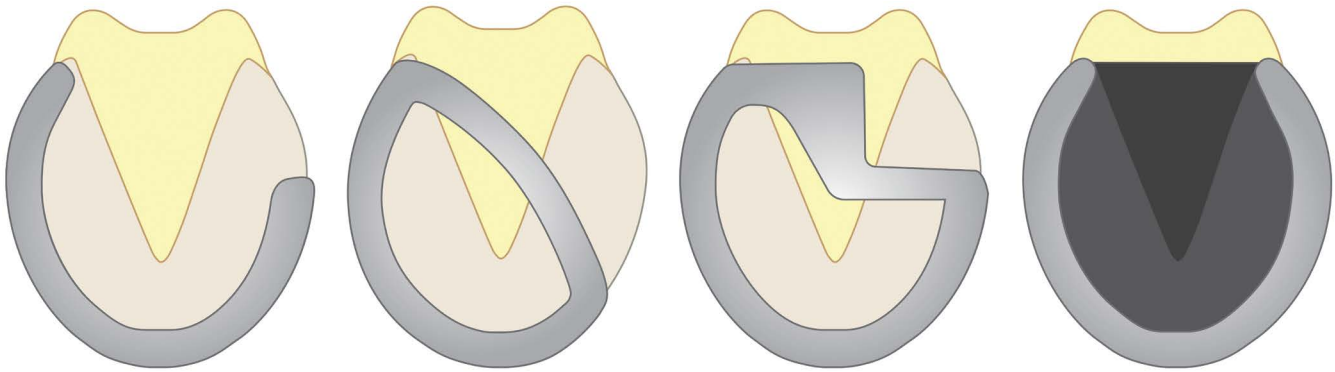


Fig. 6.4 Shoeing options to relieve pressure from seat of corn/bar/heel region. From left: ¾ shoe; ¾ bar; half heart bar ('Z-bar'); digital/frog support pad.

Prognosis
- Excellent. Return to ridden exercise (after acute bruising) usually possible within 1–3 days.

Dorsopalmar foot imbalance (collapsed/flat heels)

This condition involves low/collapsed heels +/– an overlong toe. It can affect both front and hind feet but secondary lameness is most common in the front feet. Typically bilateral but may be unilateral. May be associated with a simple imbalance of the hoof capsule or a more profound alteration of the hoof–pedal bone (P3) relationship (flat/negative solar angle of P3; 'negative plane of P3'). Usually worsens with time in training unless preventive measures taken. Low heels cause greater loading of the back of the foot during stance (and on toe at breakover). This predisposes to subsolar bruising (+/– laminar tearing at the toe). A small proportion of affected horses suffer from recurrent foot lameness.

Cause
- Thoroughbreds are predisposed due to conformation and hoof quality.
- Deformation of hoof occurs with loading during fast exercise.
- Long-term foot management (inappropriate dressing of heels) contributes to loss of balance.

Risk factors
- Dorsopalmar foot imbalance is very common.
- Risk factors include shoeing practices (frequent shoeing/overdressing of heels/underdressing of heels resulting in heels 'running forward'/shoeing close at heels to avoid lost shoes) and training itself (fast work linked to lowering of heel angle).
- Individual susceptibility may have genetic component.

History
- Typically develops over months/years.
- More rapid loss of foot shape may occur in a shorter period (weeks) with inappropriate foot care in susceptible horse; commonly during rest period from training or when away from the care of regular farrier.

Signs
- Broken-back hoof-pastern axis.
- When examined from side and solar aspects, heels may appear to run forward/collapse.
- May develop secondary lameness (bruised foot, p. 78).

Diagnosis
- Clinical findings are definitive (Figure 6.5a,b).
- External appearance of hoof is poor guide to alignment of internal structures.
- Diagnostic blocking: lameness usually abolished with palmar digital nerve block; however, important to be aware that proximal spread of local anaesthetic may abolish lameness arising from pathology up to and including the fetlock region.
- Imaging rarely needed but recommended if lameness is persistent or recurrent.
- Radiography: may reveal 'reverse rotation/inclination' of P3; palmar processes of P3 are closer to solar margin than the toe (on lateromedial [LM] projection) (Figure 6.6a,b).
- Magnetic resonance imaging (MRI): occasionally justified in poorly responsive cases to rule out other injuries. Signs of laminar disruption and concussive strain involving P3/navicular bone is typical.

Management
- Balance between practicalities of shoeing for continued training/racing (avoiding pulled shoes) and

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Fig. 6.5 **Dorsopalmar foot balance: (a) satisfactory balance; (b) low/collapsed heels.**

prevention of longer-term deterioration in shape of foot; not always compatible.

- General rule: style of shoe used should be the simplest that both successfully keeps horse sound in short/medium term and permits gradual improvement in foot balance over weeks/months. May require experimentation over several shoeings.
- Most cases manageable with correct and timely trimming (to prevent excessive length of toe and crushing or underrunning of heel tubules) combined with shoes that distribute load better through foot/sole and ease breakover. Shorter shoeing cycles, appropriate dressing back of heels +/– use of digital/frog support (Figure 6.6c) or bar shoes may be necessary in the medium term to improve overall heel shape.
- Flat- or reverse-inclination of P3: if poor response to the previous recommendations, may be appropriate to raise heels (5–6 degrees) with a graduated heel shoe or

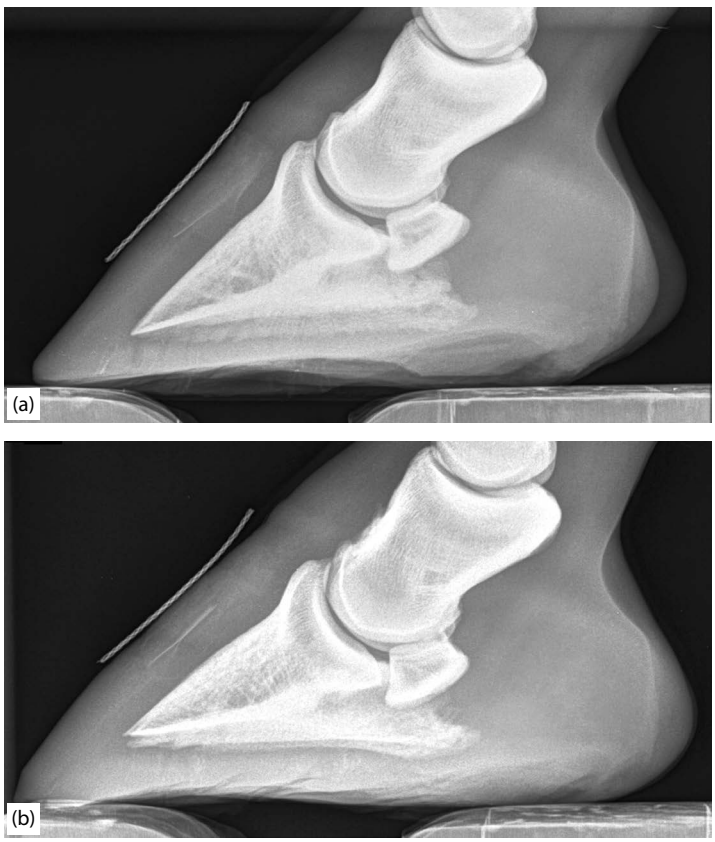




Fig. 6.6 **Dorsopalmar foot imbalance: Radiographs (LM) of front feet with (a) normal P3-solar relationship. (b) reverse inclination/rotation of P3; (c) caudal foot support pad with impression material.**

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pad for several shoeings. Can yield good medium-term soundness, although used with caution as subsequent increased pressure on coronet of heels may reduce rate of heel growth.
- Biotin supplementation: small improvements in hoof growth/quality with long-term (months) daily supplementation (20–60 mg/day).
- If poor foot balance/foot lameness is a recurrent/widespread problem in training yard, barefoot trimming (applied through lengthy periods of conditioning programme when feasible for horses to be unshod) may help to improve overall quality and shape of feet. Factors that influence practicality of this approach include daily distance travelled (and surface type) on horse 'commuting' tracks, training surfaces and local climatic conditions.

Prognosis
- Condition typically not reversible but progression can usually be halted.
- Most cases can be kept sound in training with appropriate management.

Quarter crack
Quarter cracks are longitudinal cracks of variable length in the hoof wall (Figure 6.7a,b) that can develop anywhere from the medial or lateral quarter to the heel. They may be full or partial thickness; involvement of sensitive tissue is common due to the thin hoof wall at the quarters. Cracks usually originate in the upper half of the hoof wall or close to the coronet. They are most common in the front feet, and predominantly medial.

Cause
- Typically arise from stresses in the hoof capsule (secondary to foot imbalance/limb loading).
- Occasionally secondary to hoof wall defects arising from previous injury/scarring to the germinal cells at coronet, such as following deep overreach injury.

Risk factors
- Uncommon condition.
- ML foot imbalance or poor forelimb conformation (toe-out) may predispose.

History
- Acute onset of quarter crack +/– lameness.

Signs
- Lameness is not always a feature but when present is typically mild-moderate in severity.
- Other signs include increased digital pulse, pain on palpation of coronet above the crack, and bleeding at top of crack/coronet after exercise.

Diagnosis
- Clinical findings definitive.

Management
- Lameness and assessment of instability/depth (hoof testers) guide management in acute phase.

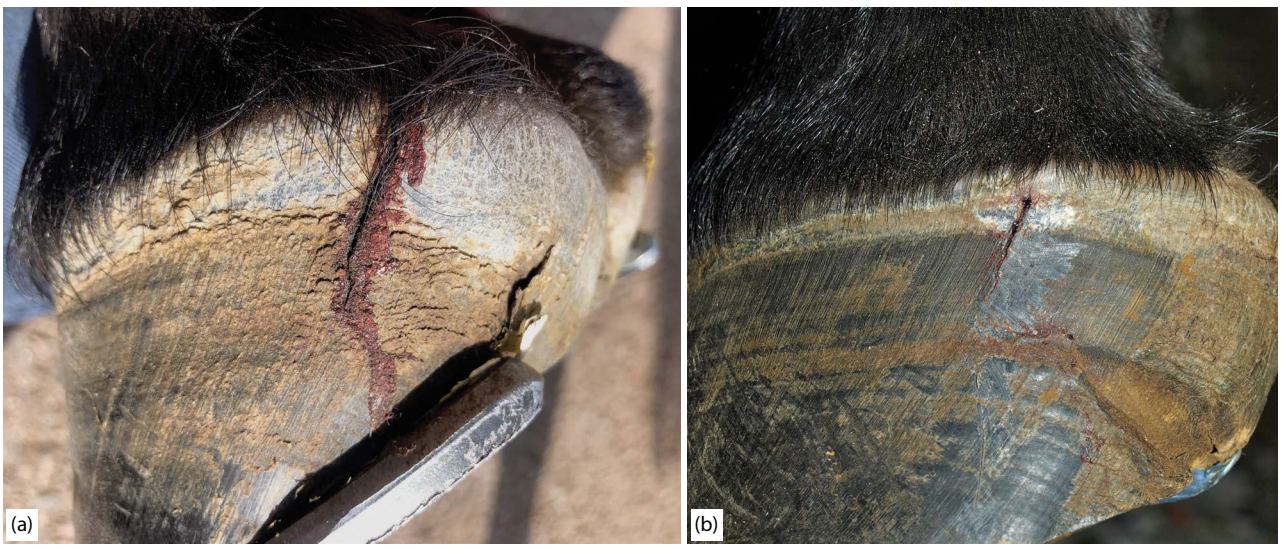


Fig. 6.7 Quarter cracks: (a) fresh crack with haemorrhage typically seen following exercise; (b) shorter crack not quite breaching coronary band.

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- Fresh cracks extending to sensitive tissue should be poulticed and allowed short period of time to resolve any associated infection before considering repair.
- Address any underlying foot imbalance.
- Primary goal is stabilization of hoof capsule to prevent progression (and resolve lameness). Usually achieved with bridging acrylic hoof patch +/– bar shoe, or hoof cast.
- Type of repair dependent on location/length of crack. If at caudal heel: bar shoe +/– floating of heel (to remove from load bearing) may be more appropriate than repair.
- Once crack is stabilized, may resume normal training.

Prognosis
- Minimal interruption to training with appropriate management.
- Recurrence rate is high if underlying foot imbalance is not addressed satisfactorily.

Subsolar abscess
Localized infection under the sole causes acute and often marked lameness. Depending on location, severity and management, infection either remains subsolar or tracks up the white line to emerge at coronet. Most common in the front feet.

Cause
- Usually secondary to repetitive bruising.
- Can arise from direct penetration of sole.
- Less commonly with compromise to white line.

Risk factors
- Any age/stage of training.
- Foot imbalance (ML or dorsopalmar) and poor shoeing may predispose.

History
- Acute-onset lameness.

Signs
- Moderate-marked unilateral lameness; worse on the turn.
- Increased digital pulse/warmth of foot.
- Moderate-marked focal pain response to hoof testers.
- +/– focal swelling/pain at coronet (if infection about to burst out proximally).

Diagnosis
- Clinical findings usually definitive.
- Evidence of infection can usually be found on inspection of solar surface of foot (Figure 6.8a,b).

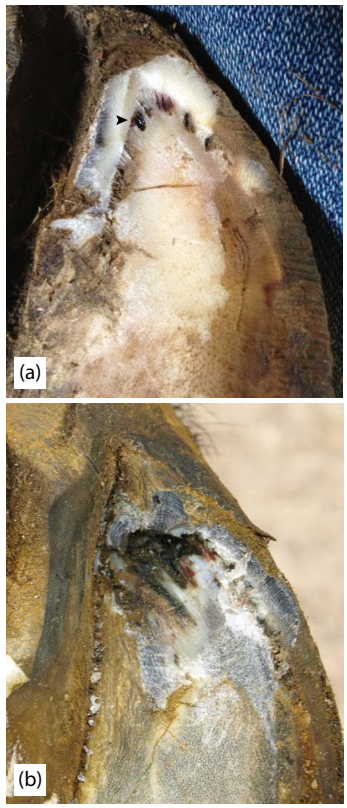


Fig. 6.8 Subsolar abscess. (a) 'open' tubules with black exudate typical of infection; (b) more extensive infection in medial solar angle ('seat of corn') region.

Management
- Infection usually self-limiting but drainage is desirable to speed recovery.
- Shoe should be removed and sole pared in area of greatest response to hoof testers.
- If focal defect found, establish drainage with loop knife (Figure 6.9).



Fig. 6.9 Drainage of pus established from medial solar angle.

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- Osmotic poultice as required (until infection has subsided) +/– stand foot in shallow tub of salt water/diluted disinfectant once/twice daily.
- +/– poulticing over coronet if indicated.
- Stable rest +/– walking until infection resolved.
- If sensitive sole becomes exposed/prolapsed (Figure 6.3), lameness may continue beyond resolution of infection and is best resolved with shoeing.

Prognosis
- Most cases resolve rapidly following drainage and can be reshod within 2–5 days.
- Rarely, long-standing/deep abscessation may result in focal infection of the adjacent portion of the pedal bone (septic pedal osteitis, p. 85).

Foot penetration/puncture
Foot penetration by foreign objects typically affects the sole; less commonly the frog. Effects of penetrating injury are determined by location and depth; subsolar abscess (p. 83) is a common outcome. Shifted shoes are the most frequent cause and bleeding indicates penetration of sensitive corium by nails or toe clip (Figure 6.10a,b). Penetrations by other objects (building nails/screws) are rare but when they occur are often in the frog or sulci. These are potentially more serious injuries, as infection of deep vital structures of foot (navicular bursa/DIPJ/digital tendon sheath/DDFT) may result.

History
- Acute-onset lameness or penetrating object detected.



Fig. 6.10 Solar penetrations typical of shifted shoe, with haemorrhage indicating subsolar involvement. (a) shoeing nail; (b) toe clip.

Diagnosis
- Clinical findings (length/orientation of penetrating object) generally sufficient to determine risk to deep structures of the foot.
- If object is no longer in foot, paring of sole/frog and use of hoof testers to locate entry point.
- Penetrations of the frog often difficult to find because of the soft horn and absence of bleeding.
- Radiography: risk assessment in cases where object is still in place.
- MRI: most useful modality in challenging cases but only in post-acute phase to detect DDFT involvement or sequestration of flexor aspect of P3. Generally not useful for detection of synovial sepsis in acute phase (initial days/week).
- Synoviocentesis: (DIPJ +/– navicular bursa +/– digital tendon sheath) indicated if synovial sepsis suspected.

Management
- Spread shoe/shoeing-nail penetration: any solar defect (including toe clip) from which bleeding is noted is best opened with a loop knife to establish drainage and the foot poulticed +/– systemic antibiotic medication. Care taken during exploration to minimise size of solar defect, as large defects (>3–4 mm) frequently result in prolapse of sensitive corium and prolonged lameness beyond resolution of infection.
- Systemic +/– regional antibiotic perfusion may be warranted if concern over potential for infection of P3.
- Suspected synovial penetration: referral to hospital facility. Synovial involvement (if confirmed with synoviocentesis) is an emergency that requires early diagnosis and aggressive and appropriate management (navicular bursoscopy, lavage +/– debridement) for good outcome. If initial synoviocentesis not indicative of synovial infection, manage as for deep subsolar penetration; poor clinical progress >5–7 days may merit MRI to assess involvement of DDFT/P3.

Prognosis
- Shoeing-nail or toe clip penetration: minimal interruption to training (typically <5 days).
- Synovial sepsis following frog penetration: poor prognosis for return to full athletic soundness (<30%) and high mortality rate unless early surgical intervention (regardless of structure involved).

Nail bind ('quicked' hoof)
Nail bind is lameness/inflammation arising from close proximity of a shoeing nail to sensitive tissue of the foot.

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It does not always result in secondary infection and may occur in any foot.

Risk factors
- Any age/stage of training.
- Horses with thin-walled feet or boxy feet at greater risk.

History
- Lameness in immediate (within 1–2 days) post-shoeing period.

Signs
- Mild-moderate lameness.
- Increased digital pulse/warmth of foot.
- Hoof tester pain response frequently focal to a single nail.
- Clench of suspect nail may be high on hoof wall.

Diagnosis
- Clinical findings strongly indicative although may sometimes be less definitive than subsolar abscess.

Management
- Mild lameness: remove affected nail.
- Moderate lameness (or no improvement following nail removal): removal of shoe and treatment as for subsolar abscess (p. 83).

Prognosis
- Minimal interruption to training with appropriate management.

Septic pedal (P3) osteitis

Septic pedal osteitis is infection of P3. It is usually focal and limited to solar margin of bone. Occasionally associated with sequestrum formation. May affect any foot.

Cause
- Occurs secondary to infection within the hoof capsule in proximity to P3.
- May follow long-standing subsolar abscess where effective drainage has not been established. May also occur following deep foot penetration (nail).

Risk factors
- Any age/stage of training.
- Inappropriate or delayed treatment of subsolar abscess or foot penetration.

History
- Persistent/recurrent foot lameness (over weeks) following subsolar abscess.



Fig. 6.11 Septic P3 osteitis. Radiograph (D60^{0}Pr-PaDiO) of irregular lysis of solar margin at toe (arrowheads) of P3.

Signs
- Same as for subsolar abscess (p. 83).

Diagnosis
- Radiography: signs of bone infection may not be visible for 1–3 weeks after initial injury/infection. Focal radiolucent defect of P3 in region of solar defect (Figure 6.11).

Management
- Mild clinical/radiological findings: antibiotic medication (2–4 weeks) may resolve infection and avert need for surgery.
- Most cases with advanced radiological findings require open debridement: standing surgical procedure accessing infected solar margin of P3 through sole (Figure 6.12a). Post-surgery hospital shoe/plate permits daily antiseptic dressing until the solar defect heals (Figure 6.12b).
- Maggot debridement therapy is a treatment option for surgical cases.

Prognosis
- Good prognosis for return to full soundness.
- Non-surgical cases: return to training determined by level of soundness.
- Surgical cases: return to ridden exercise generally possible in 6–10 weeks.
- Recurrence of infection following surgery in a small proportion of cases.

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Fig. 6.12 **Septic P3 osteitis. (a) excavated sole with evidence of pus adjacent to P3. (b) healthy granulation tissue during healing phase (hospital plate) following debridement of P3.**

Pedal bone (P3) fracture

Fractures involving P3 occur in several configurations. The causes and management vary according to injury type (Table 6.1).

History

- Acute-onset lameness.
- May initially be mistaken for other causes of foot pain; investigation usually prompted by failure of lameness to resolve with poulticing/reshoeing.

Signs

- Clinical findings non-specific for fracture configuration but as a general rule greater severity of lameness with articular fractures.
- Sagittal/parasagittal fractures are articular: severe acute lameness.
- Wing fractures: mild-moderate lameness, may be intermittent.
- +/– increased digital pulse (usually mild).

Table 6.1 **Description and cause of various fractures involving P3**

FRACTURE TYPE	DESCRIPTION	CAUSE
Palmar/plantar process ('wing') fracture	Oblique or sagittal fracture of wing to medial or lateral solar margin (Figure 6.13a,b). May be articular/non-articular. Predominantly in forelimb. Most common configuration in racehorses. Fractures of left forelimb lateral or right forelimb medial wing most common when racing counter clockwise.	Usually an acute exercise-related overloading injury.
Sagittal/parasagittal fracture	Midline/near-midline fracture (Figure 6.13c). Invariably articular. Forelimb or hindlimb.	Typically due to direct trauma (such as kicking wall for hindlimb fracture) but may also be exercise related.
Extensor process fracture	Fragmentation of extensor process (insertion of common digital extensor tendon) at dorsoproximal aspect of P3 (Figure 6.13d). Invariably articular. Fragment size varies. Predominantly in forelimb.	Likely to be stress injury but acute injuries also occur.
Frontal fracture	Rare. Frontal fracture line extending for variable distance along dorsal aspect (Figure 6.13e). Non-articular. Predominantly in forelimb.	Cause unknown; likely to be acute overload/traumatic event such as a fall.
Solar margin fracture	Fragmentation of dorsal solar margin of P3 (Figure 6.13f). Fragments typically small/thin. Predominantly in forelimb. May not be clinically active at time of detection; of questionable importance other than as marker of possible prior foot pain.	Associated with flat feet +/– chronic foot soreness and likely to arise from repetitive concussion; rarely, secondary to acute impact/penetration injury.

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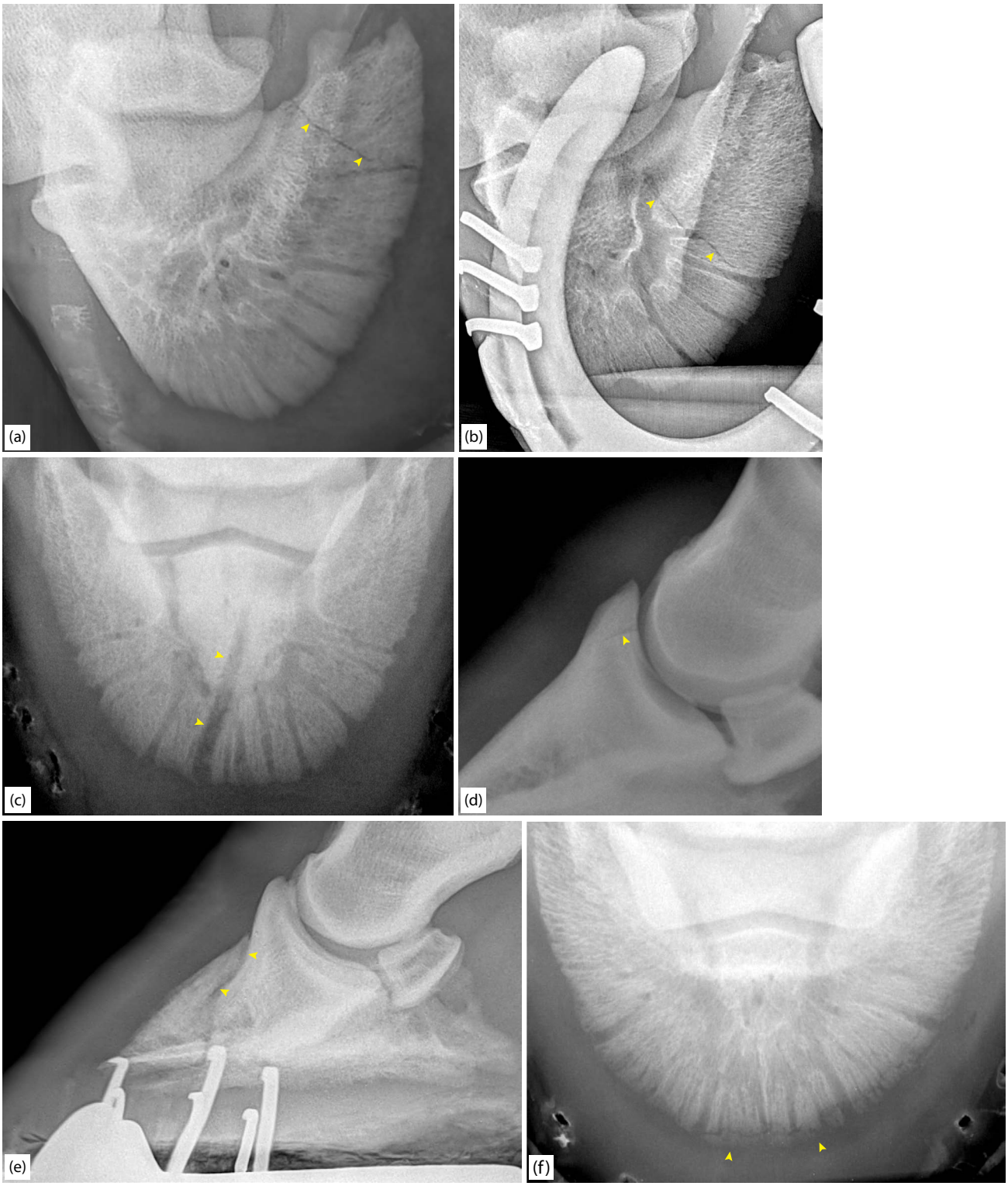


Fig. 6.13 **P3 fractures. Radiographs of (a) non-articular and (b) articular fractures through lateral wing of P3 (D50^{0}Pr45^{0}L-PaDiMO). (c) mid-sagittal (articular) fracture (D60^{0}Pr-PaDiO). (d) extensor process (articular) fracture (LM). (e) frontal (non-articular) fracture with slight displacement (LM). (f) solar margin fracture (D60^{0}Pr-PaDiO).**

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- Variable response to hoof testers but often resent strong percussion of hoof wall or sole.
- +/– effusion of DIPJ depending on injury type.

Diagnosis

- Clinical findings non-specific and generally do not permit differentiation from other causes of foot lameness.
- Diagnostic blocking: lameness usually improved by blocking of foot (but may not be completely abolished).
- Radiography: in acute phase, fracture line usually narrow and multiple projections may be required for detection. Wide/irregular lucency or fragment with rounded/sclerotic margins implies longer-standing injury.
- High incidence of P3 fractures in foals means that small/
  long-standing palmar/plantar process fragments are common in radiographic screening of 'normal' adult feet.
- Bone scan/MRI/computed tomography (CT)/ positron emission tomography (PET): rarely required but may be of use if results of conventional imaging ambiguous.

Management

- Conservative management in most cases: stable rest (8–12 weeks) followed by walking.
- Bar shoe +/– quarter clips, or hoof cast recommended during rest phase to immobilize fracture by preventing heel expansion (also for return to training for wing fractures).
- Sagittal fractures with articular incongruity: surgical (lag-screw) fixation technically challenging and associated with complication risk but may give best chance of long-term athletic soundness.
- Majority remain visible radiologically long term: return to exercise dictated by clinical rather than radiological improvement.

Prognosis

- Prognosis for return to full use:
  - Non-articular (wing) fractures: excellent (80–90%).
  - Articular wing fractures: good (approximately 70%).
  - Articular sagittal/parasagittal fractures: good (>70%).
  - Extensor process fractures: fair (approximately 60%).
  - Frontal fractures: fair.
- Overall prognosis best for hindlimb fractures.

Pedal bone (P3) osseous cyst-like lesion ('bone cyst')

Osteochondrosis lesion that typically becomes clinically active in early training. Most common location is sagittally in proximal portion of P3 and in the middle third of the joint surface; size and communication with DIPJ varies. Usually unilateral and solitary and predominantly in forelimb. Prevalence unknown but may be associated with lameness.

Cause

- See Osteochondrosis (p. 267).

Risk factors

- Rare condition.
- Risk factors for development of condition unknown.

History

- Found in any age of horse, but most commonly first detected at yearling/2-YO stage when exercise increases.
- Acute or intermittent lameness in early cantering phase of training.
- Often initially mistaken for foot bruise/infection.

Signs

- Clinical findings and diagnostic blocking pattern similar to other foot conditions.
- Mild-moderate unilateral lameness, often transient/ intermittent.
- Increased digital pulse; variable or negative response to hoof testers.
- +/– effusion of DIPJ.

Diagnosis

- Diagnostic blocking: lameness usually abolished/ improved by blocking of DIPJ or palmar digital nerve block.
- Radiography: upright pedal (DPr-PaDO) (Figure 6.14) and weight-bearing DPa (Figure 6.15a,b) projections have highest diagnostic yield; usually visible as a smooth-margined depression or circular lucency +/– surrounding sclerosis a variable distance from the joint margin.
- Presence of arthritic change or irregular density/ thickness of subchondral bone (DIPJ) is non-specific but may be strong indicator if cyst not radiologically evident (Figure 6.16).
- MRI/CT: diagnostic modalities of choice for both diagnosis (greater sensitivity than radiography) and surgical planning.

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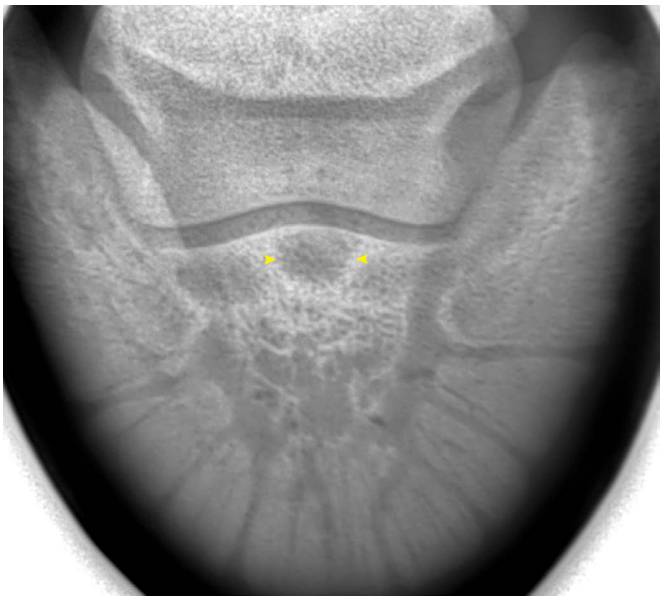


Fig. 6.14 **P3 subchondral bone cyst. Circular lucency (DPr-PaDiO).**

Management

- Surgical debridement of the cyst offers best chance of return to athletic soundness, but individual circumstances (including surgical access to lesion) frequently mean surgery reserved for cases responding poorly to conservative management.
- Conservative management: for paddock or non-racing use is the management option of choice; return to full athletic use also possible. May also be treatment of choice for cases in which MRI findings indicate active inflammation of P3/'immaturity' of cyst in a young horse. Stable rest/walking for 10–16 weeks +/– intra-articular medication.

Prognosis

- Conservative management: prognosis for return to full racing soundness guarded to fair (approximately 60% horses with conservatively managed P3 cyst will race; significantly less likely to race than maternal siblings). Periodic intra-articular medication often necessary on return to training.
- From the limited data available, prognosis for racing (with conservative management) does not appear to be strongly influenced by age at diagnosis, forelimb or hindlimb, size or radiographic features of cyst.
- Surgery: good-excellent prognosis for athletic soundness.



Fig. 6.15 **P3 subchondral bone cyst. (a) small, and (b) large lucency with surrounding sclerosis (standing DPa).**

Navicular bone fracture

Rare and sporadic cause of lameness. Configuration of fracture may be oblique and sagittal (either side of midline) (Figure 6.17a,b) or may involve lateral or medial wing with varying degrees of comminution and distraction of fragments (Figure 6.17c,d). Rarely, avulsion fractures of distal border (Figure 6.17e) (latter may be clinically insignificant). Complex fractures involving associated articular margin of P2 +/– P3 may also occur (Figure 6.18).

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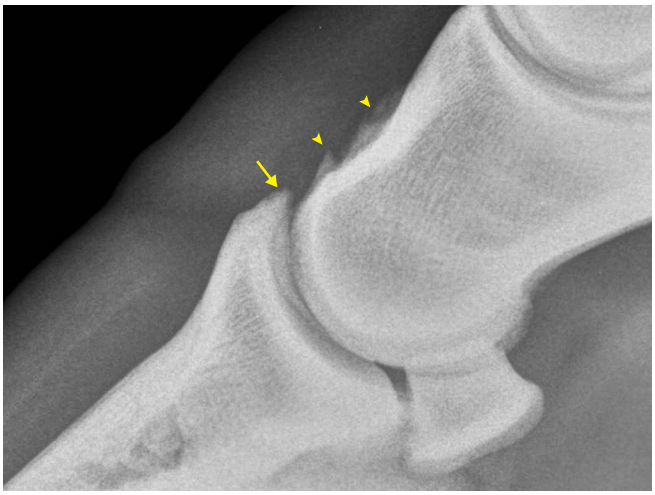


Fig. 6.16 **DIPJ osteoarthritic change. Radiograph (LM) of lysis of the extensor process (arrow) and modelling of dorsal P2 (arrowheads).**

Cause

- Forelimb fractures: typically sustained at exercise and presumed to be acute overload injury; also following trauma.
- Hindlimb fractures: usually attributed to direct trauma such as kicking a wall.

Risk factors

- Rare condition.
- Risk factors unknown.
- Role of pre-existing navicular pathology in exercise-related injuries not known.

History

- Acute and usually severe lameness.
- First noted either in stable or following/during exercise.

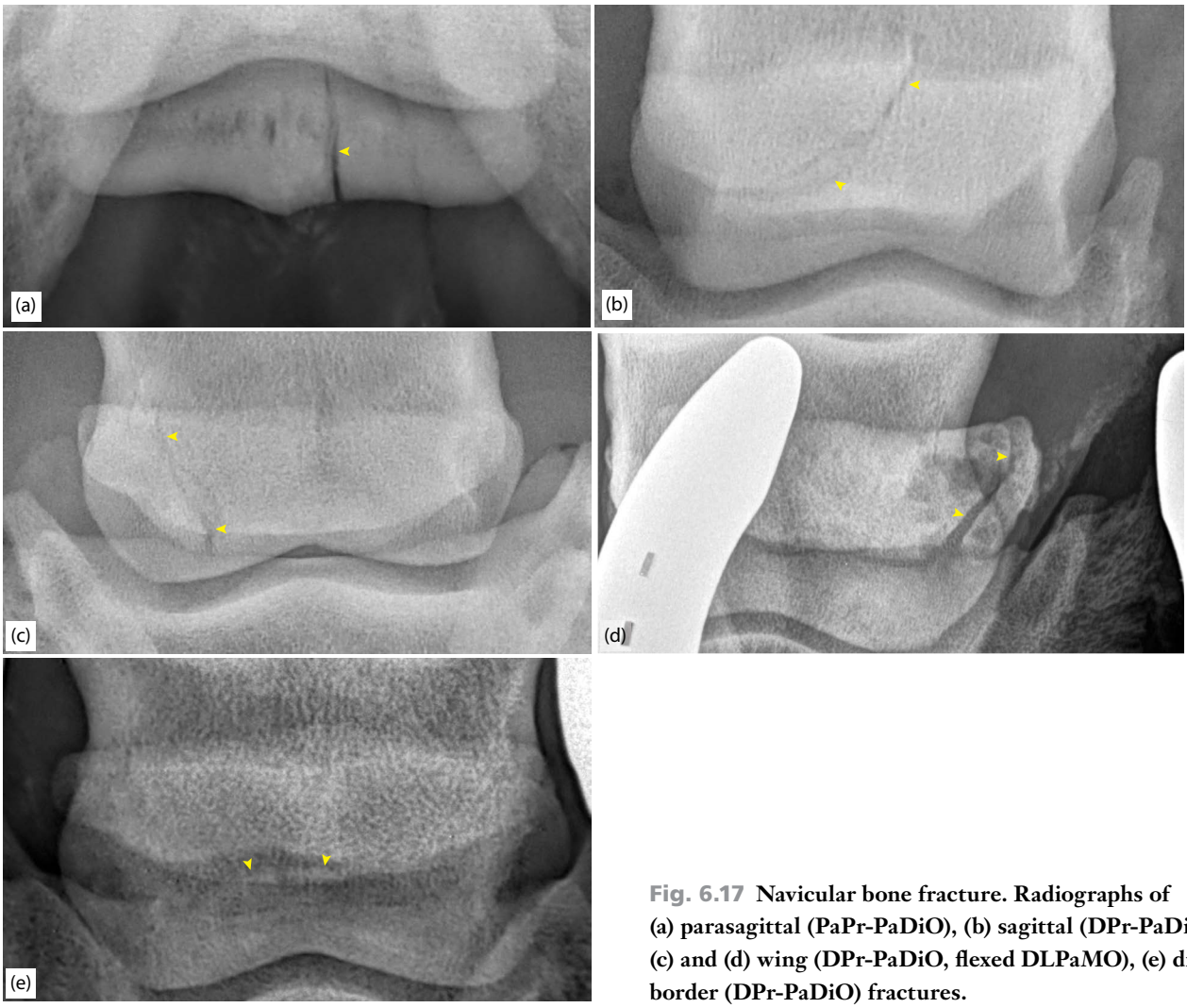


Fig. 6.17 **Navicular bone fracture. Radiographs of (a) parasagittal (PaPr-PaDiO), (b) sagittal (DPr-PaDiO), (c) and (d) wing (DPr-PaDiO, flexed DLPaMO), (e) distal border (DPr-PaDiO) fractures.**

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Fig. 6.18 Complex navicular wing and P3 injury (frontal CT) with multiple fragments present.

Signs
- Moderate-marked unilateral lameness.
- Signs referable to foot may be subtle/mild: slightly increased digital pulse +/– inconsistent response to hoof testers +/– effusion of DIPJ.

Diagnosis
- Radiography: upright P3, skyline and oblique navicular projections most useful.
- Should be differentiated from bipartite/tripartite navicular bones (Figure 6.19); these are congenital or may originate from injury early in life and are often bilateral. They are generally not associated with severe/acute lameness and may be encountered as incidental finding in horses in training, although lameness frequently develops at some stage due to complex of pathologies at site including secondary soft tissue adhesions/inflammation.

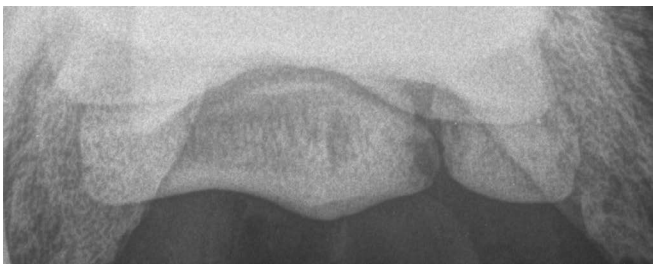


Fig. 6.19 Bipartite navicular bone (PaPr-PaDiO).

Management
- Return to racing possible with conservative management, however fracture configuration and clinical progress throughout rehabilitation is best guide to likelihood of athletic future. In most cases, expectations should be for paddock soundness only. Hindlimb fractures are considered the best candidates for return to racing.
- Stable rest (8–12 weeks) with bar shoe/wedged heel prior to walking/restricted turnout.
- Surgical repair (lag screw fixation) is technically challenging but may be considered for paddock salvage/light ridden use in some cases of non-comminuted sagittal fracture.

Prognosis
- Guarded prognosis for return to racing regardless of management. Best athletic outcomes seen with hindlimb wing fractures, and worst outcomes generally associated with forelimb mid-body/sagittal fractures.
- Usually regain sufficient soundness for non-athletic/paddock use.

White line disease ('seedy toe')

A disease of the white line in which progressive disruption of the horny layers (beginning at the solar margin and tracking proximally) causes separation within the hoof wall. Usually involves dorsal wall (typically toe region); width and proximal extent of involved tissue (at time of detection) can vary widely between individuals. Most common in forelimb and usually unilateral, but may affect several feet.

Cause
- Opportunistic fungal +/– bacterial invasion of white line.
- Mechanical (e.g., poorly balanced feet) or environmental factors make the white line vulnerable to hoof wall separation/infection.
- Lower density of horn tubules in the inner hoof wall means that this layer of the hoof is less rigid and more susceptible to damage from mechanical stress than the outer wall.
- Separation slowly progresses up hoof wall, largely without causing signs of inflammation as it is confined to inert keratin.

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Risk factors
- No strong associations with type of bedding and occurs in both wet and dry environmental conditions.
- Incidence increases with age.

History
- Often first detected at routine shoeing.

Signs
- Usually no external sign of disease on hoof wall, unless disease is extensive.
- Area of discoloured/widened white line +/– hollow cavity beneath the hoof at toe, extending for variable distance up dorsal wall (Figure 6.20a,b).
- +/– mildly increased digital pulse.



Fig. 6.20 Seedy toe. Typical appearance of dorsal hoof wall defect following partial (a) and full (b) resection of underrun horn.

- Lameness usually only a feature with extensive lesions or secondary infections.
- Origin of lameness frequently difficult to determine conclusively: may be due to subsolar/subcapsular infection +/or laminar pressure/instability. Best initial approach generally to uncover cavity and eliminate possible infection followed by remedial shoeing.

Diagnosis
- Clinical findings definitive.
- Radiography: sometimes useful in advanced cases to plan treatment. LM projection to highlight proximal extent of gas shadow and evaluate any displacement of pedal bone. Secondary modelling/lysis of P3 is a common feature.

Management
- Dictated by severity and stage of season.
- Small/early separations often resolved easily with good farriery: correction of predisposing conditions (hoof distortions/foot imbalance).
- Treatment involves resection of the dorsal hoof wall overlying the hollow lesion back to bordering healthy tissue, cleaning/disinfecting exposed tissue, unloading the affected area and supporting remaining hoof capsule.
- Affected area can be left open to grow out; regular attention at routine trimming to debride any further tracts.
- Range of disinfectant/medicated topical treatments in use to treat debrided area; currently little scientific basis to recommend any product over another.
- Topical treatment without resection of little use.
- Support: approach to shoeing dependent on individual circumstances (foot balance and shape; extent of lesion; level of exercise): options range from natural-balance or square-toe shoe to move breakover back for milder lesions, to bar or open-toe shoe +/– bridging acrylic hoof repair (or hoof cast), or digital/frog support pad +/– impression material following extensive resection.
- Extensive lesions best addressed electively and out of season, as radical hoof wall resection may result in variable period (weeks to months) of intermittent/ongoing lameness +/– remedial shoeing until foot stabilises.
- Oral biotin supplementation.
- Following resection, clean/dry bedding is recommended; wood shavings is bedding of choice.

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Prognosis
- Recurrence is common, but minimised by diligent routine farriery.
- Interference to training is usually minimal: occasional episodes of foot soreness/infection.

Laminitis
Laminitis is an important and potentially life-threatening disease in which breakdown of the bond between P3 and the inner hoof wall occurs. Severity varies depending on degree of initial compromise to this bond and stage of disease, and ranges from mild inflammation or realignment of P3 within the hoof capsule through to catastrophic loss of support of P3 or even shedding of hoof. Duration of acute phase of disease may be days/weeks, and recovered but seriously affected horses may display chronic or recurrent laminitis long term. Initial severity of damage and point at which P3–hoof connection is stabilized determines degree of short- and long-term disability.

Cause
- Complex cascade of events that involves vascular, enzymatic, inflammatory or metabolic factors to varying degrees.
- Vascular system of equine foot may predispose to laminitis due to presence of arteriovenous 'shunts', which allow blood to bypass the sensitive structures of the foot under certain circumstances (thus depriving lamellae of blood supply).
- Normal growth of hoof wall past P3 is facilitated by enzymes: inappropriate activation of these enzymes by inflammatory mediators is involved in breakdown of the bond between dermal and epidermal lamellae.
- Developmental phase of laminitis: damage can occur 48–72 hours before clinical signs become apparent.
- Weakened P3–hoof bond: weight-bearing (+/– pull of DDFT) rotates/displaces P3 towards (and sometimes through) sole, with subsequent acute/chronic damage to blood supply and sole.
- Usually worse in forelimbs because of greater load, although all four feet can be affected.
- Inflammation, tearing of lamellae, loss of blood supply all contribute to foot pain; also altered pain signalling may result in 'neuropathic' pain.

Risk factors
- Racehorses are at lower risk of developing laminitis than the general horse population, but when it does occur is frequently serious.
- Supporting limb laminitis: most important type of laminitis seen in racehorses. Severe unilateral lameness (due to infection/orthopaedic injury) may result in heavy loading of supporting limb: causes arterial compression and subsequent lack of blood supply to the hoof of the weight-bearing leg. May develop insidiously as initial lameness masked by that of opposite limb. Occurs in weeks/months after initial insult and affects 10–20% of horses at risk. Greater risk with full limb casts/transfixation pin casts/greater bodyweight (BWT)/longer duration of casting.
- Colitis/pneumonia/endotoxemia: severe systemic infections requiring hospitalization may trigger the inflammatory cascade that results in loss of basement membrane integrity in hooves.
- Corticosteroid use (systemic and intra-articular) has previously been suspected as a risk factor for laminitis. While sporadic cases are sometimes encountered subsequent to corticosteroid administration, current high-quality statistical evidence does not support a causative link. Intra-articular use of corticosteroid in the Thoroughbred racehorse is not associated with increased risk of developing laminitis.
- Carbohydrate-overload laminitis: exceeding the foregut's capacity to digest starch (grain) or sugars found in pasture can lead to changes in hindgut microbial populations. Subsequent release of endotoxin can cause enzymatic breakdown of basement membrane. Rarely a cause of laminitis in racehorses.
- Endocrinopathic (associated with equine metabolic syndrome/pituitary pars intermedia dysfunction/insulin dysregulation) laminitis, a major disease in many pony and non-Thoroughbred breeds, is not a significant problem in racehorses.

Signs
- Lameness/footsoreness involving front (+/– hind) feet, variable severity (mild to marked); worse on hard surface
- Obel grading system:
  - Grade 1: alternately lifting feet; not lame at the walk.
  - Grade 2: stiff and resists turning at the walk and lame at the trot.
  - Grade 3: lame at the walk; stilted gait; resists lifting feet.
  - Grade 4: will not move unless forced.
- Increased digital pulses/warm feet.
- Hoof tester pain response greatest over sole near apex of frog.
- Depression around coronet +/– convexity of sole may indicate 'sinking' of P3.

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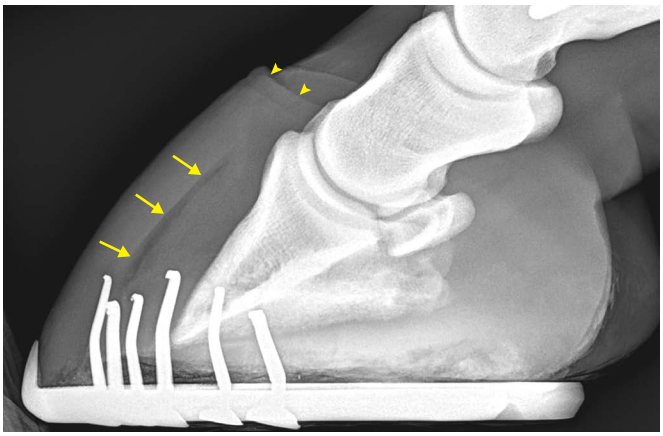


Fig. 6.21 Laminitis. Radiograph (LM) of 'sinking' P3: Characteristic 'ridge' appearance of coronary band (arrowheads), gas shadow beneath dorsal hoof capsule (arrows) and distal displacement/tipping of P3.

Diagnosis

- Clinical findings usually indicative.
- Radiography (LM projection): allows assessment of P3–hoof alignment/displacement (Figure 6.21). Serial radiography can be used to monitor progression.
- PET emerging as potentially useful imaging modality to assess digital perfusion.

Management

- Level of intervention determined by severity.
- Remove/treat inciting cause.
- Examine sole and identify/treat any subsolar abscess (may occur secondary to rotated P3).
- Distal limb cold therapy may be useful during prodromal phase.
- Mechanical support of feet to stabilize P3: redistribute loading away from dorsal laminae and reduce tension in the DDFT.
- Foot support options depend on individual circumstances: simplest (mild cases) is barefoot on soft/conforming bedding +/– solar and frog supports through to full sole support systems/foam pads or shoeing with raised heels (glue-on shoes preferable).
- Analgesia: managing pain is vital but important to recognise that pain has a protective role in limiting further damage (mechanical integrity of foot/hoof capsule is weakened during acute laminitis). Appropriate analgesia must be combined with confinement/restriction of activity to prevent further damage. Non-steroidal anti-inflammatory drugs (NSAIDs; phenylbutazone) are key analgesics (but no evidence that they actually alter disease progression). Multi-modal analgesia (NSAIDs + combination of other drugs/techniques, e.g., lidocaine, opioids; epidural, cryotherapy) has a role particularly in hospitalized cases.
- +/– acepromazine (0.02–0.06 mg/kg IM 2–3 times/day, or oral dosing to achieve mild sedation) for possible vasodilation and to limit movement.
- Mild laminitis: stable confinement/deep bedding/phenylbutazone until clinical improvement.
- Broad guideline for confinement: approximately 1 week confinement for each day of acute lameness.
- Failure to control pain/'sinking' of P3 with penetration of sole/recurrent severe abscessation/loss of hoof capsule may warrant euthanasia.

Prevention

- Supporting limb laminitis: preventive measures at time of initial surgery/treatment in at-risk horses; pain management plus heel wedges (20 degrees)/solar support/caudal breakover on supporting limb. Forced walking may be desirable for some conditions (e.g., cellulitis/lymphangitis).
- Endotoxemia cases: continuous distal limb cold therapy (1°C; preferably to mid-cannon level) during risk period for endotoxemia limits delivery of trigger factors to lamellae by reducing blood flow to foot. Thought to be beneficial only during developmental phase.

Prognosis

- Prognosis for survival +/– return to athletic use is correlated to severity of pain and speed of progression.
- Mild cases with no/little P3 rotation: good prognosis for return to full soundness.
- 'Sinking' of P3 associated with high mortality.
- Mortality rate after onset of supporting limb laminitis is high (50–75%).
- Degree of P3 rotation is not a reliable predictor of survival, but has implications for long-term soundness.

Keratoma

A keratoma is an aberrant, firm, slow-growing keratinous mass that develops within the foot between the hoof wall and P3. Rarely, may involve the sole. It causes lameness due to impingement on the internal structures of the foot, or due to the loss of the integrity of the barrier to ascending infection that the normal hoof provides. Usually in forelimb, solitary and unilateral and generally found under dorsal hoof. Pressure from enlargement of the mass

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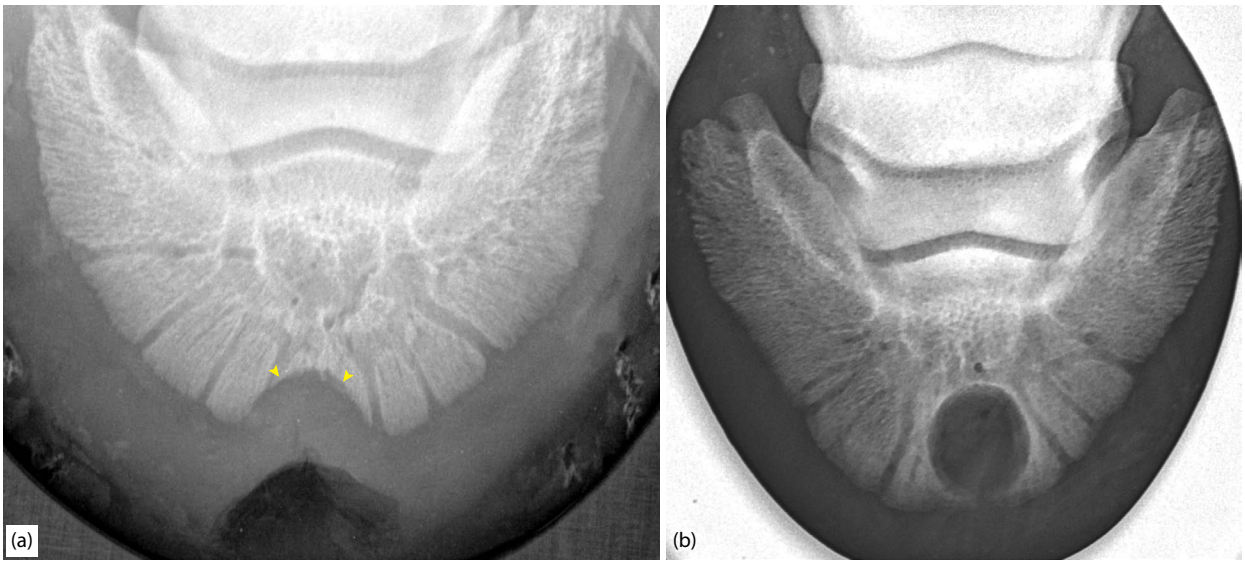


Fig. 6.22 Keratoma. Radiographs (D60^{0}Pr-PaDiO) of smooth-margined radiolucencies at (a) dorsal solar margin and (b) within dorsal P3.

may cause gradual deformation of the hoof wall, deviation of the white line and/or focal resorption of P3. Disruption of the white line may predispose to secondary infections.

Cause

- Underlying cause not known but thought that previous trauma to hoof or chronic irritation may contribute.

Risk factors

- Rare condition.
- Any age/stage of training.
- Risk factors unknown.

History

- Chronic/recurrent foot lameness.
- May initially be mistaken for other causes of foot pain (bruising, subsolar abscess).

Signs

- Mild-moderate lameness.
- +/– mild increase in digital pulse.
- Focal deviation of white line is common.
- Distortion/buttressing of hoof wall in long-standing cases.

Diagnosis

- Clinical findings and diagnostic blocking pattern similar to other foot conditions.
- Radiography: usually diagnostic, although some lesions are not visible. DPr-PaDO projection: smooth-edged radiolucent defect in P3 margin (differentiate from normal 'notch' or crena at the toe of P3) +/– new bone on mid-dorsal margin of P3 on LM projection (Figure 6.22a,b).
- CT (Figure 6.23): diagnostic modality of choice to assist surgical planning, as allows superior visualization of sensitive/insensitive laminar tissue. MRI also of use but variable signal intensity of lesion and surrounding tissues can complicate interpretation.



Fig. 6.23 Keratoma. CT (volume render) image of space-occupying soft-tissue lesion in P3.

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Management
- Surgical removal is treatment of choice.
- Surgical approach dependent on location of lesion: partial- or full-length hoof wall resection.
- Post surgery: application of hospital plate that stabilizes the hoof and permits daily dressing.
- Return to training is dependent on healing of hoof wall/solar defect; 2–4 months out of ridden exercise is typical.

Prognosis
- Prognosis for return to full use is good-excellent (>80%) with surgical removal.
- Recurrence is uncommon.
- Conservative management usually unsuccessful due to continued growth of mass and recurrent lameness.

Thrush
Thrush is an infection of the frog/sulci in one or more feet caused by anaerobic bacteria that break down hoof horn and progressively invade sensitive tissue.

Cause
- The bacteria responsible (most commonly *Fusobacterium necrophorum*, one of the causes of foot rot in sheep) are opportunistic invaders and are found in normal faeces and soil.

Risk factors
- Predisposing factors include conditions that lead to deep frog clefts (sheared heels, boxy foot), poor foot care/trimming and damp stable environment.
- Commonly seen in horses being stable rested.

History
- Usually detected at shoeing.
- May present with acute or chronic foot lameness.

Signs
- Black, foul-smelling discharge from sulci or frog.
- Frog horn usually underrun (Figure 6.24).
- +/– mild increase in digital pulse.
- +/– subtle lameness.
- Advanced infection or infections in feet with sheared heels may extend into sensitive tissue between bulbs of the heel, causing soreness/filling of the lower pastern.

Diagnosis
- Clinical findings definitive.



Fig. 6.24 Thrush: Underrun horn of frog.

Management
- Trimming of frog/sulci to expose underrun/infected tissue to air.
- Topical application of copper sulphate crystals, bleach (10% hydrogen peroxide solution) or povidone–iodine (10% solution) daily until healthy.
- Address any foot imbalance/hygiene factors thought to be causative.

Prognosis
- Excellent; does not interfere with training.
- Resolves in days/weeks with appropriate management.

THE FETLOCK AND PASTERN

Applied anatomy
The fetlock joint is the articulation between the third metacarpal/metatarsal ('cannon', MC3/MT3) bone and the first phalanx ('long' pastern bone, P1) (Figure 6.25a,b). It is a high-motion joint, subject to large forces during fast exercise and, in combination with the flexor tendons/suspensory apparatus, plays an important role in the energy efficiency of equine locomotion.

The distal condyles of the cannon bone are separated by a prominent sagittal ridge that articulates with a corresponding central groove in P1. Together with strong collateral ligaments, this ridge restricts rotational motion (although at full loading there is some lateral torsion of P1 relative to the cannon). The medial condyle is slightly larger than the lateral. The dorsal aspect of the condyles has a round profile and articulates with P1, whereas the palmar/plantar aspect is flatter and articulates with the proximal sesamoid bones (PSBs) (Figure 6.26): the dorsal

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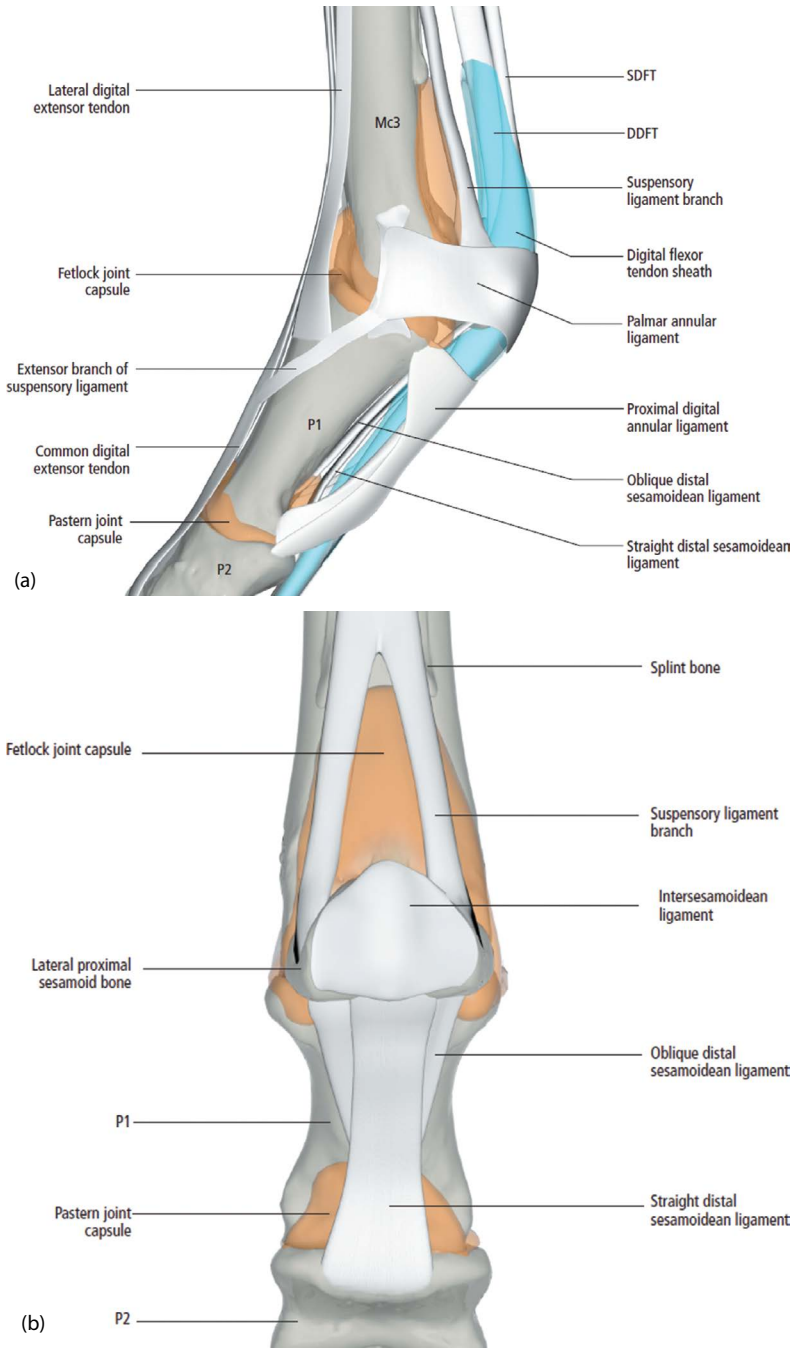


Fig. 6.25 **(a) Major structures of the fetlock and pastern (lateral view); (b) major structures of the fetlock and pastern (palmar view) with flexor tendons removed.**

and palmar/plantar aspects are separated by the transverse ridge. The paired PSBs, each interposed in the suspensory apparatus, sit at the back of the joint and serve to provide strength to the suspensory ligament and flexor tendon unit, which would otherwise be weak in compression. The suspensory ligament splits into two branches in the mid-cannon region, each branch fusing with its respective PSB and being continued functionally by the distal (straight, oblique, cruciate and short) sesamoidean ligaments below the back of the fetlock; weaker extensor branches run forward on the pastern to join the common digital extensor tendon. The thick intersesamoidean ligament, which connects the axial borders of the medial and lateral sesamoid bones, forms a groove over which the flexor tendons run at the back of the joint, constrained by the proximal annular ligament. Both the superficial digital flexor tendon (SDFT)

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Fig. 6.26 Articulation (arrowheads) of proximal sesamoid bones (PSB) with metacarpal condyles (transverse CT).

the DDFT broaden at this point in their course to apply greater surface contact with the suspensory apparatus.

At the level of the fetlock, the SDFT forms a ring (manica flexoria) around the DDFT then it bifurcates below the fetlock into its terminal branches, which insert above and below the proximal interphalangeal joint (PIPJ, 'pastern' joint). Emerging from this bifurcation the DDFT continues down into the back of the foot. High at the back of the pastern, deep to the flexor tendons, the distal sesamoidean ligaments (DSLs; straight, oblique, cruciate, short) attach the base of the sesamoids and intersesamoidean ligament to P1 and P2, thus stabilizing the PIPJ. The PIPJ is a relatively low-motion articulation between P1 and P2 and is the site of few problems in the racehorse.

The fetlock joint has both dorsal and palmar/plantar pouches. In the dorsal pouch a bi-lobulated synovial pad is found on the dorsal aspect of the distal cannon and is thought to act as a cushion. The palmar/plantar pouch ('articular windgall') is found between the back of the cannon bone and the suspensory ligament branches (SLBs). Close to this pouch is another synovial space, the digital tendon sheath ('tendinous windgall/windpuff'). The tendon sheath surrounds the flexor tendon bundle from the lower quarter of the cannon through the back of the fetlock and into the mid-pastern. By contrast, the PIPJ is a relatively low-volume articulation and is constrained by collateral ligaments and the flexor tendon bundle/tendon sheath. There are no direct communications between any of these synovial structures.

Functional adaptation and development of injury

Movement of the fetlock joint is primarily extension (during weight-bearing) and flexion (during limb protraction). At maximal loading (at full gallop) the fetlock hyperextends (Figure 6.27): the back of the fetlock is capable of striking the ground. At full extension P1 rides up around the dorsal aspect of the condyle and its dorsoproximal margin can impinge on the lower cannon above the articular surface; at the same time the PSBs move distally around the articular surface of the cannon and their lower margin can extend beyond the transverse ridge. In addition to distal movement, the sesamoid bones appear to rotate slightly in relation to each other, possibly due to tension exerted by soft tissue attachments. Extension of the joint and movement of the sesamoid bones is counterbalanced by the suspensory apparatus of ligaments and tendons.

The fetlock joint in both health and disease is the most important musculoskeletal site in the racehorse. Functional adaptation of the joint occurs in response to the cyclic biomechanical stresses of training: the subchondral bone thickens and the bony 'struts' (trabeculae) underlying it that provide structural support align to peak loading direction and also thicken through bone deposition. These adaptive changes occur at specific locations in the joint and have a protective effect by increasing resistance of the subchondral bone to mechanical fatigue. Damage, repair and adaptation is a natural cycle and at the microscopic level nearly all racehorse fetlocks have 'superficial microcracks' in the junctional zone between the articular cartilage and the subchondral bone of the lower cannon that is classified as normal 'wear and tear'. Adaptation of the sesamoid bones also occurs. In some circumstances, however, the equilibrium between damage and repair/adaptation is lost, and subchondral bone microdamage accumulates to the point of causing pathology. The factors that tip healthy adaptation into degenerative processes and mechanically fatigued bone in any given joint or horse are not understood fully, but it is known that bone repair (remodelling) at the cellular level is reduced during periods of intense training (subchondral bone microdamage in the juvenile racehorse is unlikely to accumulate below training speeds of 11 m/s). With insufficient ongoing repair, microdamage can accumulate and progress to clinical injury, including fracture. Pathologies can occur in isolation or concurrently in the fetlock; complex fetlock fractures encountered at racing speeds can involve combinations of condylar, pastern and sesamoid injuries

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Fig. 6.27 **Fetlock in neutral weight-bearing stance (left) and at extension under exercise load (right).**

and are frequently catastrophic. While much microdamage originates at the level of the subchondral bone, pathology involving the articular soft tissues is also commonly encountered in racehorse fetlocks and can include cartilage 'wear lines', partial (fibrillation) to full thickness cartilage loss, and dorsal impact injuries such as inflammation of the synovial pad and articular fragmentation.

Examination

Synovial effusion of the fetlock joint or digital tendon sheath is usually readily apparent with the limb weight-bearing. Observation of the limb from in front or behind the horse may reveal prominence of a SLB or sesamoid bone. It is useful to determine whether soft-tissue thickening is solely medial or lateral, confined to the fetlock or above or extending below the fetlock into the pastern.

With the leg raised in passive flexion, careful palpation of the structures around the fetlock joint is undertaken to determine the presence of any repeatable pain response. Key sites are the dorsoproximal aspect of P1, the SLB/sesamoid bone interface, the medial and lateral distal cannon, the sesamoid/base of the sesamoid regions and the flexor tendon bundle. Pain responses should be compared with the opposite limb. Forced flexion of the joint allows assessment of synovial pain and any restriction of range of motion. It should be noted that a feature of some of the most important fetlock pathologies is that the joint may be clinically unremarkable to palpation (i.e., not effused, and painless to palpate/flex).

Proximal phalangeal fracture

P1 fracture is one of the most important injuries affecting the racehorse fetlock. Several main configurations of P1 fracture occur; most common is the 'split pastern' (mid-sagittal fracture). May affect the forelimb or hindlimb. Classification of fracture type is determined by length, plane and direction of fracture line and the presence of comminution:

- Incomplete mid-sagittal fracture: fracture line propagates from the sagittal groove (typically dorsal half of groove) extending for a variable length down into P1. May be 'short' or 'long', with full extent not always discernible with initial radiographic imaging (Figure 6.28a–d).
- Very short incomplete mid-sagittal fissure fracture: may represent a prodromal phase of the more common 'split pastern' in some animals. Fracture at mid-sagittal site as previously mentioned but confined

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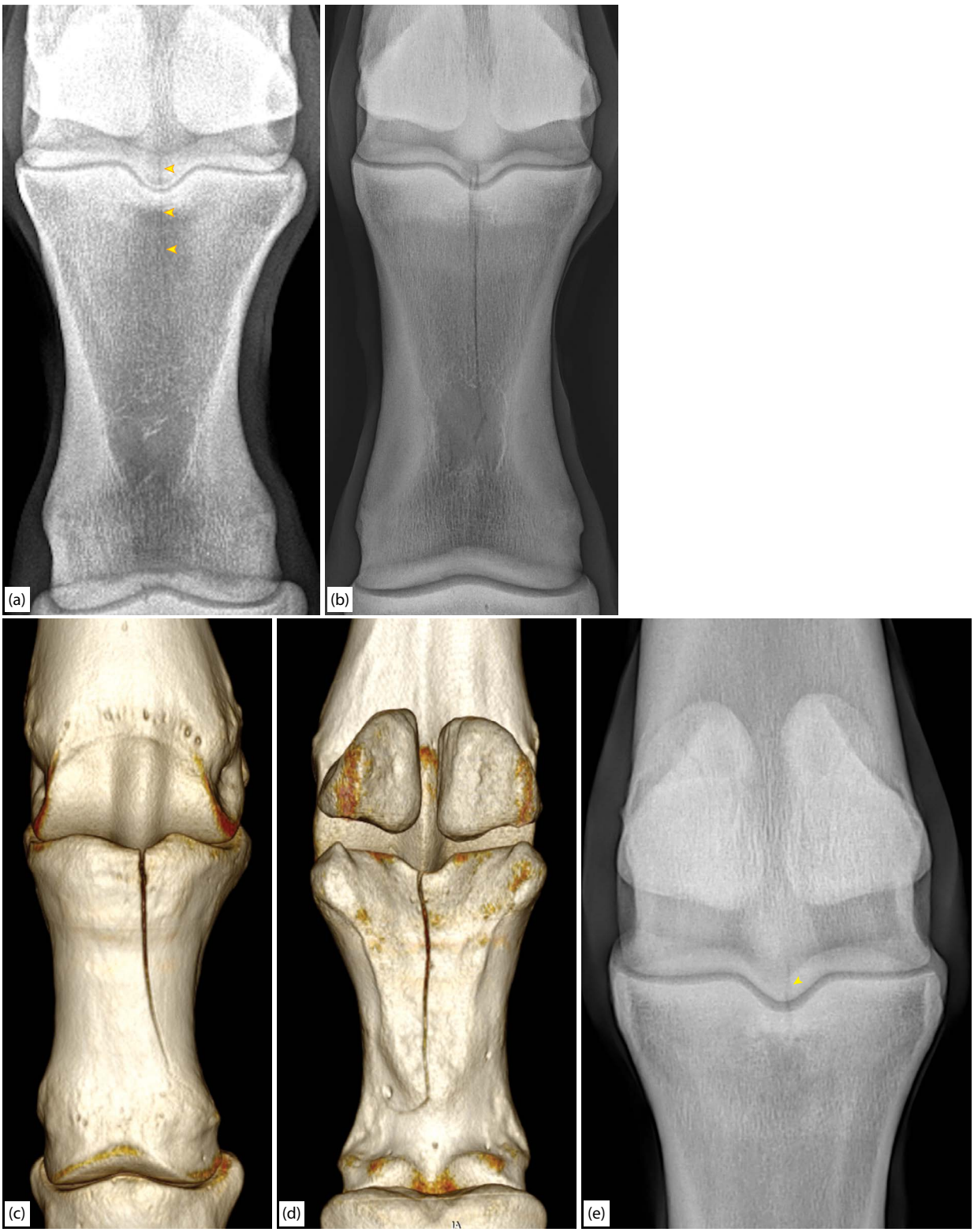


Fig. 6.28 **P1 fractures. (a) and (b) incomplete sagittal fracture (DPa). (c) dorsal and (d) palmar aspect of a long incomplete sagittal fracture (CT/volume render). (e) linear lucency at proximal articular margin of P1 typical of very short incomplete sagittal fracture (DPa).** *(Continued)*

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Fig. 6.28 *(Continued)* **(f) complete mid-sagittal fracture (DPa) extending between fetlock and pastern articulations. (g) DPa and (h) DLPMO projections of complete fracture exiting laterally. (i) and (j) comminuted fractures.**

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to proximal (articular) site; typically extends only a few millimetres distally and may remain radiologically silent or indistinct (Figure 6.28e).

- Complete mid-sagittal fracture: fracture propagates from sagittal groove either all the way through P1 to the PIPJ (Figure 6.28f) or exits laterally (rarely medially), creating two fragments (Figure 6.28g,h). Generally with some displacement.
- Comminuted fracture: complete fracture with multiple fragments; +/– intact strut of bone between fetlock joint and PIPJ (Figure 6.28i,j).
- Frontal fracture: fracture line in dorsal plane, propagates from articular surface and extends in dorsodistal direction. Generally incomplete and predominantly in hindlimb (Figure 6.29a–d).

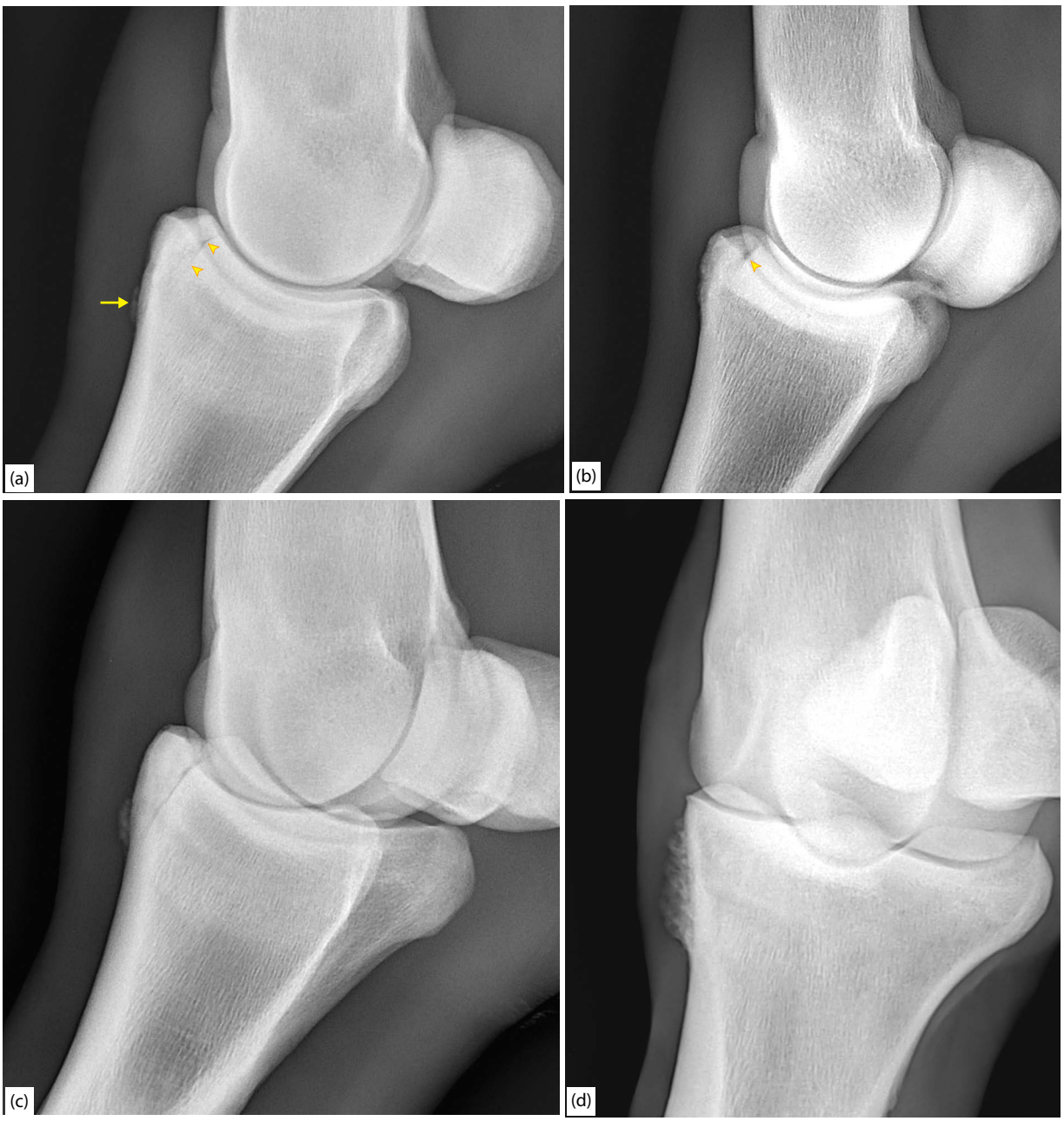


Fig. 6.29 P1 frontal fracture. Radiographs (LM) of frontal P1 fractures of (a) acute and (b) chronic appearance. Fracture evident as linear lucency (arrowheads) in dorsoproximal P1, with periosteal proliferative change at the distal extent (arrow). Oblique projections (c) may be required for injury detection, and presence of periosteal proliferation (d) is definitive even in the absence of an obvious fracture line.

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Cause
- High shear strains during full loading in sagittal groove region of proximal P1.
- Mid-sagittal fracture: generally considered an acute monotonic injury from single supraphysiological loading event. Stress fracture pathology responsible for some cases.
- Comminuted fracture: considered a severe manifestation of complete mid-sagittal fracture, although circumstances that lead to comminution are poorly understood.
- Frontal fracture: stress/fatigue injury.

Risk factors
- Mid-sagittal fracture: one of the most common fetlock fractures sustained by racehorses.
- Frontal fractures: rare.
- Encountered in horses of all ages.
- Generally during cantering/fast exercise phase of training.
- Other risk factors unknown.

History
- Mid-sagittal fracture (complete/incomplete): acute onset lameness that develops during or immediately after exercise (either on track or on return to yard).
- Very short mid-sagittal fissure fractures may have similar (but milder) presentation, or may display acute or insidious-onset mild lameness at exercise.
- Comminuted fracture: severe acute lameness on track.
- Usually no history of pre-existing lameness.
- Frontal fracture: either acute lameness after exercise or mild lameness worsening with continued exercise over days/weeks. Chronic injuries are also frequently encountered as an incidental finding without lameness.

Signs
- Lameness, presence of joint effusion and palpable pain varies with injury type/severity (Table 6.2).
- Typically severe lameness and obvious pain response to palpation of dorsoproximal P1; exceptions are hindlimb and some short incomplete injuries and frontal fractures, which may present with milder/more ambiguous signs.
- Consistent/repeatable focal pain response to firm digital pressure at midline dorsoproximal P1 site at/just below fetlock joint margin (with limb raised) is highly specific for P1 injury. Presence of this sign should be considered strong evidence of likely fracture pathology regardless of imaging findings.

Diagnosis
- Clinical findings strongly indicative.
- Radiography: to confirm configuration and guide management (generally unnecessary for comminuted fracture). Multiple DPa/Pl projections may be required to detect/assess fracture line (in the case of frontal plane fractures: may be best visualized on LM/slightly obliqued LM projections). CT recommended to assist surgical planning for long mid-sagittal fractures with suspected distal propagation.
- Very short mid-sagittal fissure fractures are not always radiologically evident and can be a diagnostic challenge. In addition to DPa/Pl projections, flexed DPa/Pl projections should be obtained; indistinct linear lucency +/– associated thickening of subchondral bone plate are possible signs of injury (Figure 6.30a,b). Repeat radiographic examination at 7–14+ days is recommended if initial screening unremarkable: development of periosteal reaction at dorsoproximal P1 does not always occur, but when

Table 6.2 **Signs associated with P1 fractures**

INJURY	LAMENESS	JOINT EFFUSION	RESPONSE TO FLEXION	PAIN ON PALPATION (DORSOPROXIMAL P1)
Mid-sagittal (forelimb)	Severe	Yes	Yes (marked)	Yes (marked)
Mid-sagittal (hindlimb)	Moderate-severe	Variable	Variable	Variable
Mid-sagittal (very short fissure/incomplete)	Mild-moderate	No	No	Yes (mild-moderate)
Comminuted	Severe	Yes (+ instability/crepitus)	Not required	Not required
Frontal plane	Mild-moderate; occasionally clinically silent	Mild (+/– palpable buttressing P1)	No	Variable

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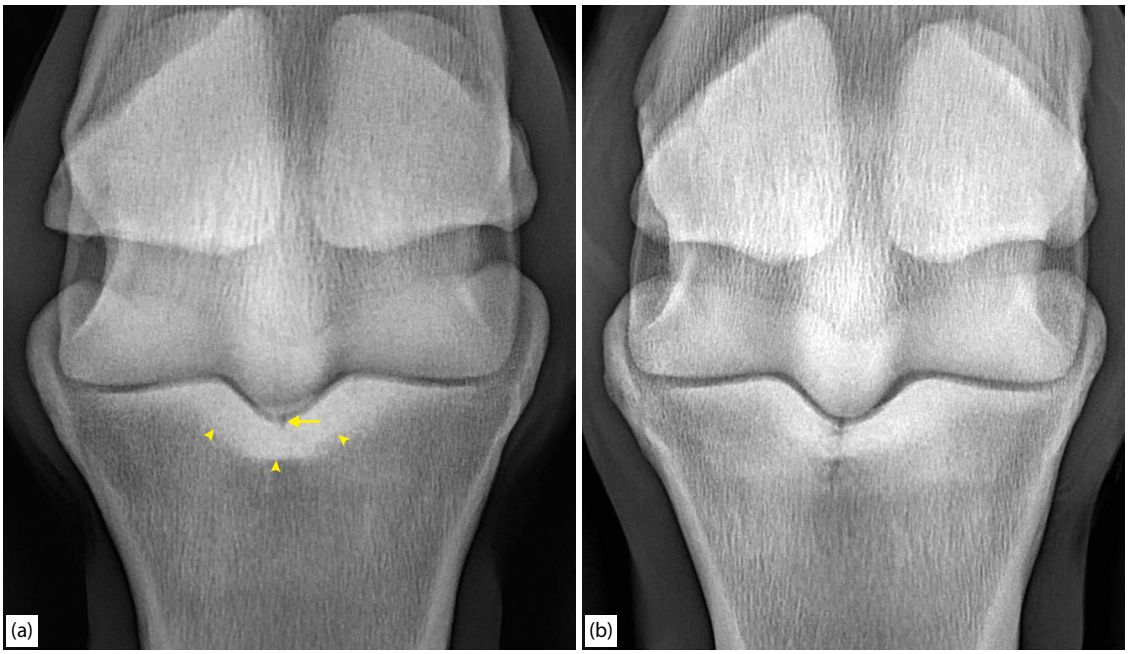


Fig. 6.30 **Flexed DPa/DPl radiographs (a, b) of very short incomplete P1 sagittal fractures. Focal/linear lucency (arrow) may be indistinct but when associated with thickened subchondral bone plate (arrowheads) may be sufficient for provisional diagnosis.**

it does is a strong indicator of injury (Figure 6.31). If radiological findings are inconclusive, MRI/bone scan/PET/CT is recommended for definitive diagnosis (Figure 6.32a–c).

- Diagnostic blocking (of distal limb) is contraindicated for all cases in which P1 injury is a possible differential diagnosis; high risk of serious/catastrophic deterioration of injury when assessing block result at trot.

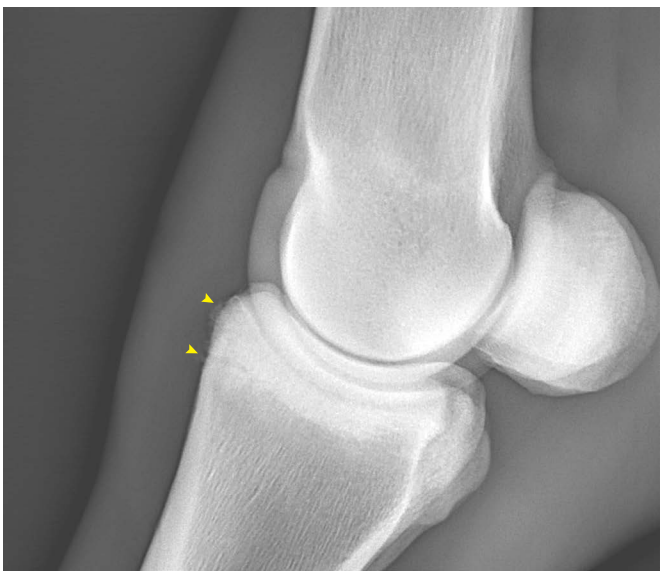


Fig. 6.31 **Very short incomplete P1 sagittal fracture: periosteal proliferative change at dorsoproximal P1 (LM).**

Management

- See Table 6.3.
- First aid: application of immobilizing splint/compression boot or Robert Jones bandage for transport or until initial radiographic assessment.
- Clinical suspicion of P1 injury but initial radiographic screening unremarkable: stable confinement (+ immobilizing bandage) with repeat radiography at 7–14 days (or advanced imaging: MRI/PET/CT).

Condylar fracture

Condylar fracture (MC3/MT3) is one of the most important injuries of the racehorse fetlock. Fracture typically originates from accumulated microdamage in the subchondral bone of the parasagittal groove and unless detected at the 'prodromal fissure' phase may progress to more serious (complete or incomplete) and potentially catastrophic injury. The fracture propagates proximally into the cannon usually from either the lateral (most common: >80%) or the medial parasagittal groove and may be incomplete or complete (Figure 6.33a,b). A proportion of fractures originate in the condyle rather than in the parasagittal groove (Figure 6.34). The spectrum of injury at time of diagnosis varies but the majority encountered in training are incomplete non-displaced injuries of variable length. They are most common (80%) in the forelimb. While lateral condylar fractures usually have a simple configuration, medial injuries are frequently incomplete

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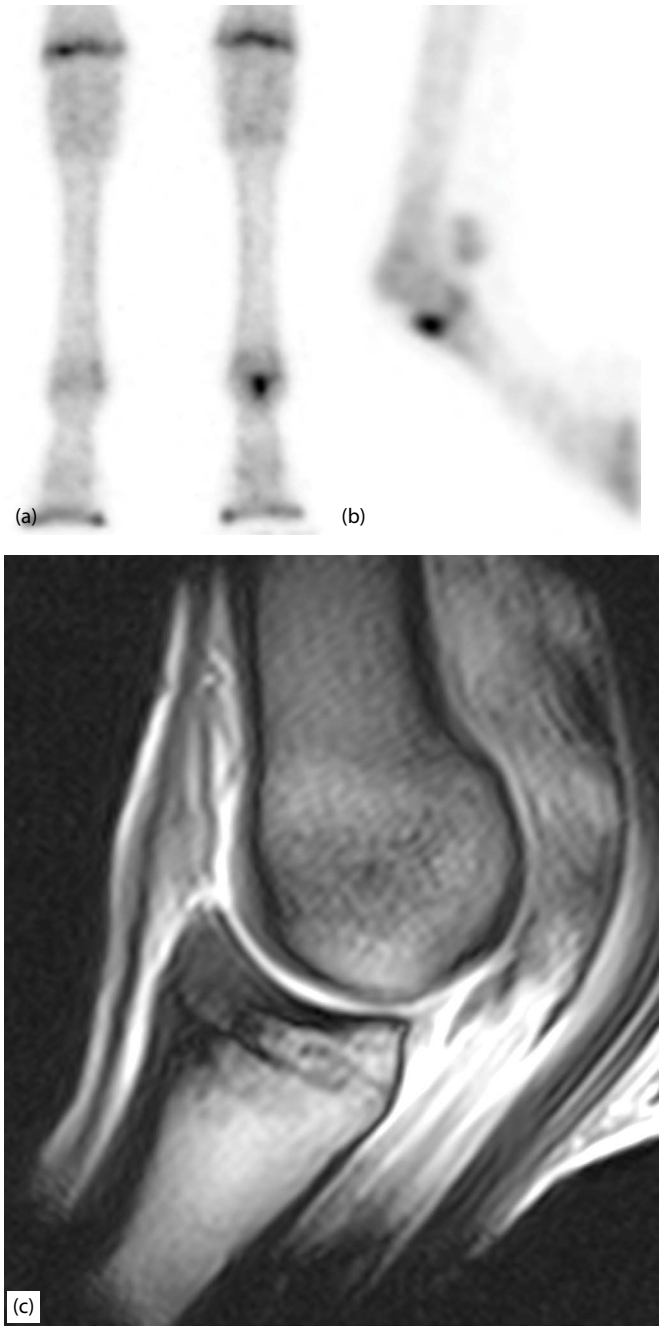


Fig. 6.32 Dorsal (a) and lateral (b) scintigrams, and (c) standing low-field MRI of radiologically silent P1 sagittal fracture; latter showing fluid-based signal in dorsoproximal aspect of P1.

and spiral proximally (Figure 6.35) and are associated with a high risk of displacement. Comminution at the articular margin occurs in approximately 15% of cases. Concurrent fracture of P1 may occur rarely (more common in hindlimb than forelimb) and is presumed to result from asymmetric loading of P1 immediately following loss of condylar congruity (Figure 6.36). Concurrent axial fracture of the ipsi-axial PSB is not uncommon in cases of displaced lateral condylar fractures and is considered a potentially catastrophic presentation.

Serious condylar fracture is one of the most readily preventable orthopaedic injuries of the racehorse. Few horses sustain complete/catastrophic injury without a preceding period during which detection of prodromal pathology is possible, with appropriate vigilance for lameness or imaging defects. Detection of injury at the 'prodromal'/'unicortical fissure fracture' phase is associated with reduced morbidity and mortality, shorter rehabilitation periods and better athletic outcomes.

Cause

- Cyclic loading during full training causes site-specific microcracks in the parasagittal groove: failure of normal bone repair processes to keep pace with damage may lead to accumulation of these microcracks and extension into the subchondral bone: variably termed 'microcrack coalescence'/'parasagittal crack arrays'/'unicortical' fissure fracture (Figure 6.37a–b).
- If training/racing continues, clinical fracture can occur when cannon subjected to high loading at faster paces (Figure 6.37c).
- Period of time elapsed between 'prodromal' phase of subtle lameness and progression to acute severe complete/incomplete fracture (if it occurs) can be highly variable between individuals and likely to be affected by many intrinsic (horse-level) and extrinsic (speed, surface, training loads) factors, as well as the presence of concurrent pathologies: can range from days to months. Some sub- or mildly clinical fissure fractures appear to withstand training/racing for a considerable period and develop a degree of chronicity, likely due to increased strain resistance at site of injury afforded by adaptive thickening of subchondral bone plate.
- Size and extent/depth of 'prodromal' lesion may have an important future role in risk determination.
- Some fissures may develop a 'bone cyst' appearance on radiographic/CT imaging, which is indicative of chronicity. These lesions ('pseudocysts') have an atypical appearance (Figure 6.38) and may be clinically silent but remain at increased risk of fracture propagation.
- Possible that some condylar fractures are sustained as monotonic injuries (i.e., without overt prodromal pathology); however, it is likely that these represent only a very small/atypical subset of the condition.

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Table 6.3 **Management of P1 fractures**

INJURY TYPE	MANAGEMENT	PROGNOSIS
Mid-sagittal: Short incomplete	Surgery generally not necessary for satisfactory repair but is recommended to assist healing/minimise risk of reinjury. Conservative management: stable rest (8–12 weeks)/walking +/– immobilizing bandage in acute phase depending on initial lameness.	Good prognosis (approximately 70%) for return to full use. Small proportion of conservatively managed cases heal incompletely or reinjure on return to training.
Mid-sagittal: very short (fissure) incomplete	Determined by individual circumstances. Conservative management (8–12 weeks walking) sufficient for many cases but moderate risk of incomplete healing/injury recurrence (greater risk than seen with other configurations). Surgery (osteostixis +/– surgical implant) gives best chance of uninterrupted return to racing and is treatment of choice.	Good prognosis for return to full use if managed surgically, although as for condylar fissures (p. 113) persistence of articular fracture may be problematic in some cases. Most horses managed conservatively return to training but recurrence of lameness at canter stage not uncommon.
Long incomplete (non-displaced)	Surgical reduction: treatment of choice for athletic soundness. Satisfactory outcome possible with stable rest/immobilizing bandage (8–12 weeks guided by radiography); small risk of displacement in initial weeks (greater risk with hindlimb fractures: typically warrant surgery).	Good prognosis (approximately 70–80%) for return to full use. Average time to return to racing 9 months.
Long incomplete (displaced)	Surgical reduction.	Good prognosis (approximately 70–80%) for return to full use.
Complete	Surgical reduction.	Complete (to lateral cortex): good prognosis (approximately 70%) for return to full use. Complete (to pastern joint): guarded prognosis (<50%) for return to racing.
Comminuted	Immediate euthanasia on humane grounds if severely comminuted/no intact strut. Surgical reduction (incorporating transfixation cast/external fixation device) under exceptional circumstances for salvage of horses with breeding value.	Hopeless prognosis for athletic use. Comminuted with intact strut: guarded-fair (>60%) prognosis for survival/paddock use with surgery.
Frontal:		
• Dorsoproximal incomplete	Conservative management: stable rest (8–12 weeks)/walking. Rehabilitation guided by radiography. Selected cases (if subclinical/long-standing) may continue in training without rehabilitation.	Good/excellent prognosis for return to (or continued) full use.
• Mid-articular	Surgical reduction.	Guarded prognosis for athletic soundness.

Risk factors

- Lateral condylar fracture is one of the most common causes of racetrack catastrophic distal limb injury (most common hurdling/steeplechase fatal injury).
- Encountered in horses of all ages.
- Clinical injury generally occurs during fast exercise/racing phase of training.
- Fast ground increases likelihood of injury.
- Catastrophic lateral condylar fracture (racing): greater risk for horses that have done no fast work during training; also for those that start racing as 3 or 4 YOs.

History

- Frequently display subtle-mild pre-existing lameness (prodromal injury/'fissure fracture' stage) in days/weeks prior to fracture.
- Incomplete/complete fracture presents as acute-onset severe lameness; usually develops during or immediately after exercise (either on track or on return to yard).

Signs

- Prodromal/unicortical injury ('fissure fracture'):
  - May be clinically silent/free from lameness.
  - Lameness, if present, is unilateral and subtle-mild.
  - Lameness may only be apparent (or may be more apparent) when ridden.
  - Usually no fetlock joint effusion or localizing signs (although can develop 'silently' in joints carrying other pathologies that do cause joint effusion).
- Incomplete fracture:
  - Moderate-marked lameness.
  - Mild-moderate fetlock joint effusion.

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Fig. 6.33 **Incomplete lateral metacarpal condylar (a) and complete lateral metatarsal condylar (b) fractures (DPa/Pl). Fracture in (b) exits laterally with proximal comminution.**

- Complete fracture:
  - Severe lameness.
  - Marked fetlock joint effusion +/– oedematous thickening over affected side of lower cannon.
  - Marked resentment to fetlock flexion.
  - Catastrophic fractures may be open/comminuted with discontinuity of bone column in mid-cannon.



Fig. 6.34 **Incomplete fracture propagating from mid condylar position in hindlimb fetlock (flexed DPl).**

WHAT TO DO WITH A CLINICALLY SILENT CONDYLAR FISSURE?

- When a fissure/suspected fissure is detected through a screening process (such as at pre-purchase) in a sound horse, it is generally not known how long the lesion has been present.
- Information about activity/chronicity of fissure is important to guide management/rehabilitation and specifically determine likelihood of imminent progression of injury with continued training. Radiological/CT appearance alone may not be sufficient to make these determinations.
- Horses with radiologically subtle fissures (or those with chronic/settled appearance) are best fully assessed with functional imaging (MRI/PET).
- Horse should be removed from high-speed exercise until diagnosis is established (may walk/trot).
- Surgery (lag screw across lower cannon) frequently recommended; however, efficacy in assisting healing/preventing reinjury is uncertain. Most fissure fractures only just breach subchondral bone and safe placement of an implant so close to the joint is not possible.
- Simple rest/rehabilitation is an acceptable option; however, essential to utilise imaging (radiography/CT/PET/MRI) to monitor healing and guide return to full training.
- If fissure remains radiologically 'static', or if initial imaging suggests chronic non-healing lesion, a stronger case can be made for surgery as an intervention that may limit future risk of serious propagation in the event of poor radiological resolution.



Fig. 6.35 **Medial condylar fracture (hindlimb) showing spiralling proximal propagation of fracture line (DPl).**



Fig. 6.36 **Concurrent (catastrophic) lateral condylar and P1 fracture (frontal CT).**

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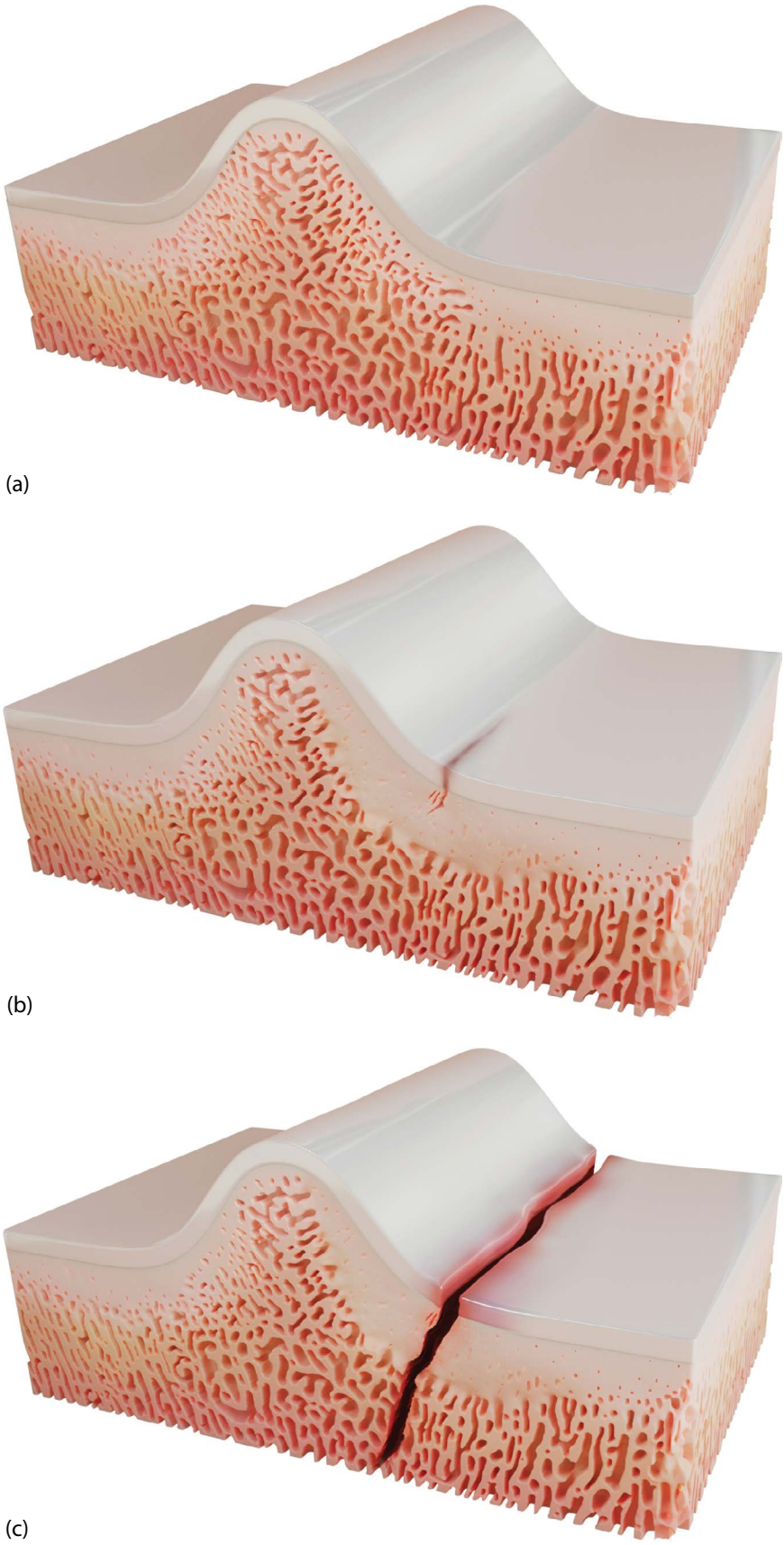


Fig. 6.37 **Progression of pathology leading to complete condylar fracture. (a) normal 'conditioned' distal cannon with some thickening of subchondral bone plate in condylar region; (b) prodromal fissure(s) in subchondral bone of parasagittal groove, with surrounding increased bone density; (c) complete fracture.**

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Fig. 6.38 **Chronic fissure in parasagittal groove with 'pseudocystic' appearance (CT).**

Diagnosis

- Prodromal injury: diagnostic blocking, radiography +/– MRI/CT/PET may be required to localize injury. Majority (>80%) of clinically active prodromal injuries are radiologically detectable with good technique, but (with few exceptions) only on flexed dorsopalmar/plantar radiographic projection (Appendix 4, p. 473); multiple projections may be required (Figure 6.39). If radiologically unremarkable but clinical suspicion still exists, MRI/CT/PET or delayed (generally at 7–14 days) radiographic re-evaluation (Figure 6.40a,b) is recommended. Radiological features indicative of injury include some/all of following (repeatable on multiple projections): distinct or indistinct focal lucency (of any shape) in parasagittal groove region; increased radiodensity distal cannon centred on parasagittal groove +/– sagittal region (rather than condyle) (Figure 6.41a); increased conspicuity metacarpal/tarsal vessels (Figure 6.41b) (latter finding not specific to condylar fracture).
- MRI (standing low-field), CT or PET: required for diagnosis when prodromal injuries are radiologically silent or ambiguous; MRI also highly useful to determine activity/risk of propagation of condylar fissures detected with radiographic screening (Table 6.4).
- Bone scan is an inferior diagnostic modality for detection of condylar fracture due to poor differentiation of bone activity patterns from other subchondral pathologies (Figure 6.43). Not recommended.
- Incomplete/complete fracture: clinical findings strongly indicative, confirm with radiography. Multiple oblique projections to determine extent of injury in cases with spiral configuration. Displaced lateral condylar fractures: recommended that standing +/– flexed D20°MPaLO radiographic projections obtained to assist detection of concurrent axial sesamoid fracture.

Management

- See Table 6.5.
- Injuries detected at prodromal ('fissure fracture') stage with no or mild lameness require shorter rehabilitation periods and conservative management (removal from ridden exercise) is sufficient for healing in most cases. Risk of progression to complete fracture is negligible providing horse does not canter/gallop.
- Prodromal fissure fractures: Surgery (surgical implant in lower cannon) may be prescribed but is not the treatment of choice for all/most cases; placement of implant across fracture site typically not possible due to proximity to joint. Best viewed as prophylactic measure against serious fracture propagation during future training in the event

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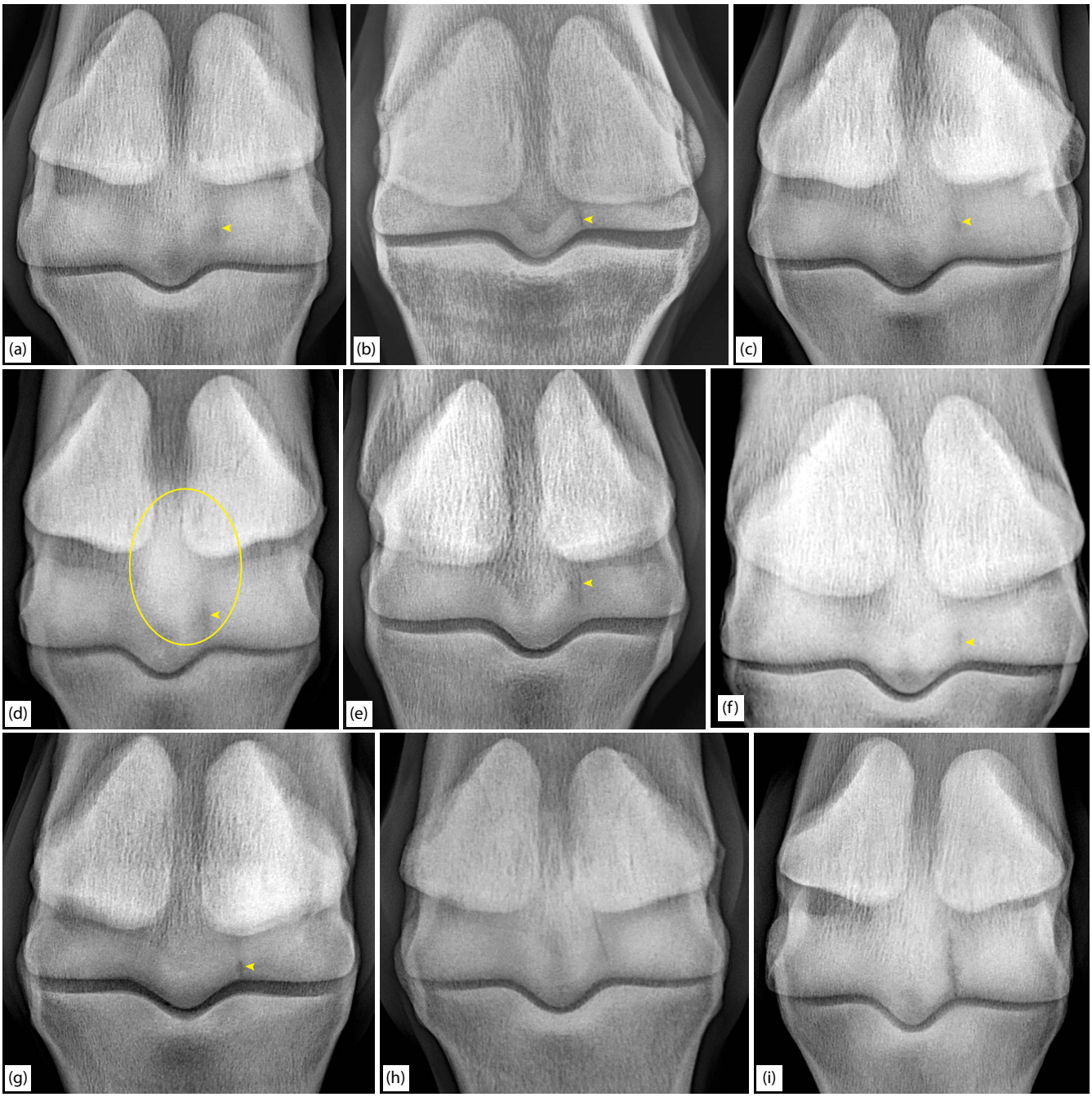


Fig. 6.39 (a–i))Examples of range of appearance of prodromal 'fissure' fractures (arrowheads) on flexed DPa/DPl radiographic projection. Increased opacity in parasagittal groove/midline region (circled) is a variable feature and associated with chronicity.

of inadequate healing of original injury. Efficacy of surgery in assisting healing of primary lesion remains undetermined/doubtful, and articular defect +/– lameness may persist long-term (Figure 6.44), regardless of surgery/rehabilitation. Best surgical candidates are those with chronic +/– deep fissures where uncertainty exists about likelihood or speed of healing with conservative management, or those with concurrent/ongoing orthopaedic issues that complicate risk assessment during training (therefore elimination of future risk from condylar fissure advantageous).

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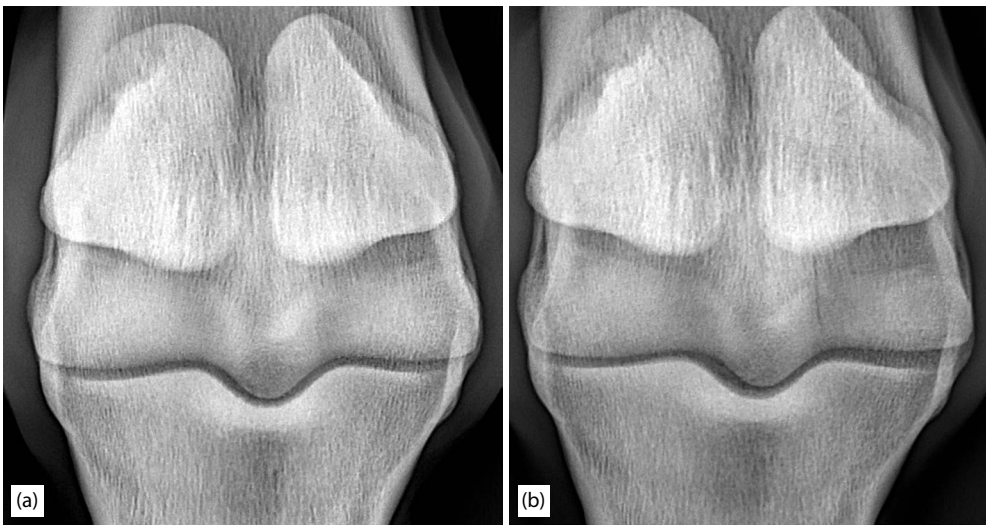


Fig. 6.40 Initial (a) and delayed (b) flexed DPa radiographs showing development of radiologically obvious fissure 3 weeks after acute onset lameness.

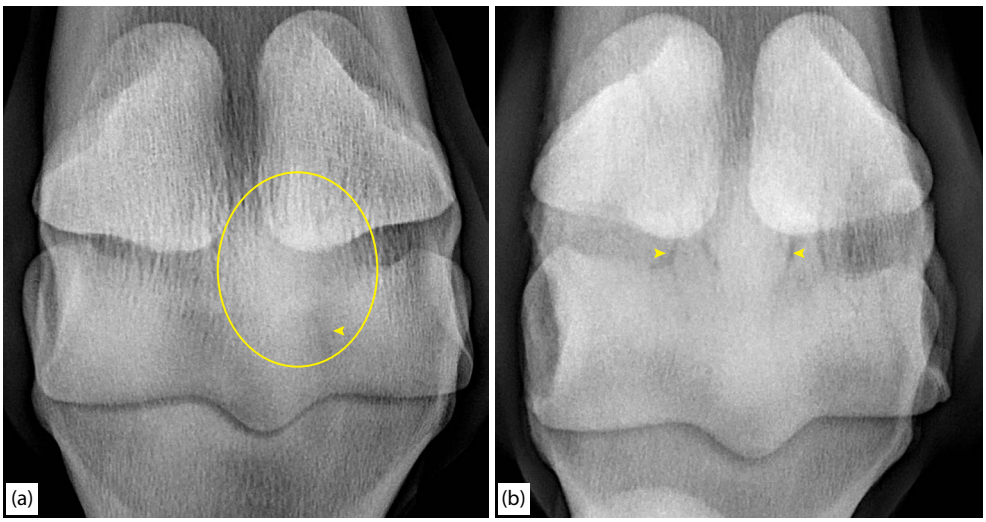


Fig. 6.41 (a) Increased density in region of parasagittal groove/sagittal ridge (circled) +/– associated lucency (arrowhead) is a characteristic feature of fissure fracture on flexed DPa/DPl radiographs. (b) increased conspicuity of vessels (arrowheads) indicates serious chronic distal cannon pathology but is not specific to fissure fracture.

Table 6.4 **Prodromal condylar injury: MRI grading of severity and risk of fracture propagation**

MRI FINDINGS	RISK
Cortical fissure visible within the subchondral bone, no/minimal increased density/thickening of the surrounding SCB plate and no T2*/STIR hyperintensity in surrounding bone (Figure 6.42a).	May be developmental. No/low immediate risk of fracture if remains in high–intensity exercise.
Cortical fracture extending minimally into the cancellous bone with mild local reactive sclerosis and/or very mild, STIR/T2* hyperintensity in the cancellous bone immediately surrounding the fracture.	Moderate immediate risk of fracture if remains in high-intensity exercise.
Cortical fracture extending minimally into the cancellous bone with any degree of local reactive sclerosis with moderate/marked STIR/T2* hyperintensity diffusely within the cancellous bone of the affected condyle (Figure 6.42b).	High immediate risk of fracture if remains in high-intensity exercise.
Cortical fracture extending a variable depth into the cancellous bone and/or exiting the endosteal surface of the subchondral bone plate with marked generalized STIR/T2* hyperintensity in one or both metacarpal/tarsal condyles (visible radiologically as short incomplete parasagittal condylar fracture).	High risk of complete fracture even in medium-intensity exercise.

STIR, short tau inversion recovery; S. Powell (unpublished data).

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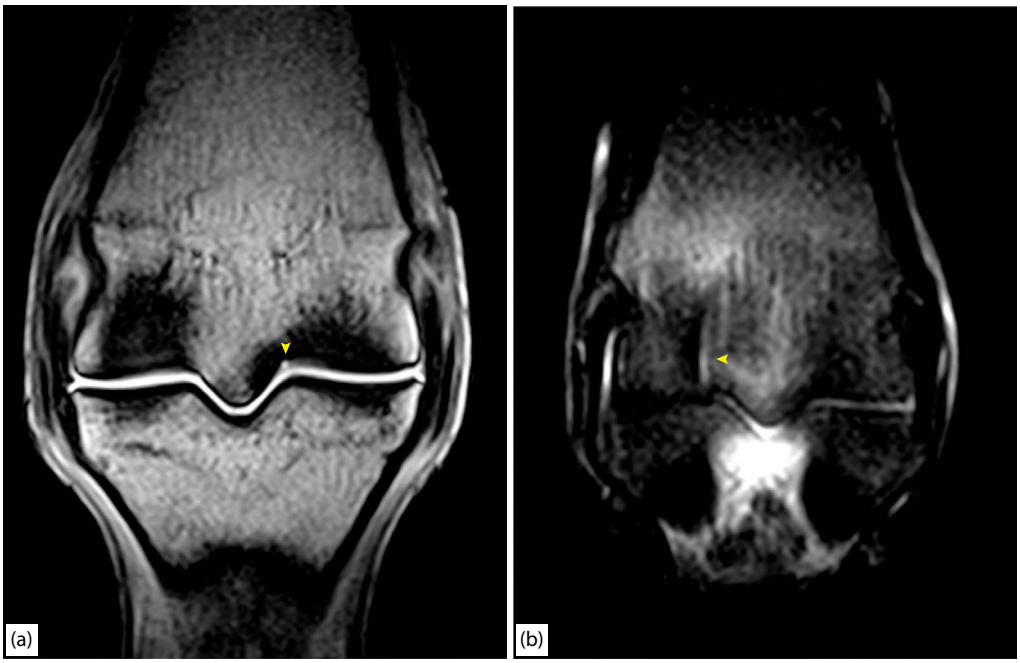


Fig. 6.42 Low-field MR images of a 'low-risk' (a) and a 'high-risk' (b) cortical fissure.

Table 6.5 **Management of condylar fractures**

INJURY TYPE	MANAGEMENT	PROGNOSIS
Prodromal/unicortical 'fissure' fracture	Removal from ridden exercise (may walk; generally no need for stable rest) for 4–12 weeks (determined by initial staging and severity of injury). Immobilizing bandage not required. Radiographic monitoring +/– MRI to assess healing/guide management if early return to cantering desired. Surgery (single implant) optional but may lower risk of future serious propagation in the event of poor healing and strengthens confidence around safety upon return to full work.	Likely progression to incomplete/complete fracture if not removed from fast exercise. Conservative: good/excellent prognosis for return to full use; small proportion may heal incompletely or reinjure. Surgery: good/excellent prognosis for return to full use, although persistent articular defect in small proportion of cases can cause recurrent lameness.
Short incomplete (non-displaced)	Surgical reduction for best chance of good athletic outcome. Conservative management: stable rest (8–12 weeks)/walking +/– immobilizing bandage in acute phase). Rehabilitation guided by radiography.	Excellent (>80%) prognosis for return to full use with conservative management or surgery.
Long incomplete (non-displaced)	Surgical reduction is treatment of choice for athletic soundness. Satisfactory outcome possible with stable rest/immobilizing bandage; small risk of displacement in initial weeks and OA long term.	Good-excellent prognosis for return to full use with conservative management or surgery.
Complete (displaced/ non-displaced)	Surgical reduction.	Good (approximately 80%) prognosis for return to full use, although less likely to race than horses with incomplete fracture. Guarded (>50%) prognosis for racing following surgery for horses with articular comminution.
Complete with concurrent axial fracture of proximal sesamoid bone	Surgical arthrodesis if paddock future desired; otherwise euthanasia on humane grounds.	Poor/hopeless prognosis for athletic soundness. Paddock salvage possible with surgery.
Open/comminuted	Usually warrants immediate euthanasia on humane grounds. Surgical reduction under exceptional circumstances for salvage of horses with breeding value.	Can be salvaged with surgery but poor-guarded prognosis for life (>40% non-healing due to infection).

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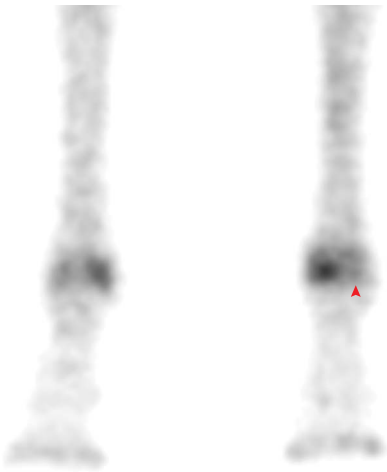


Fig. 6.43 **Scintigram (dorsal view) of front fetlocks of horse with active fissure fracture in lateral parasagittal groove of left fore distal cannon (arrowhead). IRU of unrelated non-fracture pathology (POD) in medial condyle of same fetlock is of much greater intensity than of the fracture site.**

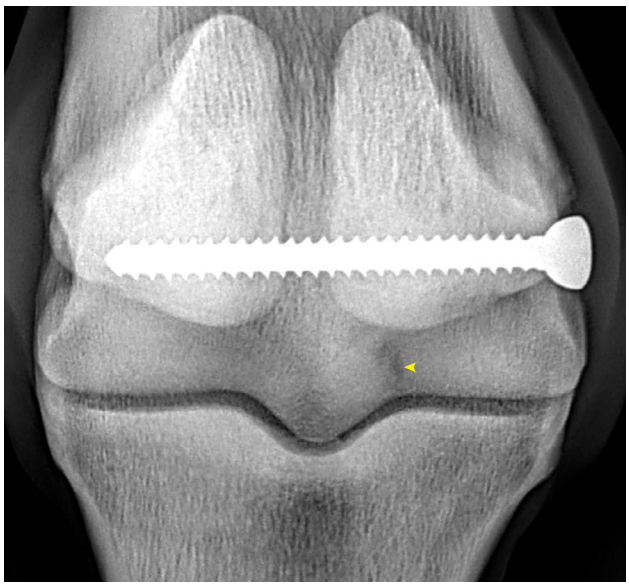


Fig. 6.44 **Clinically active persistent fissure/articular defect (arrowhead) despite surgical placement of lag screw.**

- Failure to detect injury (or failure to rehabilitate injury) at prodromal stage will typically result in progression to incomplete/complete fracture during high-intensity exercise; however, when this might occur is difficult to predict without advanced imaging. Some horses may continue to train/race for significant periods before serious injury ensues.
- Field management of incomplete/complete fracture: application of immobilizing splint/compression boot or Robert Jones bandage for transport to surgical facility or until initial radiographic assessment (Table 4.1, p. 38).
- Radiologically evident fissures in the parasagittal groove (at the predilection site for condylar fracture) that are clinically and physiologically inactive (as determined by MRI) can be difficult to manage because they represent a persistent defect that may predispose to serious injury. When 'static' and presumed to be either developmental or long-standing, there can be little certainty that resolution/healing will occur within any practical time frame. Management is determined by individual circumstances and guided by regular reassessment.

MANAGING THE PERSISTENT CONDYLAR FISSURE

- Persistence of radiologically visible articular defect is an occasional problem for both surgically and conservatively managed condylar fractures.
- May be clinically silent, or associated with chronic lameness +/– joint inflammation.
- If there is an existing surgical implant, preferable to leave in place: gives confidence that serious propagation of fracture unlikely to ensue.
- Further rest generally not helpful; continuation of training usually the only option, although presence of/severity of lameness will guide management and may necessitate retirement in a proportion of cases.

Prognosis

- If not catastrophic, the majority (>2/3) of horses may return to racing regardless of fracture configuration. Successful athletic outcome generally determined by congruity/quality of healing of articular margin of fracture.
- Fractures with spiral configuration that extend into proximal cannon remain at risk of catastrophic deterioration for initial 3–4 weeks following injury regardless of surgical intervention and should be managed with care (Figure 6.45).
- Prognosis for return to racing is diminished if there is concurrent PSB fracture or articular fragmentation.
- In general, prognosis for return to racing is better for hindlimb than forelimb injuries, and better for incomplete than complete injuries.
- 50–60% return to racing following surgery for medial condylar fractures (forelimb/hindlimb).
- Ongoing lameness (on return to training) in small proportion of cases following surgery: may be due

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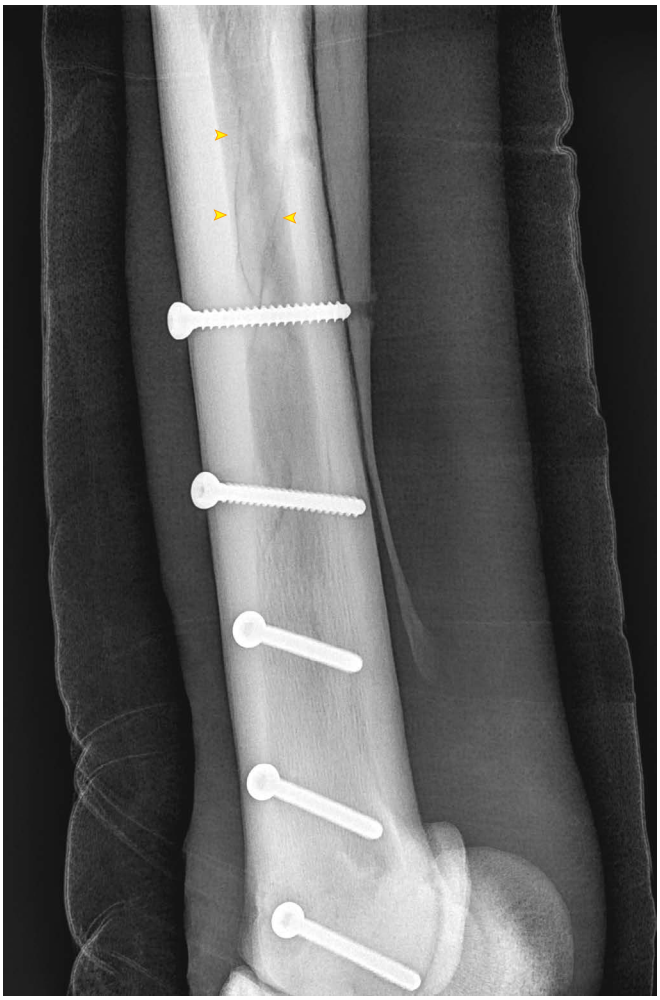


Fig. 6.45 **Potentially serious spiral propagation (arrowheads) of fracture into proximal cannon during post-surgical rehabilitation (not catastrophic in this case; returned to racing).**

to poor healing (or initial fragmentation) at articular margin; ingress of synovial fluid can lead to formation of 'bone cyst'-like defect. Response to subsequent intra-articular medication is variable. Generally preferable to leave implants in situ (rather than remove) as gives degree of confidence that serious fracture will not ensue. Risk of fracture propagation is low but can occur even in presence of surgical implants.

Proximal sesamoid bone fracture

PSB fracture is one of the most important injuries involving the racehorse fetlock and the leading cause of catastrophic race day fracture in several racing jurisdictions. May occur during training/racing or can be encountered as long-standing radiological lesions from previous foal injury. Several configurations of fracture occur, each with its own particular pathological processes and risk factors, and can largely be considered distinct injuries. Fracture type, severity and location of affected sesamoid (forelimb/hindlimb, medial/lateral) determines management and outcome of injury:

- **Apical fractures**: most common configuration (Figure 6.46a,b). Occur in proximal one-third of the bone. Associated with partial disruption of SLB (Figure 6.46c,d). Occur more frequently in hindlimb. Almost always articular.
- **Mid-body fractures**: when uniaxial usually affects forelimb medial sesamoid (Figure 6.47a–c). Biaxial (mid-body medial + apical/mid-body/oblique lateral PSB) fractures are potentially catastrophic due to loss of suspensory support of fetlock. May be comminuted and usually associated with distraction of fragments (Figure 6.48a,b).
- **Abaxial fractures**: true avulsion fracture of variable size/configuration (Figure 6.49a–d) with moderate-severe associated disruption of SLB (Figure 6.50). Predominantly in forelimb and medial sesamoid. May be articular or non-articular.
- **Basilar fractures**: fragment of variable size from base of sesamoid (Figure 6.51a,b); partial displacement typical. Predominantly affects forelimb medial sesamoid. Usually articular.
- **Axial fractures**: predominantly in forelimb. Invariably associated with concurrent (displaced) condylar fracture of cannon bone (Figure 6.52).
- **Other**: atypical configurations occur but are rare (Figure 6.53a,b).

Cause

- Fractures (in horses in training) are likely to arise as acute event preceded by cumulative microdamage/modelling of sesamoid (as a result of repetitive loading at high speeds). May also occur as acute monotonic overloading injuries. The site of articulation between the sesamoid bones and the condylar regions of the lower cannon experiences high-magnitude compressive forces during high-speed exercise, while the palmar/plantar aspect is subject to high tensile strains from suspensory attachments.
- Sesamoid bone and suspensory ligament strengthen at different rates during conditioning.
- SLBs more likely to injure early in training (or in untrained juvenile) and sesamoid bones later in training (if overloaded).
- Training induces non-uniform changes in bone density through the sesamoid that may make the

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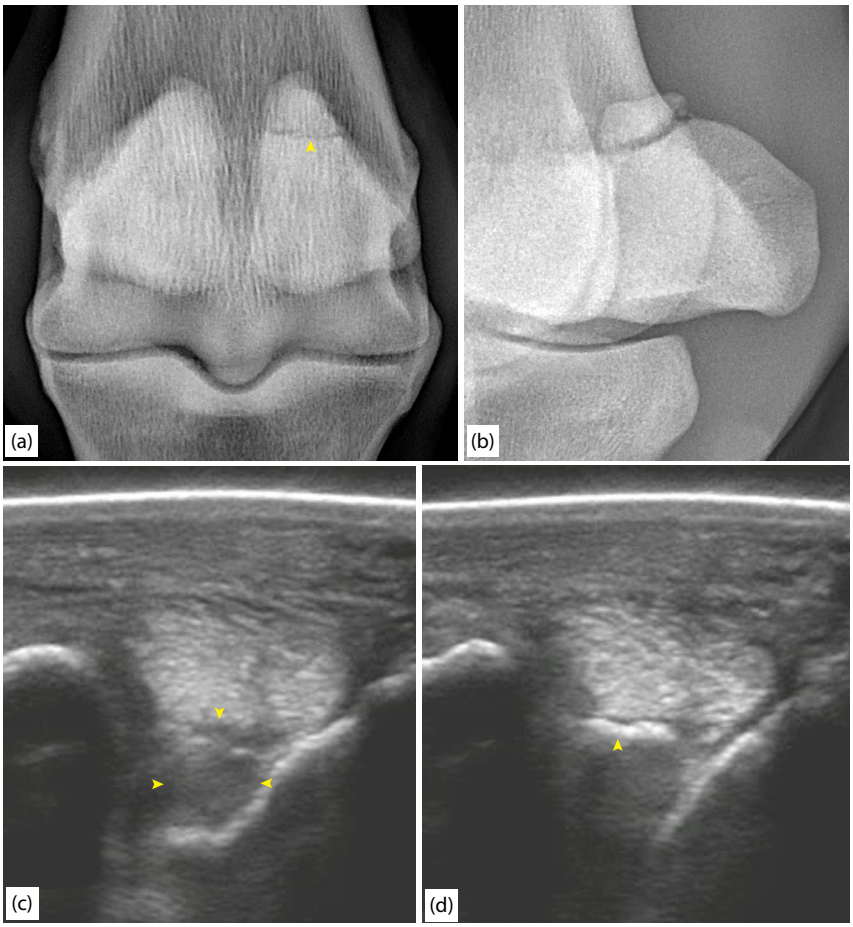


Fig. 6.46 (a) and (b) Apical PSB fracture (DPl/DLPlMO); (c) ultrasonogram of associated disruption to suspensory ligament branch (arrowheads); (d) fracture fragment evident more distally (arrowhead).

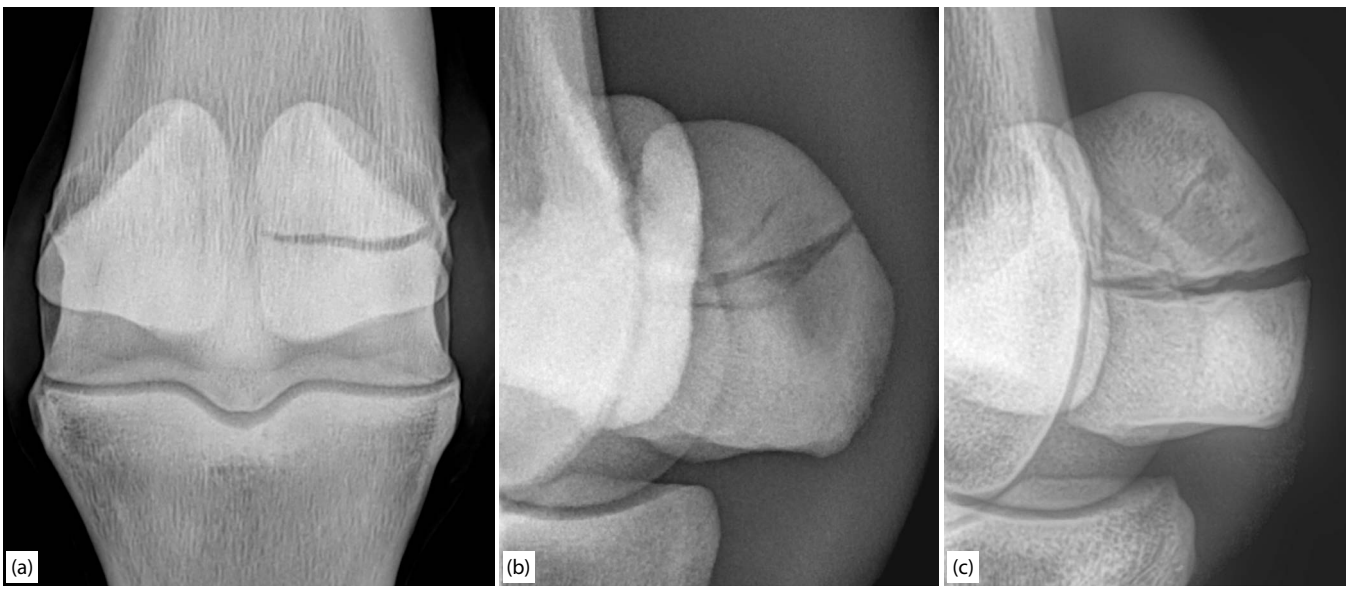


Fig. 6.47 (a-c) Mid-body PSB fractures (DPa/DLPaMO).

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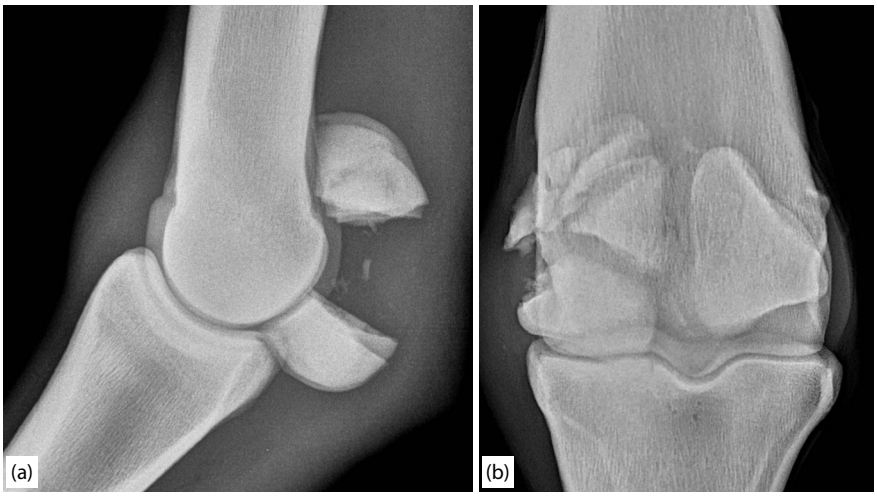


Fig. 6.48 Biaxial (a) and comminuted uniaxial (b) PSB fractures.

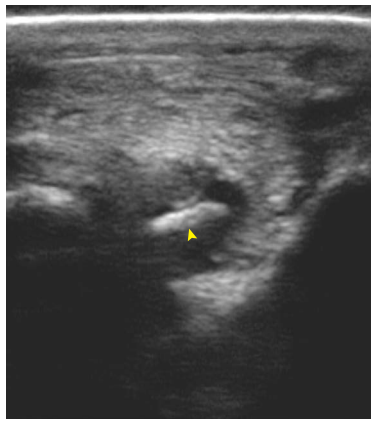


Fig. 6.50 Ultrasonogram (transverse) of distal SLB with avulsion fragment of PSB (arrowhead).

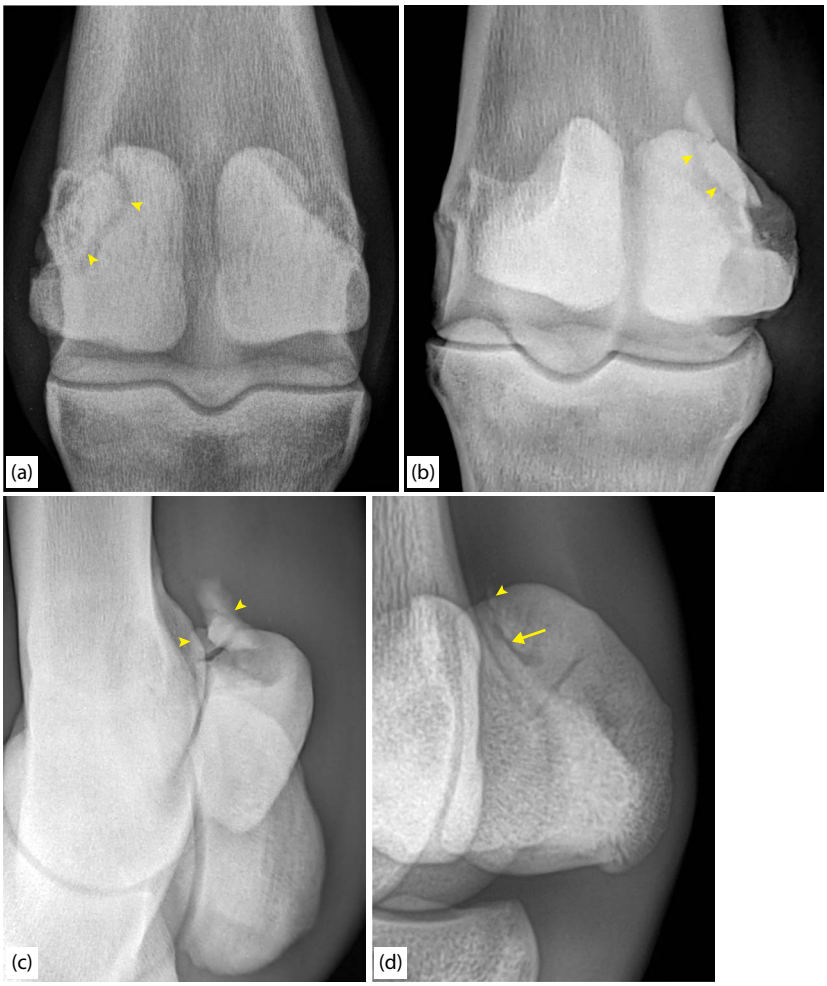


Fig. 6.49 Abaxial/avulsion fractures of PSB (arrowheads indicating avulsed fragments). (a, b) Typical configuration; (c) comminuted fracture best demonstrated on proximodistal 'skyline' radiographic projection and (d) atypical small avulsion fracture with additional lucent residual defect in parent bone (arrow).

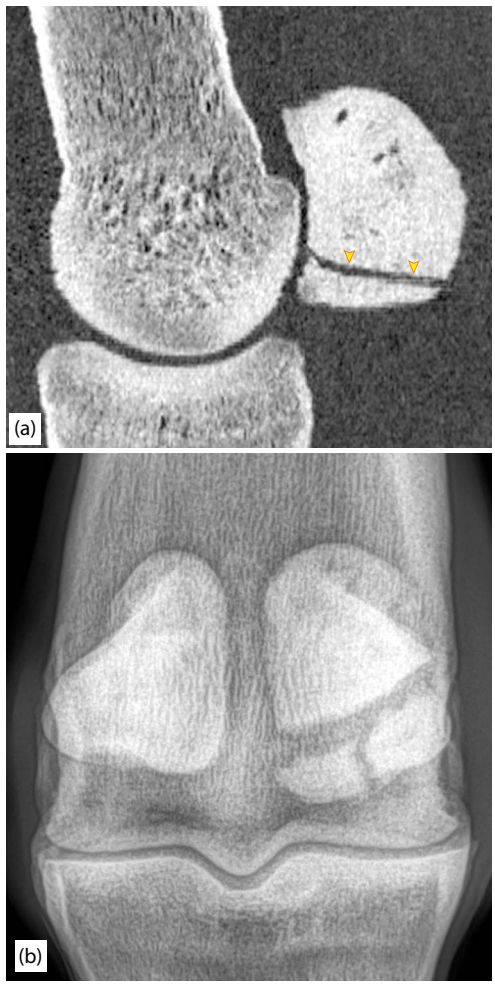


Fig. 6.51 Basilar PSB fractures: (a) sagittal CT image of typical configuration (arrowheads); (b) comminuted (DPa).

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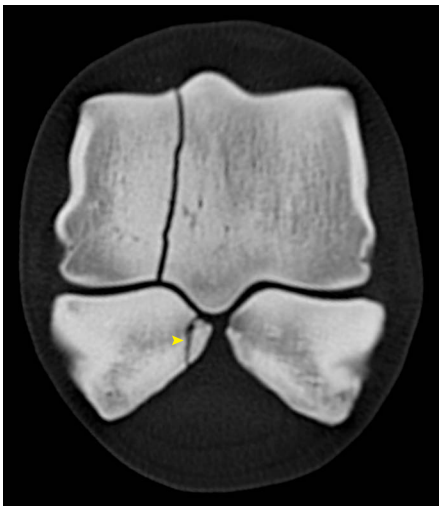


Fig. 6.52 **Axial fracture of forelimb PSB (arrowhead) with concurrent lateral condylar fracture of MC3 (transverse CT).**

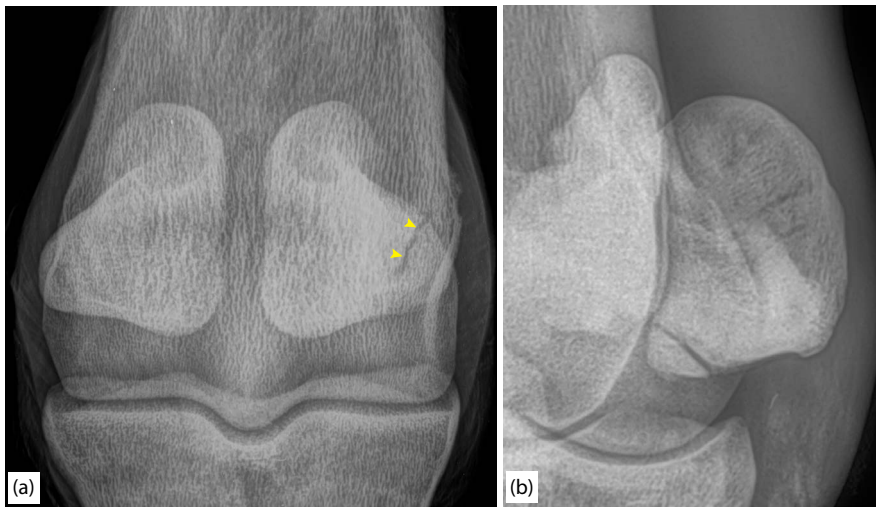


Fig. 6.53 **Atypical PSB fractures: (a) non-displaced abaxial (arrowheads); (b) oblique basilar.**

zone just below apex of sesamoid a structurally weak point, while the mid-body becomes relatively denser and thereby predisposed to the types of cumulative microdamage seen in the lower cannon.

- Mid-body fractures generally propagate from focal sites of cumulative microdamage with typical site being on the dorsal abaxial (subchondral) mid-body face of the bone (Figure 6.54), when the fetlock hyperextends maximally at high speeds. These subchondral lesions share some characteristics with palmar/plantar osteochondral disease (POD) of the lower cannon (p. 119). Less commonly, possible that some injuries propagate from prodromal resorptive lesion on palmar (non-articular) aspect of PSB.
- Apical (most commonly) and basilar fractures occur in foals and weanlings (majority occur in first 3 months of life); typically heal or settle then remain clinically inactive until training commences.

Risk factors

- Biaxial mid-body fractures are an important cause of catastrophic racetrack injury (particularly on dirt and synthetic tracks): typically in older horses and more likely in horses that have higher cumulative training at high speed or have been campaigned intensively during season of injury. More common in entire males than fillies or geldings.

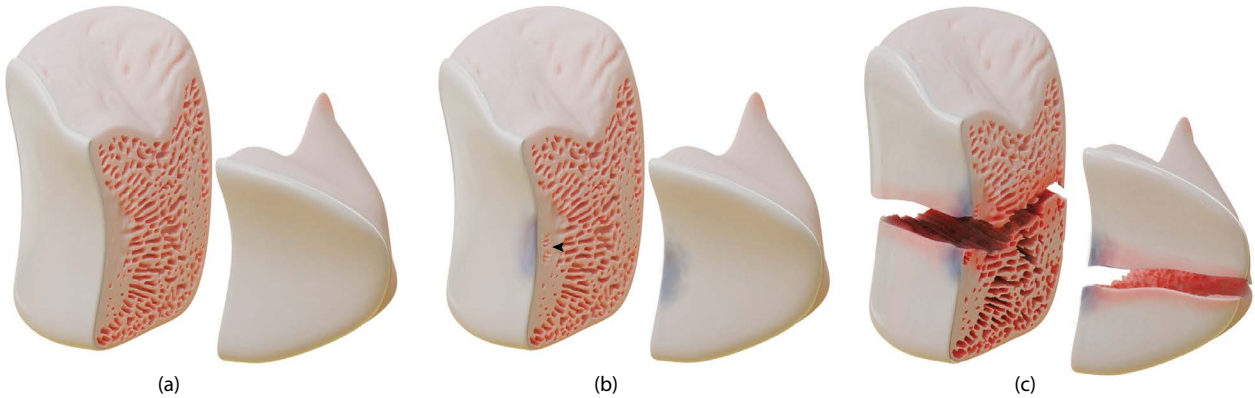


Fig. 6.54 **Progression of pathology commonly implicated in mid-body PSB fracture: (a) normal 'conditioned' PSB with thickened subchondral bone on dorsal articulating facet; (b) development of focal lytic lesion in subchondral bone (arrowhead), with overlying cartilage degeneration; (c) complete fracture through weakened zone under high exercise loads.**

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- Risk factors for other configurations poorly understood.
- Sesamoids with radiological changes consistent with 'sesamoiditis', and those with prominent vascular channels are not associated with greater risk of fracture than unaffected horses.
- Fractures sustained by foals are associated with turnout, and hard/dry ground is likely to be a contributing risk factor.

History

- Acute-onset lameness during or after racing/fast work.
- Long-standing (settled) injuries may be detected at routine radiography (Figure 6.55a–c).

Signs

- Clinical findings vary with fracture configuration and severity.
- Mild-marked unilateral lameness.
- Focal swelling and pain on palpation of affected sesamoid.
- +/– fetlock joint effusion.
- Biaxial fractures: severe lameness and dropping (hyperextension) of fetlock due to complete disruption of suspensory apparatus.
- Occasionally, radiologically silent injury presumed to be subchondral bone insult/'bruise': moderate-marked unilateral lameness (with reluctance to place heel of affected limb to ground) without other palpable clinical abnormality of fetlock region.

Diagnosis

- Radiography: fractures usually well-defined on standard and elevated oblique projections.
- Standing and flexed D20^{0}MPaLO radiographic projection useful for detection of axial fractures.
- Ultrasonography: assessment of suspensory/DSL attachments not essential but may assist management.
- PET is the modality of choice for detection of prodromal fracture and other bone injuries: intense increased radiopharmaceutical uptake (IRU) typically noted on dorsal abaxial subchondral face of bone (Figure 6.56a,b).
- Bone scan: not necessary for overt fracture, and poor resolution limits sensitivity and specificity (and overall usefulness) for prodromal/incomplete injuries. May occasionally permit detection of some atypical (and radiologically silent) pathologies such as presumed 'bone bruise', tensile strain injury or atypical prodromal fracture (Figure 6.57). Not recommended for detection of prodromal injury.
- MRI (standing low-field): may be of some use in differentiating the causes of increased scintigraphic activity but sensitivity and specificity currently undetermined (likely to be poorer than PET/CT) and is not generally recommended for detection of prodromal injury.

Management/prognosis

- See Table 6.6.

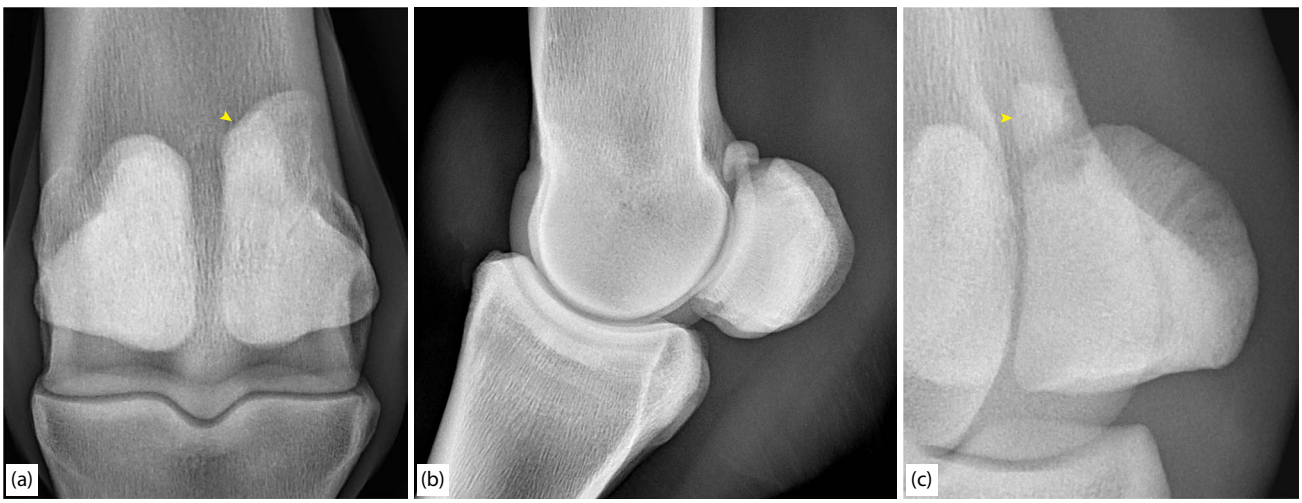


Fig. 6.55 **Typical radiological appearance of long-standing/historical PSB fractures: (a) enlarged/elongated lateral PSB (arrowhead) resulting from healed juvenile fracture; (b) and (c) clinically silent apical fractures (arrowhead).**

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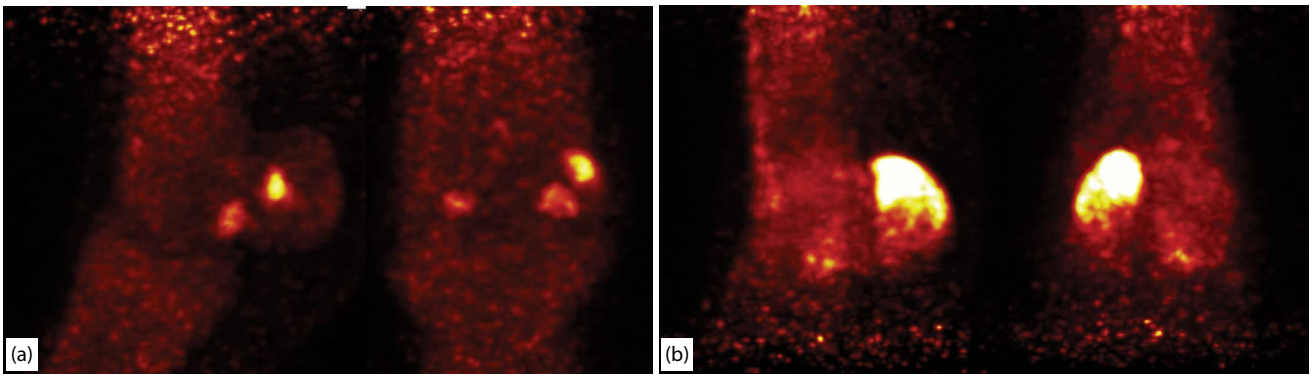


Fig. 6.56 **Lateral (left) and dorsal (right) maximal intensity projections (18F-NaF PET) of forelimb fetlocks showing (a) marked focal uptake consistent with 'pre-fracture' stress remodelling/osteolysis at dorsal abaxial site of PSB; (b) marked diffuse uptake in proximal half of PSB associated with acute lameness but of uncertain origin; may be consistent with 'bone bruise', tensile strain injury or atypical prodromal fracture. Mild focal palmar metacarpal condylar uptake is also present in image (a).**

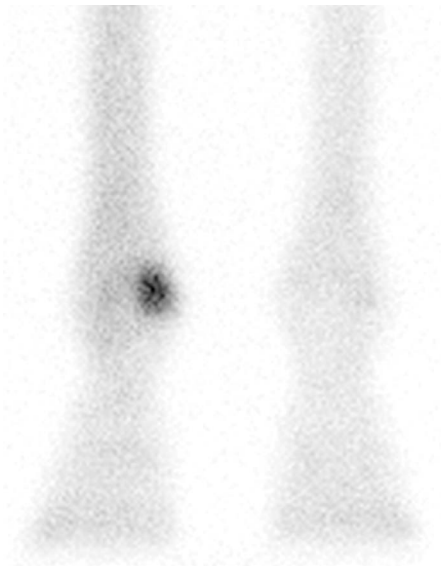


Fig. 6.57 **Plantar scintigram of marked IRU in medial PSB associated with severe acute lameness and presumed 'bone bruise' or tensile strain injury.**

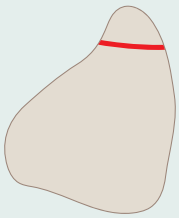
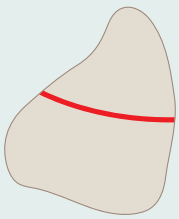
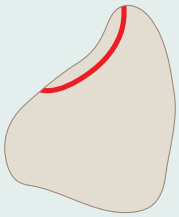
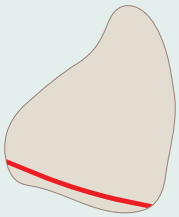
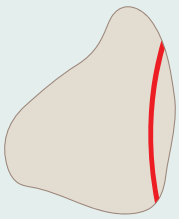
Palmar/plantar condylar osteochondral disease (POD/'condylar stress reaction')

POD is a chronic and, in many cases, progressive syndrome of subchondral bone +/– cartilage damage affecting load-bearing sites at the back of the fetlock. The term encompasses a spectrum of pathology which is an extension of the normal adaptive response to bone loading at predetermined sites in the palmar/plantar metacarpal/metatarsal condyles, and manifesting as microscopic trabecular fractures/osteolysis/osteonecrosis, which may be recoverable through to irreversible macroscopic collapse of the joint surface. Very few animals are affected by the latter. Despite the condition's name it is recognised that cartilage lesions develop much further along the pathological spectrum and that in the earlier stages pathology is confined to subchondral bone. When clinically apparent it may be associated with generalised loss of action (usually when multiple limbs are affected) or overt lameness. Some degree of POD is extremely common in the horse in training, does not always necessitate veterinary attention and frequently remains undetected throughout a racing career, but in a small proportion of individuals it may cause significant lameness or be career-limiting.

Most frequently occurs in hind fetlocks and is usually bilateral but may occur in any limb. There is no apparent left- or right-sided bias regardless of track geometry. Affected joints typically have biaxial lesions (hindlimb pathology greatest in lateral condyle; forelimb distributed between medial and lateral condyles). Affects the palmar/plantar aspect of the condyle, approximately 5–8 mm palmar/plantar to the transverse ridge. Pathology encountered can range from increased bone density/hardening (osteosclerosis) of condylar bone to crescentic/ovoid-shaped regions of 2- to 4-mm diameter of subchondral bone collapse +/– subsequent cartilage damage in advanced cases (Figure 6.58a–c). Progression of pathology is unpredictable and not always linear, and aside from the most severe cases (joint surface collapse) there is often a poor correlation between imaging findings, clinical presentation and outcome. Subchondral bone damage precedes cartilage erosions: overlying cartilage remains intact/viable prior to subchondral bone plate collapse. Associated subchondral (and cartilage) lesions may also develop on the opposing articular face of the PSB.

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Table 6.6 **Management and prognosis for acute proximal sesamoid bone fractures**

FRACTURE TYPE	MANAGEMENT	PROGNOSIS
Apical 	Surgical removal of fragment gives best chance of return to racing. Conservative management is acceptable alternative: stable rest/walking for 8–12 weeks.	Size/geometry of apical fragment does not influence prognosis but location does. Conservative management: generally a good prognosis for racing. Surgery: excellent prognosis for hindlimb or lateral forelimb fractures. Medial forelimb sesamoid: guarded prognosis for return to racing (<50%) regardless of management.
Mid-body 	Uniaxial fractures: consider surgical fixation if athletic future desired. Lengthy rehabilitation (up to 1 year) regardless of management. Biaxial fractures (or uniaxial comminuted fractures): generally justify euthanasia; salvage for paddock use possible with surgery (fetlock arthrodesis) or long-term splinting.	Uniaxial: fair prognosis (60–70%) for return to some athletic use following surgical fixation; guarded prognosis for racing. Biaxial, and uniaxial or biaxial comminuted fractures are a career-ending/life-threatening injury.
Abaxial 	Surgical removal of fragment gives best chance of return to racing. Conservative management acceptable alternative with lower success rate.	Surgery: good prognosis (>70%) for return to racing. Fractures involving apical and abaxial margins have less favourable outcome.
Basilar 	Surgical removal of fragment gives best chance of return to racing (when fragment does not involve entire base of bone). Conservative management acceptable alternative with lower success rate.	Surgery: good prognosis (>70%) for return to racing. Conservative management: guarded prognosis (<50%) for racing. Factors with negative influence on outcome include large fragment size (full dorsopalmar width), displacement (>3 mm) and comminution.
Axial 	Not usually treated (concurrent condylar fracture may be repaired by surgical arthrodesis for paddock salvage only).	Prognosis for athletic use poor/hopeless.
Prodromal/ incomplete fracture	Continued training without rest period carries risk of sesamoid bone fracture. Conservative management: stable rest/walking for 6–10 weeks.	Excellent prognosis for return to full use. No current information on long-term recurrence rates.

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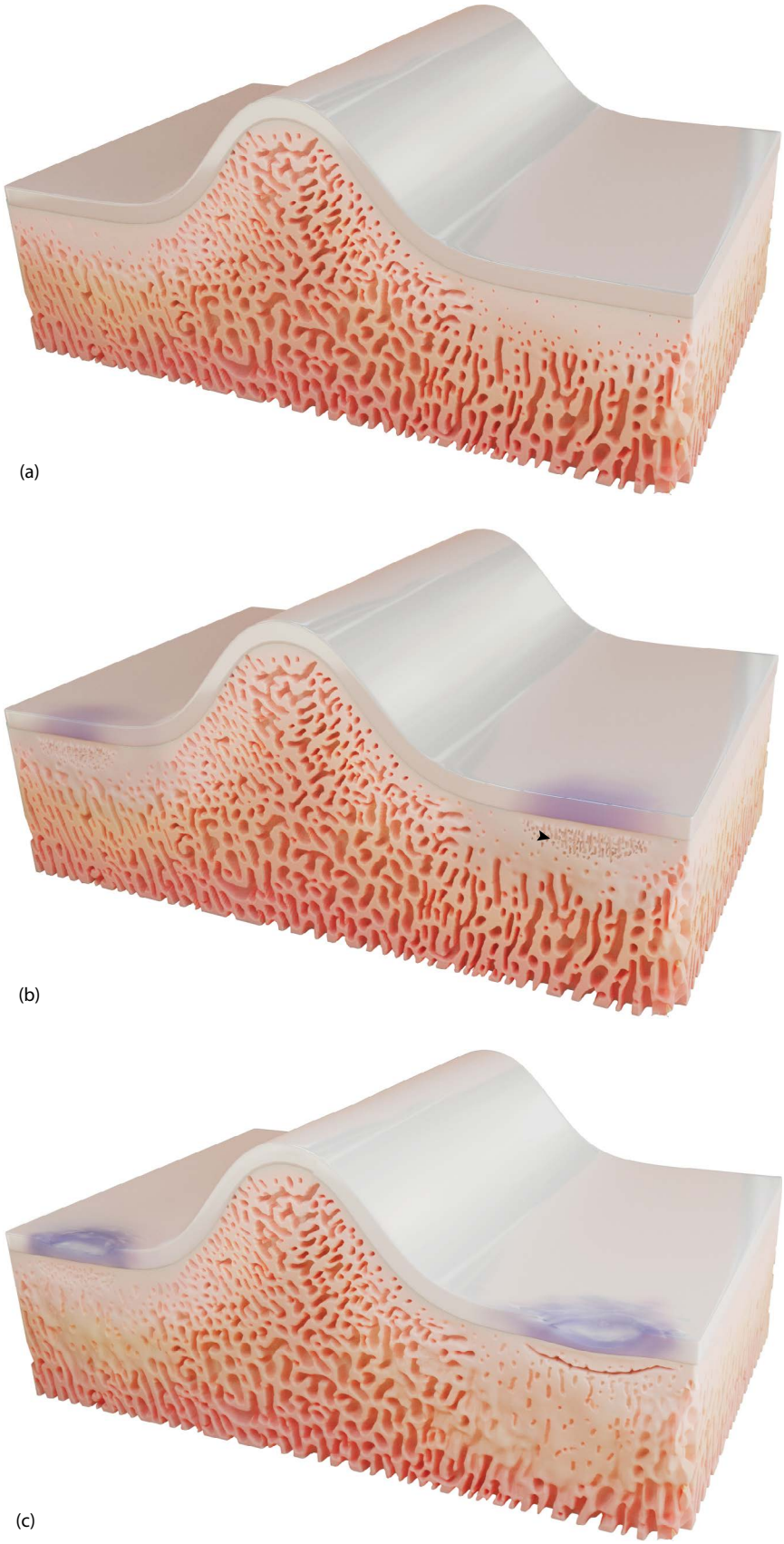


Fig. 6.58 **Progression of pathology associated with POD in distal cannon. (a) normal 'conditioned' distal cannon with adaptive thickening of subchondral bone plate in condylar region; (b) subchondral osteolysis/ osteonecrosis (arrowhead); (c) marked condylar sclerosis with transverse subchondral fracture, collapse of subchondral bone plate and overlying cartilage degeneration/cavitation. Most cases encountered in clinical practice do not progress beyond (b) regardless of training load/management.**

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Cause

- Adaptive remodelling in the subchondral bone at the back of the fetlock (condylar regions of cannon) occurs in response to training stresses and is largely protective against those stresses, but accumulation of microdamage can lead to focal fatigue of bone and secondary damage to overlying cartilage.
- There is a broad association between severity of POD and other degenerative changes in the fetlock (such as cartilage loss, dorsal impact injuries).

Risk factors

- Very common condition.
- Overall prevalence unknown; however, majority of racehorses in training have POD lesions (and up to 30% of racehorses submitted for postmortem for other reasons have lesions of moderate-marked severity).
- Influence of possible factors of importance such as conformation, bodyweight, foot balance, stage of career, training speeds, athletic ability, track surfaces, etc., remain poorly researched.
- While in general incidence appears to increase with age and cumulative high-speed training, 2 YOs are also commonly affected.

History

- Insidious onset of poor action (if bilateral) or lameness, typically developing over period of months.
- Non-specific signs: trainer may report that horse 'doesn't trot' (but usually satisfied with faster paces); may display unwillingness to jump off at start of canter.
- Action may slowly worsen as horse progresses to fast work/racing phase of training.
- +/– secondary thoracolumbar back pain/epaxial muscle spasm; many affected horses may have received treatment for a sore back.
- Lameness (particularly unilateral manifestation) may sometimes be most pronounced in initial weeks of training following a long rest period (and then improve as training progresses).
- May also be encountered as incidental imaging finding (i.e., not associated with lameness/poor performance) at pre-purchase/regulatory screening.

Signs

- May be clinically silent.
- Hindlimb pathology: characteristic bilateral action at trot: short striding/'rolling' of quarters/close or plaiting gait.
- Unilateral lameness (if present) can be of mild-moderate severity.
- Severely affected: may display multi-limb lameness and attempt to break directly from walk to canter.
- Lameness often improves ('warms up') rapidly with exercise.
- Fetlock joints typically free from palpable abnormality. Rarely associated with fetlock effusion until end-stage disease.

Diagnosis

- Diagnosis should take into account history, clinical presentation, diagnostic blocking and imaging: interpretation of these factors in isolation may lead to misdiagnosis or inappropriate management choices. Clinical presentation strongly indicative but non-specific; imaging severity may be poorly correlated with lameness (and eventual outcome) in many cases.
- Diagnostic blocking: lameness improves in most cases to intra-articular blocking of fetlock joint/s (low 4- or 6-point nerve block may be required for complete abolition but non-specificity means they should be interpreted with caution if used in isolation). Hindlimb cases may improve/switch following lateral plantar metatarsal nerve block.
- Radiography: relatively insensitive modality for detection/assessment of condition but useful for initial screening to exclude other pathology (in particular condylar fracture). Findings (if present) include increased/irregular radiodensity of condyle(s) (flexed DPa/DPl projection) (Figure 6.59a,b) and rarely, focal mid-condylar (Figure 6.60a,b) or condylar/parasagittal (Figure 6.61) lucency. Subchondral fracture (preceding subchondral bone collapse) manifested as a crescentic linear lucency in the affected condyle (Figure 6.62, and corresponding to Figure 6.58c), or the resulting disruption to articular outline (flexed DPa/DPl and elevated oblique projections) found in some chronic cases (Figure 6.63a–c) are very rarely encountered but pathognomonic for the condition. The latter are not always associated with lameness and can represent a settled/static state of the condition. Osteoarthritic change is not typically detected radiographically; however, some secondary changes (osteophytes on base/apex of PSBs; 'flattening' of the outline of the palmar/plantar condyles) (Figure 6.64) may be associated with more advanced lesions.
- Bone scan: characteristic pattern of IRU through affected condyles (Figure 6.65a–c), although correlation

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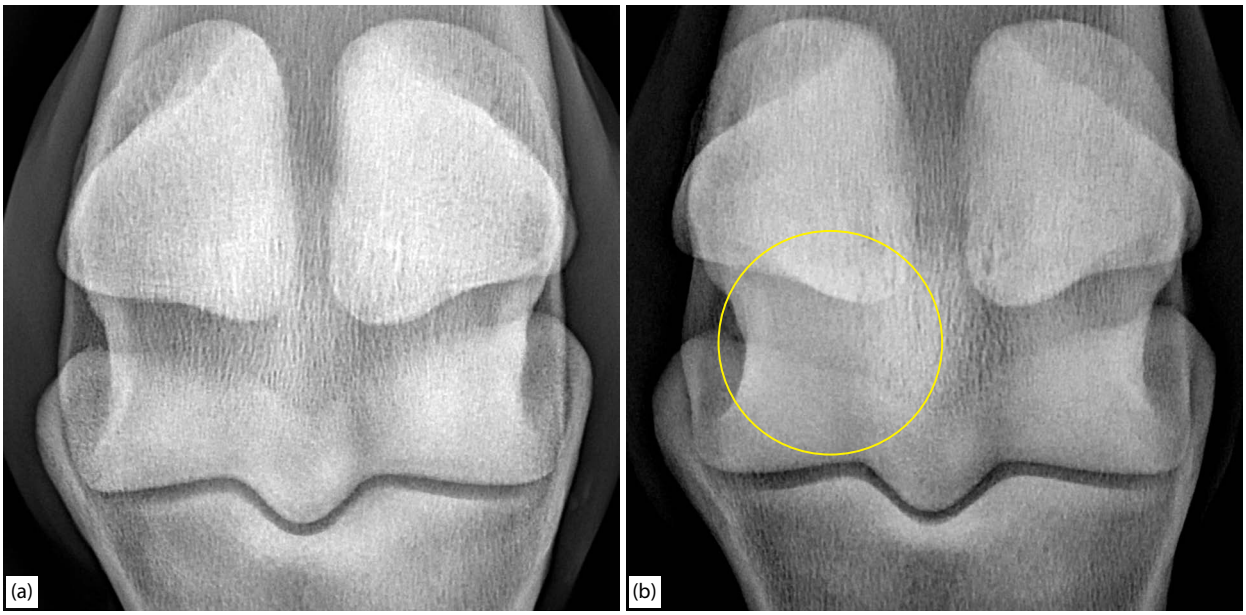


Fig. 6.59 Hindlimb fetlocks with (a) no radiologically evident condylar densification; trabeculation is visible in all regions of distal cannon where there is no superimposition of other bones. (b) increased radiodensity of lateral condylar region (circled), with loss of visible trabecular pattern. Latter finding is common in trained racehorses and may represent adaptive change +/– POD 'pathology' (flexed DPl).

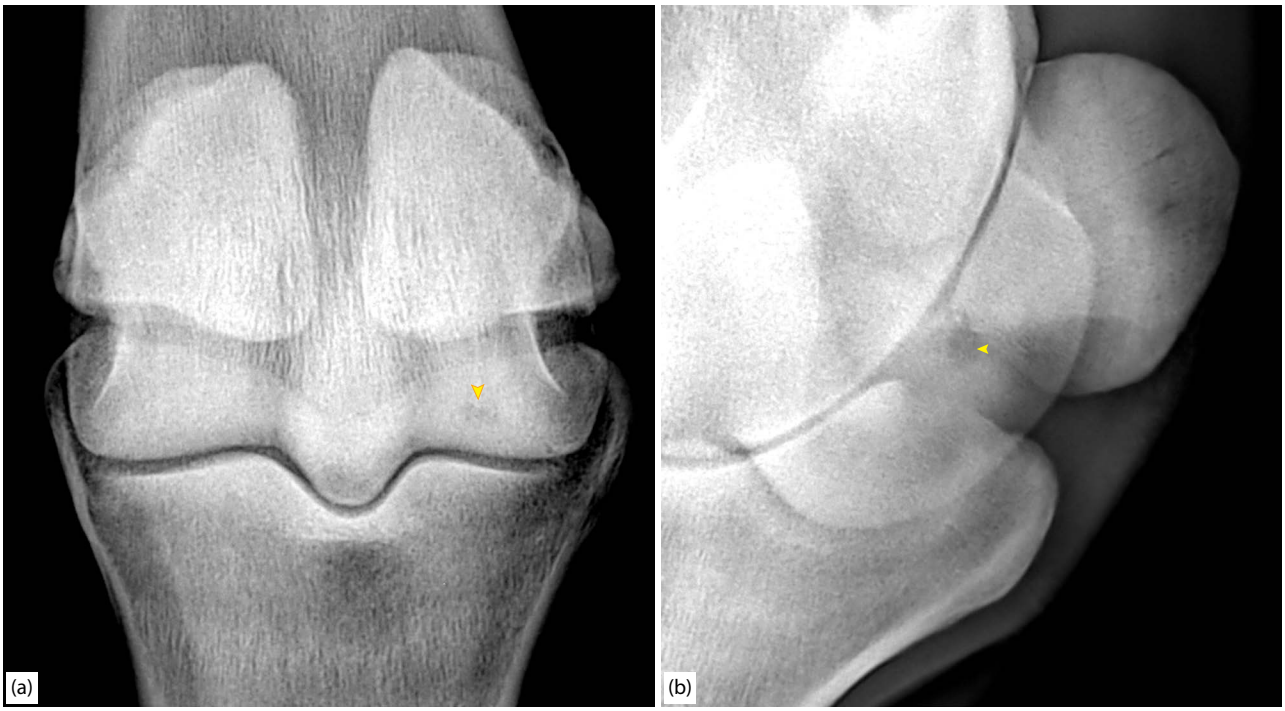


Fig. 6.60 Atypical feature of plantar osteochondral disease of fetlock. Focal radiolucency (arrowhead) in lateral metatarsal condyle on flexed DPl (a) and D45^{0}PrL-PlDiO (b) radiographic projections. Presence of this finding is not always clinically important (may be an incidental finding).

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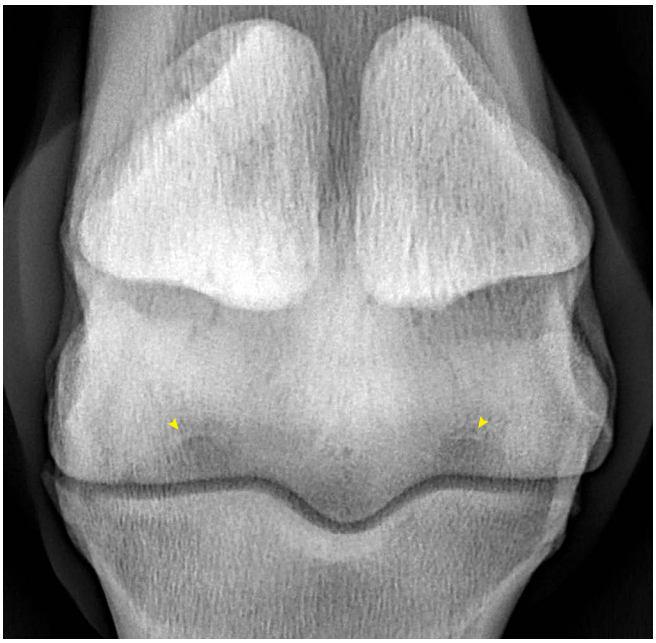


Fig. 6.61 Advanced palmar osteochondral disease of fetlock. Marked radiological findings (flexed DPa): Severe lysis/collapse of subchondral bone in both metacarpal condyles (arrowheads), increased radiodensity of distal cannon, increased conspicuity of metacarpal vessels, modelling of articular margins.



Fig. 6.62 Palmar osteochondral disease of fetlock: Transverse subchondral bone fracture that developed subsequent to this horse being removed from active training. Corresponds to pathology illustrated in Figure 6.58c.

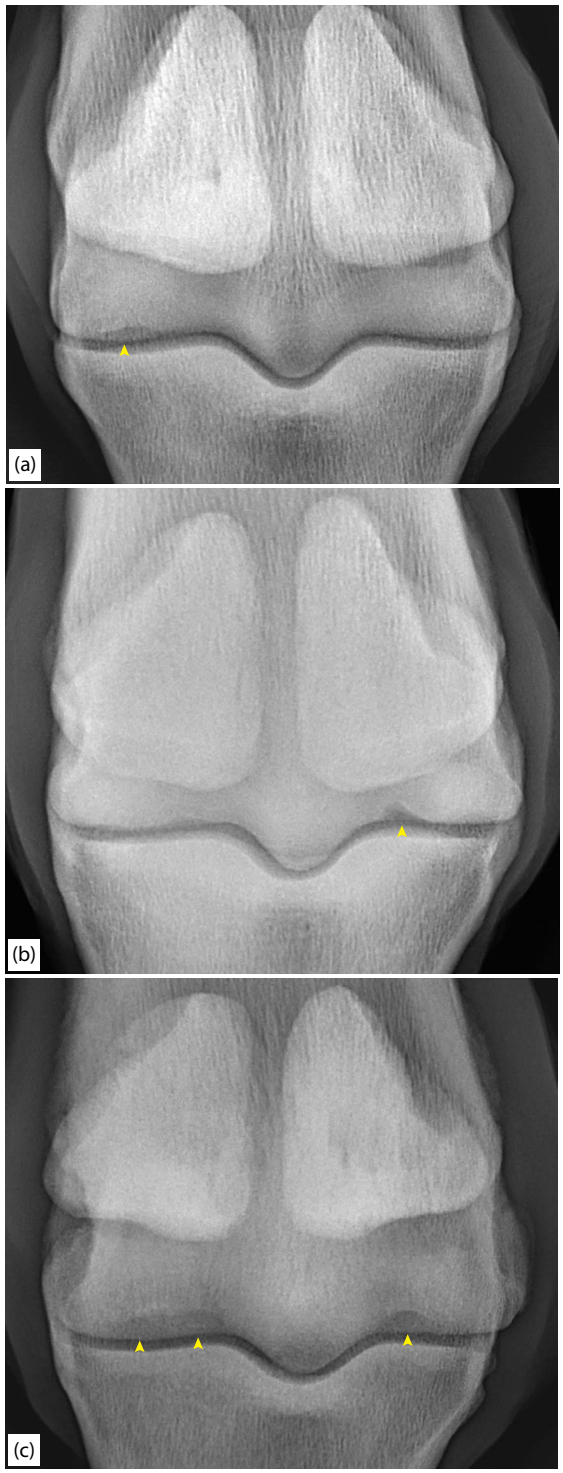


Fig. 6.63 (a–c) Palmar osteochondral disease of fetlock: Altered articular margin(s) (arrowheads) of affected condyles secondary to osteolysis +/or subchondral bone collapse is an atypical finding. May be encountered across spectrum of presentations from clinically silent/unimportant (presumed long-standing/settled) to markedly lame/'untrainable' (flexed DPa).

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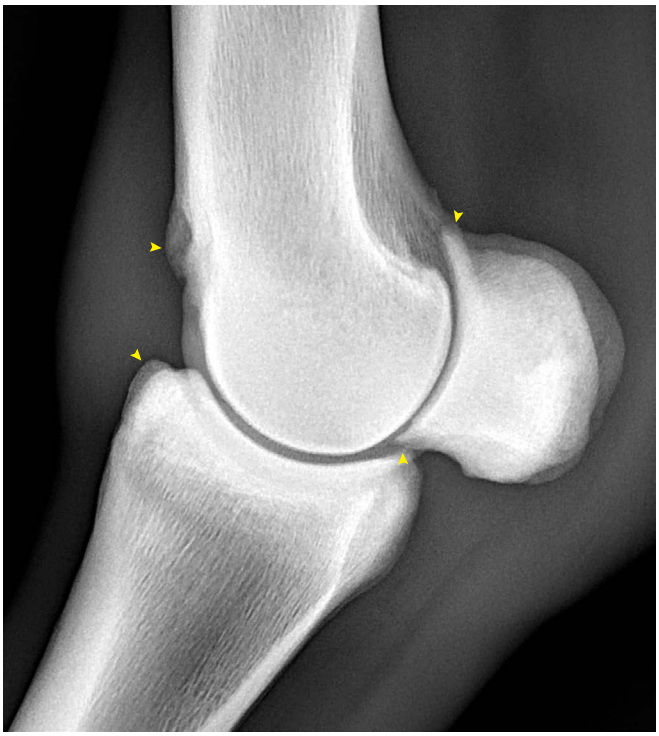


Fig. 6.64 **Secondary osteoarthritic change (modelling of articular margins – arrowheads) is a non-specific finding seen in some cases of advanced palmar/plantar osteochondral disease (LM).**

with lameness/stage of pathology is weak and does not reliably permit differentiation from other fetlock pathologies (condylar fracture, PSB subchondral injury). IRU usually greatest in lamest limb. Primarily useful to rule out injuries at non-fetlock sites and is a poor prognostic (and diagnostic) aid unless used in combination with other diagnostic modalities.

POD OR CONDYLAR FISSURE?

- Multiple subchondral pathologies may co-exist in fetlocks (Figure 6.67).
- Presence, location and severity of these pathologies determine relative contribution to risk of future serious injury.
- POD and condylar fracture/fissure lesions in lower cannon most commonly found at predilection sites that are in close proximity but anatomically distinct.
- While radiography has limitations, in the great majority of first opinion cases it is usually possible to distinguish between these two possible pathologies using good technique and clinical judgement.
- Small proportion of cases may require advanced imaging (MRI/CT/PET) for further definition (also to assess sesamoid activity).
- Although condylar fractures may propagate through existing POD lesions, and POD lesions may exist in/near predilection site (parasagittal groove) for condylar fractures, these are rare occurrences and best considered as atypical events.

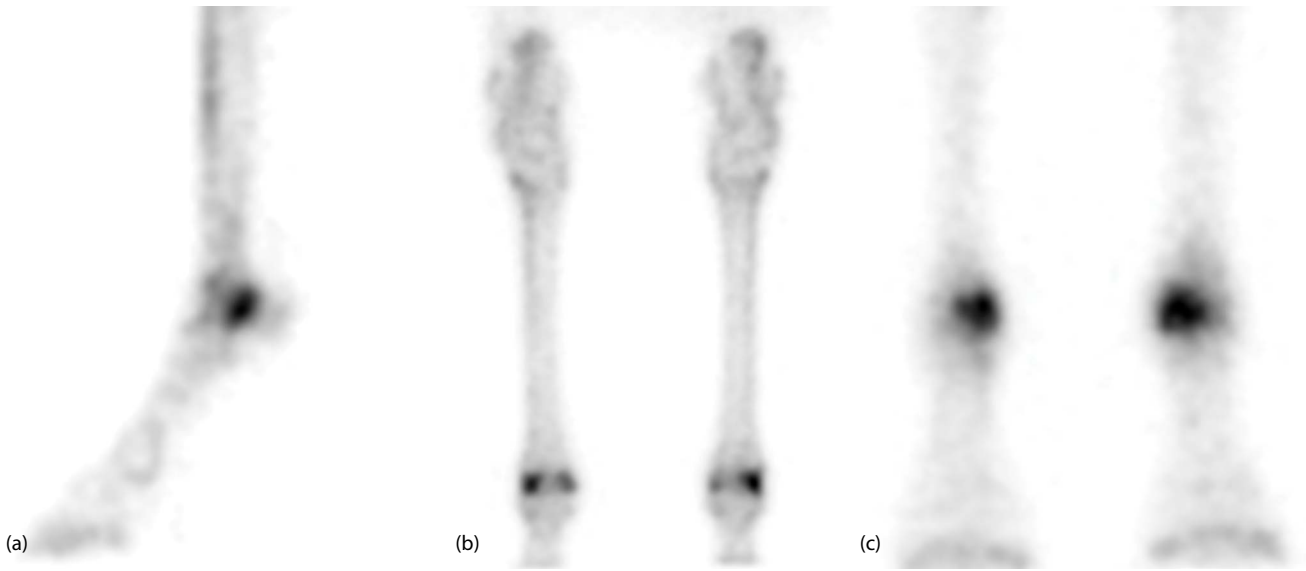


Fig. 6.65 **Scintigrams showing typical IRU associated with POD: (a) focal palmarodistal IRU (lateral view); (b) focal plantarolateral IRU (caudal view of hindlimbs) that requires differentiation from PSBs; (c) intense distomedial IRU affecting front fetlocks (dorsal view).**

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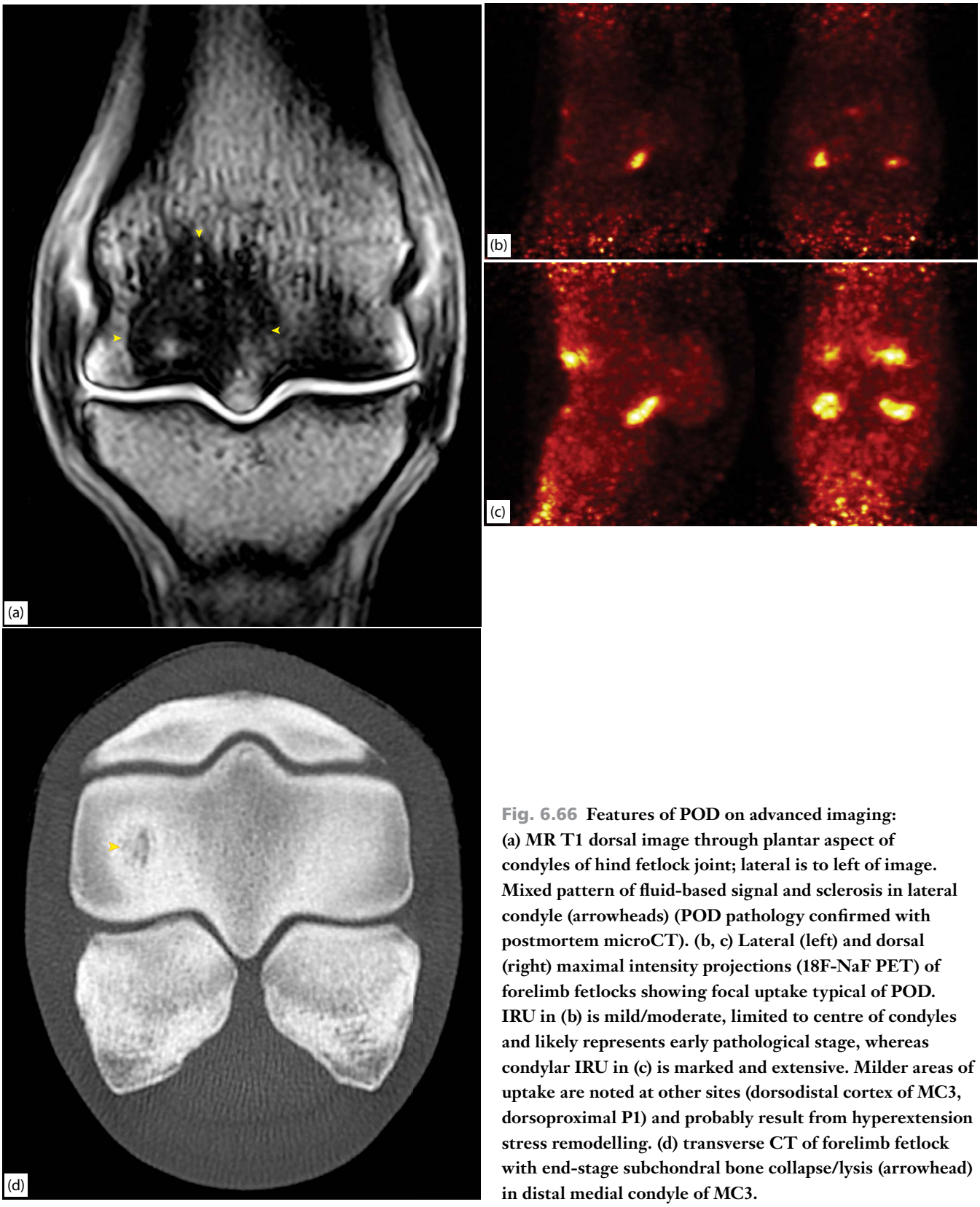


Fig. 6.66 **Features of POD on advanced imaging: (a) MR T1 dorsal image through plantar aspect of condyles of hind fetlock joint; lateral is to left of image. Mixed pattern of fluid-based signal and sclerosis in lateral condyle (arrowheads) (POD pathology confirmed with postmortem microCT). (b, c) Lateral (left) and dorsal (right) maximal intensity projections (18F-NaF PET) of forelimb fetlocks showing focal uptake typical of POD. IRU in (b) is mild/moderate, limited to centre of condyles and likely represents early pathological stage, whereas condylar IRU in (c) is marked and extensive. Milder areas of uptake are noted at other sites (dorsodistal cortex of MC3, dorsoproximal P1) and probably result from hyperextension stress remodelling. (d) transverse CT of forelimb fetlock with end-stage subchondral bone collapse/lysis (arrowhead) in distal medial condyle of MC3.**

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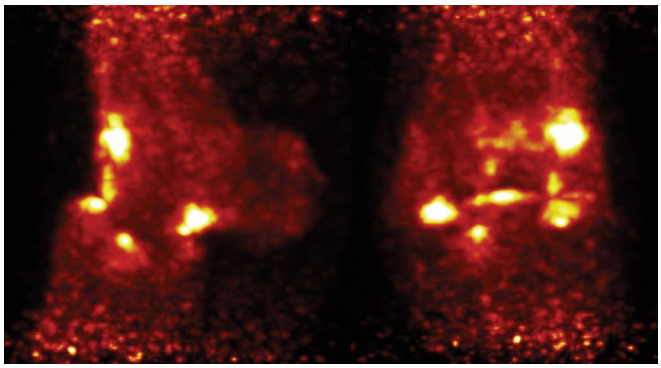


Fig. 6.67 Lateral (left) and dorsal (right) maximal intensity projections (18F-NaF PET) of left fore fetlock (medial is to the left in dorsal projection) showing IRU at multiple sites. The palmar metacarpal uptake is moderate/severe, worse medially and represents POD. There is also activity at sites of hyperextension remodelling (dorsolateral distal MC3, dorsomedial articular margin of proximal P1) as well as subchondral bone of dorsodistal MC3 and mid-proximal P1.

- MRI/PET/CT: diagnostic modalities of choice to stage lesion (Figure 6.66a–d) and differentiate from prodromal condylar fracture (p. 109).
- POD lesions are separate and distinct from condylar fractures; however, concurrent pathology at the same location on the condyle is seen very occasionally in the European Thoroughbred.

Management

- Wide range of management approaches may be applied to horses diagnosed with POD, from continued training (without treatment) to retirement.
- Management should be tailored to the individual: the same lesion that in one horse proves career-ending might in the next horse not result in any lost training at all. Prescribing rest/rehabilitation based purely on imaging and without considering individual circumstances is associated with unnecessary career wastage. Important to recognise that most racehorses (including elite performers) carry some degree of POD pathology throughout their careers and that veterinary intervention is sought in only small subset of affected horses.
- Most cases are safely managed empirically without advanced diagnostic imaging (MRI/PET/CT); however, initial risk assessment for other pathology (prodromal condylar fracture) is useful/important if continued training is desired: necessity determined by history/clinical findings such as rapid deterioration of action.
- In general, it is prudent to manage cases of acute-onset or rapidly deteriorating unilateral lameness (that arise in full training) more conservatively than slowly progressive bilateral lameness. Reduction in training intensity (to light ridden exercise) may be preferable to removal from training altogether; recommendations for time period out of cantering/fast work determined by individual clinical severity and career goals.
- Mature horses displaying unilateral/bilateral lameness early in a training cycle (e.g., following a spell for unrelated reason): continuation in exercise programme (without medical intervention) + regular clinical re-evaluation is generally recommended (rather than further rest), as lameness diminishes over time in many cases. If persistence/deterioration of lameness: diagnostic imaging warranted.
- Treatment options are limited; intra-articular medication (Table 5.7 p. 71) may be used to manage lameness but is not disease-modifying or curative: many horses respond favourably; however, quality and duration of effect varies widely, likely due to wide spectrum of disease, wide variation in tolerance of individual animal and many external variables.
- Currently no scientific support for therapeutic usefulness of any nutritional supplementation.
- Modification of training regime (reduced work on uphill or synthetic tracks), use of treadmill/swimming exercise may be incorporated into management; efficacy undetermined and individual clinical effect likely to be limited.
- As with other conditions that frustrate through inconsistent outcome, a culture of interventions based on plausible/speculative effects has developed around POD. Includes aspirin, bisphosphonate medication, systemic chondroprotective medications, extracorporeal shockwave treatment (ESWT), remedial shoeing; however, little/no current scientific/empirical support for their use.
- Short (<6 months) periods of rest currently considered to be of little benefit and may be contraindicated.
- Prescription of rest requires careful consideration. Short rest periods followed by rapid resumption of training are associated with significant risk of serious deterioration of joint integrity: removal from training can initiate resorptive phase of bone repair leading to weakening/collapse (insufficiency fracture) of subchondral bone plate (Figure 6.62). If horse is removed from active training, stable rest

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should be avoided and careful/slow resumption of loading highly recommended. Effective rehabilitation of advanced subchondral pathology is an uncertain outcome regardless of length of time out of training.

Prognosis

- The factors involved in progression (or not) of POD pathology are poorly understood, including whether there is a threshold beyond which repair of lesions does not/cannot occur.
- Even with the benefit of advanced imaging to stage lesions more precisely, prognosticating for the individual is difficult for reasons explained earlier.
- Unpredictable condition: most horses remain static or display slowly deteriorating signs over one or more seasons; however, sustained clinical improvement may also occur with continued training in many horses. Presumptions about likely structural behaviour of 'overtrained' bone do not always concur with clinical progression.
- Progression to severe end stage pathology (subchondral bone fracture/collapse) (Figure 6.58c) with continued training is very rare: cannot be effectively predicted, and risk of this outcome should not influence management advice for majority of cases.
- Continued training in medium to long term usually possible.
- Is not statistically associated with increased risk of developing condylar fracture, although horses with advanced POD + other pathologies arising from chronic fetlock overextension should be subjected to more intense ongoing risk assessment whilst they remain in full training.
- Horses with marked pathology may have careers shortened or troubled by lameness; however, this is not universal and many horses (including elite performers) race satisfactorily for several years after diagnosis. Scientific evidence of association between POD and poor performance is weak/conflicting. Studies in which lesions have been reliably graded are lacking. Management advice should be tailored to individual circumstances.

Synovitis

Primary synovitis (without overt underlying orthopaedic injury) is a sporadic finding and may affect one or more fetlocks. Most common in the forelimb. Joint effusion is of variable severity and usually self-limiting, although some remain palpably abnormal throughout future training.

Cause

- Undetermined in most cases.
- Acute or repetitive overextension/overloading may cause inflammation of joint capsule or support structures.

Risk factors

- Common condition.
- Any age/stage of training but most commonly in 2 YOs in cantering exercise.
- Risk factors poorly understood but possible influence of conformation/BWT/maturity relative to workload.

History

- Acute or chronic (days/weeks) onset of joint filling.

Signs

- Distension of dorsal and palmar joint pouches (Figure 6.68) +/– periarticular oedema in acute phase.
- Variable response to forced flexion of joint: painful in some cases.
- +/– pain on deep palpation of joint capsule margins: may require care to differentiate from SLB desmopathy (p. 146).
- Lameness is rare, and if present is usually subtle-mild.
- Rarely, lameness of moderate severity and marked joint effusion.



Fig. 6.68 Fetlock joint effusion, with prominence of dorsal pouch.

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Diagnosis
- Clinical findings usually sufficient.
- Imaging rarely required but warranted if lame (to rule out orthopaedic injury), or if synovitis severe or non-responsive to anti-inflammatory measures.
- Ultrasonography/radiography/MRI as dictated by clinical requirement/perceived fracture risk. Ultrasonography is the modality of choice for detection of dorsal osteochondral fragmentation of P1 (p. 129).

Management
- Topical anti-inflammatory therapy: osmotic dressing/cold therapy.
- If no lameness: continued training possible, although short period of reduced exercise (walk/trot) during acute phase may be beneficial.
- If poor response to initial anti-inflammatory measures (and if orthopaedic injury not suspected): intra-articular medication +/– continuation of training.

Prognosis
- Primary fetlock synovitis rarely causes significant interruption to training.
- Articular fragmentation may occur in small proportion of cases but prognosis for continued training is good.

Dorsal osteochondral fragmentation ('chip' fracture)
Osteochondral fragments or 'chips' in the fetlock typically arise from the dorsomedial or dorsolateral aspects of the joint margin of P1. They vary in size, are usually solitary and may be displaced/non-displaced. Typically associated with other overextension pathology (soft tissue +/– subchondral bone) in joint, such as impingement of dorsal synovial pad and fibrocartilage, and tearing of joint capsular attachments. Predominantly in forelimb; unilateral or bilateral.

Cause
- Results from acute or repetitive overextension of fetlock and dorsal impact of P1 on lower cannon.
- Frequently part of an overloading syndrome that may include palmar (p. 119) or dorsal (p. 132) osteochondral disease/chronic proliferative synovitis (p. 131)/suspensory ligament desmopathy (p. 146).

Risk factors
- May occur at any age.
- Usually during cantering/fast work phase of training.
- Risk factors poorly understood: possible intrinsic factors such as conformation/BWT, and extrinsic factors such as track surface and condition.

History
- Frequently a clinically silent incidental finding at routine imaging.
- May present with acute joint effusion +/– lameness.

Signs
- Acute phase: transient joint effusion (synovitis) +/– pain response to joint flexion +/– mild-moderate lameness; subsides over days/weeks.
- Fragmentation can also occur with little/no evidence of joint inflammation.

Diagnosis
- Clinical findings alone do not permit differentiation from primary synovitis (p. 128) but relatively mild lameness distinguishes from proximal phalangeal fracture (p. 99).
- Ultrasonography: excellent sensitivity and specificity for detection of fragments; diagnostic modality of choice.
- Radiography: modelling of dorsal articular margin +/– displaced/non-displaced fragment(s) (DLPaMO, LM and DPa projections most useful) (Figure 6.69a–f). Has traditionally been imaging modality used for diagnostic (or screening/pre-purchase) detection of fragmentation; however, sensitivity is inferior to ultrasonography.

Management
- Different management options available; dictated by clinical severity and training objectives. Radiological severity should not be prescriptive, as size/number/location of fragment(s) is not associated with subsequent clinical or racing outcomes. Overly simplistic to base management decisions on presence/absence of chip as correlation with specific pathology affecting joint may be poor.
- Conservative (non-surgical) management: period of reduced exercise (walking/trotting for 1–4 weeks) in acute phase +/– systemic/topical/intra-articular anti-inflammatory medication to control synovitis.
- When rest period undesirable: topical and/or intra-articular anti-inflammatory therapy may permit continuation of training. Response should guide management; training in face of lameness/synovitis may lead to long-term osteoarthritic change.

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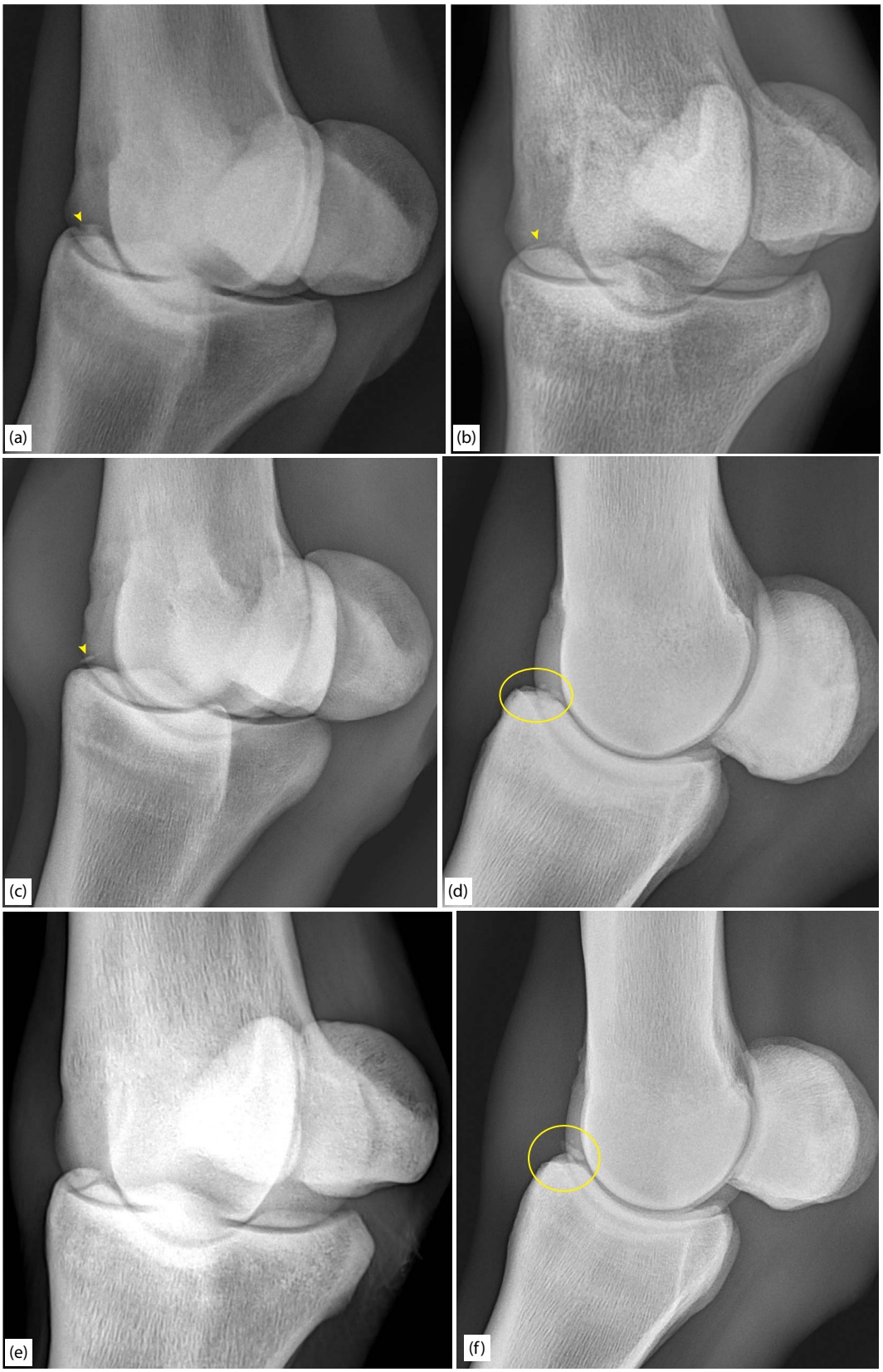


Fig. 6.69 **(a–f) Radiographs of fetlock dorsal osteochondral chip fracture/fragmentation. Variation is seen in size and degree of displacement of fragment(s) (arrowheads), and degree of modelling of dorsoproximal articular margin of P1 (circled).**

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- Conservative management is not statistically associated with any increased risk of long-term lameness or osteoarthritis (OA), and is not associated with significant negative effects on career performance parameters. Empirically appears to be the treatment of choice for most cases.
- Surgical removal has traditionally been considered the treatment of choice, but scientific basis for this is weak and does not account for concurrent articular pathologies (Figure 6.70a,b). However, may still be recommended for some large chips or for cases that respond poorly to rest/medication. Associated with longer return to racetrack times than conservative management.
- Long-standing fragments are generally not clinically active but may be markers of other overextension pathology in fetlock region.

Prognosis

- Excellent: generally results in little interruption to training and no deleterious effect on long-term soundness.
- Surgical removal also carries excellent prognosis for return to racing but generally not necessary. Mean time to return to full training following surgery approximately 12 weeks.
- Small proportion of cases with marked initial synovitis/lameness require greater care in handling and longer rehabilitation periods, but prognosis for return to full soundness remains good.

Chronic proliferative ('villonodular') synovitis

Chronic inflammatory condition of dorsal soft tissues of forelimb fetlock joint. Synovial pad within dorsal pouch of fetlock normally protects front of lower cannon but may become inflamed/thickened with repetitive trauma from overextension. Most commonly unilateral but may be bilateral.

Cause

- Repetitive overextension of fetlock causes 'pinching' of synovial pad by top of pastern.
- Frequently part of an overloading syndrome that may include dorsal osteochondral fragmentation (p. 129)/ palmar (p. 119) or dorsal (p. 132) osteochondral disease/suspensory ligament desmopathy (p. 146).

Risk factors

- Most commonly in horses ≥3 YO.
- Risk factors poorly understood.

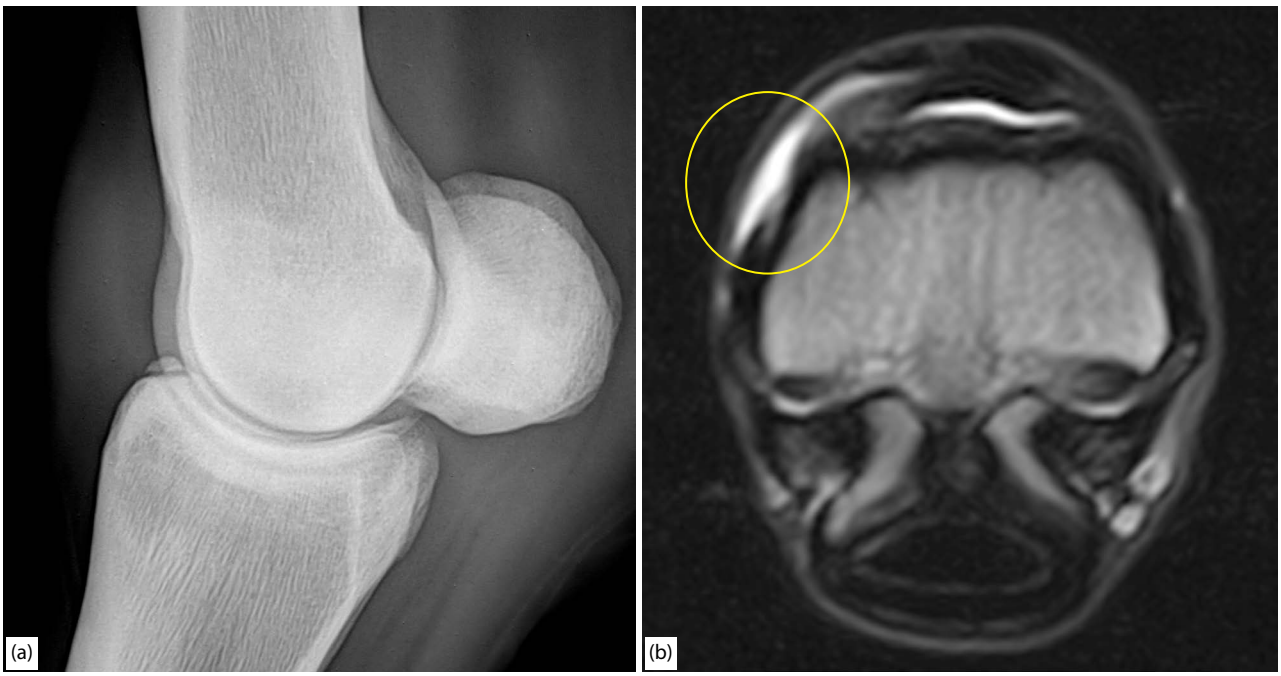


Fig. 6.70 Case of acute forelimb fetlock synovitis in which dorsal chip fracture of P1 was identified on radiographs (a); concurrent low-field MRI (b) however confirmed primary injury site to be remote to this (tear to dorsolateral joint capsule-increased signal, circled) with chip fracture site being relatively 'silent'.

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History
- Chronic condition with insidious onset and progression; develops over long period (months/years).
- Typically exacerbated by fast exercise.

Signs
- Visual prominence of dorsal pouch of front fetlock joint(s).
- +/– joint effusion.
- Lameness an inconsistent feature and if present is typically mild.
- Flexion of joint resented.

Diagnosis
- Clinical findings usually definitive.
- Radiography: typically associated with radiological feature known as 'supracondylar lysis' ('cutting back'/relative concavity of lower dorsal cannon just above the condyles on LM projection). Frequently with bony proliferation immediately above the depression, at proximal attachment of joint capsule (Figure 6.71). (Note that 'supracondylar lysis' is poorly defined and may be encountered in horses with no current/previous fetlock pathology).
- Ultrasonography: employed infrequently. Mean thickness of synovial pad in affected joints 9–11 mm.

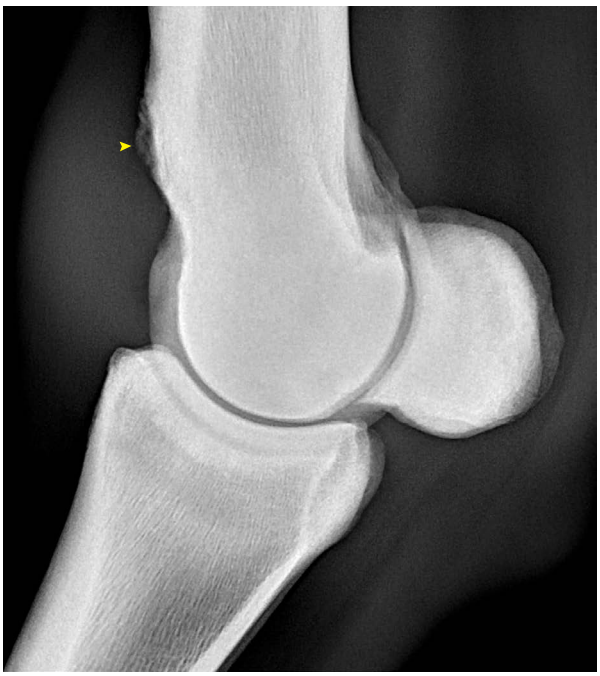


Fig. 6.71 Radiological (LM) features typically associated with chronic proliferative synovitis: periosteal proliferative change at dorsodistal MC3.

Management
- Intervention/rest not always necessary, but usually some treatment indicated by the time the condition is clinically obvious.
- Intra-articular medication +/– cold therapy may alleviate inflammation sufficiently to permit continued training.
- Surgical excision is treatment of choice for cases that do not respond favourably to medical management but rarely required/undertaken.

Prognosis
- Although medical/conservative management is symptomatic and not curative, many cases are manageable and continue to train/race with little interruption.
- Surgery: good prognosis for return to racing (approximately 80%). Recurrence rare.

Dorsal osteochondral disease (MC3/MT3)

Overextension injury of fetlock joint that can cause unilateral lameness; affects dorsal aspect of lower cannon bone. Predominantly an injury of the forelimb. Spectrum of pathology from increased density (osteosclerosis) of bone at dorsal sagittal ridge (most common location) or metacarpal condyles, to regions of subchondral bone collapse and associated cartilage damage. Osteochondral damage involves the extra-articular fibrocartilage of the dorsal recess of the joint +/– dorsal synovial pad. Often in association with adaptive change at dorsoproximal margin of P1.

Cause
- Presumed to result from repetitive overextension of fetlock and dorsal impact of P1 on lower cannon.
- May be seen in association with other fetlock overload/overextension pathologies including dorsal osteochondral fragmentation/POD/chronic proliferative synovitis.

Risk factors
- Uncommon condition.
- All ages/stages of training but typically during cantering/fast exercise phase.
- Risk factors poorly understood.

History
- Chronic- or acute-onset forelimb lameness.
- Temporary improvement with rest but recurs on return to cantering.

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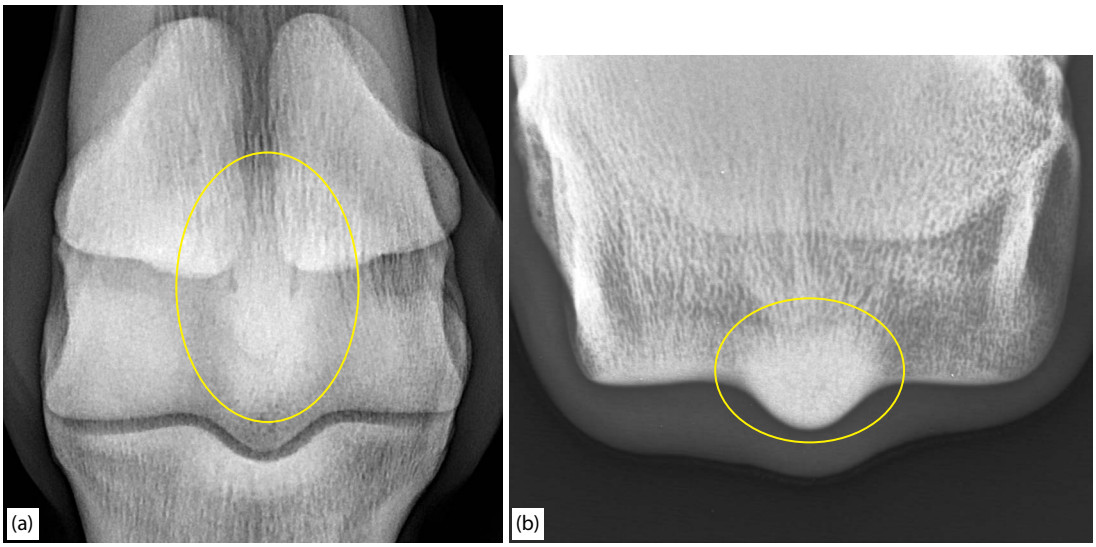


Fig. 6.72 **Dorsal osteochondral disease of fetlock: Radiological features are not specific/definitive but may include (a) increased radiodensity in mid-sagittal region of distal MC3 (circled) +/– increased conspicuity of metacarpal vessels (flexed DPa), and (b) increased radiodensity (circled) of dorsal sagittal ridge of MC3 ('skyline' DPr-DDiO).**

Signs

- Unilateral lameness of mild-moderate severity.
- Unlike most cases of palmar/plantar osteochondral disease (p. 119), joint effusion or thickening of soft tissue structures on dorsal aspect of joint may be a feature.

Diagnosis

- Clinical findings non-specific.
- Diagnostic blocking: lameness improves in most cases to intra-articular blocking of fetlock joint; low 4-point nerve block may be required for complete abolition of lameness.
- Radiography: pattern of increased/irregular density through dorsal sagittal ridge region (Figure 6.72a,b) may be a non-specific feature; however, radiography is a non-definitive diagnostic modality for this condition and utilised primarily to rule out other pathologies.
- Bone scan/PET: IRU in affected distal/dorsodistal cannon (Figure 6.73a,b).
- Ultrasonography: most sensitive modality to detect cartilaginous damage at dorsal aspect of joint.
- MRI: diagnostic modality of choice (Figure 6.74) for subchondral/cancellous bone injury in this region.

Management

- Clinical/imaging severity determines necessity for rest period.
- Continued training aided by intra-articular medication possible; however, quality and duration of response varies (often poor).

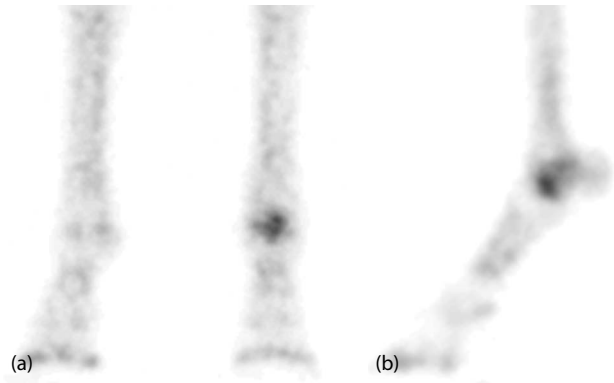


Fig. 6.73 **Dorsal osteochondral disease of fetlock: Dorsal (a) and lateral (b) scintigrams with focal IRU in dorsodistal MC3 of left forelimb.**

- Modification of training regime (treadmill/swimming) may be beneficial.
- Stable rest/walking for 6–10 weeks +/– intra-articular medication often sufficient for resolution.

Prognosis

- Good prognosis for continued training.
- Progression to stress fracture does not appear to be a feature of the condition (safe to continue in full training).

Transverse fracture

Non-articular fracture of the lower cannon (third metacarpus/tarsus, MC3/MT3); almost invariably involves the

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Fig. 6.74 Dorsal osteochondral disease of fetlock: Fluid signal (likely representing bone inflammation/haemorrhage secondary to trabecular microfractures) is biased towards the dorsal aspect of the joint in this case (arrowheads); the palmar aspect is relatively spared (MR T1 sagittal image).

forelimb but can (rarely) affect hindlimb. Fracture occurs in a transverse or obliquely transverse plane in the distal metacarpal/tarsal diaphysis; predilection site is above the growth plate. Injury may predominantly involve the palmar/plantar (most common) or dorsal cortices, or be circumferential. Most commonly unilateral but bilateral injuries also occur. Severity of injury (stress/incomplete/complete) at time of initial diagnosis varies.

Cause

- Underlying mechanism unknown but currently considered to be a stress or insufficiency injury arising from dorsopalmar bending forces on the cannon.

Risk factors

- Rare condition.
- Risk factors poorly understood.
- Unraced horses appear at greater risk.
- Generally encountered in horses in trotting/early cantering phase of training, or returning to training after a period of rest.

History

- Acute- or chronic-onset lameness.
- +/– thickening around/above affected fetlock in days prior to detection of injury.
- Occasionally encountered as catastrophic complete fracture sustained during fast exercise/racing.

Signs

- Initial lameness variable: mild-marked.
- +/– fetlock joint effusion.
- Palpable abnormalities (if present) include soft-tissue/bony thickening on palmaromedial/lateral aspect of lower cannon +/– pain response to firm palpation.

Diagnosis

- Radiography: some cases display radiological abnormalities at initial examination (LM and oblique projections most useful) but many require repeat radiography.
- Periosteal callus on palmaromedial/lateral or dorsomedial/lateral cannon (above level of physis); +/– palmar endocortical thickening (Figure 6.75a,b).
- Periosteal +/or endosteal callus (sometimes profound) +/– horizontal fracture line (or radiopacity) may develop 7–14+ days after injury (Figure 6.76a–c).
- MRI: rarely required but may assist early diagnosis if delayed radiographic examination (to assess callus formation) is undesirable.

Management

- Risk of progression to complete fracture determined from initial severity of lameness/imaging findings.
- Initial radiographic screening unremarkable: stable confinement (+/– immobilizing bandage depending on severity) and repeat radiography at 7–14+ days.
- Mild initial clinical and radiological signs: conservative management; stable rest/walking for 8–12 weeks.
- Moderate-marked initial lameness: stable rest with immobilizing bandage for initial 3–4 weeks; rehabilitation guided by radiographic monitoring.
- Surgical fixation considered for injuries at risk of catastrophic deterioration (rare).

Prognosis

- Stress or incomplete fracture: good prognosis for return to full use.
- Severe initial lameness: risk of catastrophic failure with conservative or inappropriate management.

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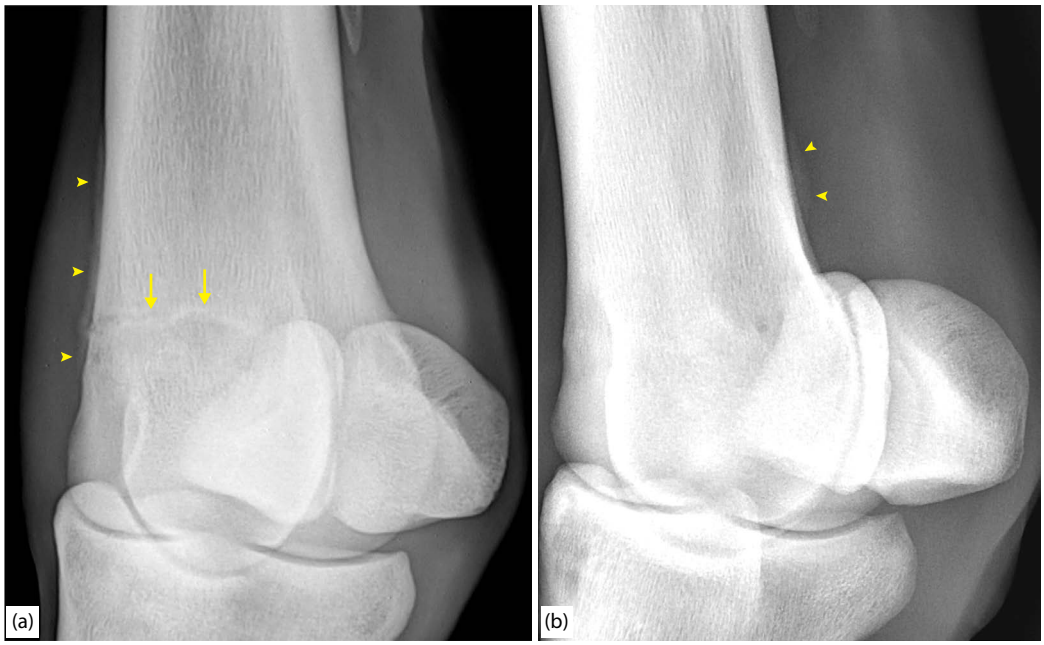


Fig. 6.75 **Transverse metacarpal fracture: Radiographs (DMPaLO) demonstrating (a) horizontal linear radiopacity (arrows) with early periosteal proliferative change (arrowheads) extending along the dorsal aspect of the cannon; (b) subtle periosteal proliferative change (arrowheads) at palmarolateral distal MC3; latter finding can be readily missed with inappropriate exposures but becomes more pronounced as callus matures.**

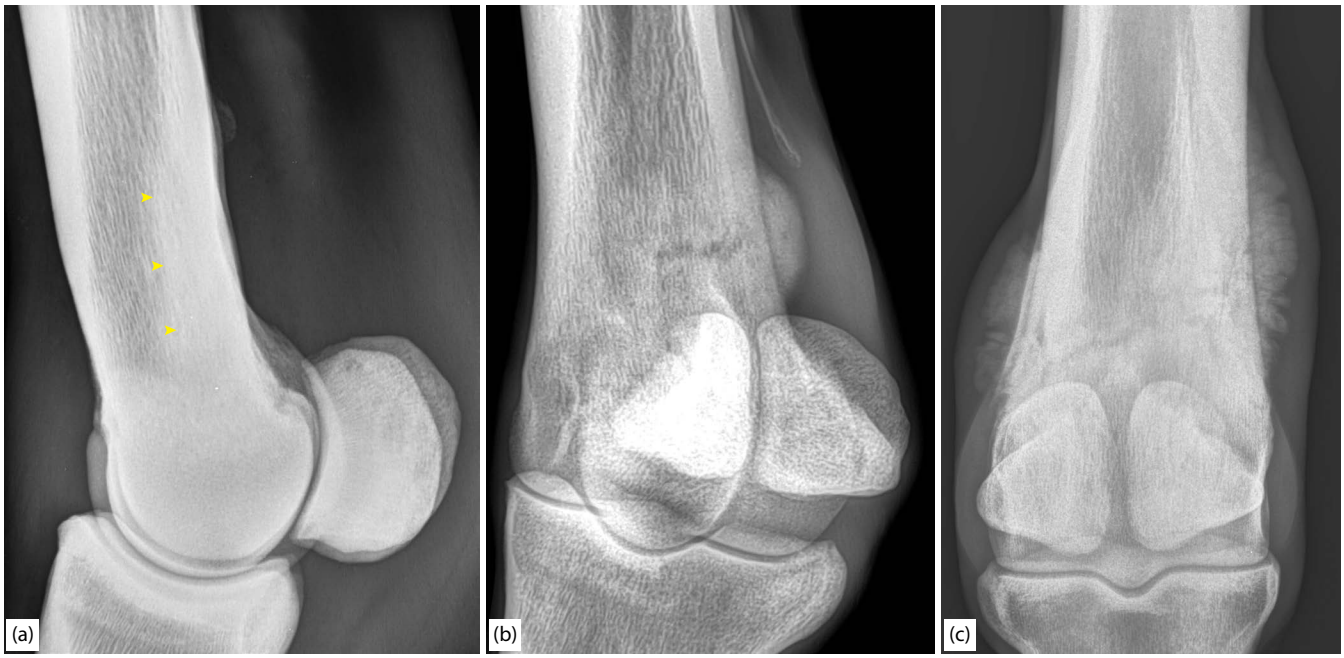


Fig. 6.76 **Transverse metacarpal fracture: Marked radiological features that may manifest at delayed repeat examination. (a) endosteal callus/thickening in palmarodistal MC3 (arrowheads); marked focal (b) and extensive (c) periosteal proliferation +/– lucent fracture line.**

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Osteochondrosis: osteochondritis dissecans (OCD)

Developmental defect of cartilage/subchondral bone. The fetlock is frequently the site of osteochondritis dissecans (OCD) lesions in the horse; these most commonly involve the middle or dorsal third of the sagittal ridge (any limb) and are characterized by flattening or osteochondral fragmentation. Severity varies. Less common are lesions involving the distal sagittal ridge of the forelimb, and fragments of various size at the back of the joint in association with the medial (typically) or lateral palmar/plantar process (Figure 6.77) The latter may also occur in any limb and are not always distinguishable from ununited palmar/plantar processes or overextension injuries sustained early in life (Figure 6.78). Regardless of origin most of these lesions are clinically insignificant. Occasionally, significance is attributed to axial (articular) plantar process fragments based on appearance and clinical presentation. Rarely, clinically important subchondral bone lesions can develop at the dorsal proximal sagittal ridge OCD predilection site of a forelimb fetlock. Unilateral or bilateral.

Cause

- See Osteochondrosis (Overview) (p. 267).

Risk factors

- Sagittal ridge lesions common: prevalence in sales yearlings approximately 20–40%.



Fig. 6.77 **Plantar process fragment (arrowhead) of hindlimb fetlock with corresponding lucency at parent site on proximal P1.**



Fig. 6.78 **Juvenile plantar process avulsion/traumatic injury with comminution (in this case associated with observed fall and subsequent lameness/swelling).**

- Palmar/plantar process fragments: prevalence in sales yearlings approximately 5–7%.

History

- Typically an incidental finding at routine yearling sales radiography (subclinical at time of detection).
- More significant subchondral bone lesions at the dorsal proximal sagittal ridge site may present with lameness in early training.

Signs

- +/– joint effusion (most common with sagittal ridge OCD).
- Plantar/palmar fragments typically clinically inapparent.
- Lameness is not typical of condition (aside from dorsal subchondral bone lesions).

Diagnosis

- Radiography: sagittal ridge OCD may be detectable on DPa projection (Figure 6.79a) but is best assessed with flexed LM (Figure 6.79b). Dorsal lesions visible on standing LM projection (Figure 6.79c). Palmar/plantar process lesions are detected on oblique projections.

Management

- Very rarely of clinical importance and treatment is generally unnecessary.

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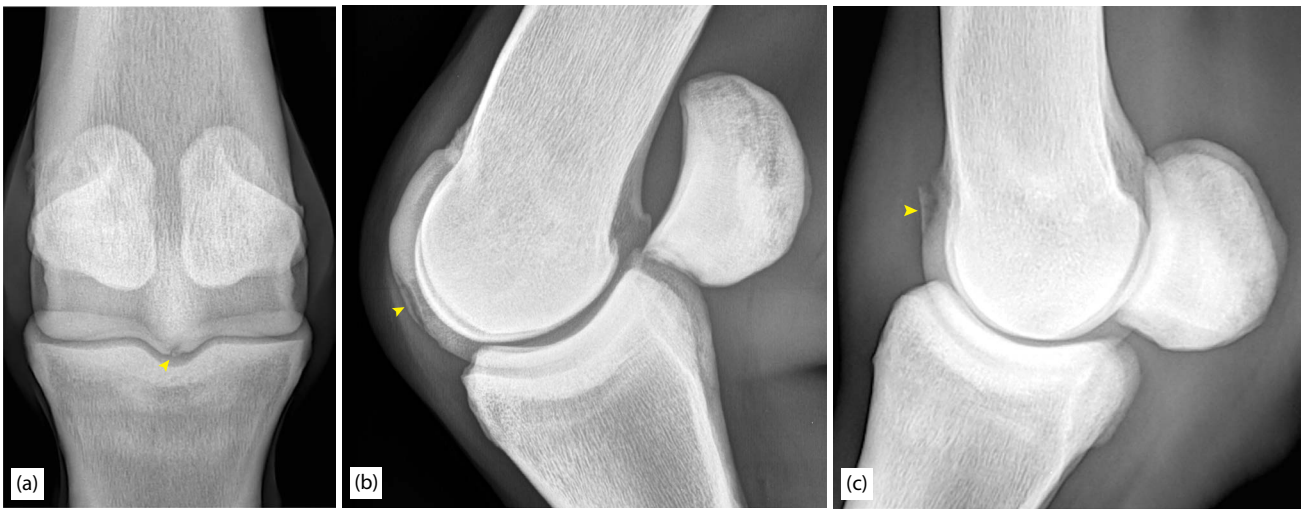


Fig. 6.79 **Fetlock OCD: (a) sagittal ridge radiolucency/irregularity on DPa radiograph (arrowhead); (b) typical osteochondral fragment (arrowhead) of mid-sagittal ridge (flexed LM); (c) dorsal osteochondral fragment (arrowhead) of sagittal ridge (LM).**

- Sagittal ridge lesions evolve/mature from early in life to yearling/2 YO stage and intervention in juveniles is generally discouraged (Figure 6.80a,b).
- Sagittal ridge lesions that are extensive, distal or associated with subchondral cyst-like lesions (Figure 6.81) may cause lameness, and may necessitate rest or medical/surgical intervention.
- If significant joint effusion/inflammation (rare) in early training: modified exercise +/or intra-articular medication.
- Surgical debridement (of sagittal ridge lesions) occasionally warranted in clinical cases (lameness) non-responsive to medical/conservative management (rare). Prognosis for return to racing following surgery in these cases is good.
- Plantar/palmar osteochondral fragments sometimes claimed to affect action at faster paces; however, no evidence base to support this (or of clinical significance in general) and may be presumed to be clinically irrelevant.



Fig. 6.80 **Fetlock sagittal ridge OCD: Evolution in radiological appearance of lesion between (a) foal and (b) yearling stage.**

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Fig. 6.81 **Large sagittal ridge radiolucency; lesions of this size/appearance are frequently clinically significant.**

Prognosis

- Generally excellent; rarely interfere with training.
- Dorsal sagittal ridge subchondral cyst-like lesions: guarded to fair prognosis but may cause interruption to early training.

Osteochondrosis: Subchondral bone cyst

Developmental defect of cartilage/subchondral bone. Subchondral bone cysts are encountered sporadically in association with the fetlock and PIPJ (pastern) joints. Bilateral lesions are seen in about 10% of cases.

Fetlock joint cysts are usually found in the forelimb and typically in the medial condyle of the cannon bone (Figure 6.82a–c), although lesions also occur in proximal P1 (Figure 6.82d) and dorsal sagittal ridge. Lesions are generally solitary, although occasionally more extensive/multifocal lesions affecting several adjacent sites/bones are encountered (Figure 6.83a,b). Cysts involving the PSBs occur (Figure 6.84) but are very rare. In the PIPJ, small midline radiolucencies in the distal articular margin of P1 are common and have generally been considered synovial fossae or normal variants (Figure 6.85a).

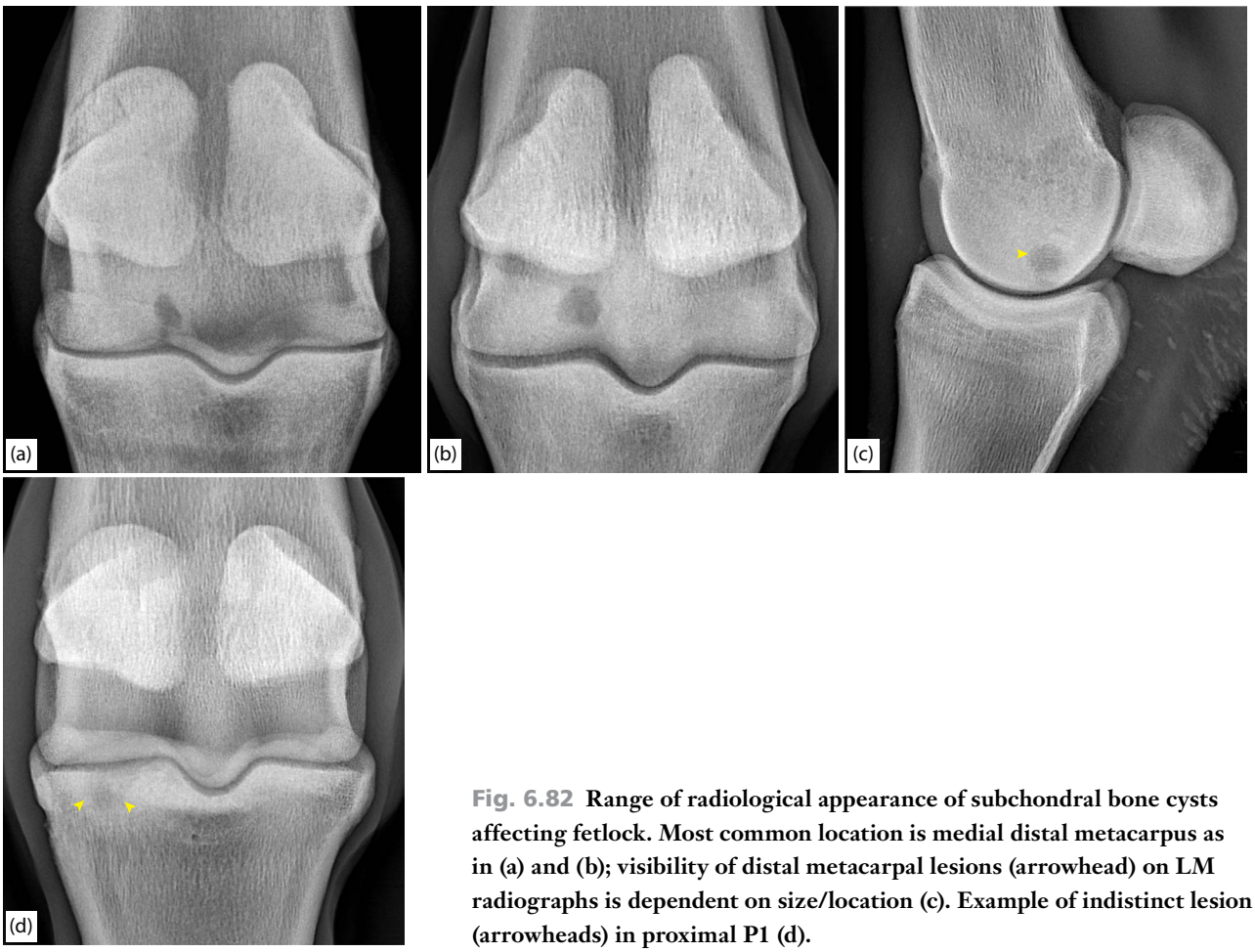


Fig. 6.82 **Range of radiological appearance of subchondral bone cysts affecting fetlock. Most common location is medial distal metacarpus as in (a) and (b); visibility of distal metacarpal lesions (arrowhead) on LM radiographs is dependent on size/location (c). Example of indistinct lesion (arrowheads) in proximal P1 (d).**

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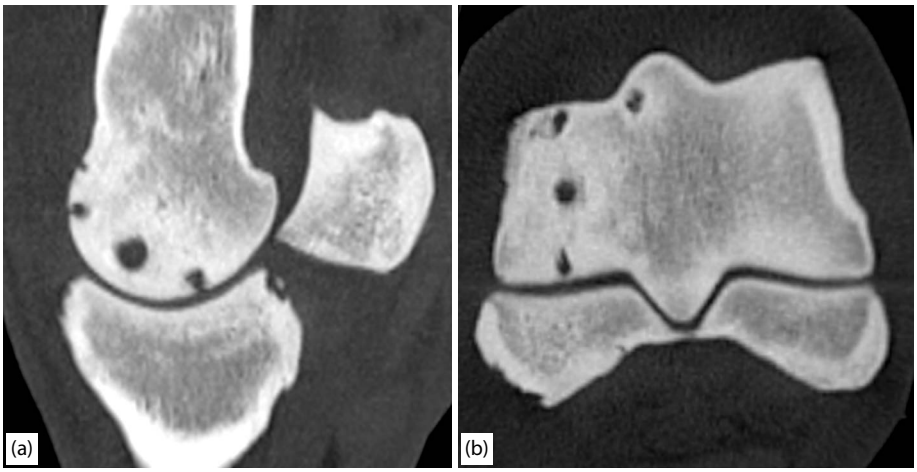


Fig. 6.83 **Atypical, multifocal developmental cystic lesions in hindlimb fetlock of yearling; this horse developed related problems but raced as a 3 YO.**



Fig. 6.84 **Cyst (arrowheads) in PSB of hindlimb. This remained clinically inactive throughout training career.**



Fig. 6.85 **(a–c) Subchondral radiolucencies of distal P1 (DPa/Pl).**

More extensive cysts (midline, or medial/lateral locations of either distal P1 or proximal P2) are potentially more relevant (Figure 6.85b,c). PIPJ cysts are most common in hindlimb.

Cause

- See Osteochondrosis (Chapter 7, p. 267).
- Some subchondral bone cysts (particularly those in PIPJ) appear to arise secondary to injury sustained early in life causing loss of integrity of joint support structures (Figure 6.86).

Risk factors

- Rare condition. Prevalence in sales yearlings: <1% (fetlock joint), <2.5% (PIPJ).



Fig. 6.86 **Subchondral radiolucency of distal P1 (circled) with concurrent entheseal/periosteal reaction (arrowheads) higher on bone, consistent with likely traumatic origin of bone cyst.**

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History
- Typically an incidental finding at routine yearling sales radiography (subclinical at time of diagnosis).
- May become clinically active when training commences: acute or intermittent lameness in early cantering phase.

Signs
- Most cases display no clinical signs.
- If clinically active: mild-moderate unilateral lameness +/– joint effusion.
- Fetlock: lameness often exacerbated by flexion.

Diagnosis
- Radiography: oval-shaped or irregular radiolucency +/– surrounding sclerosis; rarely, affected joints may also display evidence of arthritis.
- MRI: some PIPJ lesions are radiologically silent and MRI required for diagnosis.
- CT: may be useful to define extent of lesion and assist surgical planning.

Management
- Clinically silent: no treatment necessary and continue training.
- Clinically active: goal of treatment is to resolve lameness and return to full function.
- Clinically active fetlock joint cysts: intra-articular medication may be useful for short-term soundness; however, surgery (trans-cystic implant or debridement) offers best chance of return to use.
- Clinically active PIPJ cysts: intra-articular medication may be useful for short-term soundness; surgical arthrodesis offers best chance of return to use but should be viewed as salvage procedure.
- Lesions detected at foal/early yearling stage may evolve considerably without any intervention (Figure 6.87a–d), and management/treatment decisions should be guided by radiographic progress and sale/training intentions.

Prognosis
- Most do not cause lameness; however, unpredictable behaviour and can be career-threatening if become clinically active.
- Cysts in distal palmar/plantar cannon can act as point of structural weakness during high intensity training and may be associated with increased risk of condylar fracture (through defect) depending on location of lesion.

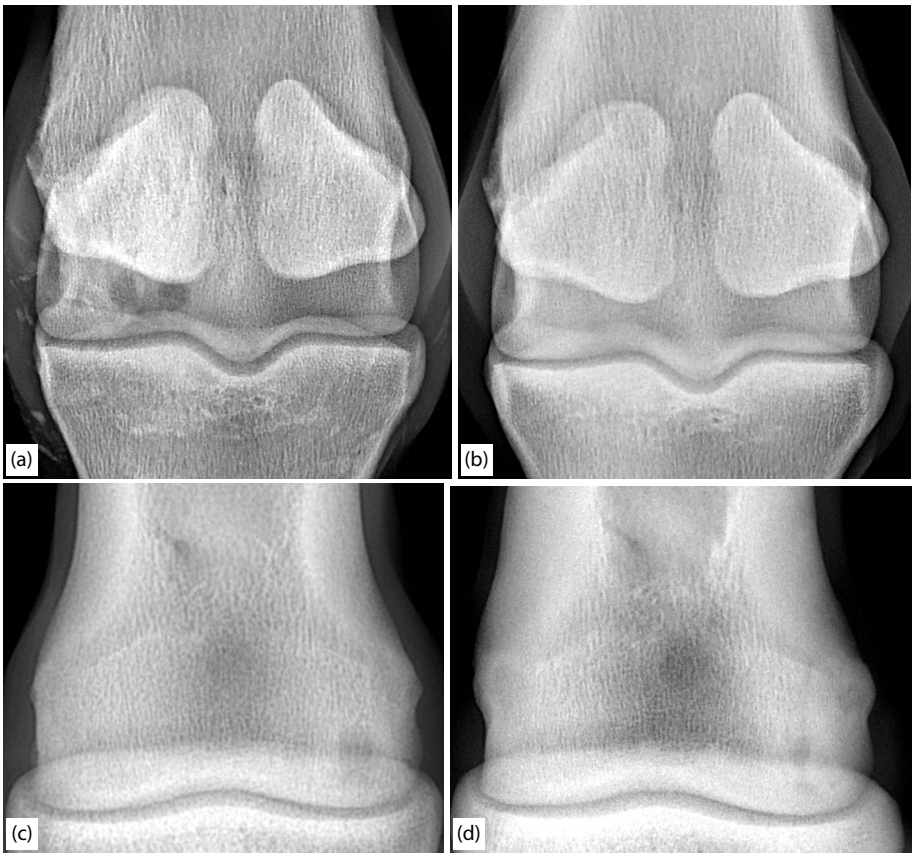


Fig. 6.87 Evolution in radiological appearance of cystic lesions in two yearlings: (a) lesion in medial condyle of forelimb fetlock, with almost complete resolution 6 months later (b); and (c) PIPJ subchondral bone cyst, becoming narrow/smaller radiolucency over 6 months (d).

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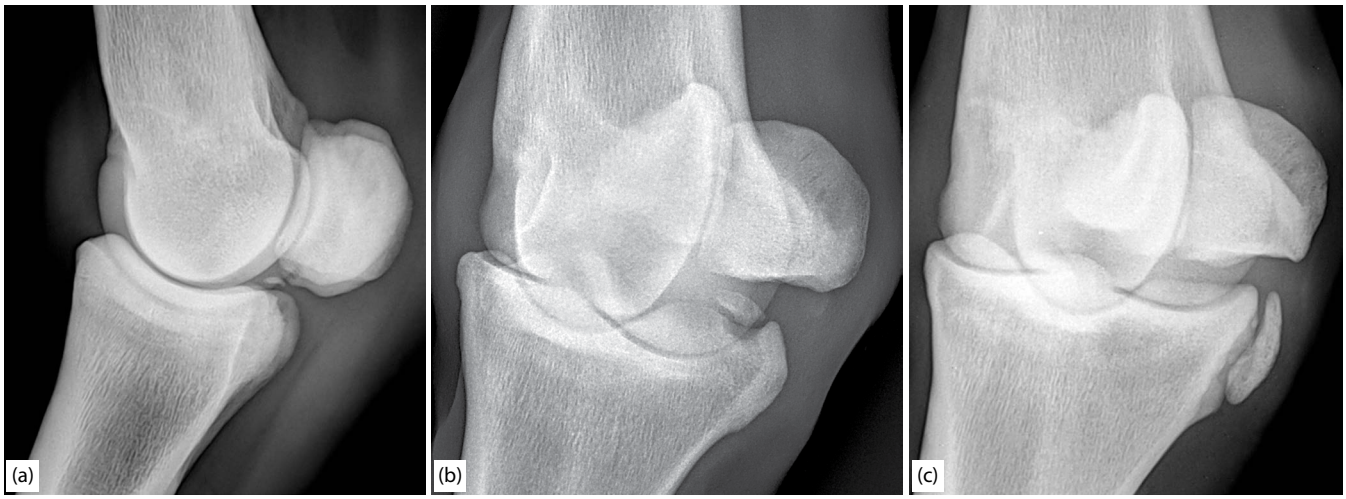


Fig. 6.88 **(a–c) Range of radiological appearances of plantar process fragments of fetlock.**

- Clinically active fetlock joint cysts: guarded prognosis with conservative management; prognosis for return to full use is good (approximately 80%) with surgery, but dependent on severity/location of lesion.
- Clinically active PIPJ cysts: poor prognosis for soundness with conservative management; prognosis for return to full use is improved but guarded with surgical arthrodesis (better prognosis for hindlimb).

Plantaroproximal P1 osteochondral fragments

Common radiological finding associated with plantar process of P1 in hind fetlocks; sometimes attributed to osteochondrosis (p. 267), however, as typically long-standing, origin is almost always obscure. Range in appearance from small distracted osteochondral fragment(s) +/– obvious fragment 'bed' on adjacent margin of P1 (Figure 6.88a,b) to apparently ununited/partly united fragments involving large portion of P1 plantar process (Figure 6.88c). Location (proximity to bone insertion of soft tissue structures including short distal sesamoidean and proximal digital annular ligaments) and appearance consistent with overextension or rotational injuries sustained early in life (rather than OCD); injury is almost invariably the origin of larger/extensive plantar process lesions (Figure 6.89a–c). Are rarely clinically significant even when large/extensive. Unilateral or bilateral.

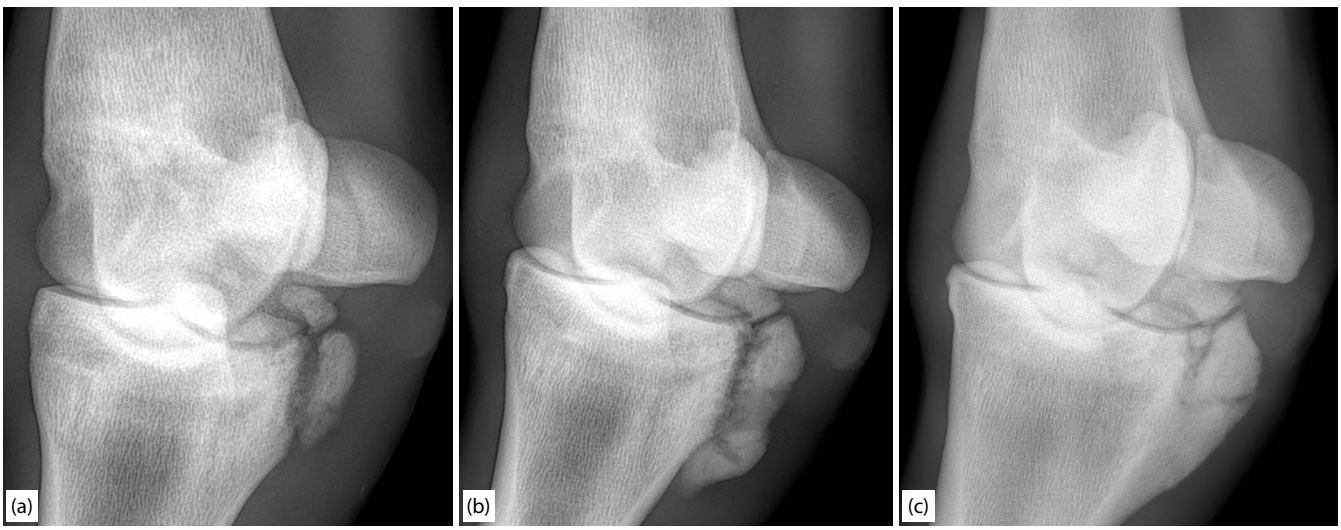


Fig. 6.89 **Radiological progression following traumatic injury involving lateral plantar process of yearling hindlimb fetlock: At time of injury (a), at 3 months (b) and at 8 months (c) post-injury.**

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Cause
- Residual radiological defect likely to have arisen from either ligament-bone avulsion or mineralisation following subclinical/mildly clinical injury sustained early (first months) in life.
- Also proposed that osteochondrosis (p. 267) may play a role in some horses.

Risk factors
- Prevalence in sales yearlings approximately 5–7%.

History
- Typically an incidental finding at routine yearling sales radiography (subclinical at time of detection).

Signs
- Typically clinically inapparent: even when radiological presence is known, not possible to detect any palpable abnormality.

Diagnosis
- Radiography: best observed on oblique projections.

Management
- Almost invariably of no clinical interest; exceptions are extensive lesions with associated clinical evidence of joint inflammation (rare).
- Sometimes claimed to affect action at faster paces; however, no evidence base to support this (or of clinical significance in general) and may be presumed to be clinically irrelevant.
- If concurrent lameness is present, care should be taken to rule out all other possible pathologies (including use of MRI/PET/CT) before attributing potential significance to plantar P1 osteochondral fragment(s).

Prognosis
- Excellent: almost without exception can be considered a radiological blemish of no clinical concern for athletic soundness.

Pastern joint (PIPJ) osteoarthritis

OA of the PIPJ (proximal interphalangeal/pastern joint) is rare and when encountered usually affects a single limb (forelimb or hindlimb). It may occur in association with osteochondrosis (subchondral bone cyst), joint margin osteochondral fragmentation or as a primary condition.

Cause
- Frequently undetermined.
- May result from acute/chronic PIPJ instability/subluxation.
- May occur in association with a subchondral bone cyst (p. 138).

Risk factors
- Any age/stage of training.
- Risk factors unknown.

History
- Chronic-onset lameness.

Signs
- Mild-severe unilateral lameness.
- Firm non-painful thickening/altered profile of affected PIPJ.

Diagnosis
- Radiography: osteoarthritic change including dorsoproximal osteophyte(s) P2, narrowing of joint space +/− altered subchondral density (Figure 6.90a,b).

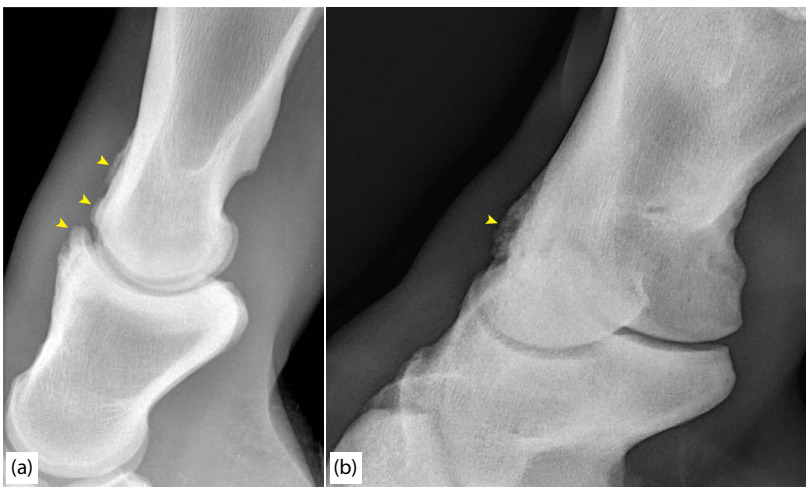


Fig. 6.90 PIPJ osteoarthritis: abundant dorsal articular and periarticular modelling on LM (a) and DLPlMO (b) projections.

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Management
- Determined by clinical severity, chronicity and stage of training.
- Early disease best managed by rest (stable rest/walking for 8–12 weeks) +/– anti-inflammatory medication.
- Mild/chronic disease: intra-articular medication +/– continued training may be possible.
- Remedial shoeing (raised heels/improved breakover) may assist management.
- Severe disease/poor response to conservative management: consider surgical arthrodesis.

Prognosis
- Guarded-poor prognosis for athletic soundness with rest/medication.
- Improved (but guarded) prognosis for return to full use with surgical arthrodesis.

Pastern joint (PIPJ) palmar/plantar fragmentation

Rare condition that may be encountered either as an incidental finding or associated with clinical injury/lameness. Fragmentation of proximal palmar/plantar margin of P2: typically at midline/axial location (at/within insertion of middle scutum); less commonly abaxial location (at insertion of SDFT branch +/or abaxial palmar/plantar ligament of pastern joint. Forelimb or hindlimb; unilateral or bilateral.

Cause
- Frequently undetermined.
- Likely to be avulsion injury of the fibrous supportive structures/ligaments at the back of the pastern joint; presumed secondary to PIPJ hyperextension.
- Midline/axial fragmentation often appears to be associated with enthesophyte formation at this site; suggests chronic ligamentous overload may play a role.

Risk factors
- Most commonly occurs early in life and detected as either incidental or clinical finding at later stage. May rarely occur as injury during training.
- Rare: prevalence in yearlings documented as 0.06%.
- Risk factors unknown.

History
- May be incidental finding (at routine radiographic screening); or may present clinically with acute, chronic or intermittent lameness.

Signs
- Frequently subclinical.
- If clinically active: unilateral lameness of mild to moderate severity.
- +/– non-painful thickening/altered profile of palmar/plantar aspect of affected PIPJ.

Diagnosis
- Radiography: displaced fragment(s) +/–/or enthesophyte at proximal palmar/plantar margin of P2. Fragment typically smooth-margined and of long-standing appearance (Figure 6.91a–c).
- Diagnostic blocking: intra-articular blocking of PIPJ recommended to confirm clinical relevance of radiological findings.

Management
- Clinically silent: no treatment necessary.

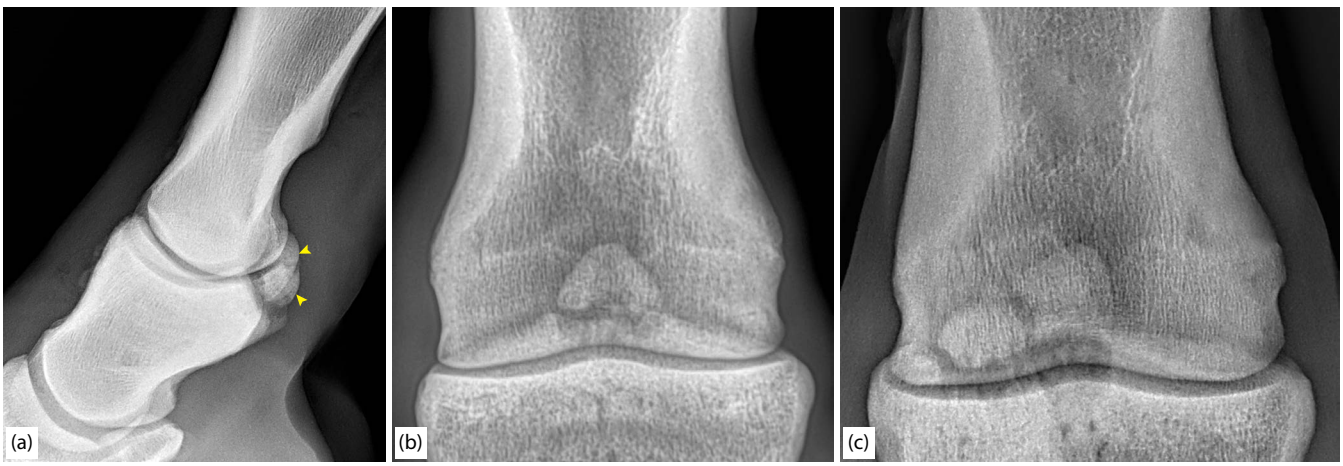


Fig. 6.91 PIPJ palmar fragmentation: Fragment(s) visible on LM (a) and DPa (b, c) radiographs. Secondary osteoarthritic change of PIPJ is evident dorsally in image (a).

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- Clinically active: intra-articular medication +/– continued training may be possible.
- Arthroscopic debridement of fragment(s) considered to be treatment of choice for young horses with significant associated lameness.

Prognosis
- Clinically silent lesions in horses in training usually remain so.
- Clinically active lesions: guarded-fair prognosis for continued athletic soundness with medication.
- Good prognosis for return to full use reported with surgical intervention.

Sesamoiditis
Widely used but ambiguous term describing a spectrum of radiological changes of the PSBs, usually referring to young (yearling/2 YO) racehorses. Condition is defined radiologically (see later) and, as described here, does not include those milder radiological changes that are considered variants of normality. Many radiological features widely reported as 'sesamoiditis' are simply the long-term residual consequence of diverse pathologies/adaptive change experienced early in life (Figure 6.92a,b) with little/no relevance to future athletic soundness. Condition characterized by areas of demineralization +/– new bone deposition predominantly on abaxial margin of sesamoid. Results in greater relative prominence of vascular channels or enlargement/change in contour of the PSB on radiography. May be seen uniaxially or biaxially in forelimb and hindlimb PSBs.

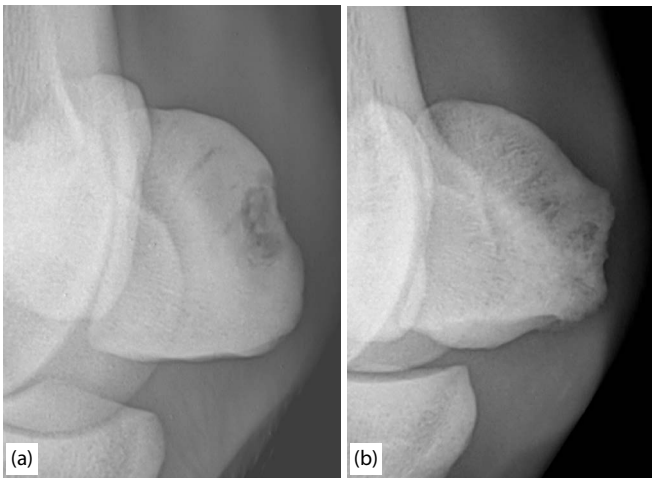


Fig. 6.92 (a, b) Examples of long-standing/juvenile clinically silent PSB irregularities.

Cause
- Underlying cause is not 'inflammation' of the sesamoid bone as such, but rather adaptive change to stresses primarily acting at interface with SLB.
- Likely association with SLB insertional desmopathy (p. 146) in some cases.
- Proximal abaxial border of sesamoid bone is site of attachment of the SLB as well as entry point for blood supply.

Risk factors
- Approximately 2–3% of yearlings affected by marked sesamoiditis (one or more sesamoids).
- Risk factors poorly understood, but nutrition and loading/conditioning early in life (foal) may be of importance. As with some other orthopaedic injuries that occur in immature horses at pasture (fracture of P3, PSBs), prevalence appears to rise with dry/firm or rapid change in ground conditions.

History
- Usually a 'radiological diagnosis' first detected at routine radiographic screening.

Signs
- Frequently no palpable/visible abnormality of PSB or fetlock.
- Firm, non-painful prominence/enlargement of affected PSB(s) may accompany more severe radiological change.
- Lameness is not a feature but may be present with concurrent, active SLB desmopathy.

Diagnosis
- Radiography: features that characterize the condition include new/immature bone on the proximal abaxial or basilar margins (at SLB and DSL attachments, respectively); enlarged (>2 mm width) or irregularly shaped vascular channels in abaxial margin; large poorly defined irregularities in bone density involving body of sesamoid bone; abnormal abaxial profile (Figure 6.93a–f). Defining significance of prominent vascular channels is frequently subjective (Figure 6.93g–i).
- Ultrasonography: assessment of associated SLB important in cases of moderate-marked radiological abnormality or if palpable abnormality (thickening/pain) at SLB/sesamoid interface.

Management
- If clinically silent and detected as incidental finding: no treatment (or change in management) required.

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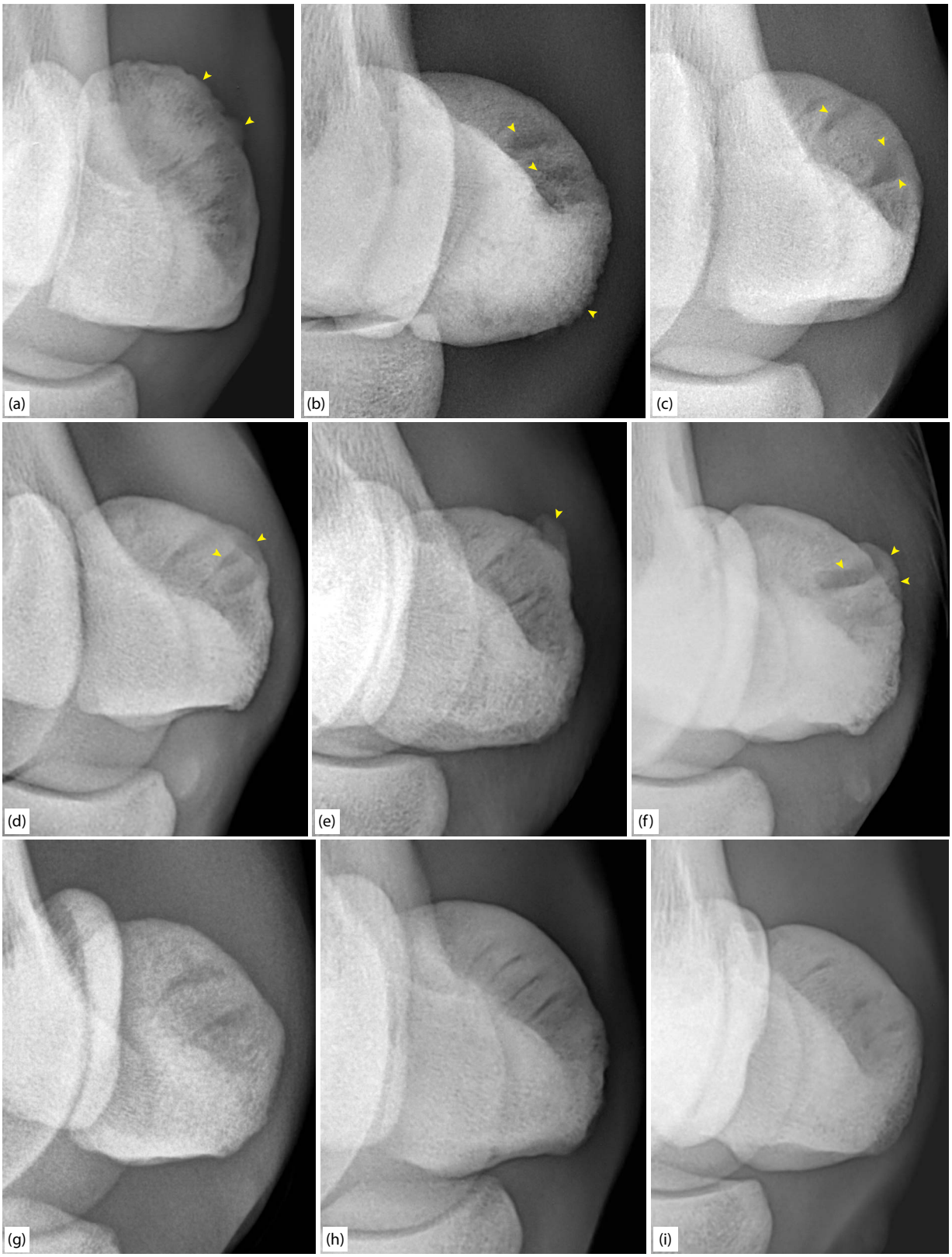


Fig. 6.93 **Sesamoiditis: Radiological features include altered abaxial contour/entheseal proliferation and enlarged/irregular vascular channels (a–f). Relative prominence of vascular channels is a subjective determination, with a range of interpretations (from adaptive to pathologic) possible for many PSBs (such as those in images g–i).**

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- Marked radiological changes: patient approach to training (to permit gradual strengthening/maturation of ligament/bone junction) generally advocated to limit risk of injury, although determined on individual basis and influenced by clinical/ultrasonographic assessment. Altered management not always advised/required.
- Concurrent SLB desmopathy (p. 146) may have greater impact on trainability and should be monitored/managed appropriately on individual basis.
- Medical (aspirin- acetylsalicylic acid/isoxuprine/bisphosphonate) therapy sometimes advocated but no scientific or empirical justification for use.

Prognosis

- Most cases of mild-moderate PSB changes detected at routine radiography do not interfere with training.
- Does not predispose to PSB fracture.
- Yearlings with moderate to severe radiological changes have fewer race starts at 2 and 3 YO, but direct association with PSB injury is unlikely.
- Horses with significant radiological changes and concurrent SLB desmopathy at greater risk of interruption to training as 2 YO if injury becomes clinically active (can be career-threatening in rare cases).
- Prognosis not influenced by whether forelimb/hindlimb affected.

Suspensory ligament branch (SLB) desmopathy

Injuries to the SLB are common and typically take the form of a focal palmar/plantar insertional tear at the interface with the PSB (Figure 6.94). More generalized/extensive damage (sometimes to the level of suspensory ligament bifurcation) is far less common and usually represents chronic injury. Clinical injury most frequently affects a single SLB, but concurrent subclinical defects are commonly present at other sites. Medial branches are overrepresented and injury is more common in the forelimbs. Injuries involving the dorsal face of the SLB are rare, and may communicate with the fetlock joint.

Cause

- SLBs are an integral component of the 'shock-absorbing' apparatus that supports the fetlock and are subject to greater strains during limb loading than the proximal portions of the suspensory ligament.
- Likely that some injuries arise from acute overextension or asymmetric loading of the fetlock

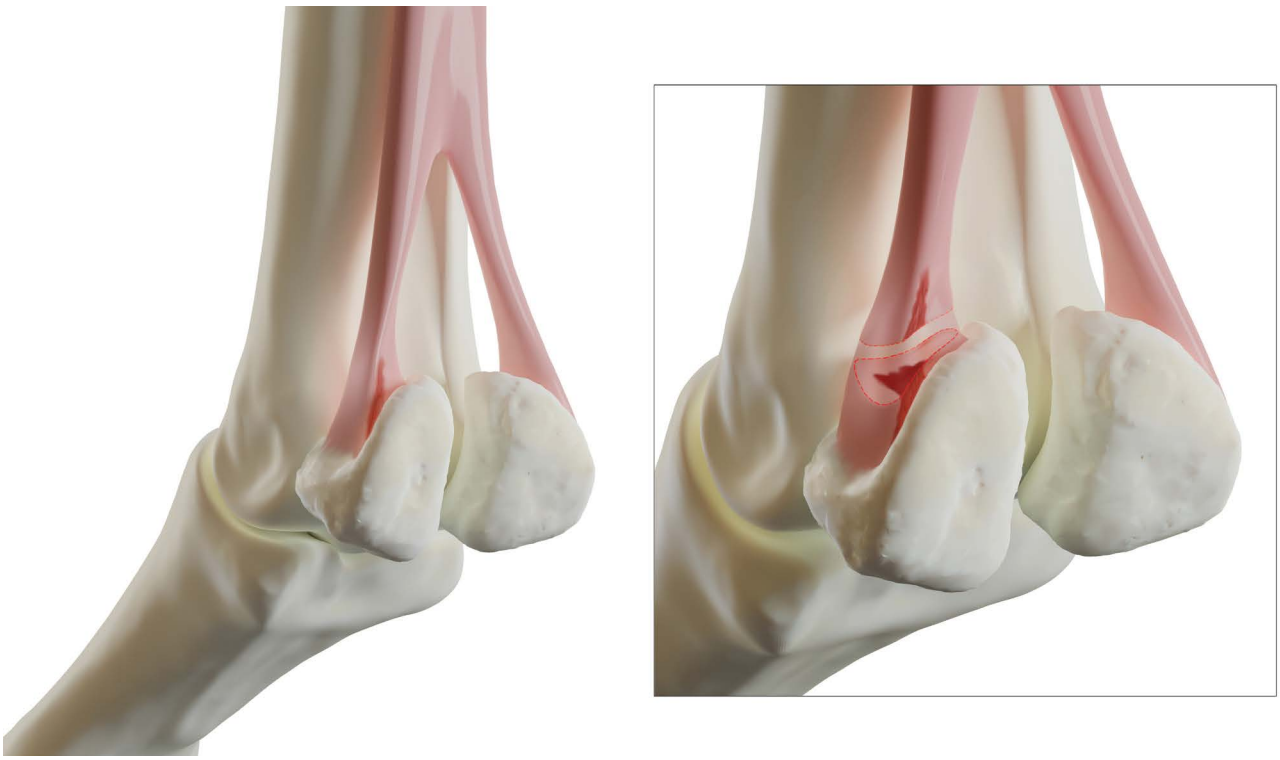


Fig. 6.94 Suspensory ligament branch desmopathy: Typical palmar/plantar location of insertional tears commonly encountered in yearlings (and young horses in training).

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and others from repetitive strain injury of ligament/sesamoid bone interface.
- Clinical and subclinical insertional lesions occur frequently in untrained juveniles (foals and yearlings) (+/– sesamoiditis, p. 144) and likely to predispose to clinical injury when exposed to increased loading in early training.
- Non-insertional injuries involving the mid- and upper portions of the SLB are less common, typically develop in older horses and in most cases appear to represent an injury syndrome distinct from 'juvenile' insertional injuries.

Risk factors
- Clinical (insertional) injury often first detected in 2 YOs during early cantering phase of training; typically preceded by pre-existing sub- or mildly clinical injuries (from yearling/foal stage) in many cases.
- Clinical injury also seen in yearlings prior to/during sales preparation (occasionally foals).
- Onset of clinical injury when >3 YO is less common; may be seen in association with other fetlock overextension pathology.
- Risk factors (insertional injuries) poorly understood but loading/conditioning early in life (foal) may be of importance; anecdotal evidence of association with dry seasonal conditions/hard ground during foal/yearling pasture turnout.
- Increased risk with moderate-severe conformational faults of fetlock (toe-in: lateral branch; toe-out: medial branch).
- ML imbalance of hoof frequently cited as risk factor but rarely a consistent feature in clinical cases.
- Risk factors for injuries of mid- and upper SLB (to bifurcation) unknown; predominantly in horses >3 YO in full race training.

History
- Sub- and mildly clinical insertional lesions may be detected during early yearling screening imaging, or pre-purchase imaging (yearlings and horses-in-training).
- Clinical injury: usually insidious onset with mild signs in early stages contributing to delayed detection in many cases.

Signs
- Prominence/enlargement of affected branch or sesamoid region, +/– overlying oedematous thickening: indicative of lesions of at least moderate severity.
- +/– focal pain response to deep palpation, particularly at level of SLB/sesamoid interface; repeatable pain response is highly specific for the presence of injury.
- +/– palpable enlargement/loss of border definition of branch with limb held in flexion. Loss of dorsal/abaxial border definition can be subtle but even in the absence of pain response is highly specific for the presence of injury.
- Hindlimb injuries frequently display diffuse periligamentous fibrosis/thickening which is evident on palpation.
- +/– fetlock joint effusion.
- Lameness is uncommon, and if present may frequently only be observed when ridden on deep track; likely that loading 'stretch' pain (on extension of fetlock) is responsible in these cases.
- Lameness (if present) is typically mild. Cases with moderate/severe lameness occur but are exceptional.

Diagnosis
- Clinical findings alone are strongly indicative and highly specific. Repeatable focal pain response to deep palpation of the SLB/sesamoid interface almost invariably indicates the presence of an injury. Palpation is a sensitive technique even for detection of subtle pathology, although a proportion of injuries are truly subclinical.
- Ultrasonography: best practical diagnostic tool and required to characterise location and severity of injury. Focal hypoechogenic lesion/enlargement/loss of border definition/periligamentous thickening/irregular sesamoid bone interface (Figure 6.95a–l). Most common lesion location is on palmar/plantar axial aspect of insertional portion of branch, in region of a normal anatomical 'cleft' formed by the diverging band of fibres. More subtle irregularity of echogenicity/fibre pattern frequently observed in clinically normal horses (Figure 6.96a–d); may represent mildest manifestation of condition on pathological spectrum and generally considered to be of little/no practical significance. Complete/incomplete sagittal 'split' in ligament extending dorsally from palmar/plantar margin may occur (Figure 6.97a,b); ultrasound does not always permit determination of articular component to injury. Scanning with leg held non-weightbearing may assist visualisation of such 'splits' in ligament. Injuries predominantly affecting the proximal portion of SLB (+/– insertional defect) extending to the level of the bifurcation are uncommon

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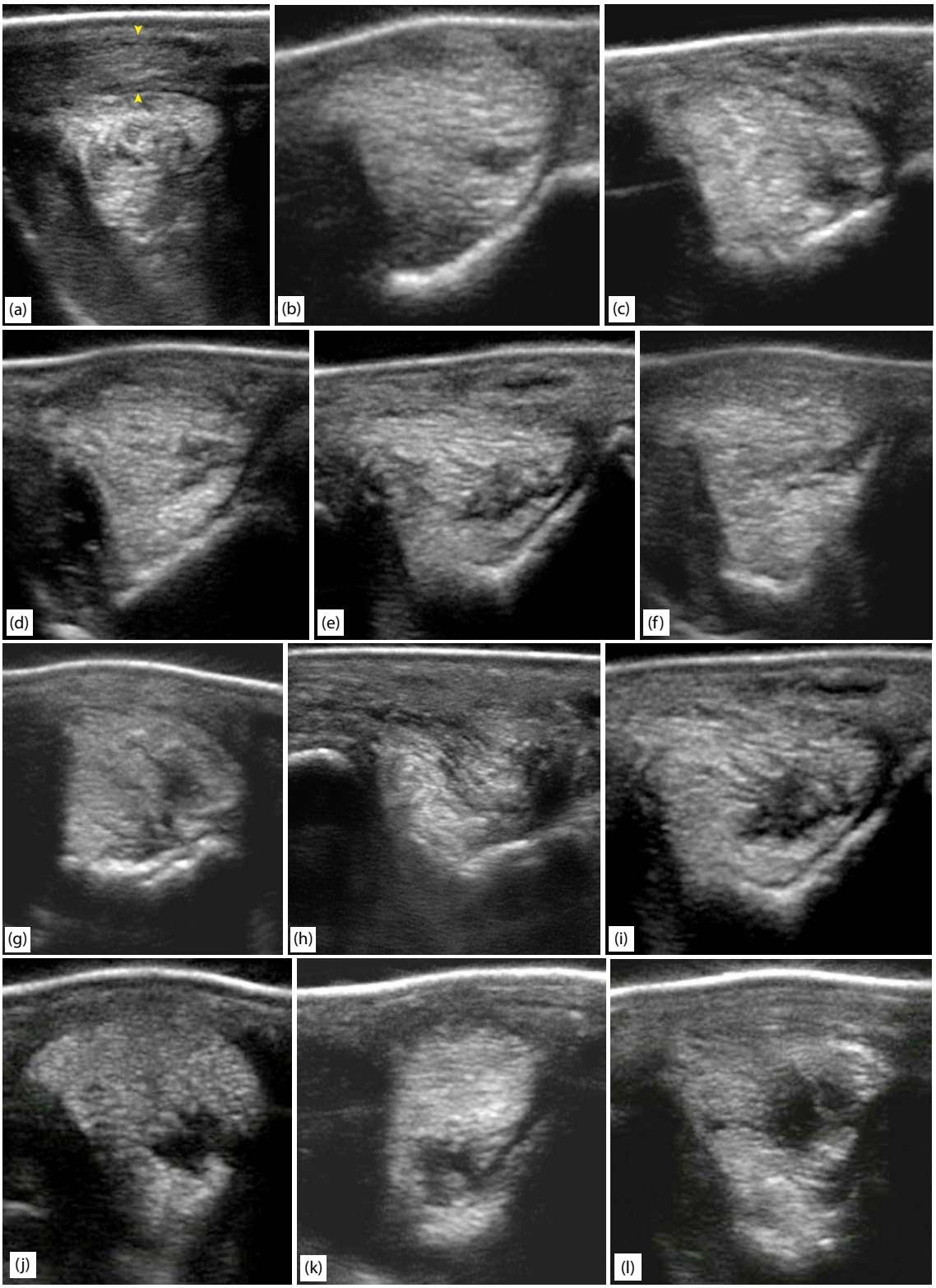


Fig. 6.95 **Transverse ultrasonograms showing range of features associated with SLB desmopathy. Periligamentous thickening (a), palmar/plantar hypoechoic defects at typical site but of varying extent/appearance (b–j), axial disruption (k). Extensive palmar disruption with extension to dorsal aspect in (l). Note that loss of border definition and disruption of fibre pattern is an additional feature of (h).**

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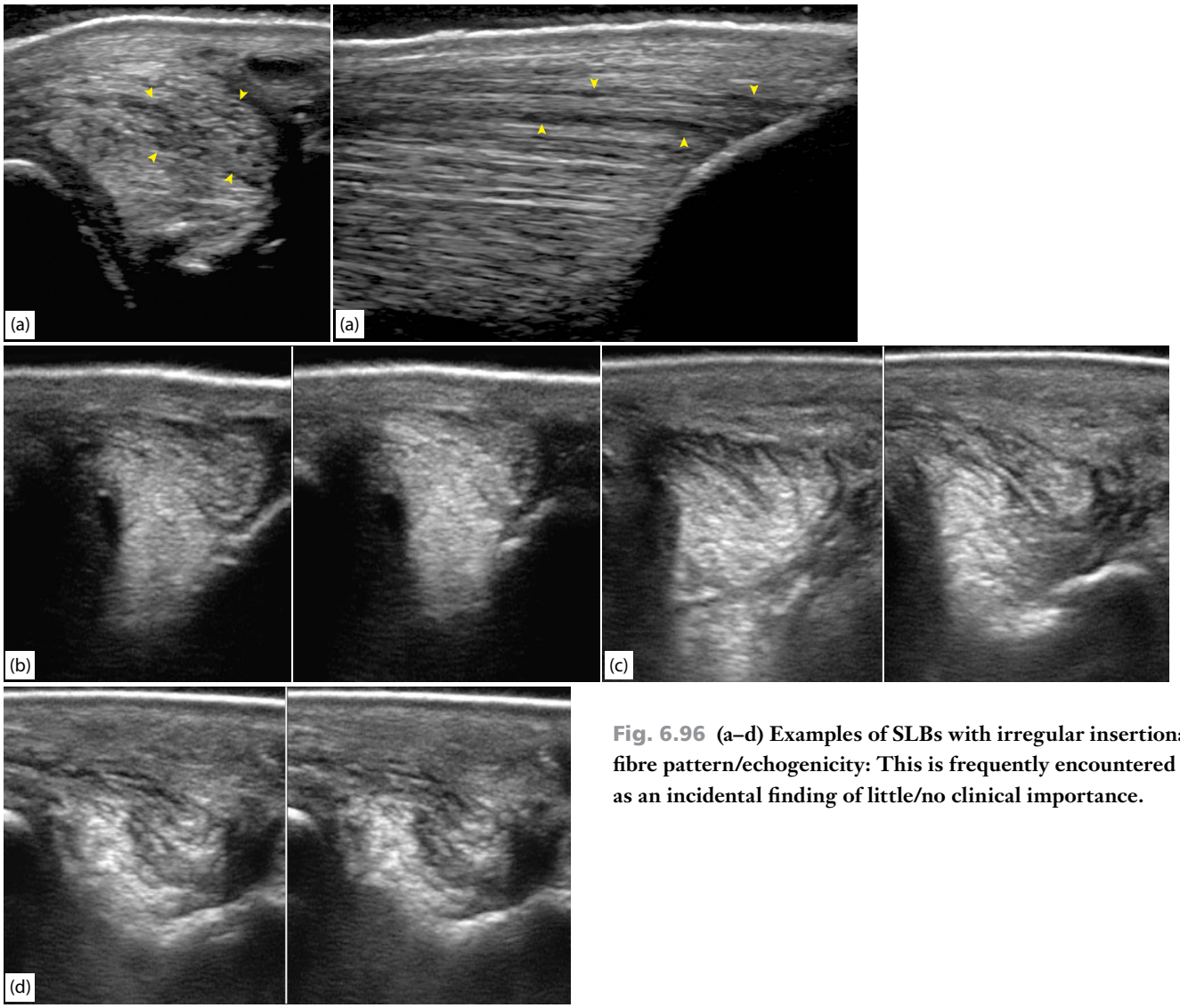


Fig. 6.96 **(a–d) Examples of SLBs with irregular insertional fibre pattern/echogenicity: This is frequently encountered as an incidental finding of little/no clinical importance.**

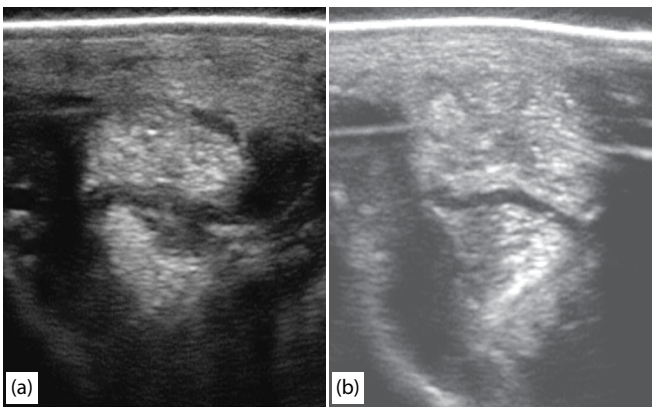


Fig. 6.97 **(a, b) Sagittal 'split' in SLBs in which injury communicates dorsally with fetlock joint. Typically associated with more severe clinical signs than with lesions confined to palmar/plantar insertional margin.**

and frequently occur with (at least initially) little evidence of clinical/ultrasonographic abnormality at the SLB-PSB interface (Figure 6.98a–d).

- Power Doppler ultrasonography (Figure 6.99a,b) can be used as adjunctive aid to assess vascularisation of lesions but prognostic value likely to be limited in most typical cases.
- Radiography: not necessary for diagnosis but may assist management, as SLB injuries with concurrent marked radiological changes (of PSB) may carry a poorer prognosis. Single or multiple enlarged vascular channels +/– entheseal modelling (p. 145) is typical but not universal.
- MRI: superior to ultrasonography at characterising injured soft tissue but rarely needed for diagnosis/management of this injury in clinical practice: typically reserved for atypical/complex cases.

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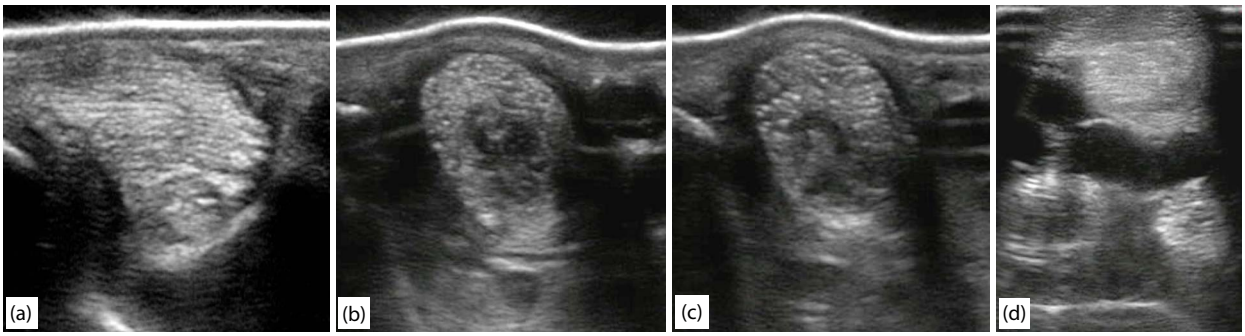


Fig. 6.98 **Transverse ultrasonograms of a typical proximal SLB injury, with relatively normal appearance of distal/insertional portion (a) and lesion apparent higher in branch (b, c) approaching the level of the bifurcation (d).**

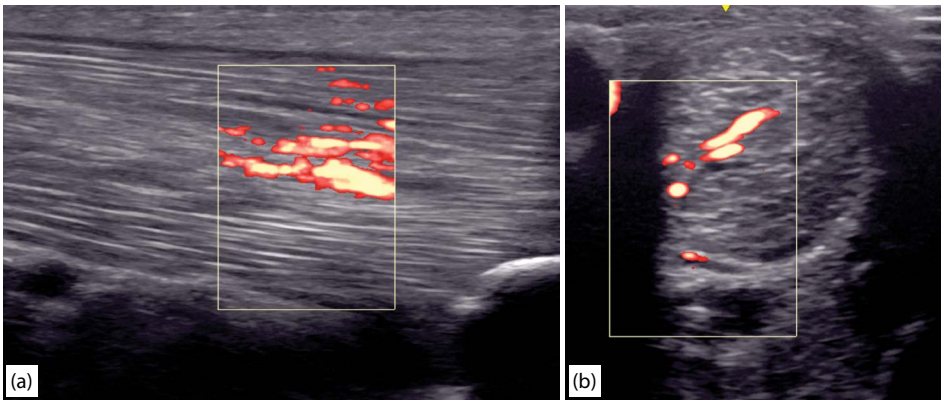


Fig. 6.99 **Power Doppler ultrasonograms [longitudinal, (a); transverse, (b)] showing vascularisation within the ligament that should not be present in a normal/uninjured SLB.**

Management

- Management dictated by clinical severity, stage of training and career goals. Tailor exercise/rehabilitation to individual.
- Prescription of (lengthy) rest periods without consideration of individual circumstances likely to result in unnecessary career wastage; most horses without associated lameness do not generally require significant rehabilitation or intervention.
- Rehabilitation is best guided by clinical progress and should not rely on ultrasonographic 'milestones'.
- SLB injury shares characteristics with some human tendinopathies: in the latter, exercise is the most evidence-based treatment (rest does not increase the tolerance of the ligament/tendon to load)
- Subclinical or subtle lesions (no lameness): no alteration in management required and training may continue; however, vigilance for clinical deterioration. Applies to yearlings with known subclinical lesions: only a small proportion ultimately develop clinical injuries in training.
- Clinical injuries early in training (when prescribing rest may have little impact on overall career): consider conservative management to improve likelihood of long-term soundness (stable rest/walking for 6+ weeks).
- Clinical injuries later in training (or when removal from training is difficult to justify): continued training may be possible +/– short initial period (2–6 weeks) of reduced exercise and anti-inflammatory therapy (topical/cold therapy). Satisfactory progress judged by maintenance of soundness (clinical +/or ultrasonographic appearance of SLB may deteriorate slightly). Training aids that reduce fetlock loading/extension (water treadmill/walker/swimming) may be beneficial. With appropriate management there is no risk of serious breakdown and little threat to long-term soundness.
- Extensive injuries, or those involving mid to proximal portions of the SLB (or bifurcation) are more likely to deteriorate unless rehabilitated and may have greater impact on overall athletic soundness; lengthy rehabilitation may be advisable.
- Shoeing: balancing foot and remedial shoeing frequently prescribed and may be beneficial in some individuals but overall usefulness likely to be very limited.
- ESWT: commonly prescribed, however, currently no scientific basis to indicate it enhances rehabilitation over simple conservative management.

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- Ultrasound-guided intra-lesional medication (biological regenerative products): currently little evidence of clinical efficacy but may be justifiable in some cases.
- Ultrasound-guided sclerotherapy: no current scientific basis to support use but anecdotal evidence of successful ablation of neovasculature; may have role in management of rare atypical/refractory cases.
- Surgery: techniques for extra-articular debridement of insertional lesions have been described but currently no scientific basis to recommend surgery over conservative management. Arthroscopic surgery can be considered for recurrent/troublesome injuries with articular component.
- High-power laser therapy (daily treatment during initial weeks): some limited scientific evidence of improved lesion healing but clinical efficacy undetermined.

MANAGING SUSPENSORY LIGAMENT BRANCH INJURIES

- Management decisions should be primarily guided by clinical features and career factors rather than scan appearance in most cases of 'typical' insertional defects.
- When considering rehabilitation, useful to 'work backwards' from training/racing objectives to determine how much time out of training may be available/possible/desirable (rather than prescribing rest based on imaging features).
- Providing attention is paid to clinical progress and training loads adapted accordingly, continuing (or resuming, after a short break) training in the face of injury should not compromise long-term soundness.
- The lameness/palpable soreness initially associated with many SLB injuries frequently 'plateaus' then diminishes over time even with continued training.
- While many interventions (regenerative therapies, surgery, ESWT etc) have been prescribed for SLB injuries, good quality evidence of efficacy in significantly improving athletic outcomes is lacking.

Prognosis

- Prediction of outcome not directly correlated to ultrasonographic severity and instead best guided by clinical history and presentation.
- Most clinically obvious injuries at predominant (PSB insertional) site settle to 'clinical stability' regardless of management but some interruption to training can often be expected in short term.
- Presence/absence of lameness and severity of palpable abnormality at initial diagnosis often a good guide to likelihood of training on without deterioration/interruption in short/medium term.
- Long-term persistence of ultrasonographic irregularity can be expected.
- Recurrent lameness and early retirement in very small proportion of cases only; risk factors for poor outcome currently unknown.
- Injuries affecting mid- to proximal branch (to level of bifurcation) generally require lengthy rehabilitation but prognosis for return to full use is good; ultrasonographic monitoring may be useful guide to management of this injury type.
- Injuries with articular involvement: more likely to recur or cause ongoing lameness if treated conservatively; prognosis for return to full use following surgery is good (>70%).
- Some limited evidence that presence of lesions of moderate severity in forelimb may be associated with increased risk of lateral condylar fracture. Presumed to relate to altered biomechanics arising from loss of integrity of suspensory apparatus; however, understanding of association and clinical relevance is incomplete (e.g., relevance to different surfaces/jurisdictions). Empirical evidence currently does not support an obvious link between SLB desmopathy and risk of fetlock fracture.

Distal sesamoidean ligament (DSL) desmopathy

Injury to the DSL(s) below the back of the fetlock most commonly involves a single structure; concurrent damage to both the straight and oblique DSL is rare. May be seen in association with other fetlock overextension pathologies, such as condylar or SLB injuries.

Injuries of the straight DSL occur in the forelimb or hindlimb (more common in forelimb); typically unilateral and rarely bilateral. Injuries of the oblique DSL may also occur in the forelimb or hindlimb; the medial ligament is most frequently involved in the forelimb and the lateral ligament in the hindlimb. Avulsion injuries (sesamoid), and complete rupture occur but are rare.

Cause

- Overextension or torsional injury of fetlock.
- Not known whether acute overload or cumulative fatigue is responsible.

Risk factors

- Rare condition.
- Usually encountered in horses in fast work/racing stage of training.
- More common in older horses.
- Risk factors poorly understood but long/slack pastern conformation may predispose.

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History

- Typically acute onset; lameness/swelling may first be noted in days following fast work/racing.
- May initially be mistaken for infection.

Signs

- Unilateral lameness of mild-moderate severity, usually worse when ridden.
- Diffuse thickening and loss of definition of back of pastern in some (not all) cases.
- Pain response to deep palpation of affected ligament; pain response more consistent with injuries to oblique DSL than straight DSL.
- Very rarely, complete biaxial rupture results in luxation of PIPJ.

Diagnosis

- Ultrasonography: loss of border definition/enlargement/focal or diffuse disrupted fibre pattern of affected oblique ligament +/– enthesopathy (Figure 6.100). Injuries to straight DSL may appear as core or marginal lesions (Figure 6.101a,b) or generalized hypoechogenicity; periligamentous thickening is common.
- Radiography: radiological abnormalities are not a consistent feature of this injury but radiographic assessment warranted if PSB fracture is a differential diagnosis based on clinical presentation. Oblique DSL injury occasionally associated with small avulsion fracture of base of sesamoid bone (Figure 6.102).

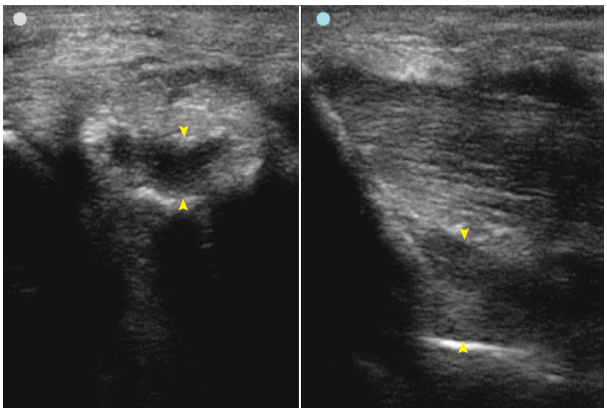


Fig. 6.100 **Oblique distal sesamoidean ligament injury: Transverse (left) and longitudinal (right) ultrasonograms showing hypoechoic defect originating from base of PSB interface.**

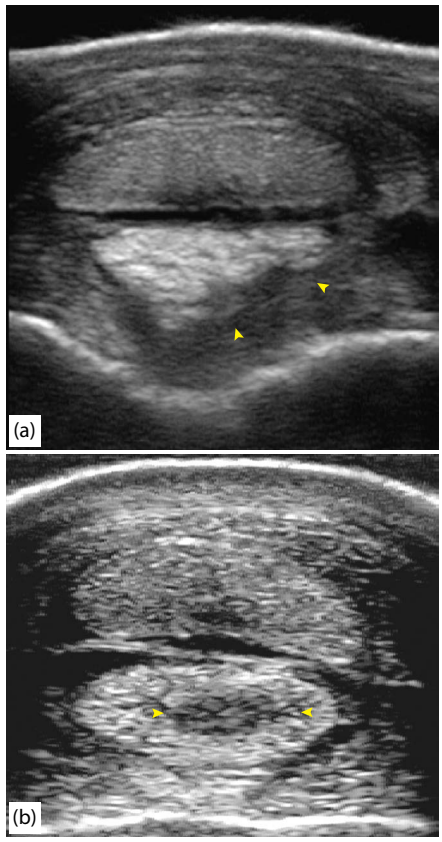


Fig. 6.101 **Ultrasonograms (transverse) of straight DSL desmopathy: (a) disruption to dorsal margin; (b) hypoechoic core lesion.**



Fig. 6.102 **Small avulsion fragment from base of PSB associated with oblique distal sesamoidean ligament injury (DPrM-PaDiLO).**

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Management
- Conservative management: stable rest, supportive bandaging and anti-inflammatory medication in acute phase, followed by controlled exercise (+/– ultrasonographic monitoring).
- 6-month rehabilitation period is typical.
- No good evidence that therapies additional to conservative management (e.g., ESWT, intra-lesional medication/biological therapies) improve outcome.
- Complete biaxial rupture: euthanasia, or surgical arthrodesis of fetlock.

Prognosis
- Mild-moderate lesions: fair to good (55–70%) prognosis for return to full use.
- Severe uniaxial lesions: often recur and carry guarded prognosis for continued soundness.
- Presence of other pathology related to fetlock overextension may affect likelihood of return to racing.
- Complete biaxial rupture: guarded prognosis for paddock soundness following surgical arthrodesis.

Superficial digital flexor tendinitis (insertional branch)

Injury to the SDFT may occur at the level of its insertion in the mid-pastern region. Generally involves only the insertional portion of the tendon (avulsion fractures are rare) but more proximal portion of branch may sometimes be affected. Most common in forelimb. Lateral branch is most commonly involved, with both branches affected in a small proportion of cases. Unilateral or bilateral.

Cause
- Overextension injury of fetlock.
- Not known whether acute overload or cumulative fatigue is responsible.
- Appears distinct from the more typical SDFT tendinitis in the metacarpal region.

Risk factors
- Rare condition.
- Usually encountered in horses in fast work/racing stage of training.
- Risk factors poorly understood but long/slack pastern conformation may predispose.

History
- Lameness at exercise/local thickening may develop over several days.

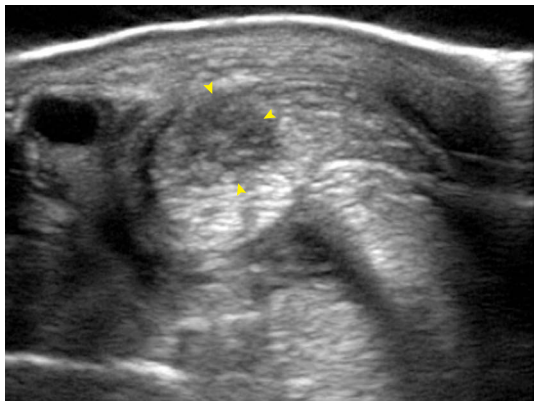


Fig. 6.103 SDFT branch tendinitis: Transverse ultrasonogram showing enlargement of insertional branch of tendon with zone of reduced echogenicity (arrowheads).

Signs
- Focal thickening at back of mid-pastern.
- Pain response to palpation.
- +/– mild-moderate lameness.
- +/– dorsal subluxation of PIPJ during stance phase.

Diagnosis
- Ultrasonography: enlargement/disrupted fibre pattern/reduced echogenicity of affected branch (Figure 6.103).

Management
- Rehabilitation as for metacarpal SDFT injuries (p. 66); controlled exercise guided by ultrasonographic monitoring.

Prognosis
- Fair prognosis for return to full use following lengthy rehabilitation.
- Long-term thickening of branch plus irregular fibre pattern on ultrasonography can be expected.
- Reinjury uncommon but may occur upon return to high-speed exercise.

THE METACARPUS/METATARSUS ('CANNON')

Applied anatomy

The anatomy of the metacarpal/metatarsal ('cannon') region (Figure 6.104) is similar in the forelimb and hindlimb, with some minor differences in relative size and position of soft-tissue structures. The cannon (MC3/MT3) bone extends from the carpus/tarsus to the fetlock and aside from the flexor tendon/ligament bundle at the

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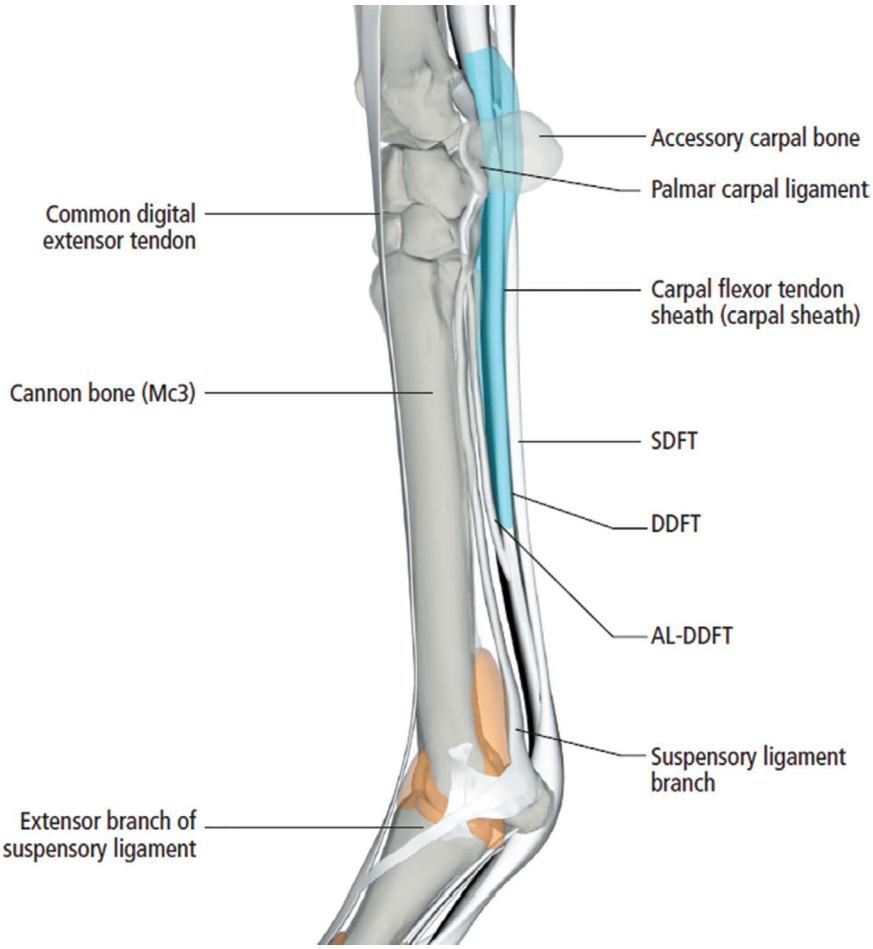


Fig. 6.104 **Major structures of the forelimb cannon (lateral view, splint bone removed).**

back of the leg is little protected by soft tissue. The small splint bones (MC/MT2 and MC/MT4) adhere to the medial and lateral aspects of the cannon, respectively, by interosseous ligaments and articulate with the carpus/tarsus at their head. The thin distal portion of each splint bone is only loosely affiliated with the cannon.

A flexor tendon bundle comprising the SDFT and DDFT descends from the level of the carpus/tarsus to the back of the fetlock. The SDFT is oval in cross section in the high cannon and becomes progressively flattened as it approaches the fetlock. Deep to the DDFT in the upper half of the cannon is the accessory ligament of the DDFT (AL-DDFT/'inferior check ligament'), which arises from the palmar carpal ligament and back of C3/C4 and merges with the DDFT mid-cannon. Deep to all these structures and lying immediately adjacent the back of the cannon is the suspensory ligament. The suspensory ligament arises largely from the top of the palmar/plantar cannon but also, in part, from the lower row of carpal/tarsal bones, and in its proximal portion consists of muscle and connective tissue as well as ligament. It is tightly confined on three sides in the upper cannon by the cannon and splint bones but emerges as a distinct ligamentous body. Just below the mid-cannon it splits into two SLBs, each diverging to attach to its respective PSB. In the forelimb, interposed between the tendon bundle and the AL-DDFT down to nearly mid-cannon, is the potential space of the carpal synovial sheath, which is only palpable when effused; the tarsal sheath is the corresponding structure in the hindlimb.

Examination

Change in profile (bowing) of the tendon bundle may be noted on visual appraisal; palpation of both limbs weight-bearing will also permit subjective assessment of any difference in heat/thickening between the legs. Assessment of individual tendons and ligaments is best performed with the leg raised; thickening/rounding and focal soreness of the SDFT is detectable with light palpation of the tendon edges and should be distinguished from thickening of the skin/subcutaneous structures. Responses should be compared with the opposite limb. The medial and lateral edges of the proximal suspensory ligament are not palpable as they are bordered by the splint bones; however,

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pressure on the ligament against the back of the cannon may elicit a pain response. The shin and splint bones are also best palpated with the leg raised; care should be taken not to apply pressure simultaneously to other sites that may confound the diagnosis. Synovial filling of the distal carpal sheath can typically be detected both with the limb weightbearing or raised.

Superficial digital flexor tendon disease ('tendinitis'/ 'tendinosis')

The SDFT is the main load-bearing tendon in the forelimb. Its elasticity (stretches by 10–16% at maximum loading during high-speed exercise) has 'shock-absorbing' and energy storing roles in locomotion. The structure of the SDFT matures by around 2 YO, with little/no adaptation or strengthening possible after maturity.

Acute or degenerative 'strain' of the SDFT in the forelimb is a potentially career-ending injury. Lesions typically occur in the cannon region, with injuries at or above the level of the carpus or in the fetlock/pastern region being rare. Disruption of tendon fibres frequently manifests as a central 'core lesion'; however, generalized/extensive/multifocal disruption and focal tears of tendon margins are also encountered. Most commonly unilateral but bilateral injuries can occur.

Cause

- Forelimb flexor tendons function with a narrow biomechanical safety margin; during fast work, peak strains within the SDFT are close to the physiological limit.
- Factors that lead to higher SDFT strains can result in clinical injury.
- Most injuries are thought to arise from damage accumulated from repetitive overloading, rather than a single overloading event. Short-term changes occur in tendon in response to high-speed exercise; tendon cells have only limited capacity to repair defects and if not permitted sufficient recovery time microdamage accumulates.
- Weight-bearing load through the lower limb and fetlock region is dampened by action of forearm muscles; muscle fatigue at closing stages of high-speed exercise can therefore lead to greater SDFT peak strains. Strength (and loading) of entire SDF tendon/muscle complex is considered an important part of injury development, rehabilitation and prevention.
- Force on limb is proportional to speed: greater risk of SDFT injury on fast ground.
- Ageing causes some structural weakening in tendon over time, which reduces resistance to fatigue; cumulative high-intensity exercise accelerates this process.
- Possible role of repeated 'overheating' of tendon matrix in centre of tendon during exercise; this may be exacerbated by use of exercise bandages.
- Damage from direct trauma or 'bandage bind/bow' may occur but is far less common than overload injury.

Risk factors

- Incidence rate in flat racing/training is approximately 3% per year.
- Risk of SDFT strain during racing approximately 0.5–0.6/1000 starts in flat races in the UK and USA.
- Primarily affects horses ≥3 YO and risk increases considerably with age.
- Racehorses in jump racing disciplines are at greater risk than flat racehorses. Risk appears to increase with frequency of jump training sessions, and with increased fence height.

History

- Acute or chronic in onset depending on severity and location of injury.
- Heat/swelling usually first noted within 24 hours of fast work/racing, but can also develop at slower cantering paces.
- Routine use of cold therapy/bandaging may mask early warning signs.

Signs

- Signs may be extremely subtle +/– variable with exercise/time of day in early stages.
- Heat +/– thickening of tendon bundle; however, significant injury may also exist in the absence of any obvious thickening/inflammation.
- +/– pain response to palpation.
- Limb examined both weight-bearing (for change in profile) and in flexion (for focal thickening/rounding of edge/pain). Opposite limb palpated for comparison.
- Location and extent of injury determines appearance: ranges from subtle to marked 'bowing' in profile (Figure 6.105). Lesions on dorsal or medial/lateral margins of tendon may present with transient filling in cannon that changes throughout day/between days in response to exercise/activity, presumably due to dispersal of inflammatory fluid.
- Lameness not a consistent feature: typically no lameness with mid-cannon lesions of mild-moderate severity.

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Fig. 6.105 **Typical 'bowed leg' appearance of marked SDFT injury.**

- Lameness more likely when injury runs into the confined spaces of carpal canal or digital tendon sheath.

Diagnosis

- Clinical signs sufficient for diagnosis in cases of moderate to marked ('bowed') injury.
- Ultrasonography: to confirm and quantify extent of injury; may be best assessed approximately 1 week after clinical injury is first noted. Likely that B-mode ultrasonography underestimates true lesion extent.
- Cross-sectional area (CSA) of normal tendon in range of 0.9–1.2 cm^2 and should be similar at same level in opposite limb, although some variation (up to approximately 15–20%) between limbs due to previous/current 'juvenile tendinitis' (p. 158) is not uncommon.
- Injury associated with reduced echogenicity and fibrillar disruption +/– generalized or focal enlargement of tendon, which may vary in severity and location (Figure 6.106a–k). Lesions may sometimes be subtle and necessitate off-incidence scanning for characterisation (Figure 6.106l–n).

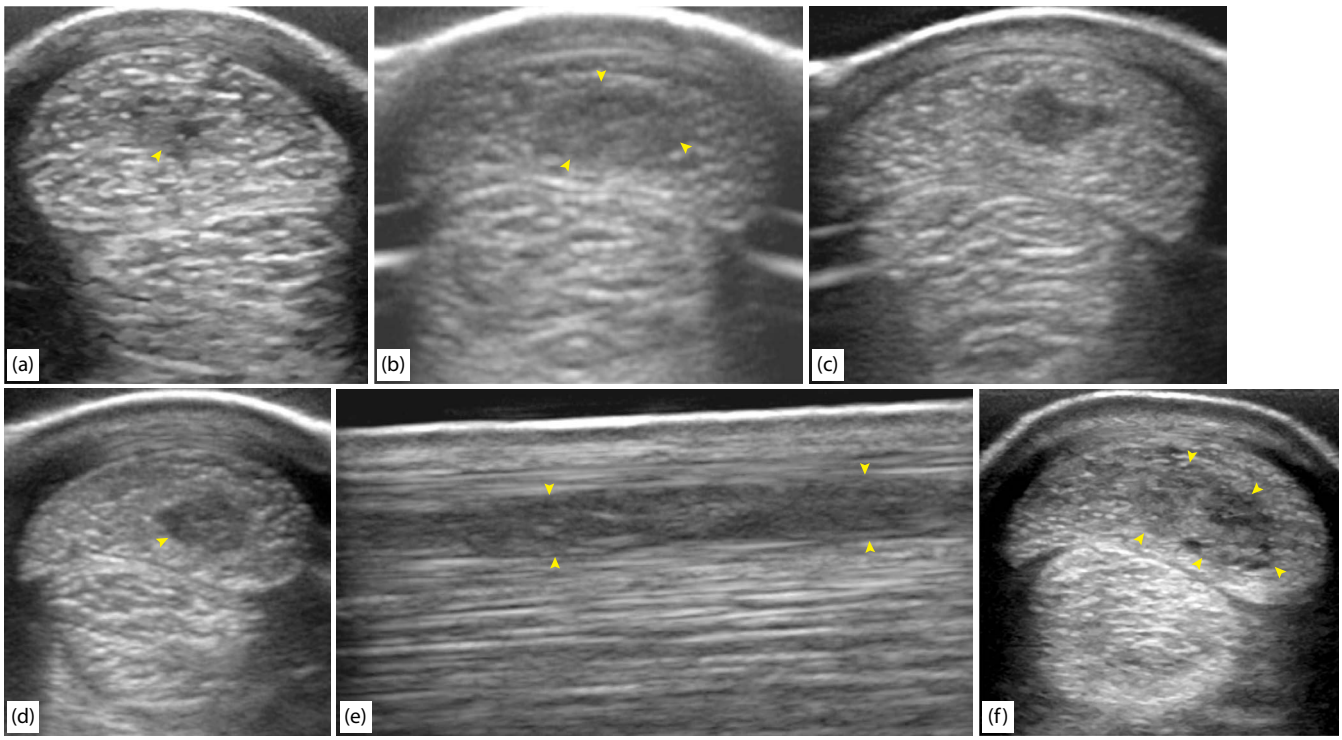


Fig. 6.106 **Ultrasonograms of SDFT injuries. Arrowheads indicating mild core irregularity (a); large zone of reduced echogenicity typical of immediate post-injury phase (b); well-defined core lesions (c, d); appearance of large core lesion on longitudinal view (e); extensive lesion during healing phase demonstrating mixed echogenicity (f);** *(Continued)*

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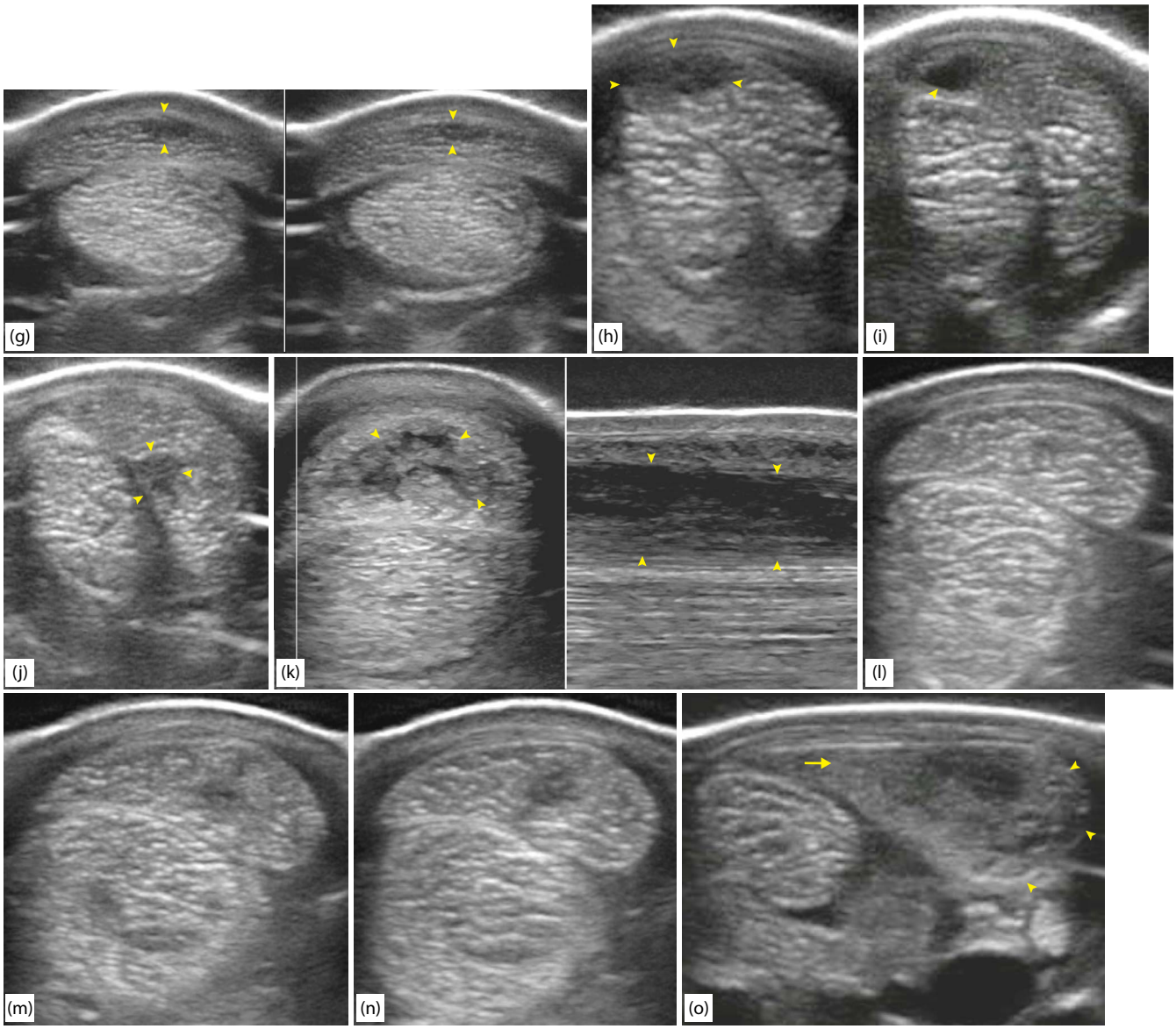


Fig. 6.106 *(Continued)* **mild/indistinct palmar lesion in distal cannon (g); lateral margin lesions (h, i); dorsal margin lesion (j); mixed pattern associated with re-injuring lesion (k); appearance of indistinct lesion may vary with transducer incidence (l–n); tendon rupture resulting in medial displacement (arrow) of tendon (o).**

DIAGNOSING TENDON INJURIES: PALPATE OR SCAN?

- Tendon injuries are not always associated with obvious clinical abnormalities (swelling, heat, palpable pain), even when tissue disruption is extensive.
- Subtle findings (mild thickening/heat) are often detectable by experienced training staff and clinicians, but may be transient or ambiguous and cannot be used to diagnose tendon injury in isolation.
- Palpation of tendon with limb raised may permit appreciation of loss of border definition +/– tenderness; should be compared with opposite limb.
- If in doubt, leg should be scanned.

Management

- See Rehabilitation and Tissue Repair (Chapter 5, p. 66).
- Tendon injuries repair with tissue that is strong but inelastic; unlike bone, tendon cannot remodel damaged tissue back to tissue with 'normal' pre-injury structure/function.
- Goal of treatment is to assist healing so that mechanical properties of the healed tendon are optimized.
- Range of management options available; all involve lengthy recuperation with graded return to loading (12+ months for return to racing).

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- Rarely, continued training without a significant rest period may be possible in some horses with minor injuries (or those located at tendon margins) providing tendon and lesion remain static clinically/ultrasonographically.
- When monitoring progression through rehabilitation, UTC ultrasonography offers advantages over conventional B-mode ultrasonography by permitting 3D characterisation of injury zone and enhanced/objective assessment of fibre alignment.
- Complete rupture is rare (Figure 6.106o): supportive (initially Robert Jones) bandaging and lengthy rehabilitation may permit salvage for paddock use.

Prevention of injury/reinjury

- Strategies with success in preventing/reducing the incidence of tendon injury have not been validated; however, awareness of risk factors associated with SDFT tendinitis provides some useful guidance.
- Avoid excessive training to fatigue and permit sufficient recovery time after racing or high-speed training.
- Avoid use of poorly prepared or inappropriate track surfaces.
- Long-term use of exercise boots/bandages may also contribute to increased risk; magnitude of this risk is unknown but should be balanced against rationale for routine use of bandages in horses that are not prone to interference injuries.
- Strategies to reduce risk of reinjury of a rehabilitating/rehabilitated tendon have also not been validated; however, it is rational to limit excessive loading of tendon.
- Possible aspects to assist with above: incorporate treadmill use in training programme; attention to rider weight; minimise horse accruing excessive body condition; ensure maintenance of good dorsopalmar foot balance.
- Possible benefit to be derived from regular post-exercise cryotherapy (such as cold water immersion): cooling the lower limb effectively can reduce enzymatic activity in tendon and potentially inhibit cell attrition resulting from high-intensity exercise.

Prognosis

- While return to racing is generally possible for most mild-moderate injuries, a high reinjury rate (20% to >60%) can be expected.
- Following return to training: guarded prognosis for continued racing soundness long term (approximately 50% of horses complete three starts and few horses race ≥5 times without reinjury).
- Better prognosis with controlled exercise (during rehabilitation) than uncontrolled pasture rest.
- Increasing severity of injury is associated with lower likelihood of return to racing, shortened racing career and drop in racing class.
- Bilateral tendinitis associated with poor prognosis (25% complete three starts following return) for racing.
- Carpal/proximal cannon SDFT lesions: poor prognosis for racing and frequently display ongoing lameness.
- Unilateral rupture: hopeless prognosis for return to athletic use; however, salvage for paddock use possible.
- Regardless of severity most injuries can sustain paddock or non-racing athletic careers (latter generally with reduced rehabilitation period necessary).

'Juvenile' tendinitis/tendinosis

Enlargement without fibre disruption of the forelimb SDFT through the cannon or carpal regions in the young horse appears to be a condition distinct from SDFT disease (p. 155), although it is likely that they share an underlying pathogenesis relating to tissue remodelling and repair in response to loading.

Condition usually affects one limb but is bilateral in approximately one-third of cases. Generally develops at yearling/early 2-YO stage, with affected tendons typically remaining enlarged long term. Tendon size in the Thoroughbred does not appear to be strongly related to body size/height/weight or sex.

Cause

- Remains an incompletely researched condition but considered to be an adaptive (or maladaptive) response to early (approximately first 6 months of training) conditioning exercise in some individuals.
- Underlying process that leads to enlarged CSA of the tendon(s) poorly understood; however, appears to be due to changes in the non-collagenous tendon matrix including increased water content.

Risk factors

- Occurs predominantly in yearlings/2 YOs.
- Occurs during early (yearling preparation or light cantering) phase of pre-training/training; development of tendon enlargement later in training career more likely to be associated with true SDFT disease than 'juvenile' tendinitis.
- Risk factors not known; factors that influence biomechanical loading of juvenile tendon (including use of horse-walkers, lunging, rider weight, track surface) presumed to be important but how they contribute to risk has not been analysed to date.

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History
- Typically chronic onset (weeks/months) of tendon thickening.
- Often first noted during pre-purchase examinations.

Signs
- Uniform non-painful thickening of one or both forelimb SDFTs. Frequently the most affected part of tendon is upper one-third of cannon region.
- Handling of leg(s) may change/fluctuate in some horses over days/weeks/months (in response to exercise, time of day and even environmental conditions) and remain static in others.
- Can often be differentiated from SDFT disease by history and clinical findings (absence of heat/pain on palpation/lameness/focal enlargement).
- May be associated with distension of distal recess of carpal synovial sheath.

Diagnosis
- Clinical findings strongly indicative.
- Ultrasonography: undertaken if differentiation from true SDFT injury desired. Affected tendons have homogenous appearance with normal/reduced echogenicity and parallel fibrillar pattern (Figure 6.107).
- CSA >1.2–1.5cm^2 is considered to be above normal range; enlargement can sometimes be profound (up to 2.5x normal CSA: 2.8 cm^2).

Management
- Risk of progression to clinical SDFT injury is considered low but should be determined on an individual basis; tendons that constantly change to handle or recurrently cause concern to training staff should be viewed with more caution and monitored regularly (ultrasound) or rested. Ultrasonographic assessment is not necessarily definitive for immediate risk of deterioration.
- Most cases can be managed empirically and do not interfere significantly with training.
- In (uncommon) cases where enlargement is profound/rapid in onset/associated with peritendinous oedema: rest or modified exercise (reduced training intensity/removal from cantering) recommended. Duration determined by severity, career goals and clinical response; walking/trotting for 6–10 weeks frequently sufficient.
- +/– medical therapy (systemic steroidal/non-steroidal anti-inflammatories) +/– topical (cold/osmotic) therapies.
- Reduction in size of tendon should not generally be expected.

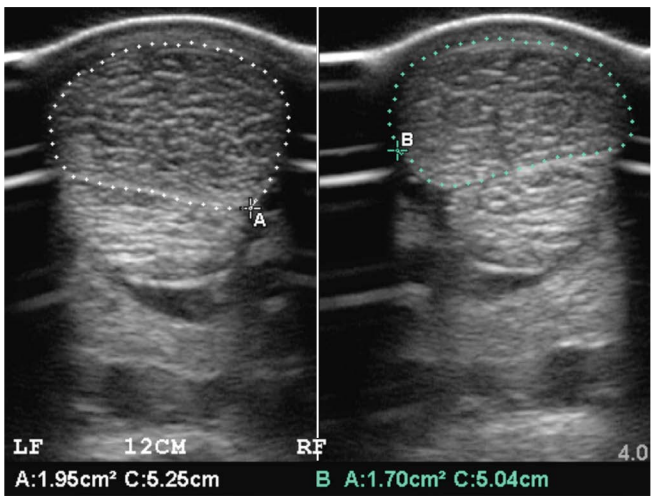


Fig. 6.107 'Juvenile' tendinitis (forelimb): Marked enlargement of both tendons.

Prognosis
- Good prognosis for continued training: as likely to race at 2, 3 and 4 YO as unaffected horses.
- Tendon usually remains thickened long term and may affect resale.
- Moderately/severely enlarged tendon(s) considered to contribute to increased risk of future clinical SDFT injury under some training conditions; quantification of risk currently not possible; however, CSA >1.4 cm^2 at any zone merits discussion and consideration of this risk in pre-purchase context.

Suspensory ligament desmopathy: Forelimb

The suspensory ligament acts along with the flexor tendons to limit overextension of the fetlock joint. Injury to the suspensory ligament can be manifested by fibre disruption, generalized enlargement or maladaptive change at the bone/ligament interface in the proximal cannon. Along with palmar cortical stress injury/fracture (p. 168), it is an important cause of subcarpal lameness. Injury usually occurs at site of origin of the ligament in the proximal third of the cannon with concurrent bone activity/pathology; injuries to the body of the ligament are less common. Unilateral (most common) or bilateral. SLB injuries are considered elsewhere (p. 146).

Cause
- Likely to result from cumulative damage incurred by repetitive strain/overload of suspensory apparatus during exercise.
- Relative strains in suspensory ligament at different paces permit some insight into injury characteristics:

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significant loading through suspensory ligament occurs at extended trot, while at high speeds (gallop) loading is redistributed through flexor tendons.

Risk factors

- Incidence rate approximately 3–5% per year.
- Typically in 2 YOs during early-mid cantering phase of training but may occur at all ages.
- Injuries in horses ≥3 YO are more likely to be chronic and involve the ligament/bone interface.
- Mid-body injuries are uncommon and almost invariably occur in older horses.
- Risk factors for development of injury (track surface/conformation/training regime) poorly understood.

History

- Unilateral forelimb lameness that develops acutely or over several days.
- Lameness often transient; horse may be sound within several hours of initial reporting.

Signs

- Mild-moderate unilateral lameness is typical of proximal injuries. Mid-body injuries frequently occur without lameness.
- Lameness (if present) usually worse when ridden +/– on soft surface +/– with affected leg on outside of circle.
- Proximal injuries frequently have no associated palpable abnormality of limb. Enlargement of proximal suspensory ligament +/– pain response to deep palpation may be a feature in some horses (should be compared with opposite limb). Strong unilateral pain response (retraction of limb +/– vocalisation-'grunt') to palpation in a lame horse should raise concerns about the possible presence of injury; however, milder pain responses occasionally encountered in apparently normal horses.
- Mid-body injuries typically demonstrate variable oedematous filling of mid-lower cannon (dorsal to flexor tendon bundle) that may disperse with exercise +/– palpable soreness of affected portion of ligament.

Diagnosis

- Lameness arising from subcarpal/lower carpal region is a diagnostic challenge due to non-specificity of regional nerve blocks and conventional imaging.
- Diagnostic blocking: frequently does not permit differentiation of subcarpal from middle carpal joint lameness. Lameness may be exacerbated by nerve blocking of foot.
- Ultrasonography: interpretation of subtle/proximal lesions may be difficult due to frequently irregular appearance of normal suspensory ligament (presence of muscle and fat tissue) in upper cannon and variability between individuals; also poor correlation with pathology. Injuries in main body of ligament generally much more obvious. Size/echogenicity of forelimb ligaments should be symmetric: signs of injury include enlargement, convexity of palmar border, loss of dorsal border definition and focal or diffuse reduced echogenicity relative to opposite limb (Figure 6.108).
- Radiography: to assess for presence of other pathologies at proximal cannon/lower carpal sites. Radiological changes occur only inconsistently with proximal suspensory ligament desmopathy: these may include increased densification of proximal medial metacarpus (DPa projection) (Figure 6.109a) or endosteal reaction (LM projection).
- Practical diagnosis is typically one of elimination of other pathology (i.e., distal limb and carpus) using a combination of ultrasonography and radiography.
- MRI is the 'gold standard' imaging modality (Figure 6.109b) but use is typically reserved for challenging cases.

Management

- See Rehabilitation and Tissue Repair (p. 51)
- Uncomplicated proximal suspensory desmopathy in 2 YOs: conservative management (stable rest/walking for 6–8 weeks) usually sufficient.
- Proximal suspensory desmopathy with concurrent disease of bone interface: requires more conservative approach (stable rest/walking for 8–12 weeks) +/– ultrasonographic monitoring.
- Persistence/recurrence of lameness following rest period (with other pathology ruled out) is rare; however, periligamentous infiltration of corticosteroid may be effective in minimising/abolishing lameness in these cases.
- Continued training in acute phase aided by local or systemic analgesic measures (or in the face of deteriorating action) risks development of more severe pathology with consequently poorer prognosis for recovery.
- While adjunctive therapies (e.g., ESWT, intra-lesional medication/biological therapies) have been prescribed for this condition, good quality evidence that they significantly improve athletic outcome is lacking.
- Suspensory body desmopathy (core lesion): lengthy rehabilitation (stable rest/walking for 12–16+ weeks) guided by ultrasonographic monitoring is

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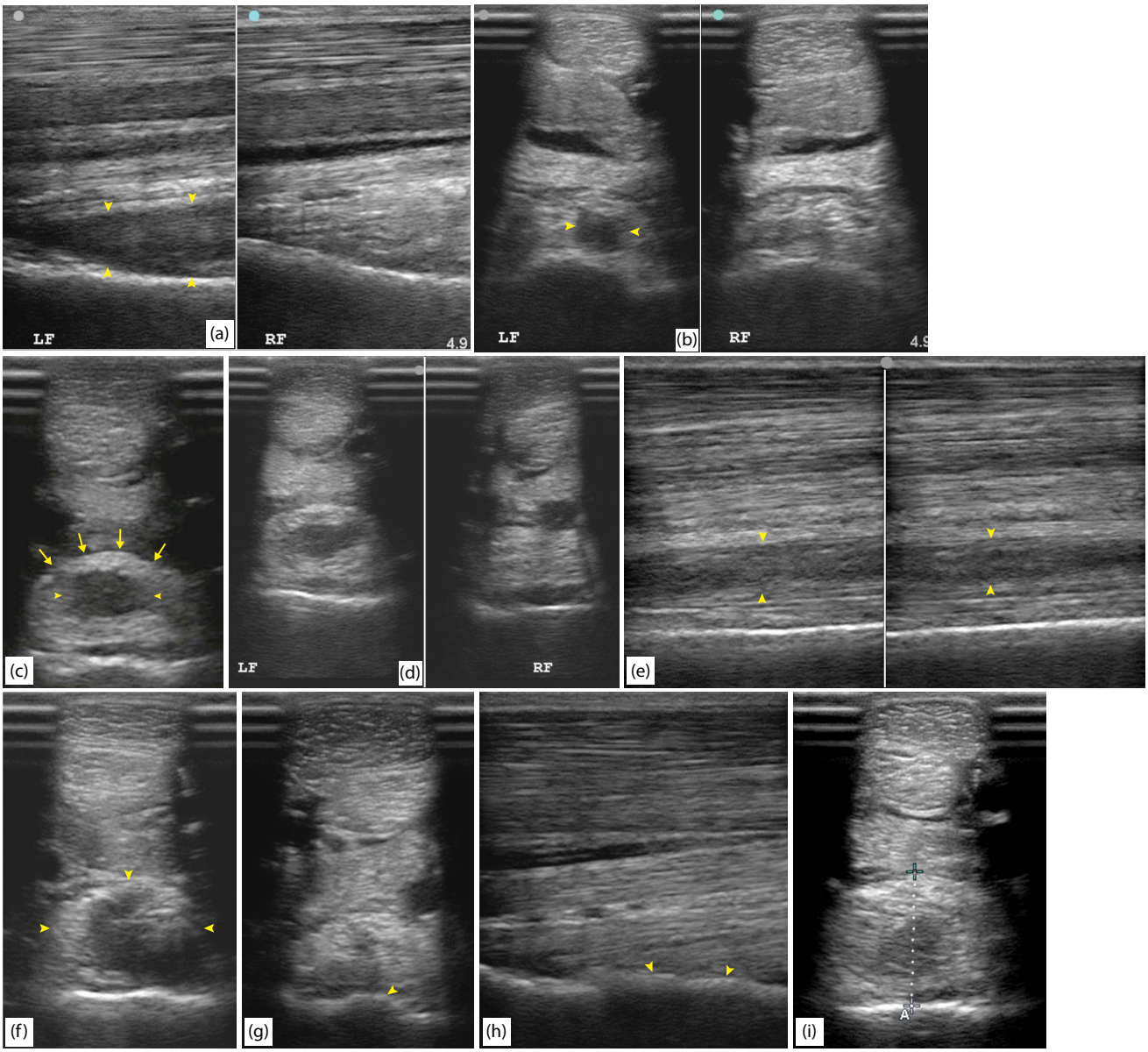


Fig. 6.108 **Proximal suspensory ligament desmopathy: Longitudinal (a) and transverse (b) ultrasonograms showing obvious hypoechoic zones (arrowheads) in proximal suspensory ligament with normal appearance of ligament in opposite limb; (c) large central hypoechoic 'core' lesion (arrowheads) with convex palmar margin (arrows) of ligament; (d) core lesion and enlargement of ligament relative to opposite limb; (e) injury (arrowheads) extending distally to mid-body of forelimb suspensory ligament (longitudinal ultrasonogram); (f) marked disruption and enlargement of ligament (outlined by arrowheads), with little normal tissue visible; irregular bone margin of palmaroproximal cannon at origin of ligament (arrowheads) on transverse (g) and longitudinal (h) ultrasonograms; sagittal measurement of enlarged ligament (i).**

recommended, although should training/racing continue in short term (weeks) following diagnosis clinical/ultrasonographic deterioration is typically slow and of limited severity.
- Shoeing: heels of affected foot should not be raised (during rehabilitation or following return to training) as this results in increased strains through the suspensory ligament (and SDFT).

Prognosis

- Proximal suspensory desmopathy in juvenile: excellent prognosis for return to soundness; recurrence rare.
- Proximal suspensory desmopathy in older horse with concurrent bone interface disease: guarded; may recur.
- Suspensory body desmopathy: guarded-fair prognosis for return to full soundness.

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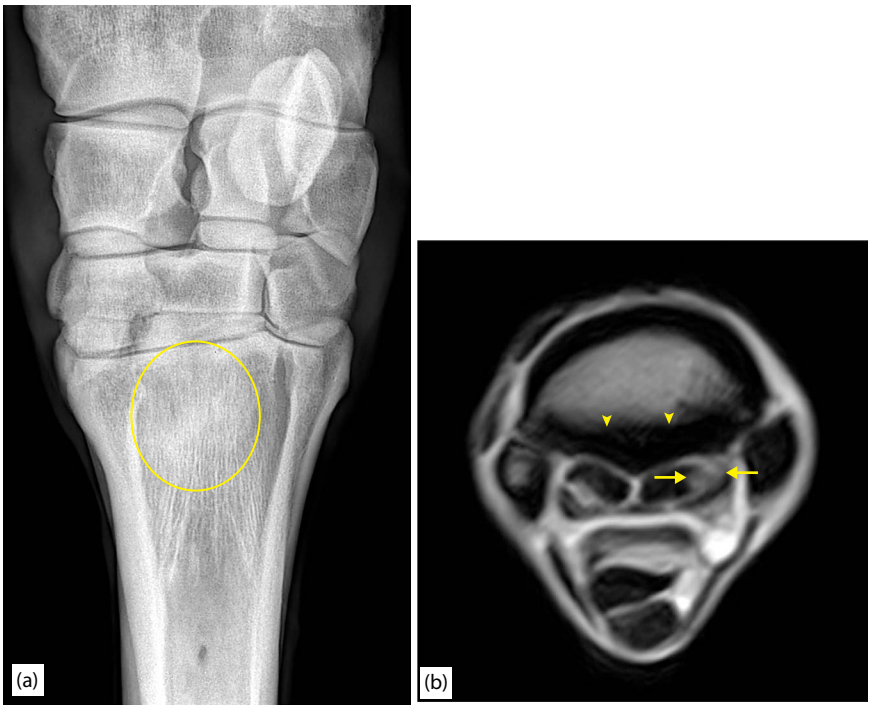


Fig. 6.109 **Proximal suspensory ligament desmopathy. Imaging features may include (a) subtle increased/irregular radiodensity (circled) in proximal metacarpus (DPa); (b) signal hyperintensity (arrows) at the enthesis of the medial lobe of proximal suspensory ligament with an extensive endosteal reaction (arrowheads) in proximal MC3 (transverse low-field MRI).**

Suspensory ligament desmopathy: Hindlimb

Less common in the racehorse than the forelimb equivalent. Injury usually occurs in proximal third of the cannon; injuries to body of ligament are rare. Proximal suspensory ligament of the hindlimb differs from the forelimb by being constrained on three sides by bone borders (cannon and both splints) and on its plantar face by a strong fascial band: enlargement of the ligament may result in a compartment syndrome with compression/inflammation of the plantar metatarsal nerves and ongoing lameness. Pathology frequently bilateral with lameness manifesting in a single limb. SLB injuries are considered elsewhere (p. 146).

Cause

- Likely to result from cumulative damage incurred by repetitive strain/overload of suspensory apparatus during exercise.
- Subsequent enlargement of ligament/compartment syndrome may cause ongoing lameness.

Risk factors

- Rare condition.
- Occurs in horses in cantering/fast work phase of training.
- Typically in horses ≥3 YO.
- Straight-through-the-hock conformation may predispose to injury.

History

- Onset of lameness may be acute or chronic.
- May display reluctance to break from stalls gate.

Signs

- Mild-moderate unilateral lameness, typically with shortened cranial phase and toe catch.
- Lameness usually worse when ridden; also stiffness, lack of hindlimb impulsion.
- Positive response to limb flexion is common.
- Large head of lateral splint and deep location of proximal suspensory ligament limit access for palpation.

Diagnosis

- Differentiating subtarsal lameness from that arising from distal hock joint(s) may be challenging due to non-specificity of regional nerve blocks and conventional imaging.
- Ultrasonography: moderate sensitivity; signs of injury include enlargement, loss of dorsal border definition and focal or diffuse reduced echogenicity relative to opposite limb (Figure 6.110a,b).
- Radiography: radiological abnormality is an inconsistent feature of injury: changes include increased densification of proximal metatarsus (DPl projection) and endosteal reaction (LM projection) (Figure 6.111a,b).
- Bone scan: often unremarkable but IRU in some cases (Figure 6.112a,b).

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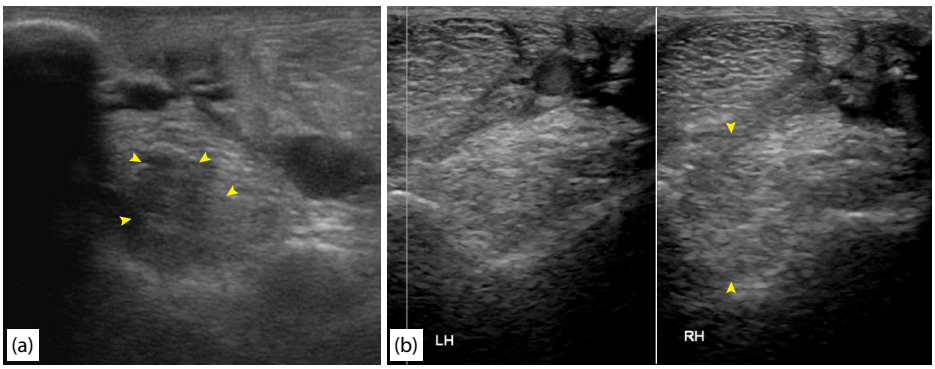


Fig. 6.110 **Hindlimb proximal suspensory ligament desmopathy: Transverse ultrasonograms showing (a) a large zone of reduced echogenicity (arrowheads) and (b) enlargement (and mildly irregular echogenicity) (arrowheads) of proximal ligament in right relative to left hindlimb.**

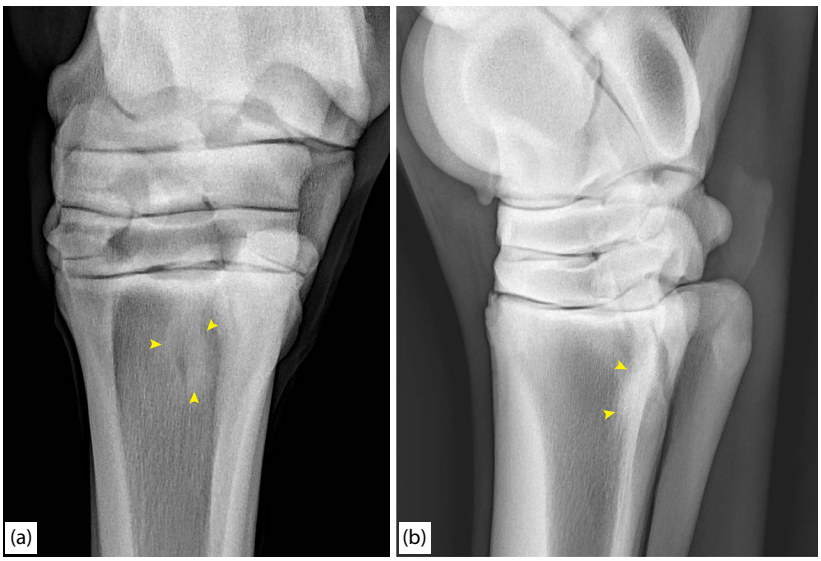


Fig. 6.111 **Hindlimb proximal suspensory ligament desmopathy: Increased/irregular radiodensity in proximal metatarsus (arrowheads) on DPl (a) and LM (b) radiographs.**

- MRI is the 'gold standard' imaging modality.
- Practical diagnosis is typically one of elimination of other pathology (i.e., distal limb and lower hock).

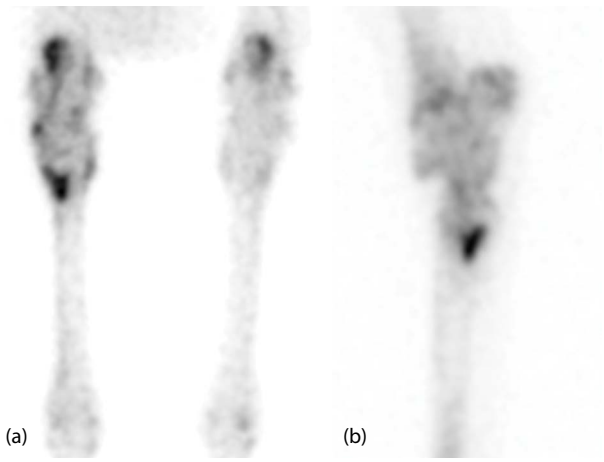


Fig. 6.112 **Plantar (a) and lateral (b) scintigrams showing marked IRU associated with bone pathology at proximal metatarsal origin of hindlimb proximal suspensory ligament.**

Management

- Acute lameness: conservative management generally recommended; stable rest/walking for 6–12 weeks.
- Low-grade or more chronic lameness (with acceptable ultrasonographic appearance): periligamentous infiltration of corticosteroid may permit continued training. Poor or short-lived response necessitates further imaging or rest period.
- While adjunctive therapies (e.g., ESWT, intra-lesional medication/biological therapies) have been prescribed for this condition, good quality evidence that they significantly improve athletic outcome is lacking.

Prognosis

- Most horses respond favourably to rest +/or periligamentous medication.
- Acute-onset lameness is associated with better long-term outcome than chronic lameness.
- Recurrence of lameness due to persistent compartment syndrome may occur in some horses: guarded prognosis for racing.

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- Surgery (releasing fasciotomy plus neurectomy of deep branch of lateral plantar nerve) carries good prognosis for return to soundness in horses with satisfactory conformation; racing following neurectomy is prohibited in most jurisdictions.

Avulsion fracture of proximal suspensory ligament origin

Small avulsion fracture with focal associated damage to proximal suspensory ligament; predominantly occurs in forelimb. May represent 'end-stage' enthesopathy in some horses.

Cause

- Avulsion fracture of suspensory ligament attachment.
- Often preceded by stress remodelling at bone/ligament interface.

Risk factors

- Rare condition.
- Occurs in horses in cantering/fast exercise phase of training.
- Risk factors poorly understood.
- Horses trained on in face of developing subcarpal lameness (aided by systemic/local anti-inflammatory medication) may be at greater risk.

History

- Typically acute-onset lameness noted after cooling-off from exercise.

Signs

- Moderate-severe unilateral lameness.
- Palpable heat/thickening in proximal cannon region.

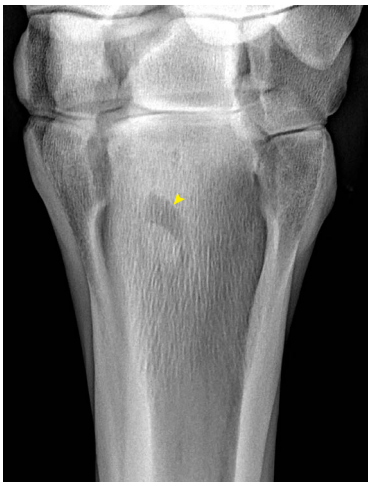


Fig. 6.113 Avulsion fracture of proximal MC3: Radiolucency readily evident on DPa radiograph.

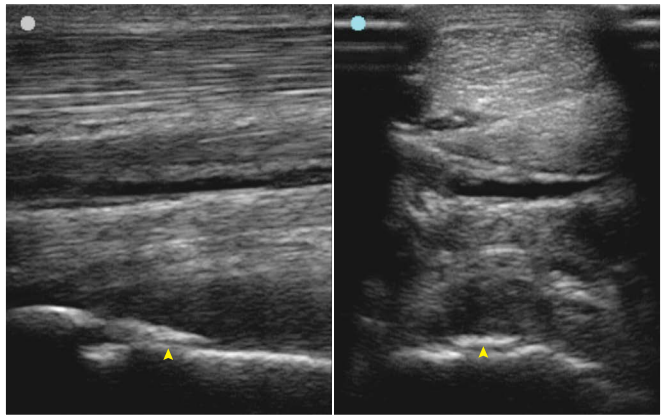


Fig. 6.114 Avulsion fragment visible on longitudinal (left) and transverse (right) ultrasonograms.

- Marked pain response to palpation of suspensory ligament origin.

Diagnosis

- Radiography: crescent-shaped lucency in proximal cannon (DPa projection) (Figure 6.113).
- Ultrasonography: fracture fragment and associated torn suspensory ligament readily detected on longitudinal and transverse images (Figure 6.114).
- Bone scan: rarely required for diagnosis. Intense focal IRU at site of injury.

Management

- Conservative management: requires longer rehabilitation period than for simple proximal suspensory ligament desmopathy; stable rest/walking for 12–18 weeks with ultrasonographic monitoring.
- While adjunctive therapies (e.g., ESWT, intra-lesional medication/biological therapies) have been prescribed for this condition, good quality evidence that they significantly improve athletic outcome is lacking.

Prognosis

- Good prognosis for return to full use with appropriate rehabilitation. Inadequate rehabilitation period associated with high likelihood of recurrence.

Accessory ligament of the DDF tendon (AL-DDFT/'inferior check ligament') injury

The AL-DDFT prevents overloading of the DDFT by sharing the load during the late stance phase. Injuries occur only sporadically in flat racehorses and predominantly in the forelimb (hindlimb AL-DDFT is small/vestigial structure). Usually unilateral. Typically affects the ligament near its junction with the DDFT in the

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mid-cannon region, and disruption at site of injury is often extensive.

Cause
- Strength and elasticity of ligament decreases with age.
- Likely to result from acute overload during high-intensity exercise, although role of chronic degeneration poorly defined.

Risk factors
- Rare condition.
- Usually an injury of older horses.

History
- Acute-onset soft-tissue thickening (cannon) +/– lameness.

Signs
- Soft-tissue thickening at junction of the mid and upper third of the cannon, immediately forward of the flexor tendon bundle.
- Mild pain response to palpation.
- +/– mild-moderate lameness.

Diagnosis
- Clinical findings strongly indicative.
- Ultrasonography: obvious enlargement/loss of echogenicity/loss of border definition of ligament (Figure 6.115).

Management
- Conservative management: stable rest/walking for 8–12 weeks (considerably shorter rehabilitation periods possible for mild injuries).

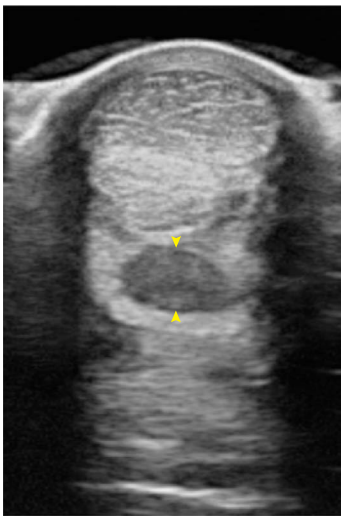


Fig. 6.115 **Ultrasonogram (transverse) showing hypoechoic core lesion in AL-DDFT in mid-upper metacarpus.**

- Systemic and topical (cold therapy) anti-inflammatory measures in initial phase.
- Adhesions to DDFT or SDFT may occur during healing; may cause transient inflammation/thickening on return to faster exercise. Of no concern unless lame.
- Rarely, chronic or recurrent lameness on return to training: surgery (desmotomy) considered for these cases.

Prognosis
- Good (>70%) prognosis for return to full use.
- Risk of recurrence low but affected horses may be at greater risk of other soft-tissue overload injuries (suspensory ligament/SDFT) on return to fast exercise.
- Permanent thickening at site of injury can be expected; cosmetic blemish only.
- Affected horses appear predisposed to AL-DDFT injury in opposite limb during subsequent training; may imply degenerative cause.

Dorsal metacarpal disease ('sore/bucked shins')

Soreness and palpable thickening of front of cannon(s) of variable severity. Predominantly a clinical problem of forelimbs only, but subclinical thickening of hindlimb cannons not uncommon (soreness/lameness associated with hindlimb cannons can occur but very rare) (Figure 6.116a,b); unilateral or bilateral. Pain/inflammation associated with acute phase can interrupt training; permanent bony thickening of affected part of cannon may result in some cases.

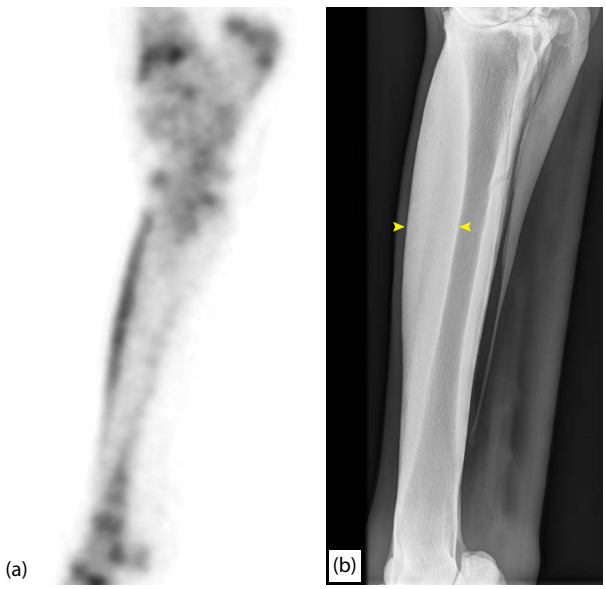


Fig. 6.116 **'Bucked shin' in hindlimb: Increased bone activity at site on lateral scintigram (a) and markedly thickened dorsal cortex on LM radiograph (b).**

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Cause
- Maladaptive modelling response of cannon bone to training-imposed stresses: at slower paces, dorsopalmar bending of cannon occurs and tensile ('stretching') forces act on the front of the shin; at faster paces, the predominant force is compression.
- Tension stimulates deposition of new bone at front of cannon bone, causing dorsomedial thickening.
- Even though greater clinical severity may coincide with the onset of high-speed exercise, cause is actually long, slow cantering phase of training, which does not prepare the cannon bone satisfactorily for fast work.

Risk factors
- Common in 2 YOs during cantering/fast exercise phase of training.
- Horses in their first training cycle (regardless of age) at greater risk.
- Prevalence varies widely with training programme/geographical location; can be as high as 20–40%.
- Greater risk on harder/faster tracks due to higher strains on forelimb.

History
- Initial palpable heat/soreness/thickening of cannon(s) frequently noted in day(s) after fast exercise.
- Some horses display reluctance to train freely at speed.

Signs
- Palpation: tenderness/heat to front of cannon; usually affects dorsomedial aspect of upper half of cannon. May be focal or diffuse.
- +/– swelling/thickening of contour of cannon at same site.
- Severity varies between individuals.
- +/– shortening of forelimb action (bilateral).
- Occasionally: reluctance/resistance to train or engage fully with fast work.
- Unilateral lameness is rare and more typically associated with progression to cortical stress fracture.

Diagnosis
- Clinical findings are definitive.
- Radiography only required if lame and cortical stress fracture suspected.

Management
- Determined by severity and horse/trainer factors.
- Mild pain with minimal 'bucking': often requires no alteration in training, although intermittent exacerbation with fast exercise/racing may necessitate short periods of reduced exercise +/– anti-inflammatory medication.
- Moderate-marked pain ('grunting' response to palpation): treat symptomatically with reduced exercise until pain diminishes. 2–4 weeks without cantering generally sufficient; may walk/trot/swim.
- Acute phase: topical anti-inflammatory measures (cold therapy/clay) useful.
- Continued training without rest may result in permanent change in profile of cannon (cosmetic blemish only). Small associated risk of development of dorsal cortical stress fracture (p. 168).
- Chronic/recurrent cases non-responsive to rest: counter-irritation (p. 58) +/– periosseous deposition of corticosteroid associated with successful resolution in some troublesome cases.

Prevention
- Exposure to faster speeds (and therefore more 'compression' force on cannon) stimulates better adaptive modelling of bone.
- Introduction of fast work at earlier stage in training programme, providing distances trained at speed are restricted (initially ≤200 m) may be useful.
- Intentionally 'bucking' shins not recommended.

Prognosis
- Excellent prognosis for return to full use.
- Interruption to training uncommon and rarely more than a few weeks.
- Very few horses subsequently develop dorsal stress fracture of the cannon and these can usually be readily differentiated clinically.

Splints (metacarpal/metatarsal exostosis)
Inflammation and enlargement of the small metacarpal or metatarsal (splint) bones. Severity varies considerably between individuals. Most frequently affects the medial splint of the forelimb; only rarely in hindlimb (when it is usually lateral). Bilateral lesions are common. Splint fractures sometimes arise as a more severe manifestation of the condition or secondary to direct trauma or fetlock overextension.

Both 'true' splints and splint fractures go through a period of active callus formation before 'setting' as a non-painful permanent bony thickening (Figure 6.117a–c).

Cause
- Medial splint bone of forelimb bears considerable load (along with cannon); splint enlargement may represent maladaptive response to these loads.

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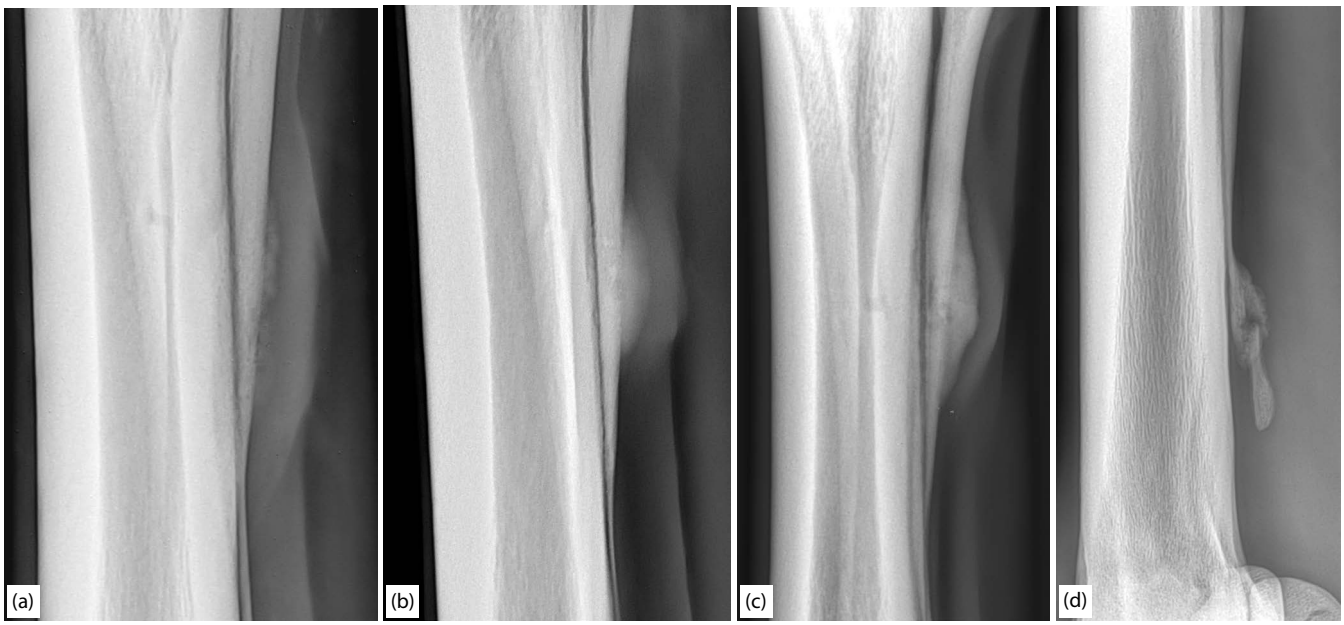


Fig. 6.117 **Splint enlargement: (a–c) radiological progression of callus maturation from acute phase (a) to 'set' (c). Distal ('button') splint fracture (d).**

- Shear forces acting on interosseous ligament between splint bone and cannon are the cause of many 'splints'.
- Direct trauma (typically interference injuries) can also cause periosteal reaction/fracture.
- Fracture of the distal ends ('buttons') of splints (Figure 6.117d): fatigue injury caused by physical deviation following SLB desmopathy (p. 146) or secondary to overextension of fetlock (fibrous attachment to PSBs).

Risk factors

- Any age/stage of training but most common in 2 YOs in early cantering phase.
- Offset carpal conformation predisposes to injury (greater load bearing through medial splint rather than cannon).

History

- Chronic- (most common) or acute-onset lameness +/– swelling; typically develops over days/weeks.

Signs

- Initially: pain on palpation +/– oedema/soft-tissue thickening at affected site.
- Followed within days by palpable thickening of underlying splint bone.
- Splint enlargement: lameness a variable feature (absent-moderate).
- Splint fracture (proximal and mid-body): moderate-severe lameness (+/– overlying wound in cases of direct trauma).
- Splint fracture (distal): mild lameness plus poorly defined thickening of the inside of the lower cannon/fetlock. Occasionally subclinical.
- Severity of lameness and palpable pain response corresponds broadly to degree of resulting splint enlargement.

Diagnosis

- Clinical findings sufficient in most cases and imaging rarely necessary.
- Radiography: generally unnecessary but warranted if concern over clinical severity, or splint fracture suspected; multiple lesion-oriented oblique projections may be required to detect fracture line (if present).

Management

- Clinical severity and lameness guide management.
- Acute phase: anti-inflammatory therapy (topical/systemic, cold therapy).
- If no lameness: interruption to training programme not necessary; however, short period (1–2 weeks) of walking/trotting beneficial to allow splint to settle.
- If lame: reduce level of exercise (to walking/trotting) until splint 'sets' (palpable pain and lameness subsides).

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- Recurrent/problematic despite rest: counter-irritant measures (p. 58) may be considered.
- Lateral splints: frequently slow-setting even when rested; light ridden exercise therefore preferable to complete rest (unless precluded by lameness). If lameness absent/mild, continued full training may be possible and does not compromise outcome providing monitored clinically.
- Fractures require considerably longer rehabilitation period than splint enlargement.
- Open/comminuted splint fractures: surgical intervention may be warranted.
- Distal splint fractures: most resolve with conservative management (painless non-union). Surgical removal rarely necessary.
- Rarely, splint enlargement may impinge upon/adhere with margin of suspensory ligament and cause chronic lameness. Definitive diagnosis difficult with radiography/ultrasound/bone scan and may require MRI (preferably high-field MRI). Management is surgical: removal of splint bone to above the level of affected site.

Prognosis
- All carry good prognosis for return to full use.
- Initial severity of lameness/palpable pain is a good predictor of length of interruption to training required.
- Medial splints: generally minimal interruption to training; 1–4 weeks out of cantering is typical.
- Lateral splints: more likely to be of low-grade clinical concern for extended period; may remain lame for many weeks/months.
- Splint fractures: may require rehabilitation period of 8–12 weeks.

Palmar cortical stress reaction/fracture
Bone fatigue injury of upper cannon, predominantly in forelimb. Along with proximal suspensory ligament desmopathy (p. 159), is an important cause of subcarpal lameness in young racehorses. Primarily affects palmar cortex/medulla in region of attachment of medial lobe of suspensory ligament, sometimes with associated inflammation of medial splint bone and interosseous ligament. Spectrum of injury ranges from stress reaction to incomplete cortical fracture. Usually unilateral.

Cause
- Stress/fatigue injury.
- Distraction forces from suspensory +/– interosseous ligaments may contribute to strains on palmar cortex of MC3.

Risk factors
- Common condition.
- Typically in 2 YOs during trotting/early cantering phase of training.
- Risk factors not known.

History
- Acute-onset lameness.

Signs
- Moderate-marked unilateral forelimb lameness.
- Frequently no localizing signs.
- +/– pain response to palpation of subcarpal region.

Diagnosis
- Confirming diagnosis can be challenging as conventional imaging (radiography/ultrasonography) frequently unremarkable or ambiguous.
- Diagnostic blocking: may not permit differentiation of subcarpal from middle carpal joint lameness.
- Radiography: radiological changes not present in all cases (up to 30% without abnormality) at time of injury but may develop over subsequent months. Increased densification +/– vertical/oblique linear lucency in medial proximal cannon (DPa projection) (Figure 6.118a,b); endosteal reaction (LM projection).
- Ultrasonography: suspensory ligament usually unremarkable; +/– periosteal irregularity proximal cannon.
- Bone scan: intense focal IRU (Figure 6.119a,b).
- MRI: the gold standard imaging modality due to inconsistent radiological/ultrasonographic features of condition. Fluid signal +/– cortical fissure (Figure 6.120).

Management
- Conservative management: stable rest/ walking for 6–8 weeks depending on severity.

Prognosis
- Good prognosis for return to full athletic use.
- Some horses subsequently develop low-grade middle carpal joint lameness later in career; may represent ongoing multi-site maladaptive response to loading of limb.

Dorsal cortical stress fracture
Fracture of the dorsal cortex of the diaphysis of the cannon bone. Predominantly in forelimb. Can develop in upper, mid- or lower cannon and usually located dorsolaterally. Typically diagnosed as a short oblique fracture line, but may progress to complete fracture of the metacarpal/tarsal diaphysis.

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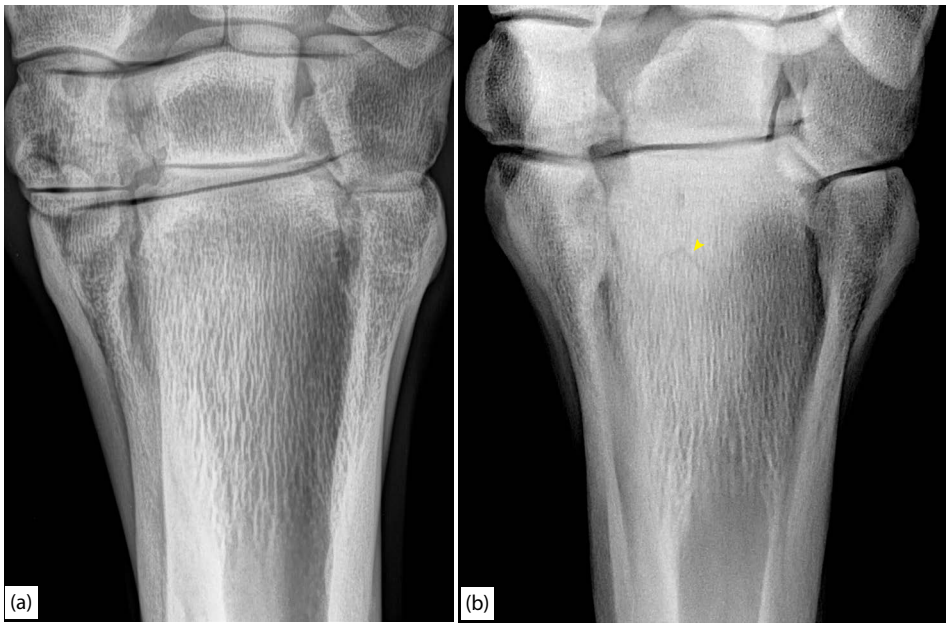


Fig. 6.118 Palmar metacarpal fatigue injury: Normal (a) and injured (b) limbs (DPa), with irregular radiolucency (arrowhead) noted in latter.

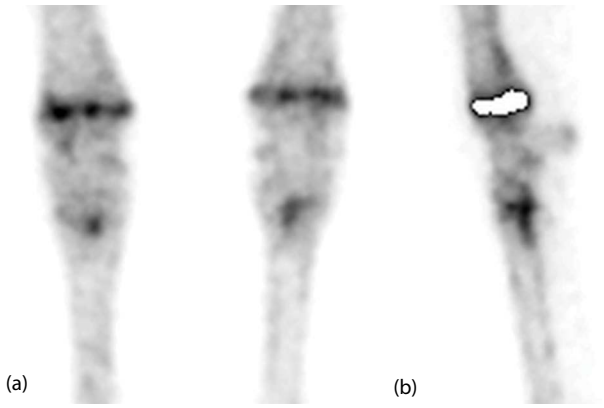


Fig. 6.119 Palmar metacarpal fatigue injury: Marked focal IRU in proximal cannon on dorsal (a) and lateral (b) scintigrams. Pathology is bilateral in image (a).

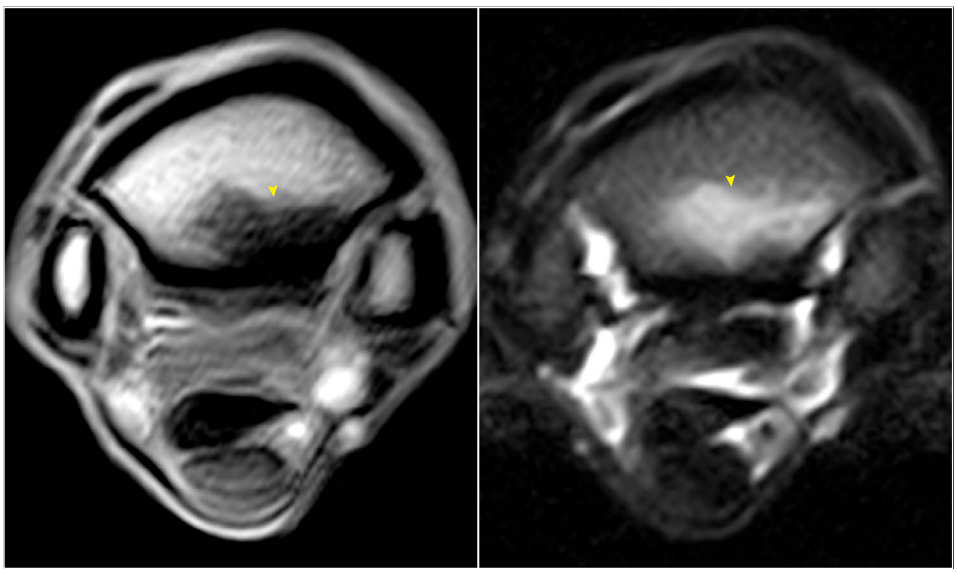


Fig. 6.120 MR T1 (left) and STIR (right) transverse image of right proximal MC3 at level of suspensory ligament origin; medial is to the right in both images. T1 hypointensity and STIR hyperintensity in palmaromedial aspect (arrowheads).

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Cause
- Stress/fatigue injury.
- May develop as progression of bucked shins (p. 165) that have received insufficient rest.

Risk factors
- Rare condition.
- Most common in 3 YOs.
- Risk factors unknown.

History
- Typically chronic-onset lameness (worsening over days/weeks), although may arise acutely following fast exercise/racing.
- May present as acute, catastrophic complete fracture of mid-cannon during fast work/racing.

Signs
- Small (≤1 cm) focal bony thickening of front of forelimb cannon (Figure 6.121a); clinically distinct from 'bucked' shin.
- Considerable focal pain on palpation.
- Mild-moderate unilateral lameness.

Diagnosis
- Clinical findings strongly indicative.
- Radiography: oblique fracture line in dorsal cortex (LM/DPa projections) +/– periosteal/endosteal reaction depending on chronicity of injury (Figure 6.121b–d).

Management
- Continued training without rest may result in complete diaphyseal fracture (catastrophic).
- Conservative management: stable rest/walking for 6–10 weeks.
- Surgery (cortical drilling/screw placement): sometimes advocated for non-healing chronic injuries.
- Complete, displaced mid-diaphyseal fracture: catastrophically severe injury that necessitates immediate euthanasia.

Prognosis
- Fair to good prognosis for return to full use with conservative management.
- Good prognosis with surgery.
- Complete, displaced mid-diaphyseal fracture: hopeless prognosis for survival.

THE CARPUS

Applied anatomy
The carpus ('knee') is a compound articulation between the forearm and cannon and comprises three joints and two rows of carpal bones (Figure 6.122a,b). The primary movement of the carpus is flexion/extension in the sagittal plane. At faster paces full extension occurs just prior to the foot landing; when the carpal joints close, the tight-packed configuration of the small bones keeps the carpus fixed and the leg rigid as the body passes over it during stance.

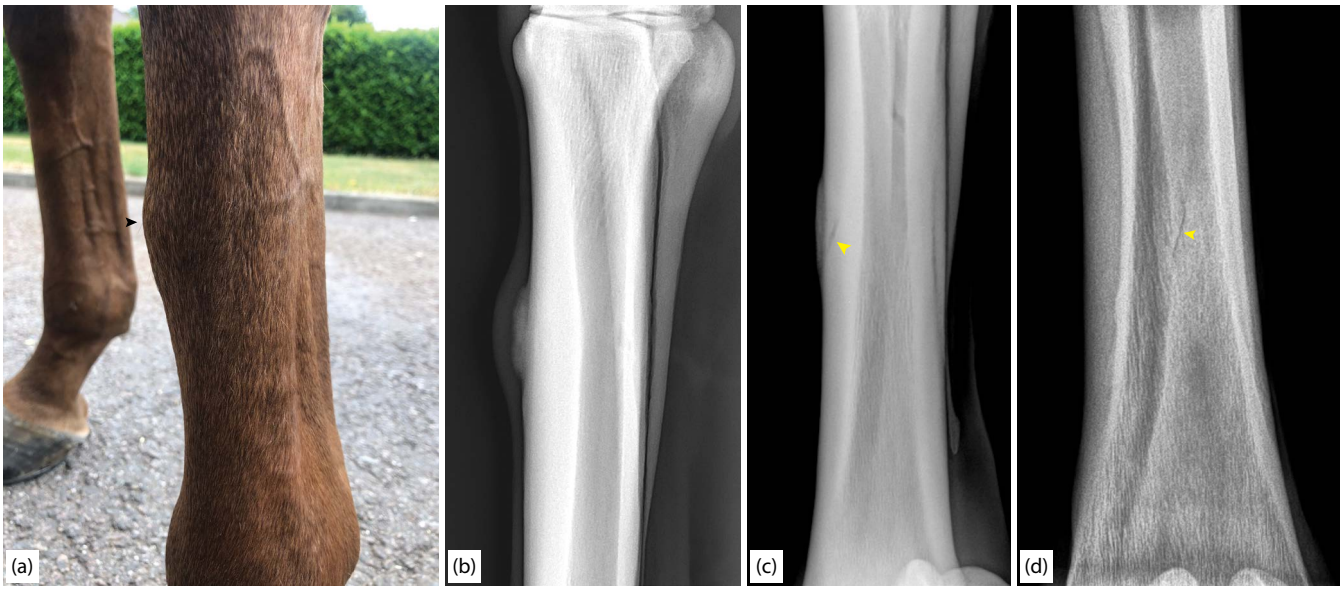


Fig. 6.121 Dorsal cortical stress fracture of MC3: Focal bony thickening of dorsal cannon (a) corresponding to bone callus at this site (b); linear radiolucency (arrowhead) is an inconsistent feature but may be visible on oblique (c), DPa (d) or LM radiographic projections.

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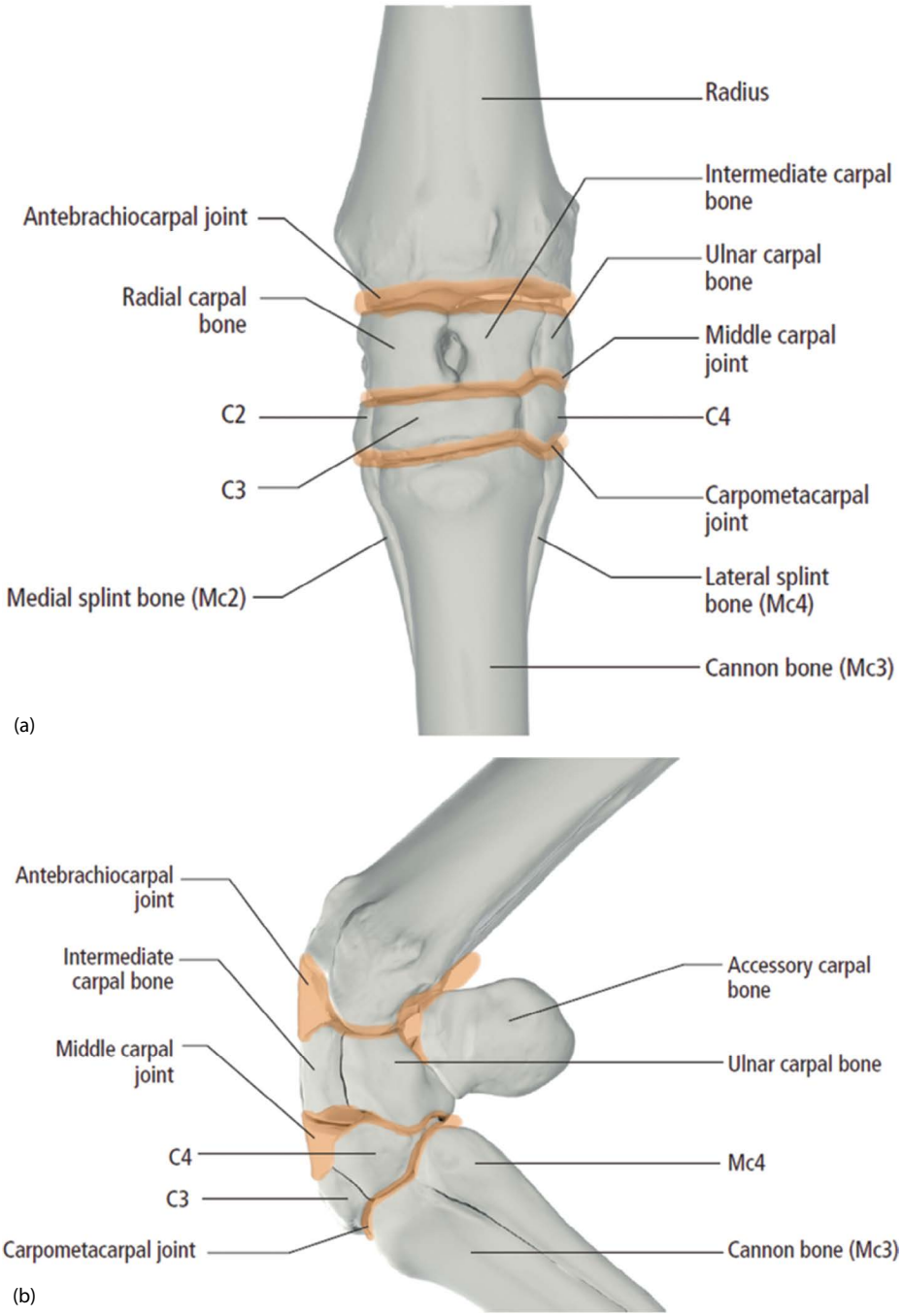


Fig. 6.122 **Major structures of the carpus: (a) dorsal and (b) flexed lateral views.**

The lowermost (carpometacarpal) carpal joint does not 'open' and is rarely associated with problems. This joint is separated from the middle carpal joint by the distal row of carpal bones (C2, 3, 4). The two high-motion (middle carpal and antebrachiocarpal) joints are separated by the proximal row of carpal bones (radial, intermediate and ulnar carpal bones). The accessory carpal bone sits prominently at the back of the proximal row, articulating with the ulnar carpal bone and lower radius. It forms part of the outer border of the carpal canal, through which the flexor tendons course.

The carpal joints are palpable at the front of the carpus between the constraining extensor tendons. All of the joints also have outpouchings at the back of the leg. The palmar pouch of the middle carpal joint is just below the accessory carpal bone and that of the

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antebrachiocarpal joint is on the outside of the lower forearm, between the back of the radius and the tendon of the ulnaris lateralis muscle.

The carpal bones are held together by several intercarpal and collateral ligaments; these provide stability and prevent overextension. Two large extensor tendons (the extensor carpi radialis and common digital extensor) run over the front of the carpus. The retinaculum that restrains the extensor tendons at the front of the carpus also forms the thick medial and palmar borders of the carpal canal. The carpal canal contains the carpal synovial sheath, which encloses the SDFT and DDFT and extends above the carpus for 8–10 cm and below the carpus to mid-cannon (with the distal recess terminating at the junction of the DDFT and the AL-DDFT). At the back of the carpal bones the dense palmar carpal ligament is the foundation of the AL-DDFT ('inferior check' ligament), which runs down into the cannon to its union with the DDFT. The AL-SDFT ('superior check' ligament) is a fan-shaped band that arises from the caudomedial radius and joins the SDFT at the back of the carpus.

The growth plate of the lower radius (immediately above the carpus) has traditionally been used to assess skeletal maturity. Bone turnover at this growth plate starts to subside rapidly at around 20 months of age, and radiological 'closure' occurs at 24–30 months (p. 14).

Load-bearing compression forces act primarily through the 'dorsal load path' at the front of the limb, and training stimulates increased density of the struts of cancellous bone (trabeculae) that are oriented proximodistally. While training adaptations allow for greater load bearing, increased bone density also causes increased stiffness. The majority of training-related pathology occurs within the middle carpal joint. The largest and most important bone in the distal row is the third carpal bone, which has two main load-bearing facets corresponding to the opposing bones in the proximal row. The radial facet (opposite the radial carpal bone) is the largest, is subject to the greatest forces and consequently is most frequently affected by training-related maladaptive changes. Together with the opposing articular margin of the radial carpal bone, these sites form the regions of the carpus most likely to sustain injury (Figure 6.123).

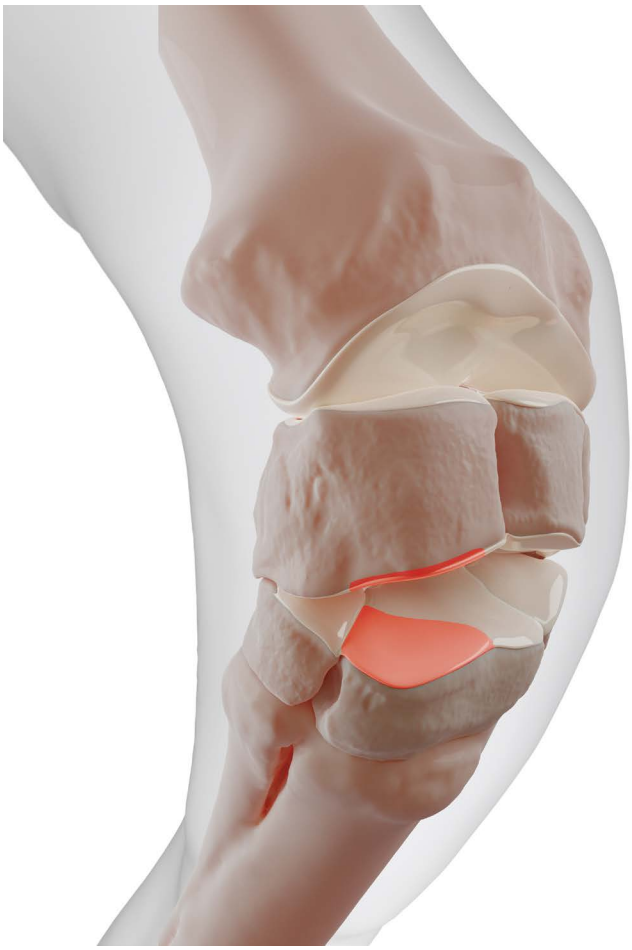


Fig. 6.123 **Dorsomedial view of partially flexed left carpus. Main load-bearing sites (opposing facets of third and radial carpal bones) at which the most important middle carpal joint pathologies occur are highlighted.**

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Synovial spaces

- Carpometacarpal and middle carpal joints: always communicate.
- Middle carpal and antebrachiocarpal joints: rarely communicate.

Examination

The conformation of the carpus (particularly offset knees) may influence the risk of injury and assessment may direct the lameness investigation. Evidence of middle carpal or antebrachiocarpal joint effusion may be subtle and is best discerned by palpation of the front of the knee with the limb weight-bearing. Joint effusion can be differentiated from subcutaneous bursae (arising from direct trauma) by observing the communication between the dorsal and palmar synovial pouches. With the limb raised, firm palpation of the bone borders most prone to injury (dorsodistal radial carpal bone/dorsoproximal third carpal bone/dorsodistal radius) may elicit a pain response, and when it does almost invariably indicates fracture/fragmentation at that site. Forced full flexion of the knee should never be painful and, if so, often indicates marked joint effusion and likely injury.

Carpal lameness

Carpal ('knee') lameness is common in all age groups and can be associated with a spectrum of pathology from subchondral bone injury to OA or fracture +/– ligamentous injury. Progression is unpredictable and considerable individual variation in presenting signs, radiological changes and outcome occurs. Many affected horses experience a phase of clinical activity after which stabilization (characterised by halting of clinical deterioration, or return to relative soundness) occurs; likely that many intrinsic and extrinsic factors determine whether stabilization or deterioration ensues with further training.

Pathology or lameness frequently affects both limbs. The middle carpal joint is the site most frequently involved in carpal lameness; pathology primarily affects the medial portions of the radial and third carpal bones (which face each other across the joint space) but can be complex and multifocal, involving subchondral bone and interosseous ligament/bone interface sites in lower carpus and upper cannon/splint regions. Antebrachiocarpal joint lameness is less common (although fractures in this joint may be more prevalent than the middle carpal joint in some jurisdictions); pathology most commonly affects the distal radius or proximal row of carpal bones (predominantly radial carpal bone; less commonly intermediate carpal bone). Fracture/fragmentation of the lateral aspect of the distal radius is more common than other sites in the antebrachiocarpal joint.

Because of the widespread use of radiography in first opinion practice, carpal pathology is often categorized by radiological appearance. Carpal lameness may be associated with a range of radiological findings; these typically include increased densification or modelling of the third +/or radial carpal bones. Correlation between radiological severity and arthroscopic evidence of OA is poor. Increased density of the third carpal bone is manifested by thickening or loss of trabeculation +/or corticomedullary definition on the DPr-DDiO ('skyline') radiographic projection (Figure 6.124a–g). There is variation between individuals in the area of the bone affected and its severity (mild/moderate/severe) as well as presence of associated lucencies (which may represent subchondral bone necrosis/injury). It is common for one limb to be affected to a greater degree. Increased density predominantly affects the 'radial facet' of the third carpal bone; however, pathology at the 'intermediate facet' (Figure 6.124h) is not unknown but considered far less significant. Osteochondral fragmentation ('chip' fracture) of the articular margin of the radial carpal (Figure 6.125a–h) and/or third carpal bone (Figure 6.126a–h) is often a progression of (or predisposed by) degenerative modelling (Figure 6.127a–i). Fragmentation involving the antebrachiocarpal joint may also arise from degenerative change or may be acute with no obvious pre-existing pathology (Figure 6.128a–h). Incomplete/complete fracture of the dorsomedial proximal forelimb cannon with carpometacarpal joint involvement may occur (Figure 6.129a–c) and is considered to represent an unusual manifestation of pathologic loading through the medial weight-bearing column of the limb. Indeed much distal carpal (middle carpal joint) lameness is probably best considered as a 'medial loading syndrome' that may incorporate a spectrum of adaptation and injury to multiple structures (subchondral bone, cartilage as well as less readily imaged sites such as interosseous/intercarpal ligaments) rather than viewed simplistically in terms of radiological appearance of the main bones.

Configurations of third carpal bone injury include proximal chip, frontal slab (Figure 6.130a–c) and sagittal slab (Figure 6.131a–e) fractures, subchondral bone necrosis/injury and cortical fissures (Figure 6.132a,b); these occur almost invariably in the portion of third carpal bone subject to the greatest load (radial facet), although rarely frontal slab fractures may involve both radial and intermediate facets. Chip fracture/fragmentation affects the proximal articular border of the radial facet, whereas slab fracture commonly occurs in the frontal plane and extends full-thickness between the proximal and distal articular margins. Cortical fissures occur in the proximal

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Fig. 6.124 **Spectrum of radiodensity encountered in third carpal bones on 'skyline' (DPr-DDiO) radiographic projection of distal row of carpus: (a) no apparent trabecular thickening; (b) mild and (c) moderate increased density of radial facet; (d) gradient of increasing density toward medial (arrowheads) aspect of bone; (e) marked density, with complete loss of visible trabecular pattern; (f) and (g) marked density with dorsal lucencies; (h) increased density of intermediate facet (arrowheads).**

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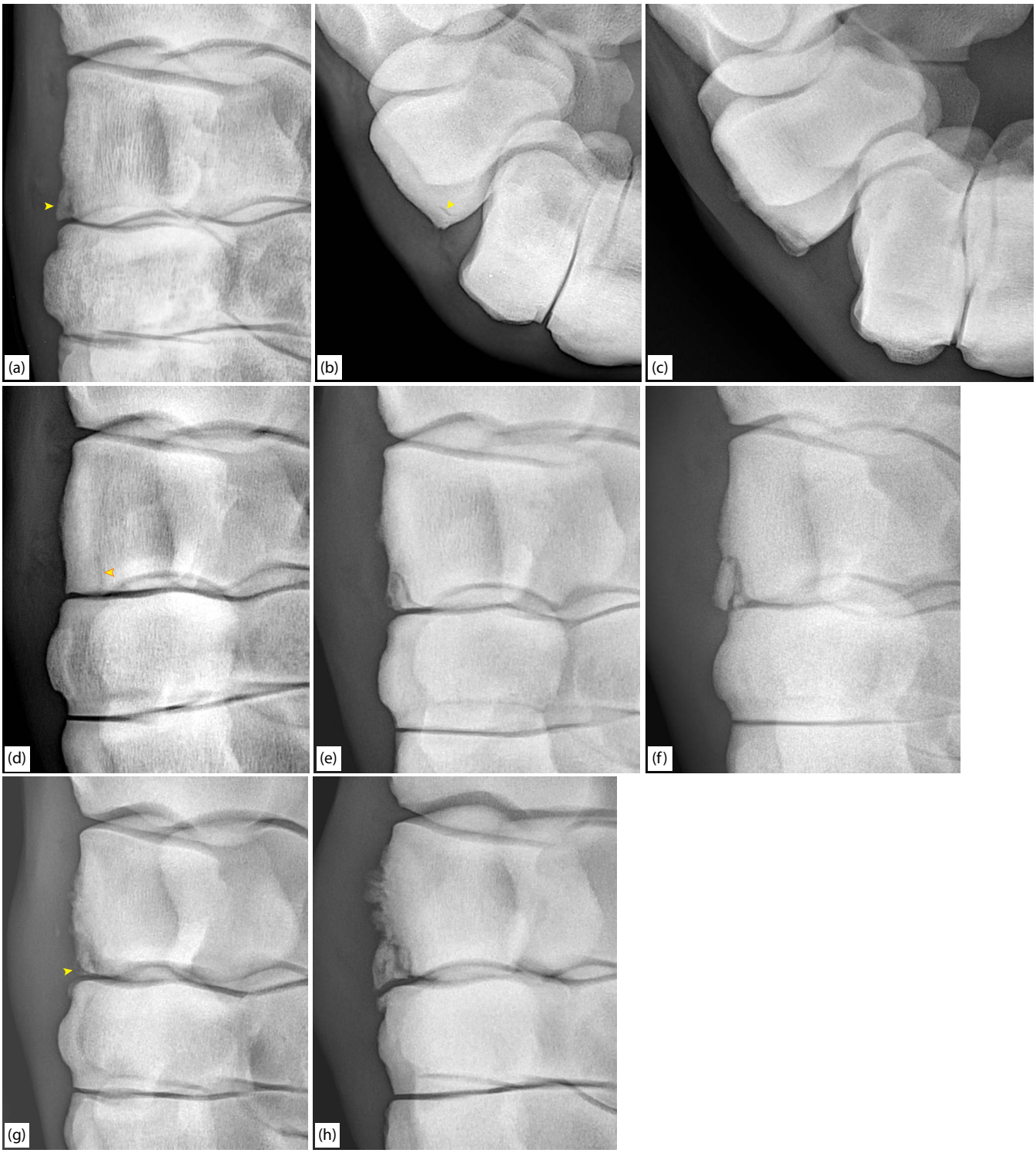


Fig. 6.125 **Osteochondral fragmentation of distal radial carpal bone. (a) modelled/partially displaced 'chip' (DLPaMO); (b) non-displaced recent fracture (flexed LM); (c) partially displaced chip of long-standing appearance; (d) indistinct linear lucency associated with large, non-displaced fracture; non-displaced (e) and displaced (f) medium-sized chips; long-standing minor (g) and major (h) fragmentation with secondary entheseal modelling of dorsal radial carpal bone.**

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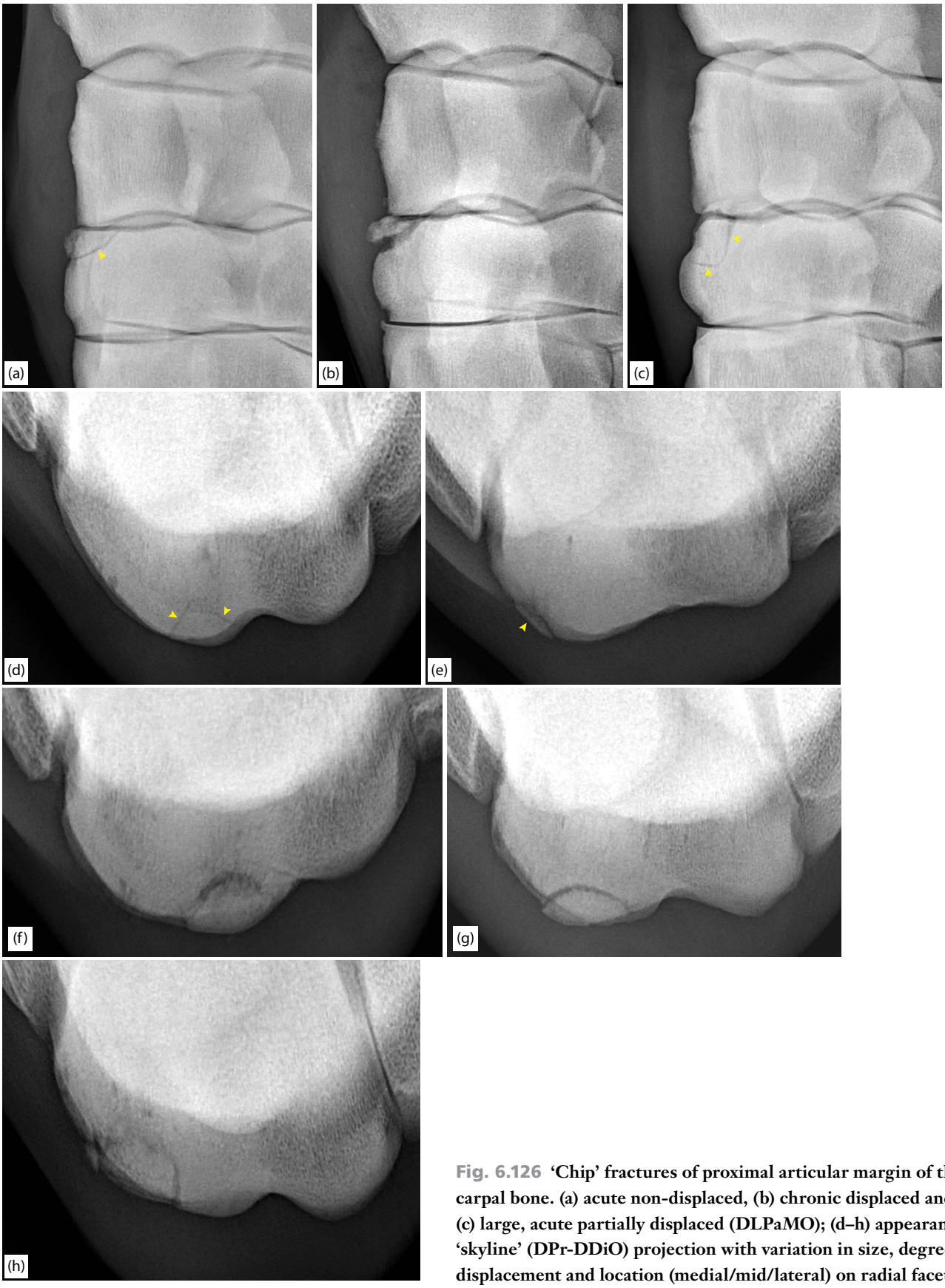


Fig. 6.126 **'Chip' fractures of proximal articular margin of third carpal bone. (a) acute non-displaced, (b) chronic displaced and (c) large, acute partially displaced (DLPaMO); (d–h) appearance on 'skyline' (DPr-DDiO) projection with variation in size, degree of displacement and location (medial/mid/lateral) on radial facet.**

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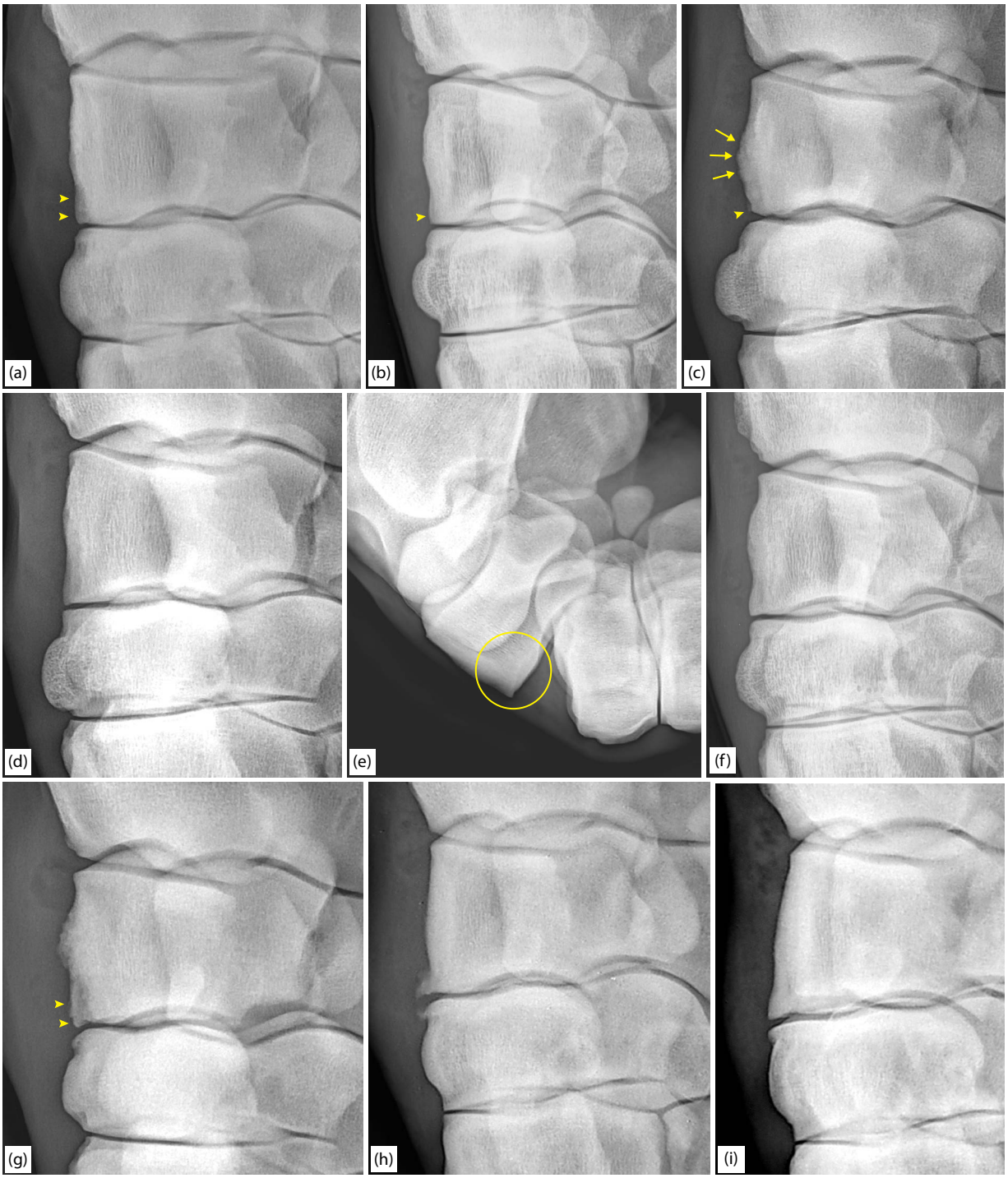


Fig. 6.127 Modelling of dorsodistal radial carpal bone (arrowheads): Range of radiological appearance (DLPaMO) encompassing adaptive and pathologic change. (a) lytic appearance generally associated with activity at site; (b) smooth margin and regular bone density associated with 'settled' modelling; (c) 'cut back' articular modelling, as well as chronic entheseal modelling (arrows) of dorsal margin of bone; (d) angular profile but settled appearance; (e) minor spurring of articular margin but notably increased subchondral density (circled) (flexed LM); (f) spurring of settled appearance; (g) lytic appearance likely due to osteochondral fragmentation (further projections required for characterisation). Modelling is a dynamic process and radiological appearance of this site may change over time: Very active modelling of opposing margins of radial and third carpal bones detected at 2 YO in (h) settled into angular but relatively benign appearance (i) by 4 YO (elite performer as mature racehorse in this case).

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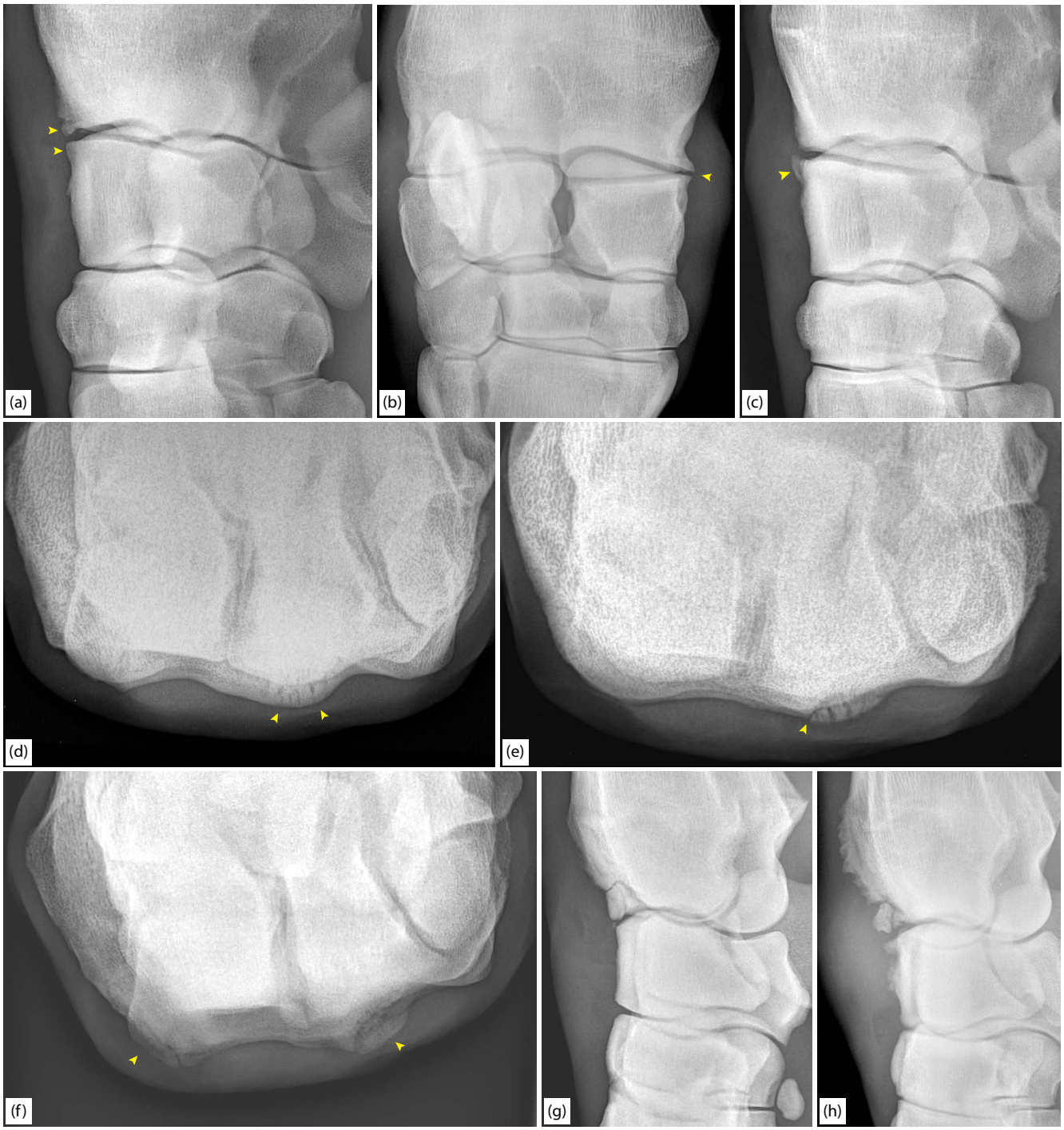


Fig. 6.128 **Antebrachiocarpal joint radiological abnormalities: Articular modelling of distal radius +/–/or proximal row (a, b); osteochondral fragmentation of proximal row (c); modelling (d) and chip fracture (e, f) of distal radius on 'skyline' radiographic projection (all left forelimb; medial to left); injury sites may be medial, mid-, lateral or multi-focal. Large, displaced acute chip fracture of distal radius (g) with similar but chronic injury (and secondary OA) in image (h) (DMPaLO).**

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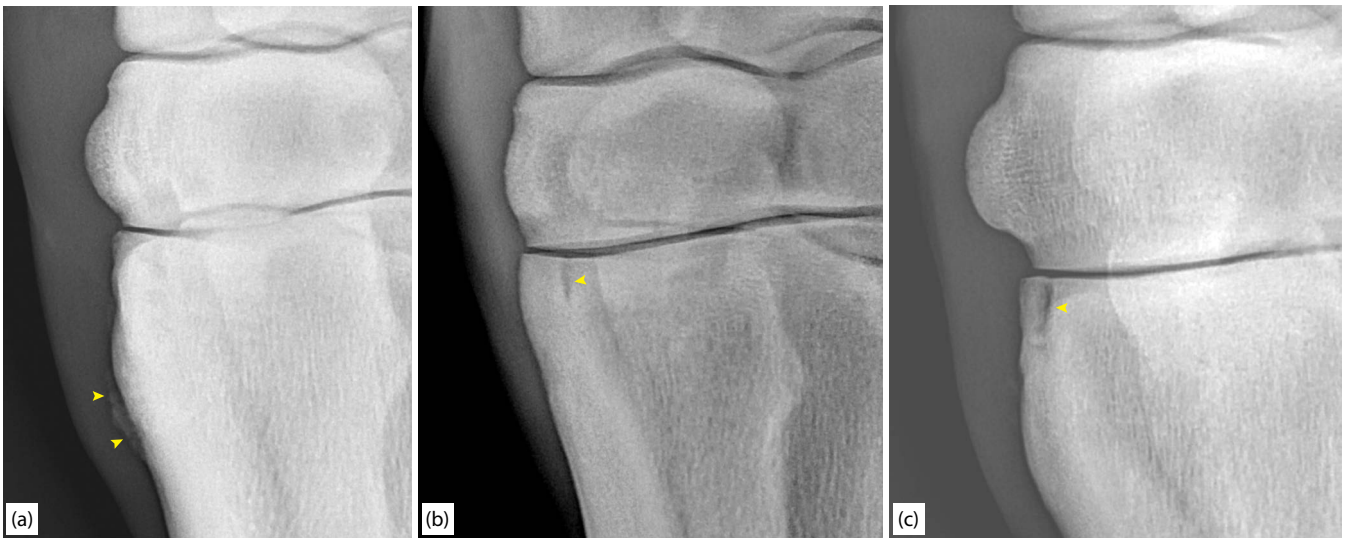


Fig. 6.129 **Fracture of dorsomedial proximal MC3 with carpometacarpal joint involvement: Radiolucent fracture line not always visible initially but distal periosteal reaction (a) is definitive. Multiple (or delayed) DLPaMO radiographic projections may be required to characterise injury (b, c).**

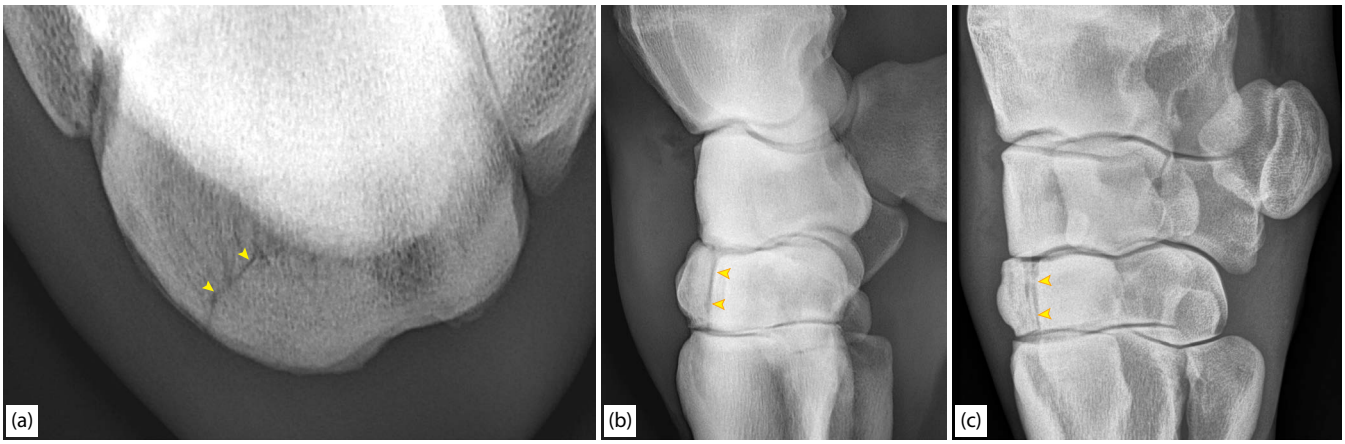


Fig. 6.130 **Third carpal bone frontal slab fracture. 'Skyline' (DPr-DDiO) (a), LM (b) and DLPaMO (c) projections all useful for diagnosis and to assess displacement.**

weight-bearing face of the bone and may require differentiation (through CT/MRI) from (benign) radiologically conspicuous vasculature (Figure 6.133). Pathology may also (less commonly) occur further back in the third carpal bone than the radiographic 'field of view', and in these cases diagnosis must rely on multiplanar imaging (CT/MRI) (Figure 6.134). The third carpal bone is the bone within the knee most commonly affected by slab or severe comminuted fractures (Figure 6.135a,b).

Modelling and/or fragmentation of the distal intermediate carpal bone (Figure 6.136) is uncommon but is most frequently encountered as an incidental finding in yearlings (at the time of screening radiographic examinations). In most cases it is clinically insignificant and its presence is not considered predictive for future carpal lameness.

Palmar carpal fracture/fragmentation is encountered only rarely. When discrete and involving the ulnar carpal bone it represents an avulsion injury of the lateral palmar intercarpal ligament. Fracture of the palmar border of the radial carpal bone (+/– ulnar or intermediate carpal bones) is usually a traumatic injury sustained during anaesthetic recovery or a fall (Figure 6.137).

Cause

- Middle and antebrachiocarpal joints are high motion.

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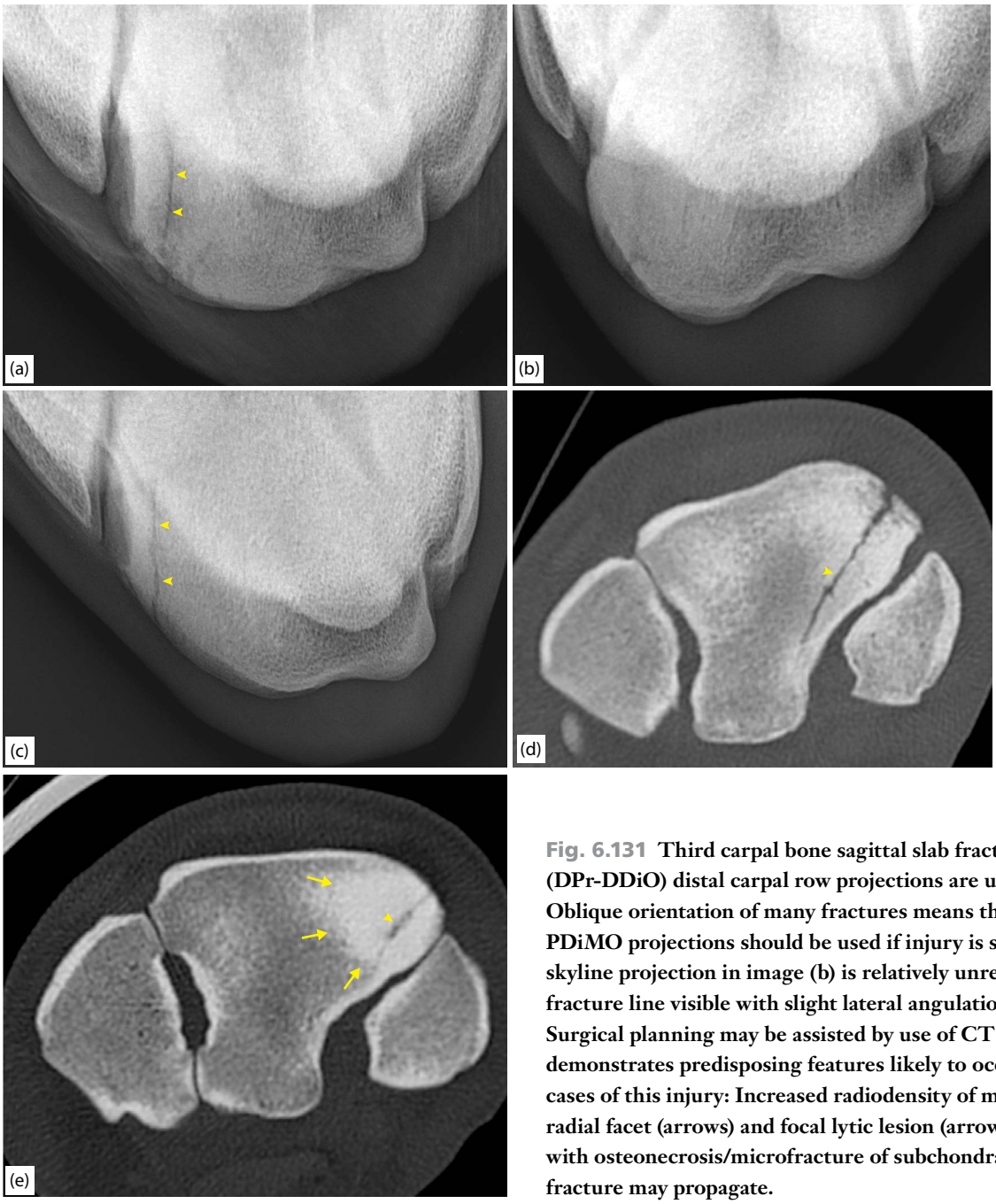


Fig. 6.131 **Third carpal bone sagittal slab fracture: 'Skyline' (DPr-DDiO) distal carpal row projections are used for diagnosis (a). Oblique orientation of many fractures means that additional DPrL-PDiMO projections should be used if injury is suspected: 'standard' skyline projection in image (b) is relatively unremarkable; however, fracture line visible with slight lateral angulation of radiograph (c). Surgical planning may be assisted by use of CT (d). CT image (e) demonstrates predisposing features likely to occur in many/most cases of this injury: Increased radiodensity of medial portion of radial facet (arrows) and focal lytic lesion (arrowhead) consistent with osteonecrosis/microfracture of subchondral bone from which fracture may propagate.**

- Dorsal margins of third and radial carpal bones subjected to considerable compressive forces during weight-bearing at faster paces.
- Loading forces in the lower knee (specifically the middle carpal joint) are greatest through the medial portion of knee and proximal cannon.
- Injury patterns suggest that during high-speed exercise on a turn, loading forces through the inside leg are greatest at the lateral aspect of the antebrachiocarpal joint; and through the outside leg are greatest at the medial aspect of the middle carpal joint.
- Training causes adaptive change; in some individuals it becomes maladaptive, leading to subchondral bone injury +/– intercarpal ligamentous tearing/avulsion +/– carpal joint instability.
- Increased density of subchondral bone/associated cartilage damage can lead to reduced compliance and greater susceptibility to fracture (chip/fragmentation/slab fracture). However, fractures frequently occur in

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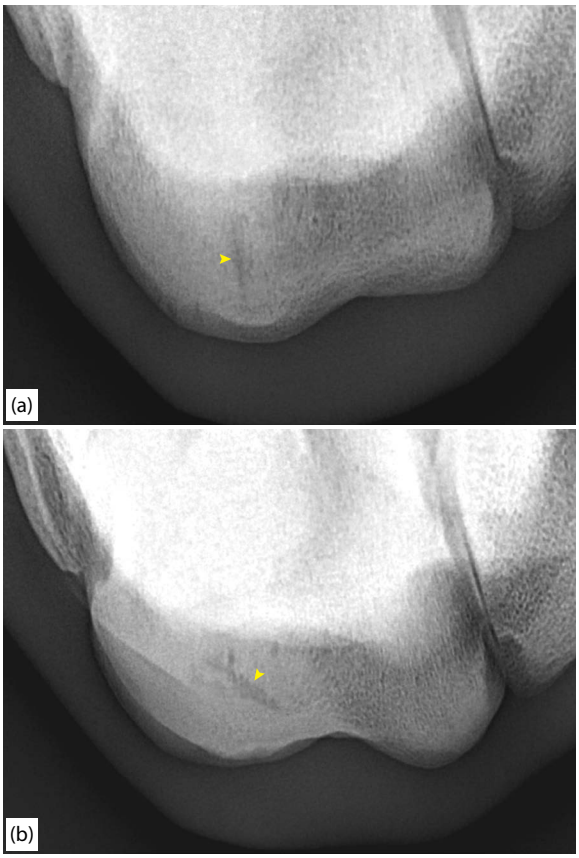


Fig. 6.132 Sagittal (a) and frontal (b) linear radiolucencies (arrowheads) in third carpal bone consistent with cortical 'fissure' fractures; the latter are generally viewed as presenting greater risk of propagating to complete fracture (unless rehabilitated).

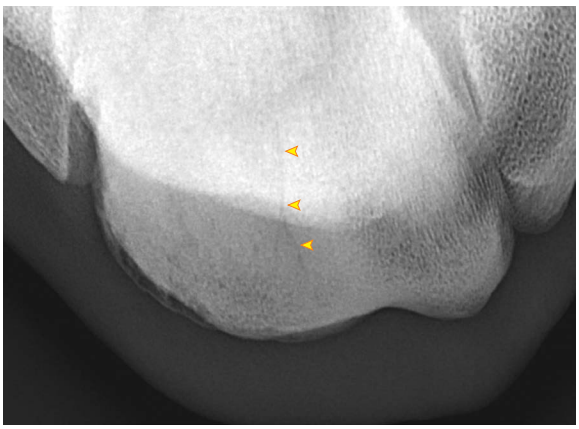


Fig. 6.133 Branching linear lucency (arrowheads) in third carpal bone with appearance of cortical fissure but actually resulting from conspicuous vasculature (confirmed with MRI); carpal lameness in this case was unrelated to any prodromal fracture.

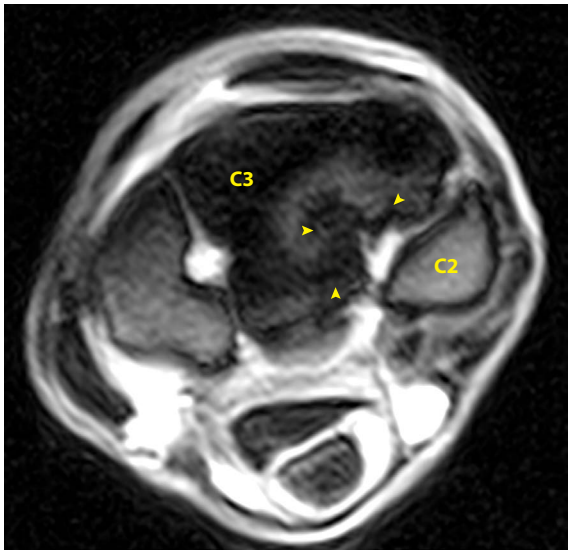


Fig. 6.134 Endosteal and periosteal changes at insertion of intercarpal ligament between C2 and C3 (arrowheads), consistent with entheseal ligament/bone strain injury at this site (transverse low-field MRI).

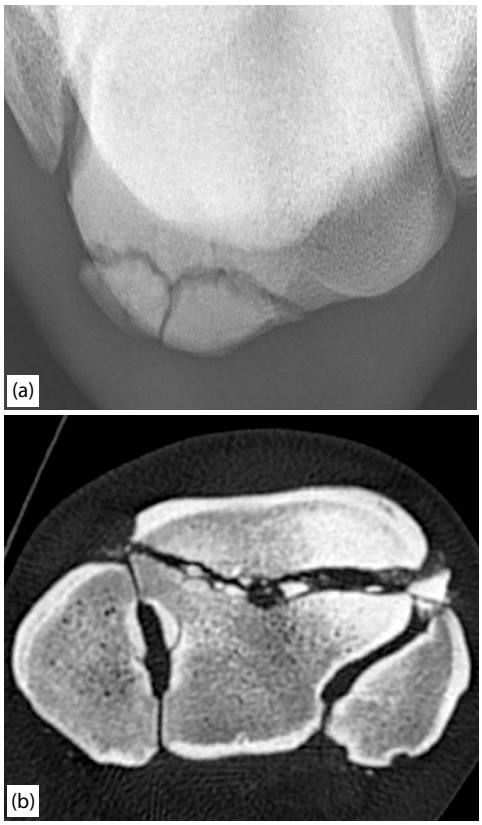


Fig. 6.135 Comminuted frontal slab fracture involving radial facet (a) (DPr-DDiO) and entire third carpal bone (b) (CT).

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Fig. 6.136 **Osteochondral fragmentation of distal border of intermediate carpal bone (flexed LM).**



Fig. 6.137 **Frontal slab fracture of intermediate carpal bone sustained during anaesthetic recovery.**

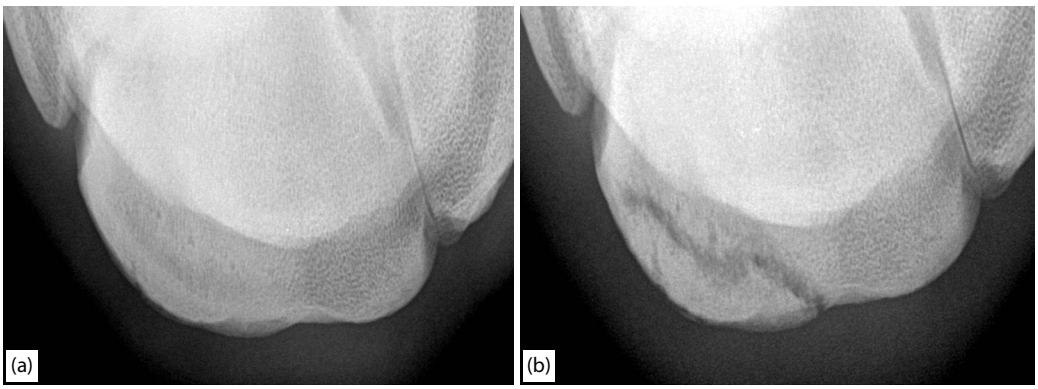


Fig. 6.138 **(a) 'Pre-fracture' appearance of third carpal bone (DPr-DDiO) showing relatively normal trabeculation/density of radial facet, and (b) frontal slab fracture of same bone 6 weeks later.**

non-sclerotic third carpal bones and the relationship is not linear or predictable (Figure 6.138a,b).

Risk factors

- Clinical signs encountered at any age/stage of training, however, overrepresentation of 2 YOs in some jurisdictions suggests that number of starts and running speed may be less important than other potential risk factors.
- Poor carpal conformation: depending on severity, 'offset' (middle carpal joint lameness) and 'back-at-knee' (antebrachiocarpal joint lameness) conformation may increase risk.
- Other risk factors for development and/or progression of carpal pathology are poorly understood but may include body type/condition (heavy), maturity, training regime and track surface.
- Prevalence varies considerably between jurisdictions; however, estimated that between 5–10% of horses in training may develop clinical carpal pathology.

History

- Many affected horses conform to the following general features:
  - Low-grade lameness that typically warms up with exercise.

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- Pathology often bilateral and may present as wide-based forelimb action rather than overt lameness.
- Insidious progression over weeks/months.
- More severe, acute lameness seen with fractures or with some subchondral bone collapse/necrosis or stress injuries.
- Slab fracture (third carpal bone): acute moderate-marked lameness, usually following fast work.
- Significant joint effusion most typically associated with osteochondral fragmentation or fracture; increased density of third carpal bone (without fragmentation/fracture) usually not associated with effused joint.

Signs

- Unilateral or bilateral lameness.
- Variable severity: subtle to severe (with slab fracture).
- +/– palpable joint effusion.
- Unless acute fracture, typically no pain/restriction to carpal flexion.
- Chip fracture/fragmentation: frequently a pain response to deep palpation of affected site/articular margin (with limb flexed), and typically with notable joint effusion.
- Frontal plane slab fracture (third carpal bone): marked effusion of middle carpal joint/pain on flexion of carpus.
- Sagittal plane slab fracture (third carpal bone): effusion of middle carpal joint typically mild/absent.
- Cortical fissure (third carpal bone): typically no joint effusion or any palpable abnormality associated with the carpus.

Diagnosis

- History and clinical findings often strongly indicative; confirmation with diagnostic blocking +/– imaging.
- Adaptive radiological changes are present in many 'normal' horses; interpret imaging findings with caution.
- Not always possible to distinguish middle carpal joint lameness from subcarpal/suspensory pain by diagnostic blocking/conventional imaging.
- Radiography (middle carpal joint): assessment of distal articular margin of radial carpal bone (DLPaMO/flexed LM projections) and third carpal bone (flexed D35^{0}PrDDiO/'distal row skyline' projection) of both forelimbs is important; oblique 'skyline' projections may be necessary for detection of sagittal C3 slab fractures (Figure 6.131b,c; Appendix 4). Increased or irregular density/loss of trabecular definition of the radial facet (less commonly intermediate facet) of third carpal bone and/or increased density/focal lysis/spurring distal radial carpal bone. Bone modelling over dorsal margin of radial carpal bone is an indicator of prior joint activity/distension (Figure 6.139). There is a broad correlation between greater severity of third carpal bone density and likelihood of current/future carpal lameness or injury, although considerable individual variation exists (Figure 6.140a,b).
- Radiography (antebrachiocarpal joint): distal radial pathology best assessed on DMPaLO and skyline distal radius/proximal row projections; modelling/spurring/fragmentation of articular margins.
- Radiography (carpometacarpal joint): best assessed on DLPaMO projection: multiple projections frequently needed to detect injury. Linear lucency in dorsoproximal cannon extending to carpometacarpal joint +/– proliferative periosteal reaction more distally on dorsomedial cannon (Figure 6.129a–c)
- While radiography is the principal ambulatory diagnostic modality used in racing practice and is satisfactory for most cases, it has limitations: much carpal pathology can only be detected with multiplanar +/– physiological imaging. Bone scan/MRI/CT/PET: useful in investigation of moderate/severe/persistent carpal lameness if radiography unrewarding (Figure 6.141a–c).



Fig. 6.139 Entheseal modelling (of long-standing appearance) at site of joint capsule attachment on dorsal radial carpal bone (DLPaMO).

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Fig. 6.140 Individual variation in clinical and radiological deterioration of third carpal bone disease: Skyline radiographs spanning three consecutive years showing minimal change in case (a) (horse that raced successfully until 9 YO) and marked deterioration in case (b) forcing retirement at 4 YO.

Management

- See Table 6.7.
- Management should be individualised as far as possible, with clinical and radiological severity, stage of training and career targets influencing approach. For most carpal lameness there is no direct relationship between clinical and radiological severity, and radiology findings are not prescriptive. In general:
  - Insidious-onset, subtle/low-grade lameness: continued training and monitor clinical progress.
  - Acute-onset or deteriorating moderate lameness: undertake imaging/blocking and determine whether rest/intervention necessary or advisable.
- Accurate radiological assessment of fracture risk (of third or radial carpal bone) with continued training is not possible unless prodromal fracture (linear radiolucency) already visible: radiological findings (including density of third carpal bone) are poorly predictive for future fracture. Advanced imaging (MRI/PET) may permit better refinement of risk assessment in selected cases.

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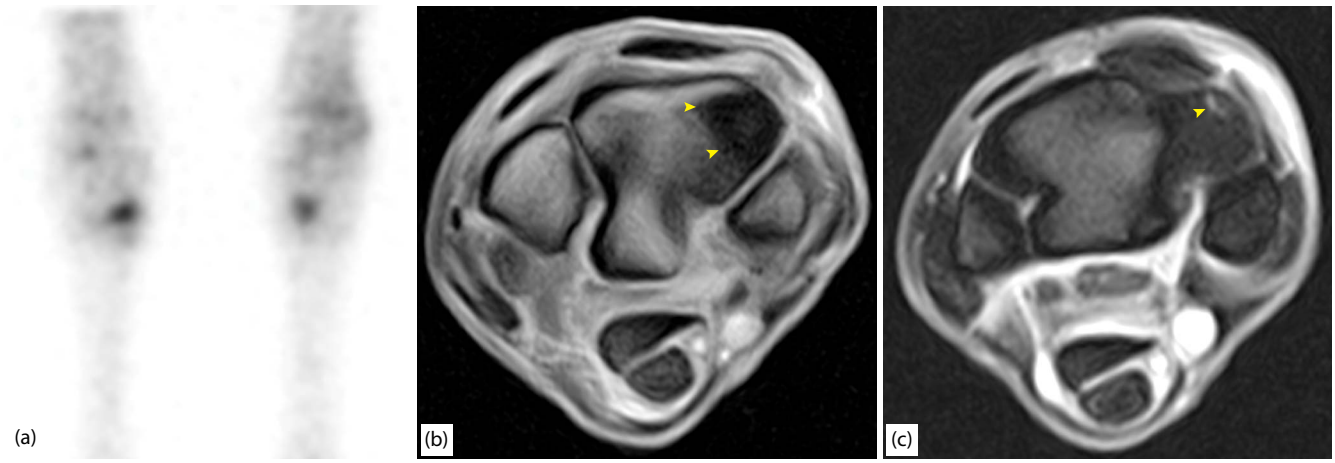


Fig. 6.141 **(a) Scintigram (dorsal) showing intense third carpal bone IRU in both forelimbs (radiography unremarkable); transverse low-field MR images featuring (b) subtle T1 hyperintensity (arrowheads) in subchondral bone plate of radial facet of third carpal bone likely representing osteolysis or early osteonecrosis, also radiologically silent; (c) frontal plane fracture (arrowhead) of dorsomedial proximal aspect of MC3 surrounded by diffuse interosseous fluid signal in the surrounding bone.**

MANAGING THE DENSE (SCLEROTIC) THIRD CARPAL BONE

- Radiological appearance of C3 is useful but awareness of limitations (radiography is not 'functional' imaging; possible involvement of adjacent or soft-tissue structures) important.
- Increased third carpal bone density is usually permanent +/– slowly progressive, and radiological improvement with rest/rehabilitation should not be expected.
- Predictive value of radiography for future C3 slab or chip fracture is very poor.
- Clinical appraisal, progression of lameness in response to training loads and career factors should guide management (rather than imaging).
- 'Point of no return' beyond which long-term athletic soundness may be compromised if horse is kept in training despite lameness/sclerosis cannot be measured objectively; whether/when to rest a horse is largely a matter of clinical judgement.
- Sclerosis + lameness of moderate (or greater) severity + worsening with high-speed exercise may merit removal from training to avoid progression to fracture: clinical instinct in these cases may be more valuable than imaging.
- Optimal rehabilitation period determined by individual factors; recurrence of lameness on return to full training can be expected in most cases.
- Intra-articular medication can be a strong masking agent and has been linked to serious carpal injury risk: in the face of deteriorating lameness + dense third carpal bone its use is discouraged.
- Pre-purchase: significance of increased third carpal bone density for future lameness determined by many factors including future racing jurisdiction/track; 'snapshot' clinical appraisal and reliance on imaging (in absence of medication and clinical history) is highly subjective and unlikely to be accurate.

- Significant lameness in juvenile horse should generally be managed more conservatively than mature horses in order to optimise long-term athletic soundness. Consider exercise modification (removal from cantering) or rest; level of exercise and time allowed dependent on individual circumstances.
- Continued training/racing in the face of non-progressive mild-moderate lameness is possible and frequently the favoured option but should be informed by imaging assessment and individual circumstances. Injury risk can generally be well managed with good clinical vigilance although radiological deterioration may occur and if so has implications for resale.
- Continued training aided by intra-articular medication: generally recommended only when already established that lameness has plateaued and following radiographic assessment. Not recommended for new/evolving lameness as may impair clinical assessment and potentiate pathology. Management then guided by quality/duration of response; if short-lived (<4–5 weeks) or poor response, period of rest may be considered.

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Table 6.7 **Management and prognosis for carpal injuries**

INJURY TYPE	MANAGEMENT	PROGNOSIS
Middle carpal joint: radial carpal bone fragmentation/fracture	Highly individualised: dependent on clinical and radiological severity/stage of training/career targets. Conservative management (rest) is recommended but continued training +/− intra-articular medication frequently possible: short initial period (1–4 weeks) of reduced exercise may be beneficial. Surgery sometimes warranted to optimize longer-term soundness (determined by chip size/configuration/displacement); however, high likelihood of ongoing carpal lameness +/− re-fracture post-surgery and should not be considered curative.	(Ongoing carpal lameness can be expected on return to full work and may require medical intervention). Good prognosis for continued training with appropriate management. Once carpal arthritis established, will always be evident radiologically regardless of management/ surgery.
Middle carpal joint (third carpal bone fracture):		(Ongoing carpal lameness can be expected on return to full work and may require medical intervention).
• Chip fracture/ fragmentation	Surgery optimizes athletic outcome; however, good/excellent outcomes possible with conservative management of selected cases.	Guarded to fair prognosis for racing soundness.
• Displaced frontal slab fracture	Radial facet: surgical reduction if athletic future desired. Radial + intermediate facets: surgical reduction for athletic and non-athletic future.	Surgery: guarded to fair prognosis for return to racing depending on configuration. Conservative management: poor prognosis for paddock/racing soundness.
• Non-displaced frontal slab fracture	Surgical reduction optimizes athletic outcome. Conservative management for non-racing use.	Surgery: fair prognosis for racing soundness. Conservative management: return to racing possible but prognosis guarded.
• Sagittal slab fracture	Surgical reduction is treatment of choice in many cases but dependent on configuration. Conservative management: acceptable alternative but radiological healing typically prolonged (expect 14–18+ weeks stable rest/walking) or incomplete.	Surgery: fair to good prognosis for return to racing. Conservative management: guarded to fair prognosis for return to full soundness; delayed healing/reinjury is common.
• Catastrophic, comminuted fracture of proximal or distal row of carpal bones	Euthanasia in most cases. Surgery (pancarpal arthrodesis) may permit salvage for paddock use, but considered heroic.	Surgery: poor-guarded prognosis for paddock use.
Carpometacarpal joint (articular fracture of dorsoproximal MC3)	Conservative management is treatment of choice: dependent on initial severity but typically 4–8 weeks walking (no/minimal stable rest).	Excellent prognosis for return to full use.
Antebrachiocarpal joint fracture/fragmentation	Continued training with minimal interruption generally possible; short initial period (1–4 weeks) of reduced exercise +/− systemic or intra-articular anti-inflammatory medication to resolve initial synovitis. If poor or short-lived response to initial management, lengthier rest or surgery can be considered (generally unnecessary).	Excellent prognosis for return to sustained athletic soundness with conservative management. No apparent correlation between radiological evidence of OA and continued/future soundness.
Palmar carpal fracture/ fragmentation:		
• Ulnar carpal bone-lateral palmar intercarpal ligament avulsion	Ulnar carpal bone-lateral palmar intercarpal ligament avulsion: surgical debridement is treatment of choice for athletic use.	Ulnar carpal bone (with surgery): excellent (approximately 90%) prognosis for return to full use.
• Multiple/radial carpal bone fragmentation post trauma	Severe injuries: euthanasia or surgery for paddock salvage.	Multiple palmar carpal fragments: guarded prognosis for return to full use (approximately 50%). Severe injuries (sustained during falls/ anaesthetic recovery) frequently have a poor prognosis for soundness/survival.

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- Antebrachiocarpal joint generally far more forgiving than middle carpal joint with respect to lameness/trainability in the presence of sometimes profound osteoarthritic change/articular margin fragmentation.
- Arthroscopy + rehabilitation may be advisable in some cases (of osteochondral fragmentation) but should not be considered curative: treated horses typically retain long-term features of carpal OA (effusion +/– lameness) upon return to training and further fragmentation/fracture is common.

Prognosis
- Most carpal lameness responds favourably to intra-articular medication or exercise modification, although duration of response may vary. Clinical stabilization/improvement also occurs over time (months) in absence of medical/training interventions in many horses.
- Small proportion of cases progress to further carpal injury with continued training.
- Small proportion remain troublesome regardless of management (rest/medication/surgery).
- Factors that influence whether pathological status (+/– radiological appearance) will deteriorate or stabilise with training are poorly understood, and currently not possible to accurately predict outcome; clinical judgement frequently more useful than imaging features.
- Prognosis may be significantly influenced by racing jurisdiction and track configuration/surface/condition: generally considered that training on tight tracks/fast surfaces is detrimental for horses with carpal pathology.
- Reinjury rate (further fracture/fragmentation) following surgery for carpal fracture may be up to 25%.

Distal radial subchondral bone injury
Poorly defined injury of lower forearm encountered occasionally in horses in full work. Primarily affects the distolateral radius. May represent a specific manifestation of injury on the pathological spectrum that includes osteochondral fragmentation of the antebrachiocarpal joint (specifically distal radial) margins.

Cause
- Presumed to arise from acute or chronic exercise-related loading.

Risk factors
- Rare condition.
- Usually encountered in horses in fast work.
- Risk factors unknown.

History
- Acute unilateral lameness.

Signs
- Moderate-marked forelimb lameness.
- Typically without palpable abnormality of limb.
- Occasionally: antebrachiocarpal joint effusion.

Diagnosis
- Diagnostic blocking: variable response to blocking of antebrachiocarpal joint.
- Radiography: usually unremarkable.
- Bone scan: marked focal/diffuse IRU in distal weight-bearing aspect of radial epiphysis (Figure 6.142a).
- MRI/CT: may permit detection of radiologically silent lesions (Figure 6.142b).

Management
- Conservative management: stable rest/walking for 6–10 weeks.

Prognosis
- Good prognosis for return to racing.

Carpal sheath tenosynovitis
Effusion of the carpal synovial sheath may have several causes and most are benign with respect to training. Mild synovial effusion encountered in early training may be an adaptive response and is often self-limiting. More severe carpal sheath effusion can also be found with injury to soft-tissue structures within or adjacent to the carpal canal (AL-SDFT, SDFT or radial head of DDFT) and may be synovial or haemorrhagic.

Cause
- Most mild/bilateral cases arise from simple synovitis with no significant underlying pathology.
- Injuries may be primary or may arise from direct trauma caused by bone irregularities of the canal borders: includes bony growths of lower forearm (distal radial osteochondromata; physeal spurring/exostosis) and accessory carpal bone fracture (p. 189).
- Osteochondroma: cartilage-capped bone growth formed by a displaced 'island' of growth plate tissue. Found in lower third of radius, proximal to growth plate; growth ceases with skeletal maturity. Sometimes difficult to differentiate from variety of physeal exostoses seen in lower radius.

Risk factors
- Mild/bilateral (and clinically insignificant) effusion commonly seen in 2 YOs as response to early training.

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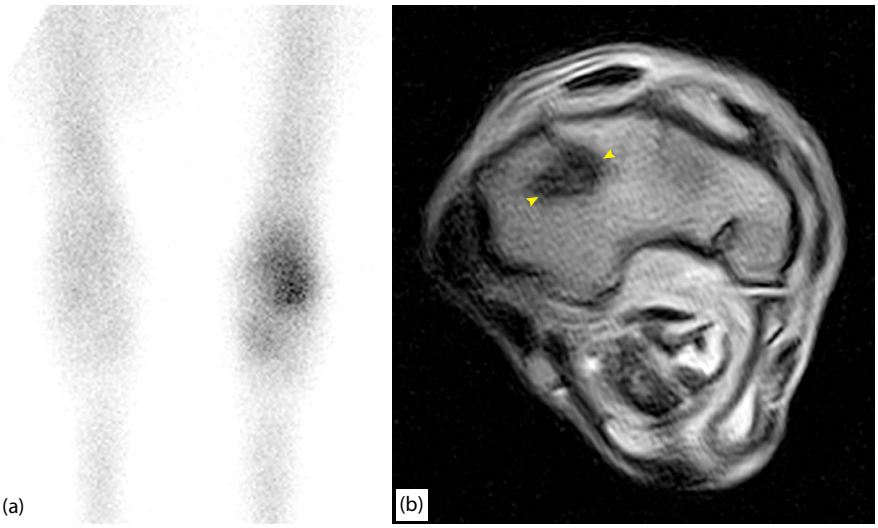


Fig. 6.142 **Distal radial subchondral bone injury: (a) dorsal scintigram showing marked diffuse IRU in distolateral radius above left carpus; (b) different case with subchondral bone lesion (confirmed arthroscopically) noted in medial articular facet of distal radius on transverse low-field MRI (arrowheads).**

- More severe pathology is rare and can occur at any age/stage of training.
- Risk factors unknown.

History

- Mild synovitis in young horses: usually insidious onset; may be mistaken for thickening of upper metacarpal flexor tendon bundles.
- Effusion arising from true injury is usually acute in onset and may follow any exercise pace.
- Osteochondroma: carpal sheath effusion and recurrent lameness.

Signs

- Clinical findings variable.
- Mild effusion: usually only distal pouch (top half of cannon) filled; non-painful thickening around upper flexor tendon bundle.
- Moderate/marked effusion: prominence of proximal pouch (lateral aspect of lower forearm) +/– transient dependent oedema through lower cannon.
- If lameness present, often direct relationship with severity of distension: marked acute effusion may be associated with severe lameness and resentment of carpal flexion.
- +/– pain on palpation of palmar soft-tissue (musculotendinous) structures above carpus.

Diagnosis

- Imaging generally only necessary if lame, or marked effusion.
- Ultrasonography: used to determine presence of lesions involving the principal soft-tissue structures (Figure 6.143a,b; see also 6.166, p. 204).
- Radiography: LM or oblique projections for detection of physeal exostosis ('spur') or osteochondroma of caudal margin of lower radius (Figure 6.144a–c).

Management

- Mild effusion/no lameness: no treatment or modification of training required.
- If marked/progressive/lame, diagnostic imaging to rule out obvious pathology.

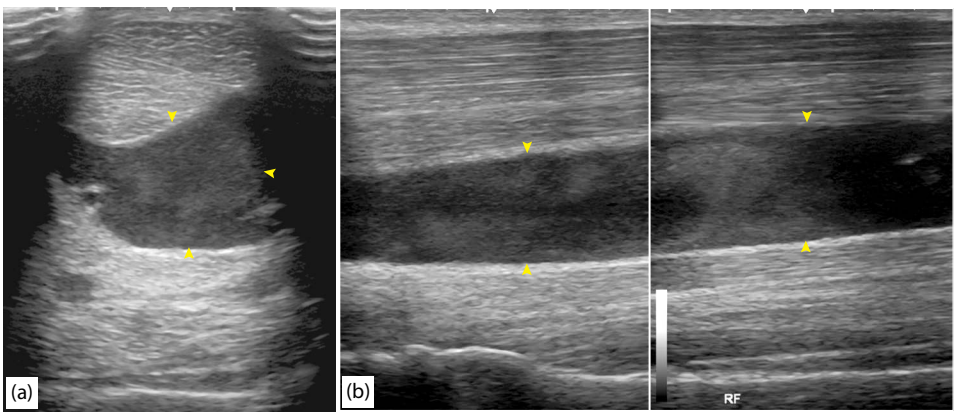


Fig. 6.143 **Carpal sheath effusion (arrowheads) resulting from haemorrhage: Transverse (a) and longitudinal (b) ultrasonograms.**

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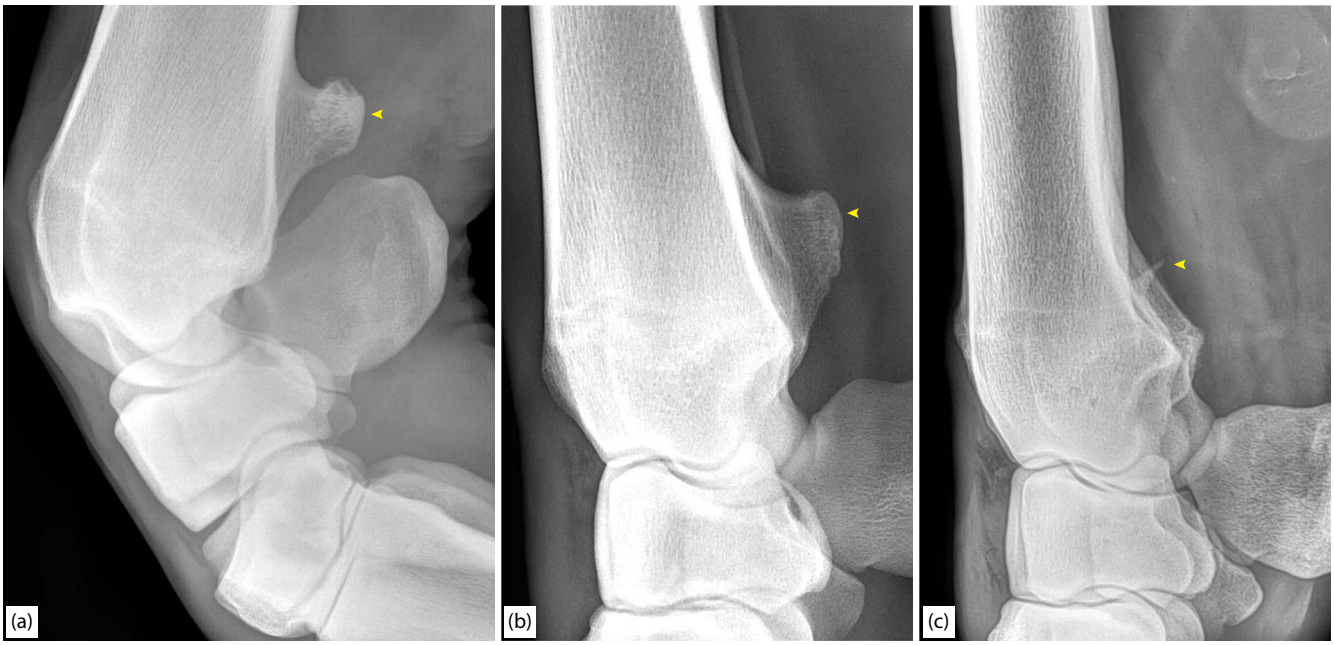


Fig. 6.144 **(a–c) Examples of distal radial osteochondroma (arrowheads).**

- If no significant pathology detected, intra-thecal medication (corticosteroid).
- Poor/short-lived response to medication should prompt further imaging or rest according to diagnosis.
- Osteochondroma or physeal spur/exostosis (if causing impingement): surgical removal along with debridement of torn DDFT tissue (if present).
- Tearing of radial head of DDFT: surgical debridement (tenoscopy).

Prognosis

- 'Adaptive', non-painful distension in young racehorses: self-limiting and rarely interferes with training.
- Tearing of radial head of DDFT: excellent prognosis following surgery.
- AL-SDFT desmopathy (p. 203): good-excellent prognosis for return to full use with conservative management (stable rest/walking for 6–10 weeks).
- Radial osteochondroma: excellent prognosis following surgery.
- Exostosis of caudal radial physis: excellent prognosis following surgery.

Accessory carpal bone fracture

Fracture of the accessory carpal bone at the back of the carpus typically results from trauma. Most common configuration is a frontal slab fracture (Figure 6.145); there is invariably displacement of the major fragments (Figure 6.146) and comminution (particularly at proximal border) may occur. Involvement of the carpal synovial sheath may result from disruption of the carpal canal border.

Cause

- Traumatic/acute injury presumed to arise from carpus being forced beyond normal range of motion (excessive extension/flexion) during racing/training fall.

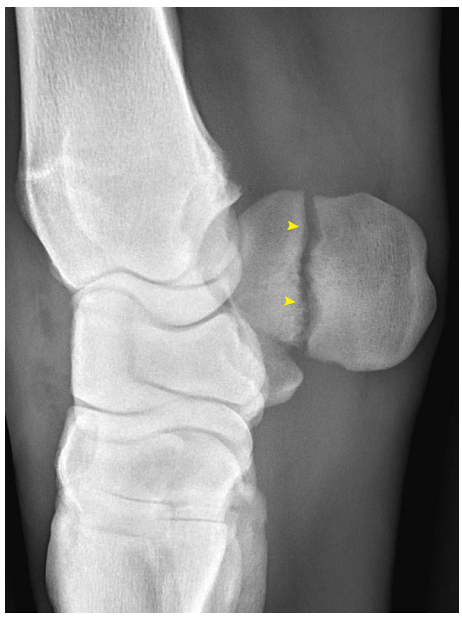


Fig. 6.145 **Accessory carpal bone fracture: Most common (frontal plane) configuration (LM).**

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Fig. 6.146 Accessory carpal bone fracture: Marked distraction of fracture fragments (flexed LM).



Fig. 6.147 Comminuted accessory carpal bone fracture: In this case the injury was clinically silent, long-standing and detected incidentally during radiographic screening of a National Hunt racehorse.

- Considered to result from distraction forces on accessory carpal bone by muscular attachments (flexor carpi ulnaris and ulnaris lateralis) +/– 'nutcracker' effect between radius and metacarpus.

Risk factors
- Rare condition.
- Any age or stage of training.

History
- Acute-onset lameness, usually following a fall.
- Occasionally encountered as incidental finding in clinically normal horse (sometimes in horses with no history of associated lameness) during radiographic imaging (Figure 6.147): presumed in these cases to be long-standing and of no future concern.
- Fragmentation of the accessory carpal bone may be encountered as an incidental finding in sound yearlings when subjected to radiographic survey.

Signs
- Moderate-marked unilateral forelimb lameness.
- Generalized (often marked) thickening of soft tissues around back of carpus.
- Carpal synovial sheath effusion usually present but may not be evident until acute swelling dissipates.
- Flexion of carpus resented.
- +/– instability/crepitus/pain on palpation of accessory carpal bone with limb in partial flexion.

Diagnosis
- Clinical findings strongly indicative.
- Radiography: required for diagnosis. Several projections (or CT) to determine fracture configuration/comminution.

Management
- Conservative management: 8–12 weeks stable rest with immobilizing bandage (full-limb Robert-Jones bandage) for initial few weeks. Radiographic union does not occur but injury generally settles sufficiently to facilitate return to athletic use including racing.
- Computer-assisted surgical fixation may optimise quality and rate of repair and improve outcomes.
- Accessory carpal bone fragmentation (and more rarely frontal plane fracture) detected in clinically normal young animals generally considered an incidental finding and no intervention required.

Prognosis
- Fair prognosis for return to racing soundness.
- Long-term chronic distension of carpal synovial sheath may occur; can be managed medically.
- Accessory carpal bone fragmentation (with or without carpal osteoarthritic radiological changes) when identified as incidental finding in yearlings appears not to be associated with any negative effects on career outcomes or racing performance.

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Radial physitis ('epiphysitis')

Inflammation of the growth plate (physis) at the lower extremity of the radius is occasionally encountered in yearlings entering training. Subsequent inhibited bone lengthening on affected side of limb can result in further deviation of carpal conformation.

Cause

- Excessive or asymmetric loading (lunging/walker exercise) of immature limb during early training.
- Compressive forces from excessive loading stimulate inflammation of growth plate.

Risk factors

- Usually occurs in yearlings during sales preparation.
- Poor carpal conformation, rapid growth, nutrition and excessive loading of limb are risk factors.

History

- Prominence of lower forearm noted in pre- or early training.

Signs

- Prominence of medial aspect of one or both lower forearms, immediately above carpus.
- Heat/pain response to palpation when active.
- +/– mild lameness.

Diagnosis

- Clinical findings definitive.
- Radiography: DPa projection to confirm/assess severity and activity; altered radiodensity of growth plate and adjacent proliferative bone reaction (Figure 6.148) on affected side of limb.

Management

- Conservative management: restricted exercise (stable rest/walking) until affected site free from pain.
- NSAID medication in initial phase.
- Feed restriction to slow growth rate/reduce BWT may be beneficial: reduce calorific intake by 25% + low-quality roughage for approximately 6 weeks. Ensure correct calcium: phosphorus ratio in diet.

Prognosis

- Prognosis good for athletic soundness.
- Training in the face of active physitis likely to result in permanent angular limb deformity (with long-term consequences for future soundness in training).

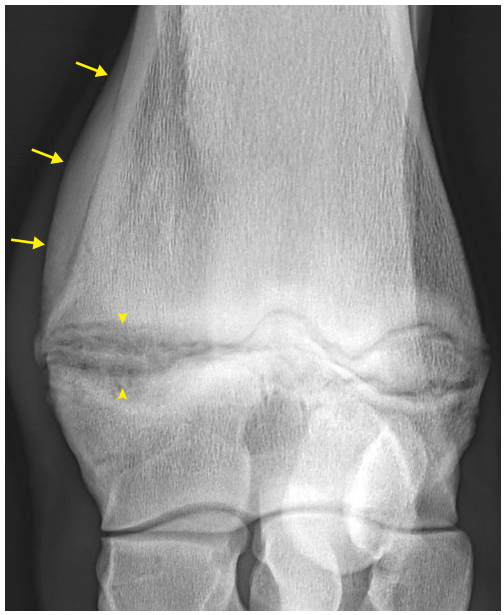


Fig. 6.148 **Radial physitis: Irregular/wide lytic appearance of medial physis (arrowheads) and new bone formation extending above physis on affected side (arrows) (DPa).**

Osteochondrosis: Carpal subchondral bone cyst ('osseous cyst-like lesion'/OCL)

Developmental defect of cartilage/subchondral bone. Subchondral bone cysts involving the ulnar carpal bone are very common while those found at all other sites (radial carpal bone, third carpal bone, proximal MC3, distal radius) are rare. Radial carpal bone cysts (Figure 6.149)



Fig. 6.149 **Radial carpal bone cyst.**

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may communicate with antebrachiocarpal +/or middle carpal joints.

Cause
- See Osteochondrosis (Overview) (p. 267).

Risk factors
- Up to >20% sales yearlings have ulnar carpal bone cysts.
- All other carpal bone cysts rare.
- Risk factors for development unknown.

History
- Typically an incidental finding at routine yearling radiography (subclinical at time of diagnosis) (p. 390).
- Cysts involving radial or third carpal bones may occasionally be found in horses with carpal lameness: generally not easy to determine contribution of cyst over other causes of carpal lameness without utilising advanced imaging.

Signs
- Ulnar carpal bone cysts: invariably clinically unremarkable.
- Most cysts at other sites also display no clinical signs, but if clinically active typically mild-moderate unilateral lameness +/– joint effusion.

Diagnosis
- Diagnostic investigation rarely necessary.
- Response to intra-articular diagnostic blocking may be variable/inconsistent.
- Advanced imaging (bone scan/MRI/CT/positive contrast arthrography) sometimes necessary to determine significance of cyst and plan treatment.

Management
- Clinically silent: no treatment necessary and continue training.
- Clinically active: intra-articular medication initially and determine quality and duration of response. If cannot be managed medically, further treatment options (surgery) dependent on individual circumstances and location/configuration of lesion.

Prognosis
- Most carpal cysts (regardless of location) do not cause lameness, and remain clinically inactive throughout life.
- However, non-ulnar carpal bone cysts may behave unpredictably and can be career-threatening if they become clinically active.

UPPER FORELIMB

Applied anatomy
The upper forelimb functions primarily as a 'shock absorber' (propulsion being the role of the hindlimb) (Figure 6.150). There is no bony articulation between the forelimb and the axial skeleton: rather the upper forelimb is attached to the trunk by the muscles and ligaments of the shoulder girdle. This is composed largely of the latissimus dorsi and serratus ventralis muscles, which connect the thoracolumbar fascia and caudal cervical vertebrae to the inside of the humerus and scapula, respectively. The pectoral muscles, running from the sternum to the inner aspect of the humerus, serve to stabilize the limb just before and during the stance phase.

The scapula articulates with the humerus at the shoulder joint. The shoulder joint is notable for having no collateral ligaments and is stabilized by the large muscle bellies that surround it; consequently, the joint capsule is not directly palpable. At the point of the shoulder the strong and bilobed biceps tendon passes over the front of the joint from its origin on the supraglenoid tuberosity of the scapula. After running through the bicipital synovial bursa over the bicipital groove of the humerus it descends to attach to the top of the radius (below the elbow joint). The biceps brachii muscle acts to extend the shoulder and flex the elbow, and its action is opposed by the triceps brachii muscle that attaches to the point of the elbow.

As with the shoulder, the elbow joint acts primarily as a hinge with movements restricted to flexion/extension. The humeral condyles articulate with the proximal radius and partly with the prominent olecranon process of the ulna, which forms the point of the elbow. The joint has cranial and caudal compartments, with muscles surrounding the cranial and medial aspects. Although the elbow joint has little soft tissue overlying it laterally, distension of the joint is not readily apparent due partly to the lateral collateral ligament, which limits rotational movement.

Synovial communications
- Shoulder joint and bicipital bursa: communication in approximately 20% of limbs.

Examination
With the horse weight-bearing, the upper forelimb, forearm and pectoral regions should be observed for muscular symmetry. Muscle atrophy may be manifested by greater prominence of some of the bony features of the upper limb such as the scapular spine. Effusion of the shoulder and elbow joints is rarely detectable clinically, although

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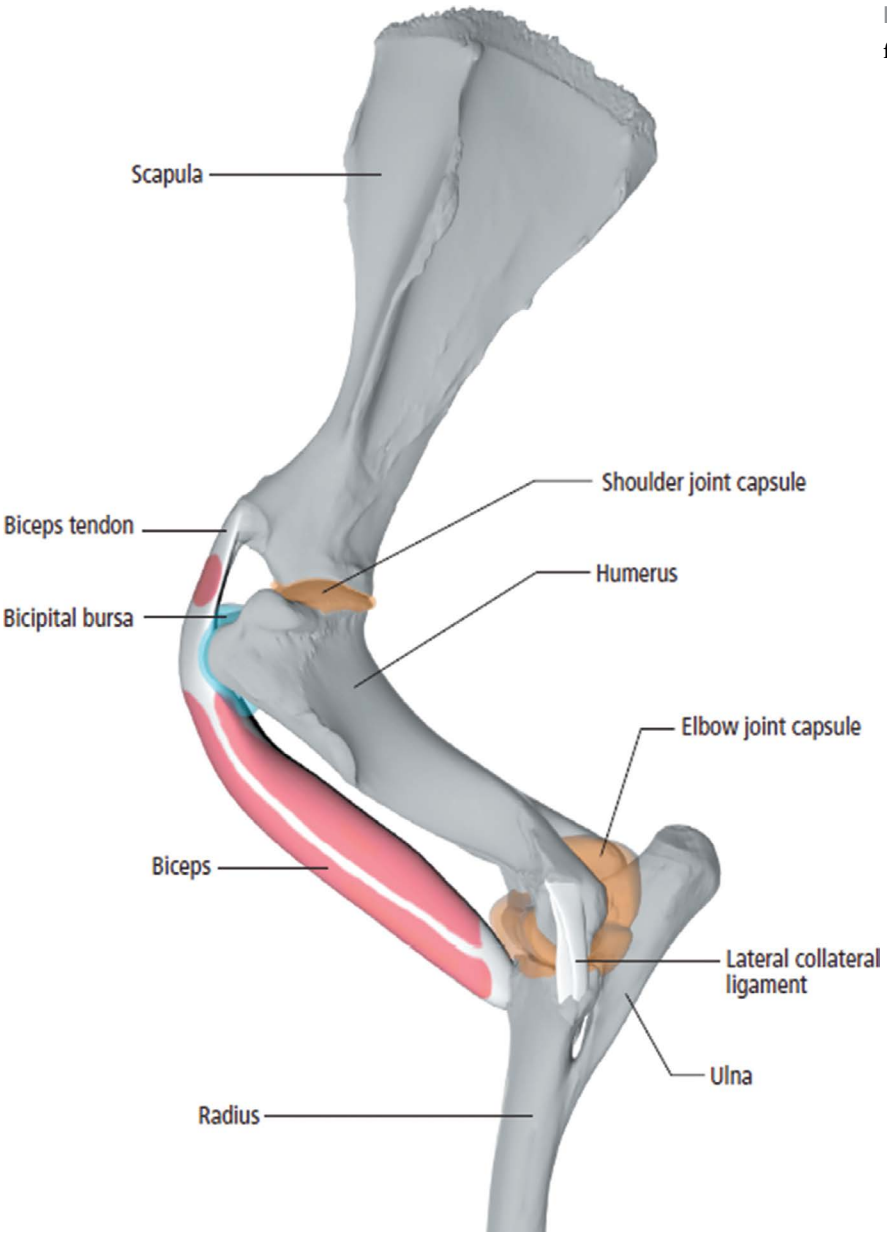


Fig. 6.150 **Major structures of the upper forelimb (lateral view).**

marked distension or inflammation of the bicipital bursa is usually apparent. Flexion and extension of the limb or deep palpation of the point of the shoulder may elicit a pain response and allow localization to the shoulder/bicipital bursa region. The biceps brachii muscle both limits and links elbow joint extension to shoulder joint flexion; drawing the limb caudally such that overextension of the elbow is achieved with shoulder flexion indicates an injury to the biceps tendon/muscle unit.

Humeral stress fracture

Despite being a bone of robust dimensions, the humerus may sustain stress injury in response to repetitive loading. The spectrum of injury encountered ranges from subclinical/mild to catastrophic (oblique or spiralling diaphyseal fracture: Figure 6.151). If complete fracture occurs, contraction of the shoulder muscles causes overriding of fragments and shortening of the limb.

Predilection sites are craniodistal (medial epicondylar region), caudoproximal, caudodistal and medial humerus. Proximal lesions are more likely to be severe or propagate to complete fracture. Lameness is typically unilateral but pathology may be bilateral.

Cause

- Stress/fatigue injury.
- Tensile strains act at distomedial and cranial humerus; compressive strains at caudal humerus during stance.

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Fig. 6.151 Comminuted, spiral humeral stress fracture (postmortem volume-rendered CT image).

Risk factors

- Any age/stage of training; however, typically encountered in horses in light exercise (trotting or cantering) rather than strong association with fast work.
- Most common in horses that are unraced or have only run once.
- Horses sustaining fatal humeral stress fractures are more likely to be unraced at time of injury than horses with non-fatal humeral injuries.
- Horses that are returning from a recent rest period (for unrelated reason) at greater risk.
- Racing surface: proximal lesions more commonly encountered on dirt than on synthetic tracks.

History

- Acute-onset forelimb lameness; usually no history of preceding lameness.
- May present with a recent history (days) of displaying noticeable but transient unilateral forelimb lameness immediately before jumping off to canter (and then subsequently cantering satisfactorily): pathognomonic for humeral stress injury.
- Incomplete (stress) injury: lameness usually first noted during warm-up for daily exercise.
- Complete fracture: acute severe lameness during exercise; horse may fall at time of injury. Must be examined on track as horse usually unwilling to move.

Signs

- Clinical findings dependent on severity of injury (incomplete/complete).
- Incomplete fracture: moderate-marked lameness characterized by reduced cranial phase to gait; often noticeable at walk. Limb usually palpably normal. Some cases may initially be free from significant lameness when trotted in hand, and only display lameness in the immediate few steps prior to jumping off to canter when exercising.
- Complete fracture: severe lameness, usually with marked swelling/sweating of proximal limb/shoulder and visible shortening of upper limb with muscle spasm (Figure 6.152). 'Guarding' of limb +/– crepitus may be observed on manipulation. Concurrent damage to radial nerve common and associated with inability to protract limb.

Diagnosis

- Complete/displaced fracture: clinical findings usually sufficient for diagnosis; radiography sometimes needed for confirmation.
- Incomplete fracture: poor sensitivity of radiography due to impaired radiological definition of upper forelimb; changes evident in approximately 50% cases and include cortical thickening, endosteal +/or periosteal proliferation at predilection sites (Figure 6.153). Ultrasonography may be useful ambulatory imaging modality: presence of bone callus +/– cortical surface disruption at predilection site. Bone scan is the modality of choice for definitive diagnosis due to high specificity/sensitivity: focal IRU (variable intensity) at predilection sites (Figure 6.154a–d).

Management

- Complete fracture: immediate euthanasia usually warranted.
- Incomplete fracture: conservative management; determined by clinical/imaging severity but typically stable rest/walking for 8–10 weeks. Caution advised during initial rehabilitation period as displacement of humeral fractures can occur at slow paces (trotting).

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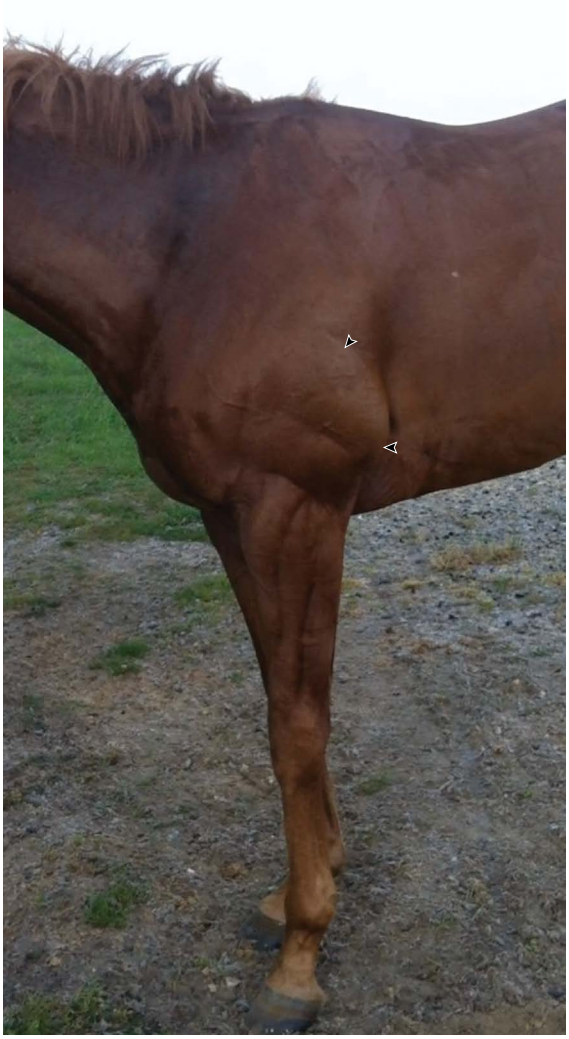


Fig. 6.152 **Complete humeral stress fracture sustained at exercise: Characteristic presentation with shortening of limb and muscular spasm (arrowheads).**

Prognosis

- Incomplete (stress) fractures: excellent prognosis for return to full use. Small proportion recur. Return to racing time does not appear to be affected by injury location (average 8 months).
- Complete fractures: generally catastrophic in adult horses and warrant immediate euthanasia on humane grounds. Number of cases of complete fracture have survived with conservative management (salvage for paddock use only); however, requires careful case selection and prognosis for survival is poor-guarded. Supporting limb laminitis (p. 93) is a significant risk and welfare of horse should be the primary concern.



Fig. 6.153 **Humeral stress fracture: Craniodistal proliferative modelling (ML radiograph).**

Scapular stress fracture

Stress injury with main predilection site at the lower end of the scapular spine. Rarely detected at subclinical/early clinical phase; typically encountered at time of complete/catastrophic fracture, suggesting that warning signs are subtle or masked by bilateral nature of pathology.

Fracture may propagate transversely across the neck of the scapula and distally to the articular surface. Complete fracture often results in severe comminution (Figure 6.155a,b). Pathology frequently bilateral.

Cause

- Stress/fatigue injury.
- Spine of scapula serves to limit mediolateral bending of body of scapula during load bearing; stress failure of this supporting strut can result in profound collapse of lower scapula.

Risk factors

- Rare condition.
- Usually encountered in horses currently in fast exercise/racing phase of training.
- Complete fracture may occur at fast or slow cantering paces.
- Most common in horses ≥3 YO.

History

- Acute-onset, unilateral or bilateral forelimb lameness.
- Complete fracture: occurs during training/racing; horse may fall at time of injury and typically regains

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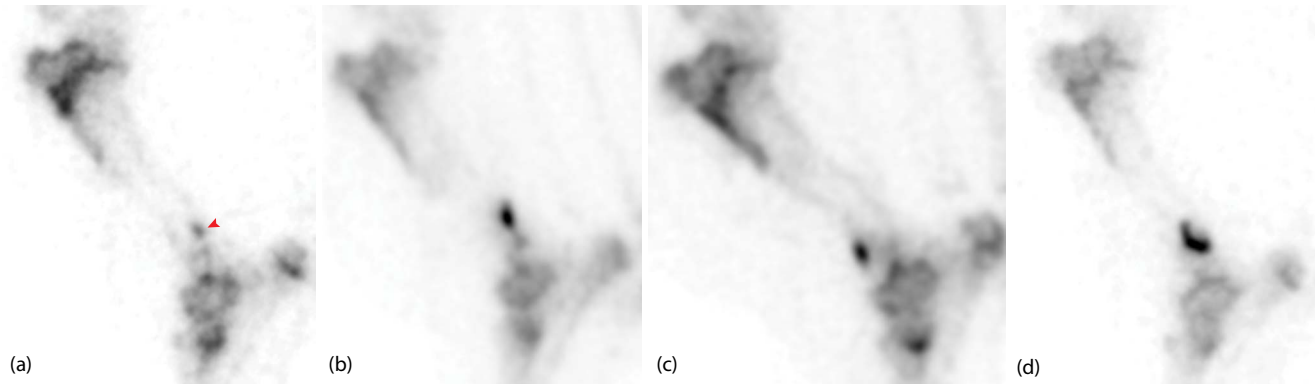


Fig. 6.154 **(a–d) Humeral stress fracture: Variation in location and intensity of IRU on lateral scintigrams.**

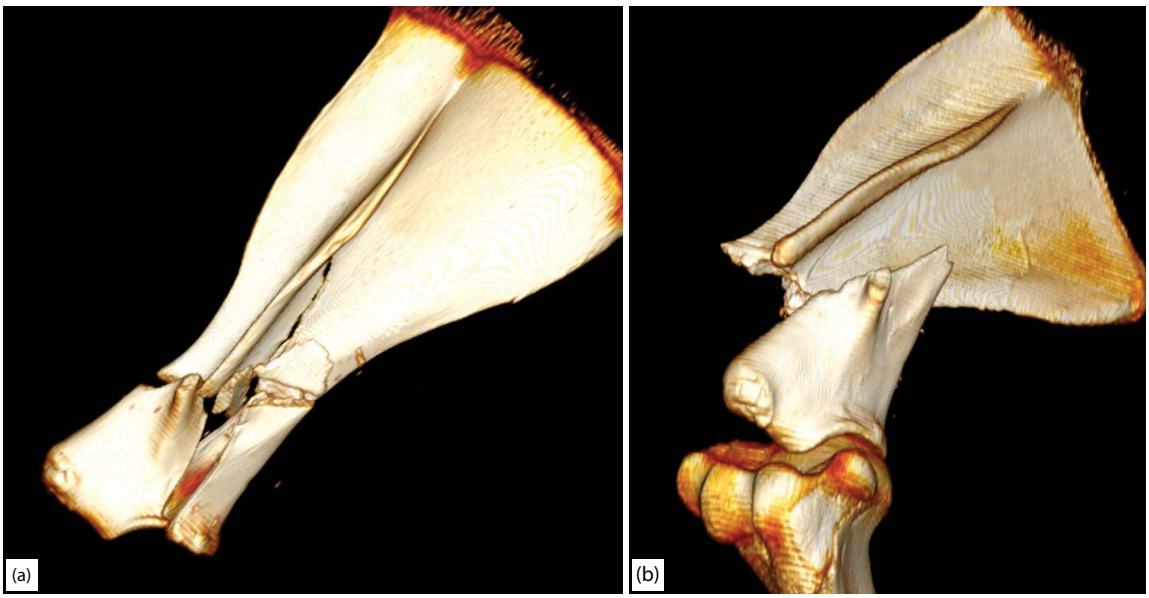


Fig. 6.155 **Scapular stress fracture: Postmortem volume-rendered CT images of (a) comminuted and (b) transverse/complete injuries.**

feet, although recumbency also encountered. Must be examined on track as horse usually unwilling to move.

Signs

- Complete fracture: severe forelimb lameness with swelling/'guarding'/crepitus/instability of lower shoulder blade region/'dropped elbow' stance. Affected limb may bear weight due to support of muscular girdle, but horse shows marked distress at any attempt at movement.
- Incomplete fracture: moderate-marked lameness characterized by reduced cranial phase to gait; often noticeable at walk. Limb usually palpably normal, although pain response sometimes elicited by palpation of scapular spine.

Diagnosis

- Clinical findings sufficient to diagnose complete/catastrophic fracture.
- Incomplete fracture: requires bone scan for definitive diagnosis: focal, often intense IRU (Figure 6.156a–d).
- Ultrasonography: callus +/or bone disruption may be detected at lower end of scapular spine/scapular neck (Figure 6.157).

Management

- Incomplete fracture: conservative management (typically stable rest/walking for 8–10 weeks).
- Complete (catastrophic) fracture: immediate euthanasia.

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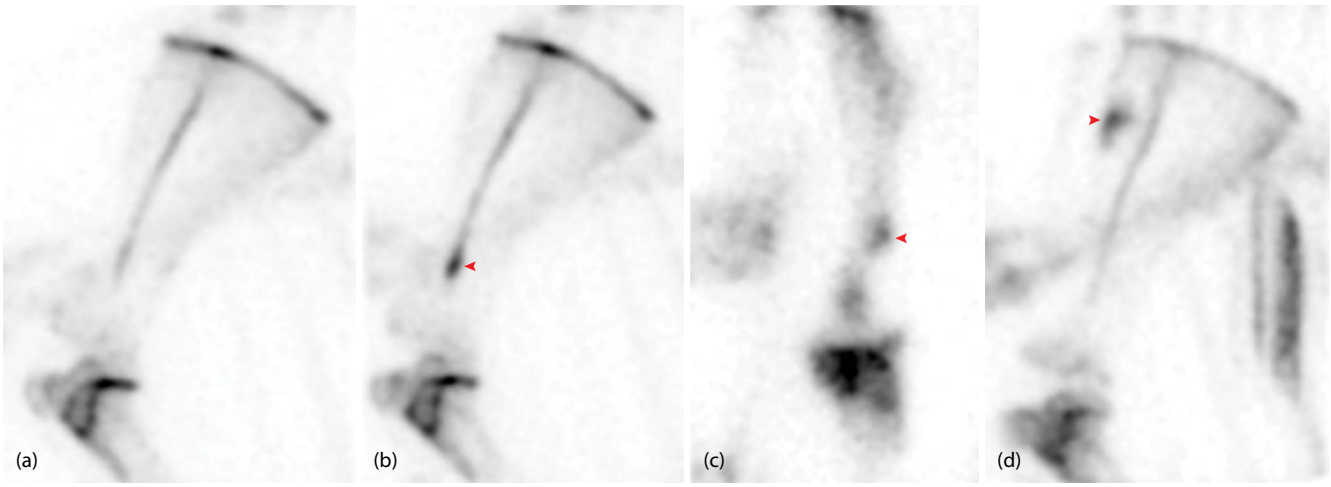


Fig. 6.156 **Scapular stress fracture: (a) lateral scintigram showing normal pattern of activity; (b) lateral and (c) cranial scintigrams showing IRU (arrowheads) in distal scapular spine associated with stress injury; (d) IRU (arrowhead) in atypical site in proximal cranial scapula.**

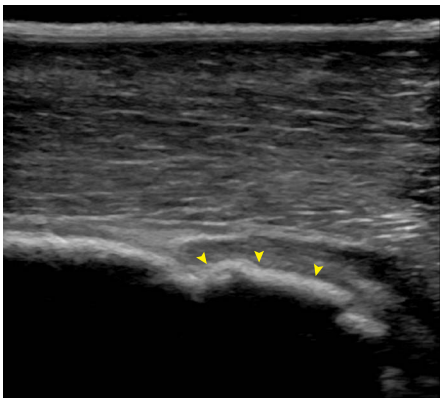


Fig. 6.157 **Scapular stress fracture: Longitudinal ultrasonogram of distal scapular spine (proximal to left) with disrupted bone contour (arrowheads).**

Prognosis
- Incomplete fractures carry a good prognosis for return to full use.
- Complete fractures are catastrophic and carry a hopeless prognosis for survival.

Radial stress fracture
Stress injury with predilection site in mid to lower third of the shaft of the radius. Severity at time of detection varies between individuals; however, complete fracture is rare. Unilateral or bilateral.

Cause
- Stress/fatigue injury.
- Mid-to-upper radius subjected predominantly to craniocaudal bending forces (cranial cortex loaded in tension, caudal cortex in compression) while lower radius undergoes largely torsional strains.

Risk factors
- Rare condition.
- Risk factors poorly understood.
- More common in young horses (2 YO).
- Usually encountered in horses in early cantering exercise.

History
- Acute-onset lameness, usually first noted during warm-up for daily exercise.

Signs
- Unilateral forelimb lameness of moderate-marked severity.
- Limb typically palpably normal.
- Occasionally pain on deep palpation/percussion of medial mid-shaft of radius.

Diagnosis
- Strong clinical suspicion of proximal limb stress fracture (radius/humerus/scapula) may warrant radiographic imaging in the first instance; however, combination of radiography and scintigraphy frequently required for diagnosis.
- Radiography: rarely definitive. Findings often subtle/indistinct at initial examination; repeat radiography at 7–14 days post-injury may be more productive.

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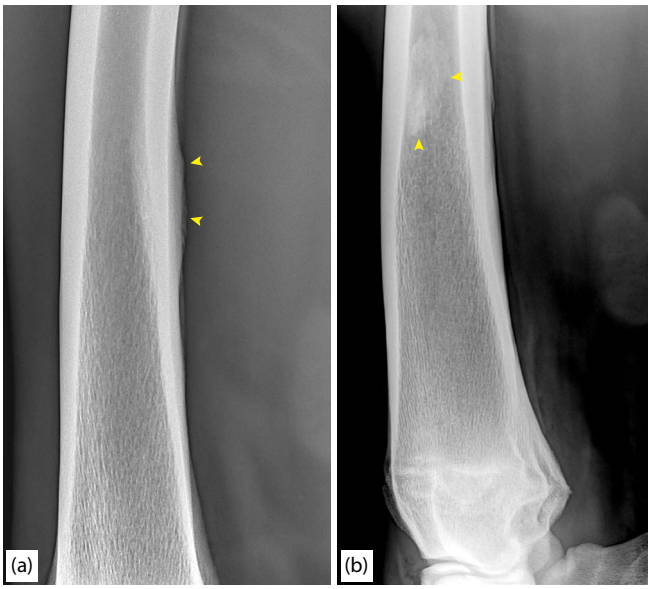


Fig. 6.158 Non-fracture radiological features encountered in radius: (a) entheseal modelling; (b) enostosis-like intra-medullary lesion (LM).

Increased radiodensity of diaphyseal medulla/endocortical callus/+/– short fracture line. Caution required as radiological irregularities (enostosis-like lesions; enthesopathy) unrelated to stress pathology may be encountered (Figure 6.158a,b).

- Bone scan: recommended if radiography unrewarding or ambiguous; focal IRU (Figure 6.159a,b); patterns of uptake may be similar to that associated with enostosis-like lesions.
- Diagnostic blocking: negative response to distal limb analgesia (blocking generally contraindicated if stress fracture suspected).

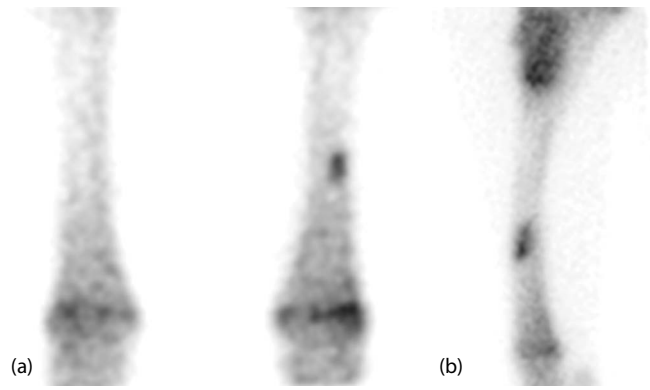


Fig. 6.159 Radial stress fracture: Dorsal (a) and lateral (b) scintigrams showing IRU in the mid-radius region; differentiated from enostosis-like lesion by apparent cortical activity (rather than intra-medullary).

Management

- Conservative management: determined by clinical/imaging severity but typically stable rest/walking for 8–10 weeks.
- If risk of deterioration/displacement, consider tying up for initial weeks pending radiographic review. Measures to minimize risk of supporting limb laminitis (p. 93).
- Complete fracture (invariably with fragment displacement): euthanasia. Surgical repair possible but poor prognosis for survival.

Prognosis

- Good prognosis for return to full use in cases of incomplete fracture.

Shoulder osteochondrosis

Developmental osteochondral defects of the shoulder joint are less common than at other sites. They develop early in life and usually first cause lameness in early training. They include OCD and bone cyst lesions. Unilateral or bilateral.

OCD lesions primarily involve the caudal half of the joint: osteochondral defects of one or both articular margins. Subchondral bone cysts may be single or multiple and are typically found in the mid-distal scapular (less commonly mid-humeral) joint margin. Both forms of osteochondrosis may be associated with secondary arthritis, depending on severity and duration of pathology.

Cause

- See Osteochondrosis (Overview) (p. 267).

Risk factors

- Rare condition.
- Usually encountered in 2 YOs.

History

- Lameness usually first noted early in training (yearling or 2-YO stage).
- Intermittent in nature, responding favourably to rest in short term.

Signs

- Unilateral forelimb lameness of variable severity; intermittent or persistent.
- Limb usually palpably normal.
- +/– boxy/upright foot on affected limb in some cases (secondary to chronic reduced limb loading).

Diagnosis

- Clinical features often suggestive of upper forelimb problem but are non-specific.

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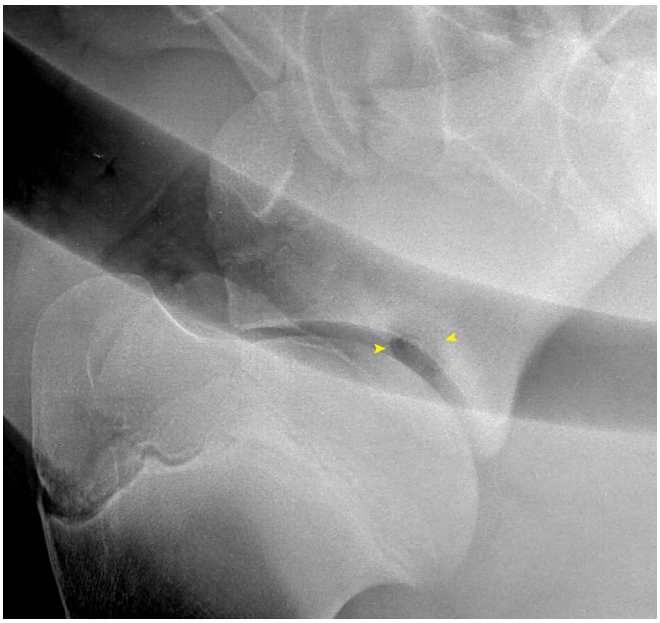


Fig. 6.160 Shoulder osteochondrosis (bone cyst): Circular radiolucency (arrowheads) with surrounding increased radiodensity in distal scapula (ML radiograph).

- Diagnostic blocking: total/partial response to intra-articular blocking of shoulder joint.
- Radiography: ML projection most useful. Signs often subtle. OCD lesions: irregular subchondral radiodensity or flattening/loss of congruity of joint margin. Cysts: subchondral lucencies with associated sclerotic rim (Figure 6.160). Most obvious feature of both types of lesions is often secondary arthritic change (modelling/lipping of caudoventral angle of scapula).

Management

- Conservative management (rest +/– intra-articular medication) in cases without marked clinical or radiographic findings. Rest period of 3–4 months out of ridden exercise.
- Surgery may give best chance of positive outcome for more severely affected cases, although not all lesions accessible.

Prognosis

- Prognosis for racing soundness poor to guarded regardless of management (<50% return to athletic use). Some manageable with periodic intra-articular medication.
- Prognosis typically linked to severity of radiological findings at initial presentation.

Biceps bursitis/tendinitis

Injuries to the biceps tendon or the synovial (bicipital) bursa that surrounds it occur sporadically. Most frequent is septic tenosynovitis of the bursa, causing progressive and severe lameness. Less common are non-septic tenosynovitis and true tendinitis/enlargement of the biceps tendon.

Cause

- Septic tenosynovitis usually the result of a penetrating injury (e.g., pitchfork) but closed/haematogenous infection can occur.
- Non-septic tenosynovitis or tendinitis may arise from traumatic stretching/flexion of shoulder during a fall or blunt trauma to point of shoulder. In some cases may arise from undefined repetitive loading factors.

Risk factors

- Septic tenosynovitis may occur at any age/stage of training.
- Tendinitis is very rare and usually in 2 YOs in cantering phase of training; risk factors unknown.

History

- Septic tenosynovitis: forelimb lameness that worsens (to become severe) over several days.
- Non-septic tenosynovitis/tendinitis: acute- or chronic-onset unilateral forelimb lameness.

Signs

- Unilateral forelimb lameness of variable severity.
- Shortened cranial phase of stride.
- +/– pain response to palpation of biceps/point of shoulder region or flexion of shoulder.
- Septic tenosynovitis: severe lameness/swelling +/– pain response at point of shoulder/+/– fever. External wound may not be readily apparent.

Diagnosis

- Clinical features usually indicative of upper forelimb origin but non-specific.
- Ultrasonography: assessment of biceps tendon (enlargement/irregular echogenicity/loss of border definition), bursa and bicipital groove (Figure 6.161).
- Diagnostic blocking: elimination of lower limb lameness; response to blocking of bicipital bursa may be partial/total.
- Radiography: useful to assess irregularities of associated bone margins: bicipital groove/enthesopathy at radial tuberosity/intratendinous calcification.
- Synoviocentesis (for suspected septic tenosynovitis).

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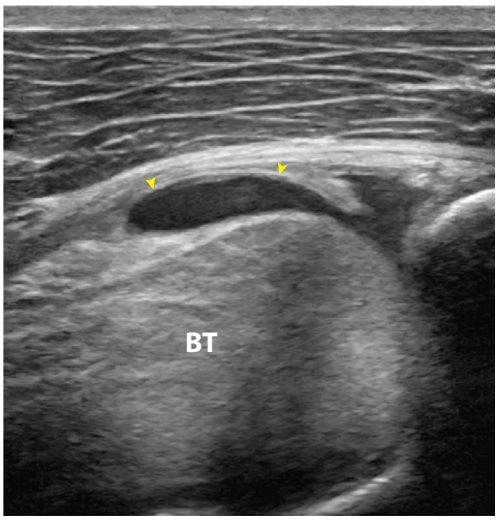


Fig. 6.161 **Ultrasonogram (transverse) of biceps bursal effusion (arrowheads); BT: Biceps tendon.**

Management
- Septic tenosynovitis: requires hospitalization and emergency tenoscopy/lavage.
- Non-septic bursitis/tendinitis: conservative management (typically stable rest/walking for 8–12 weeks) +/– intrathecal medication. Rehabilitation guided by clinical/ultrasonographic monitoring.

Prognosis
- Septic tenosynovitis: fair prognosis for return to soundness with prompt surgical management. Prognosis declines with chronicity.
- Non-septic tenosynovitis/tendinitis: good prognosis for return to full soundness.

Shoulder fracture: Supraglenoid tuberosity

The most common fracture involving the point of the shoulder. The supraglenoid tuberosity is the bony prominence upon which the biceps brachii tendon attaches. Fracture typically runs through the growth plate of the tuberosity, regardless of age (growth plate closes between 1 and 2 years of age). Fracture fragment is usually distracted cranially/distally due to pull of biceps tendon. Injuries vary in severity (simple or comminuted) and frequently have articular (shoulder joint) involvement.

Cause
- Traumatic injury.
- Occurs either through direct trauma or following over flexion of shoulder (during a fall).

Risk factors
- Rare condition.
- Sporadic injury at any age/stage of training.

History
- Acute-onset unilateral forelimb lameness.
- Usually follows a fall or collision.
- Lameness severity may improve over initial 2–3 days.

Signs
- Unilateral forelimb lameness of moderate-marked severity.
- Shortened cranial phase of stride.
- +/– pain on palpation of point of shoulder.
- Local inflammation/swelling may be minimal.
- Manipulation: concurrent flexion of shoulder and extension of elbow indicative of loss of integrity of biceps tendon/supraglenoid unit (Figure 6.162a).
- Atrophy of muscles of shoulder blade (supraspinatus and infraspinatus) may follow acute phase if concurrent damage to suprascapular nerve at point of shoulder.

Diagnosis
- Radiography (ML projection) is definitive (Figure 6.162b).

Management
- Surgery: fixation (preferred) or removal (if fragment(s) small: <30% articular surface; or comminuted) of fracture fragment offers best chance of return to soundness; full function of biceps tendon is not a necessity for athletic activity.
- Conservative management (stable rest/walking for 12–16 weeks): satisfactory for paddock use. Results in non-union and generally not considered if return to racing desired.

Prognosis
- Surgery: fair to good prognosis for return to athletic use, although lengthy rehabilitation period necessary.
- Prognosis best for injuries with minimal displacement/fragmentation/articular involvement.
- Conservative management: reasonable prognosis for survival; poor prognosis for athletic soundness and continued lameness frequently results in delayed euthanasia in these cases.

Elbow subchondral bone injury/osteochondrosis

Subchondral bone injury involving the medial aspect of the elbow joint is a poorly understood condition. The

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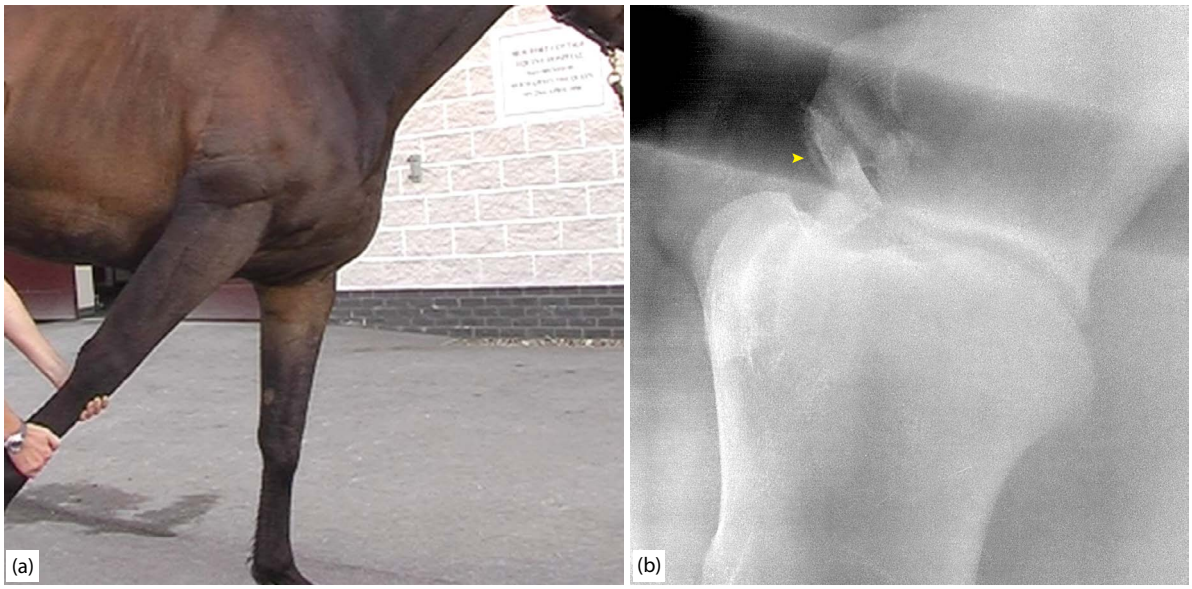


Fig. 6.162 **Supraglenoid tuberosity fracture: (a) concurrent extension of elbow and flexion of shoulder with caudal retraction of limb; (b) radiograph (ML) showing displaced fracture fragment.**

proximomedial radius appears to be the primary site of interest, but concurrent pathology in distal humerus can also be present. Typically unilateral but bilateral pathology may occur. Pathology at this site arising from presumed trauma/loading may sometimes be differentiated from developmental osteochondrosis (OCD, bone cyst) on clinical history but possible that there is overlap between the conditions. Osteochondrosis is considered to arise early in life and may not become clinically apparent until put under training loads; however, conclusions about origin and natural progression of injury and potential concurrent injury to soft tissue supportive structures of the elbow are limited by current lack of histopathological data.

Cause

- Presumed to result from overloading of medial aspect of elbow joint, but whether repetitive or monotonic loading is greatest contributor is not known.

Risk factors

- Rare condition. Risk factors unknown.
- Typically affects 2 YOs in early or full training.

History

- Typically acute-onset lameness, progressively worsening with continued exercise over short period (days).

Signs

- Typically unilateral forelimb lameness, of moderate severity.
- Lameness usually evident at both walk and trot, and characterised by shortened cranial phase of stride.
- Limb usually palpably normal.

Diagnosis

- Diagnostic blocking: positive response to blocking of elbow joint.
- Radiography: (CrCd and ML projections most useful): frequently unremarkable in initial phase and changes when noted may be subtle; secondary arthritic change may be more obvious than primary lesion. Some affected horses may have (or develop, over period of weeks/months) irregular subchondral radiodensity +/– proliferative bony reaction on proximomedial aspect of radius (Figure 6.163a). OCD: irregular subchondral radiodensity/loss of congruity or flattening of joint margins. Cysts: subchondral lucency with associated sclerotic rim (Figure 6.163b,c).
- Bone scan: moderate-marked IRU; as affected site is often on medial aspect of joint, important to obtain cranial view of elbow (Figure 6.164).

Management

- Rehabilitation advice is currently empirical and may be influenced by individual circumstances.
- Conservative management (stable rest/walking +/– intra-articular medication). Rest period dependent on clinical/radiographic severity but typically 8–12 weeks out of cantering exercise.

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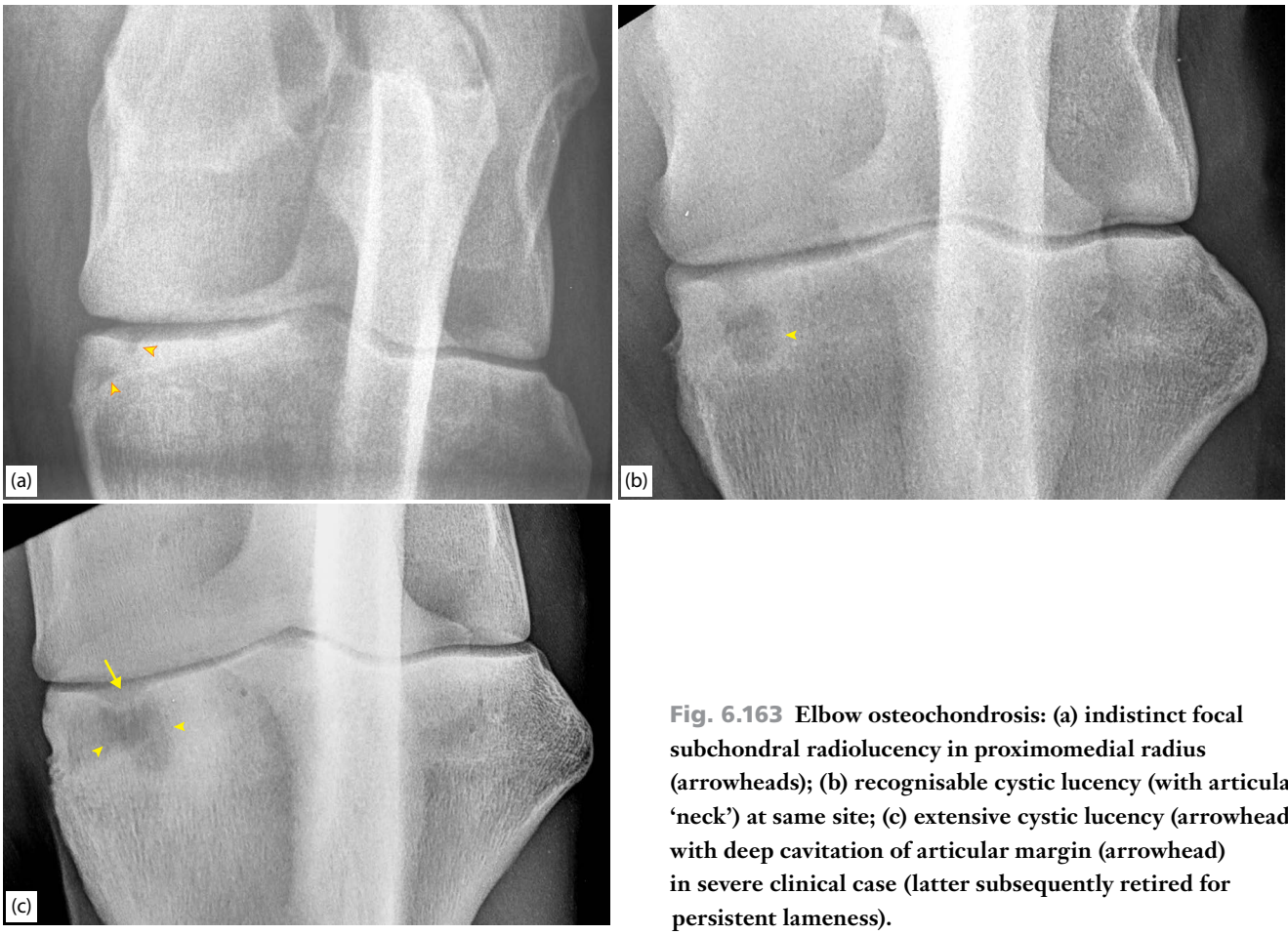


Fig. 6.163 **Elbow osteochondrosis: (a) indistinct focal subchondral radiolucency in proximomedial radius (arrowheads); (b) recognisable cystic lucency (with articular 'neck') at same site; (c) extensive cystic lucency (arrowheads) with deep cavitation of articular margin (arrowhead) in severe clinical case (latter subsequently retired for persistent lameness).**

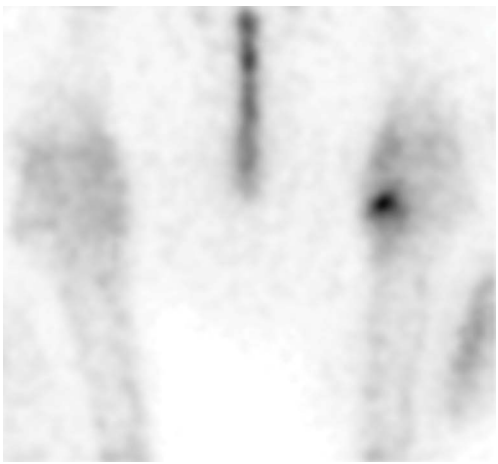


Fig. 6.164 **Elbow osteochondrosis: Intense focal IRU in proximomedial radius (cranial scintigram) characteristic of osteochondrosis/bone cyst/subchondral bone injury (lateral view frequently unremarkable).**

- Intra-articular medication + continued training less favoured due to possibility of potentiating further subchondral injury and secondary OA.
- For confirmed/suspected bone cysts: surgery should be considered if conservative/medical treatment fails; surgical implant placed across or adjacent to the subchondral lesion in cases of proximomedial radial lucencies (cyst/OCD/other) has been demonstrated to have good efficacy for return to athletic soundness.

Prognosis

- Subchondral bone injury (and OCD): fair to good prognosis for racing soundness with conservative management. Periodic intra-articular medication may occasionally be required to maintain soundness.
- Bone cyst: prognosis for return to racing soundness is guarded, but may be improved with surgery.

Capped elbow (bursitis/'shoe boil')

Focal swelling at point of elbow caused by direct trauma. Synovial bursa exists between the skin and the tendons of

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the lateral and medial heads of the triceps brachii muscle. Initial inflammation typically resolves to leave semi-permanent acquired subcutaneous bursa or fibrous pad. Typically unilateral.

Cause
- Self-trauma: usually results from elbow being struck by heel of same leg while horse lying down. May also occur following a fall.

Risk factors
- Any age/stage of training.

History
- Acute onset.
- Not associated with exercise.

Signs
- Fluctuant, fluid-filled bursa over point of elbow (Figure 6.165); painful in initial phase.
- +/– superficial abrasion and local infection.
- +/– mild lameness.

Diagnosis
- Clinical findings definitive.

Management
- Systemic and topical (osmotic/non-steroidal creams) anti-inflammatory medication in acute phase.
- Do not require rest.

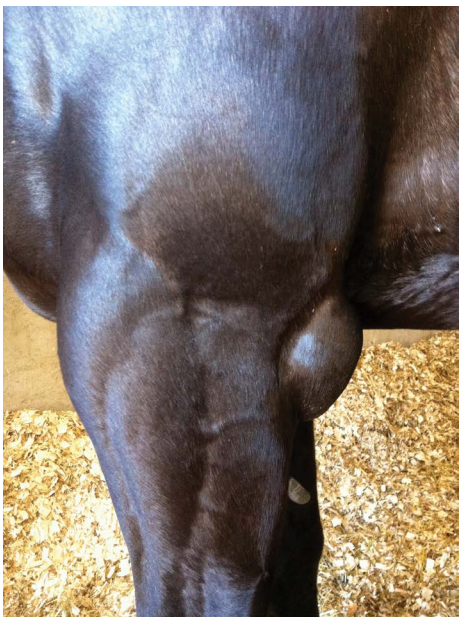


Fig. 6.165 **Capped elbow.**

- Treatment may be desired for large bursae remaining after acute inflammation has subsided. Favourable response to intra-thecal medication (corticosteroid) or surgical drainage.
- Prevent recurrence by use of pastern ('donut'/'sausage') boot on affected leg while stabled: blocks contact between shoe/heel and point of elbow when lying down.

Prognosis
- Rarely interferes with training.
- May remain a minor cosmetic blemish.

Accessory ligament of the superficial digital flexor tendon ('superior check' ligament) desmopathy

The AL-SDFT assists the SDFT and suspensory ligament in preventing overextension of the forelimb during load bearing. Strain of the ligament can cause lameness +/– effusion of the carpal synovial sheath, with which it has a close association. Typically a solitary and unilateral injury.

Cause
- May sustain injury through acute overload of limb or accumulated degenerative change.
- Loading conditions predisposing to injury, and relationship to concurrent tendon/suspensory problems poorly understood; muscle fatigue may play a role.

Risk factors
- Rare condition.
- Predominantly an injury of older (≥4 YO) horses.
- May arise during slow (including trotting) or fast exercise phases of training.

History
- Acute- or chronic-onset forelimb lameness.

Signs
- Palpable thickening (+/– pain) of lesion site caudal/medial lower forearm when limb held in flexion.
- +/– unilateral forelimb lameness of mild-moderate severity; may be intermittent.
- +/– carpal synovial sheath effusion (in proximal cannon +/– lower forearm).

Diagnosis
- Ultrasonography: enlargement/reduced echogenicity/focal fibre disruption of affected ligament (Figure 6.166).

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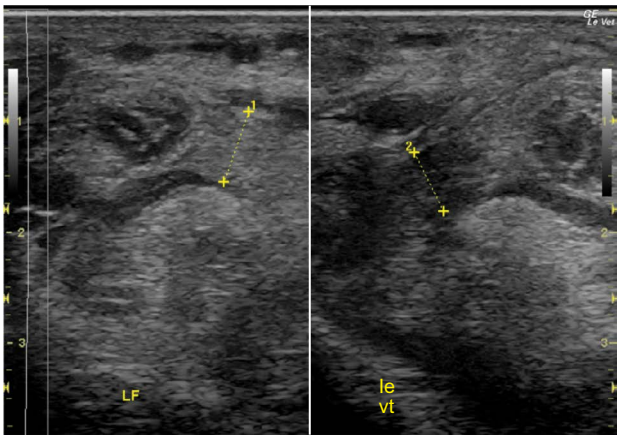


Fig. 6.166 AL-SDFT desmopathy: Transverse ultrasonograms of normal (left) and injured (right) limbs, with calliper annotations delineating AL-SDFT in each image. Note relative loss of echogenicity corresponding to disruption of ligamentous tissue in image on right.

Management

- Conservative management: stable rest/walking for 8–10 weeks with ultrasonographic monitoring to guide return to ridden exercise.
- Anecdotal evidence that swimming/water treadmill during rehabilitation may delay healing.
- Intra-thecal medication unnecessary.
- Surgical debridement may be warranted in some cases.
- Continued training with no/minimal rehabilitation period is possible in selected cases if initial clinical severity is mild.

Prognosis

- Excellent prognosis for return to racing with appropriate management (approximately 90%).
- Subsequent injury/reinjury of suspensory ligament lower in ipsilateral limb may occur on return to training; presumed to result from altered loading of flexor tendon/suspensory apparatus.

THE TARSUS (HOCK)

Applied anatomy

The tarsus (hock), in common with the carpus, is composed of multiple bones and joints and acts as the articulation between the cannon and upper limb (Figure 6.167). The lower row of tarsal bones is dominated by the third tarsal bone (T3), while the second row has the correspondingly large central tarsal bone. Along with the fused first and second and the fourth tarsal bones these make up the distal hock joints. The largest bones in the hock are the calcaneus (part of which forms the point of hock)

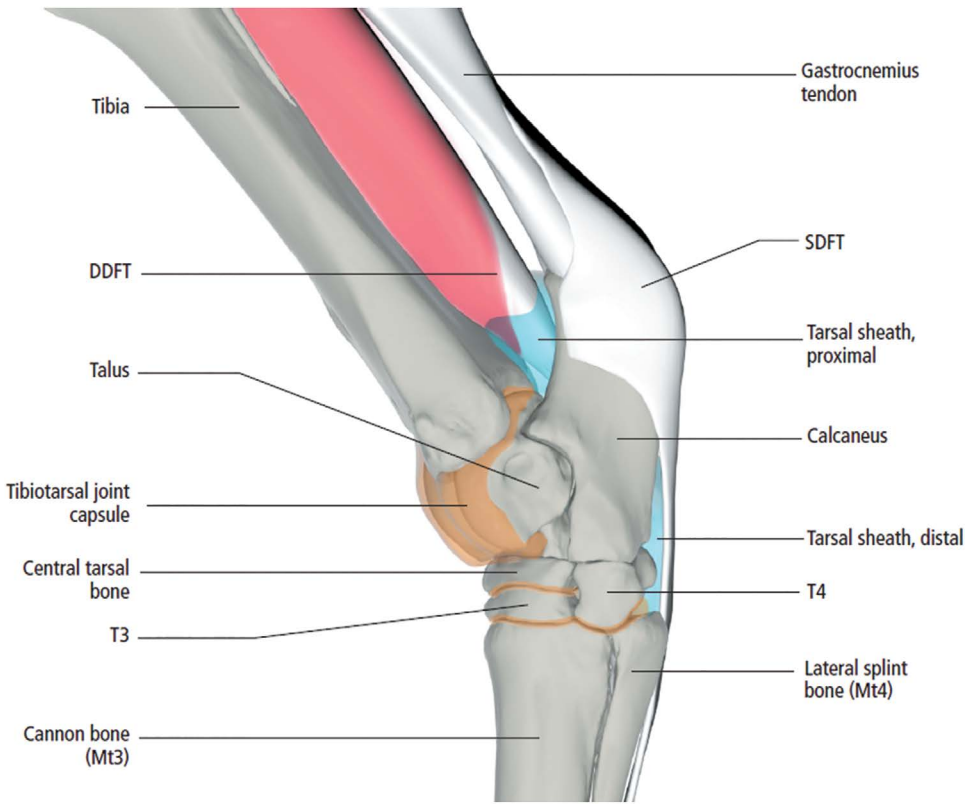


Fig. 6.167 Major structures of the hock.

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and the ridged talus. The hock has three primary joint compartments relevant to the clinician: the tarsometatarsal (TMT) and distal intertarsal (DIT) are low-motion joints associated with the distal tarsal bones, whereas the tibiotarsal joint is the large articulation between hock and tibia.

The soft-tissue structures of primary importance are found at the back of the hock. The tendons of two muscles that cover the caudal tibia merge to become the DDFT. The tendon forms just above the level of the tibiotarsal joint and courses down a groove on the medial aspect of the plantar hock to enter the cannon from a medial position. The DDFT runs through the tarsal synovial sheath, which (just as the carpal sheath does in the forelimb) extends from just above the hock to the upper third of cannon.

The 'Achilles' tendon bundle associated with the point of hock is made up of the gastrocnemius tendon and SDFT. While the gastrocnemius tendon is positioned behind the SDFT in the upper limb, as it approaches the hock it falls to the outside, then cranial to the SDFT, to insert on the point of hock. The SDFT broadens to form a cap over the point of hock then courses down the back of the cannon into the lower limb. These tendons have a close association with the calcaneal synovial bursae.

The collateral ligaments on the outside of the hock are occasionally implicated in lameness. The short lateral collateral ligament is composed of three parts (deep, middle and superficial), which arise on the lateral malleolus of the tibia and insert close to each other on the lateral aspect of the talus. The long lateral collateral ligament also arises on the lateral malleolus of the tibia (just behind the short collateral ligament) and runs nearly perpendicular to it to insert on the distolateral aspect of the calcaneus. The long lateral collateral ligament is placed under tension when the leg is in extension.

Flexion of the hock occurs entirely in the uppermost (tibiotarsal) joint, which is the articulation between the trochlea of the talus and the lower end of the tibia. There is minimal movement (primarily translational/rotational) at the small joints of the hock. The distal joints are loaded in compression. Because of the reciprocal apparatus in the hindlimb, flexion of the normal hock cannot occur in isolation to flexion of the fetlock, stifle and hip.

Synovial communications

- TMT and DIT joints: direct communication in approximately 25% of hocks; diffusion of drugs between these joints in majority (75–100%).
- TMT and tibiotarsal joints: communicate in small proportion (<5%) of hocks.
- Proximal intertarsal and tibiotarsal joints: communicate in all cases and have a common joint capsule.
- Subcutaneous calcaneal bursa communicates in approximately 40% of hocks with a synovial space made up of two compartments (intertendinous and gastrocnemius calcaneal bursae) that interpose between and around the tendons.

Examination

Conformation of the hock/hindlimb may influence risk of injury and assessment can assist lameness investigation. 'Straight through the hock' conformation is associated with reduced energy absorption and may contribute to the development of distal hock joint OA or suspensory ligament desmopathy. 'Sickle-hock' conformation may predispose to the development of 'curb'. Palpation of the limb while weight-bearing permits assessment of tibiotarsal joint or tarsal sheath effusion; however, examination of most other structures is best done with the limb raised. Firm palpation of the front of the lower hock/upper cannon may elicit a pain response in the presence of a fracture involving the lower hock bones or metatarsus.

As with the proximal cannon/lower carpus region of the forelimb, diagnostic blocking may not permit accurate differentiation of pain arising from different structures in and around the lower hock joints and proximal cannon. Accuracy of injection of TMT joint is high but by contrast that of DIT is poor: use of radiography is recommended to ensure correct needle placement for latter joint.

Distal hock joint osteoarthritis ('bone spavin')

Lameness arising from OA of the lower hock (TMT/DIT) joints is less prevalent in racehorses than in disciplines that require sharp turns or jumping. The condition is largely defined by localization of lameness to the site (and elimination of other pathology) rather than imaging, as there is a poor correlation between clinical abnormality and radiological findings (latter are common).

Cause

- Causative factors unknown.
- Loading (compression) forces are greatest on outer aspect of front of lower hock/upper cannon.

Risk factors

- Uncommon condition.
- Any age/stage of training.
- Tarsal bone collapse (foal), sickle-, straight or cow-hocked conformation may predispose.

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History
- Onset of lameness usually chronic.
- Change in gait (such as reluctance to use particular lead at canter) may be noted rather than overt lameness.

Signs
- Mild unilateral or bilateral hindlimb lameness.
- Rarely, acute lameness of moderate severity.
- Reduced hock flexion, lower and shorter limb flight during swing phase of the gait when trotting.
- Toe catch and positive response to flexion test may be features but are not specific.
- Hock palpably normal.

Diagnosis
- Exclusion of other possible local causes of lameness (proximal suspensory ligament desmopathy, tarsal bone fracture).
- Diagnostic blocking: complete or partial response to intra-articular blocking of TMT and/or DIT joints.
- Radiography (and less commonly bone scan): generally reserved for cases that fail to respond to intra-articular medication or those that display uncharacteristically severe lameness, and results of imaging should be interpreted with caution.
- Little correlation between radiological changes (spurring at top of MT3/altered density of articular margins of small hock bones/joint space narrowing) (Figure 6.168) and lameness. Radiological abnormalities (sometimes profound) are commonly encountered as an incidental/clinically irrelevant finding at pre-purchase assessment or injury screening.
- Bone spurs in lower hock (Figure 6.169) are found in up to a third of yearlings and are of little importance. May arise from OA or insertional strains of dorsal ligament/tendons; radiological differentiation not usually possible.
- Marked arthritic change involving DIT joint tends to be more significant than similar change in TMT joint (Figure 6.170a,b) and when detected in a juvenile horse may represent a risk for future development of lameness arising from this site.
- Bone scan: of limited use; variable degree of focal IRU and poor correlation between increased activity and lameness.

Management
- Rest or reduction in training intensity generally not recommended.
- Intra-articular medication as required.

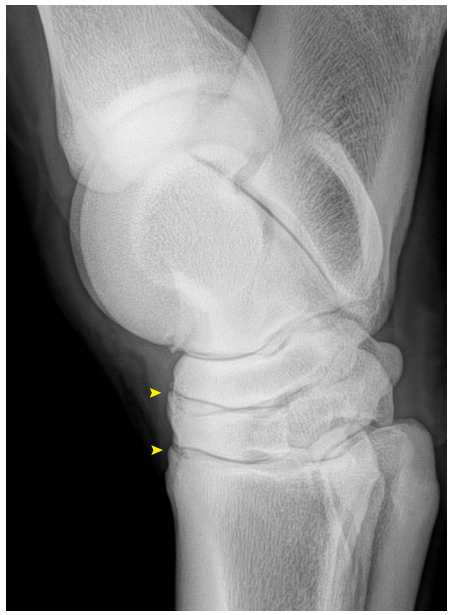


Fig. 6.168 Articular modelling (arrowheads) of TMT and DIT joints (LM).

- Unsatisfactorily response to joint medication should prompt review to ensure that original diagnosis was correct.
- Clinically severe cases non-responsive to conventional medication (rare): fusion (chemical/surgical) of affected joint/joints can be considered; however, this is a treatment of last resort and very rarely warranted or undertaken in racehorses.



Fig. 6.169 Dorsoproximal MT3 'spur' (LM).

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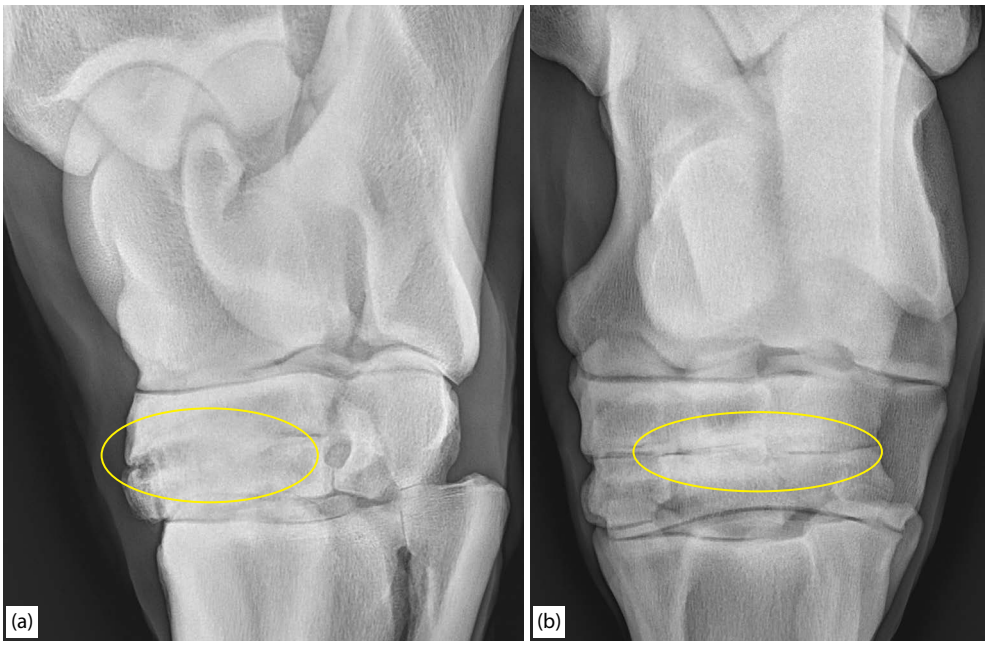


Fig. 6.170 **DIT joint osteoarthritis (severe): Markedly increased density with mixed areas of lysis and partial obliteration of joint space (circled) on DLPMO (a) and DPl (b) projections.**

Prognosis

- Rarely interferes with training.
- Most cases manageable with periodic intra-articular medication.
- Deterioration of action or unsatisfactory response to medication indicates need for further investigation.

Osteochondrosis: Osteochondritis dissecans (OCD)

Osteochondral lesions develop in the foal. Predilection sites are the distal intermediate ridge (most common), the medial and lateral malleoli of the tibia and the lateral trochlear ridge of talus (within the tibiotarsal joint); also 'teardrop' fragments distal talus (Figure 6.171a,b). Unilateral or bilateral. (See also 'Selection of the Racehorse' (p. 407)).

Cause

- See Osteochondrosis (Overview) (p. 267).

Risk factors

- Tarsal osteochondrosis lesions found in approximately 4–8% sales yearlings.
- Heritability is likely to be modest rather than high.

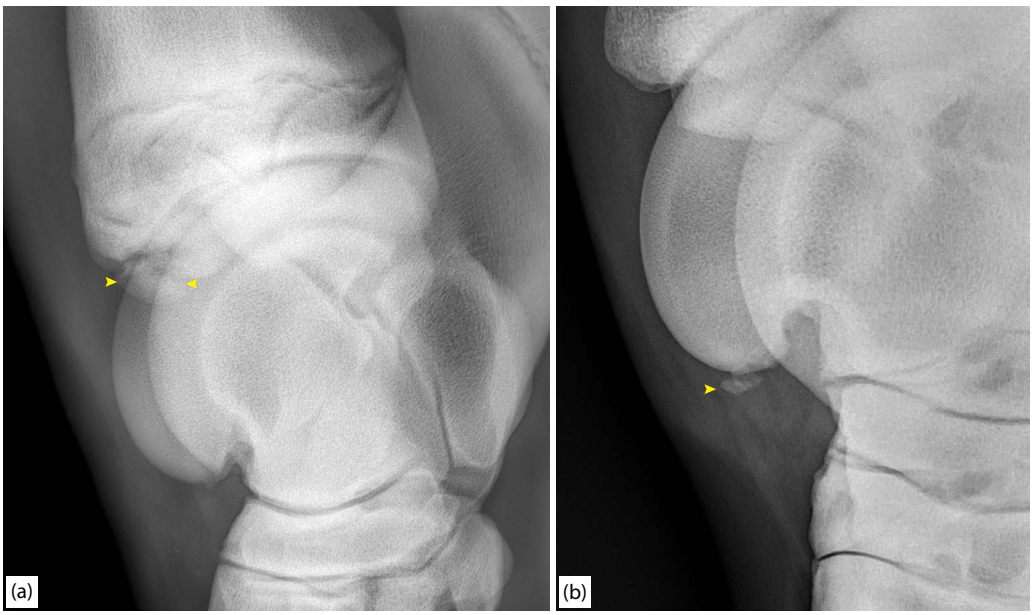


Fig. 6.171 **Tarsal osteochondrosis: Osteochondral fragments at (a) distal intermediate ridge of tibia ('DIRT'); (b) distal talus (DMPlLO).**

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History
- Typically an incidental finding at routine or pre-purchase radiography.
- Less commonly present with joint effusion +/– lameness; onset of signs usually insidious.

Signs
- Frequently clinically silent.
- +/– effusion of uppermost (tibiotarsal) hock joint: 'bog spavin'.
- Lameness an inconsistent feature; when present is usually mild.

Diagnosis
- Radiography is imaging modality of choice.
- Ultrasonography: used less frequently but can be more sensitive than radiography for some osteochondrosis lesions.

Management
- If detected at early yearling stage: surgical removal frequently advocated for all but the smallest lesions, in view of potential to affect saleability (for premier yearlings) or to cause joint effusion/lameness in training; however, not always justifiable on clinical grounds.
- If detected after yearling-sale purchase or after commenced training career: management dependent on clinical signs. No treatment required if clinically silent. In presence of lameness +/– joint effusion attributable to osteochondrosis lesion: intra-articular medication in first instance and surgery only if poor response.
- Dorsodistal fragments of the talus require no intervention and can be considered a benign incidental finding with no effect on racing performance.

Prognosis
- Rarely interrupts training.
- Good prognosis for continued (or return to) athletic soundness.
- Horses that have osteochondrosis surgery as yearlings are just as likely to race as 2 and 3 YOs as their siblings.
- Surgery resolves joint effusion in most cases.
- Size of distal intermediate ridge lesions has no effect on prognosis.

Third tarsal bone fracture

Fracture of the third tarsal bone (T3) typically occurs in the frontal plane and traverses the bone from the distal intertarsal to the TMT joints ('slab' fracture). Less commonly in oblique sagittal plane in lateral portion of bone



Fig. 6.172 T3 slab fracture of right hock: Transverse CT image showing oblique sagittal fracture (arrowheads) through zone of markedly increased radiodensity in lateral portion of bone.

(Figure 6.172). Occasionally, incomplete fracture occurs at site (Figure 6.173a,b). Unilateral. Separate condition of dorsal/dorsodistal modelling + fragmentation of T3 occurs in untrained juvenile (foal/yearling) horses: clinically unimportant and discussed further in Chapter 18, (p. 406).

Cause
- Acute injury sustained during fast exercise.
- Results from compressive forces through portion of lower hock subject to greatest loading.
- Slab fracture also encountered in juvenile/yearling Thoroughbreds, presumably sustained at pasture.

Risk factors
- Uncommon condition.
- Affects all ages; usually in horses in fast exercise phase of training.
- 'Wedge' shape conformation of T3 (Figure 6.174) may predispose to injury, although injuries do not occur only in bones with this shape. Risk is currently unquantified, but more severely affected bones (arising from neonatal 'collapsed'/incompletely ossified hocks, Figure 6.175a,b) considered to carry higher risk.
- Likely that external factors (e.g., track type/geometry, training speed) may play a role in development of injury in some cases.

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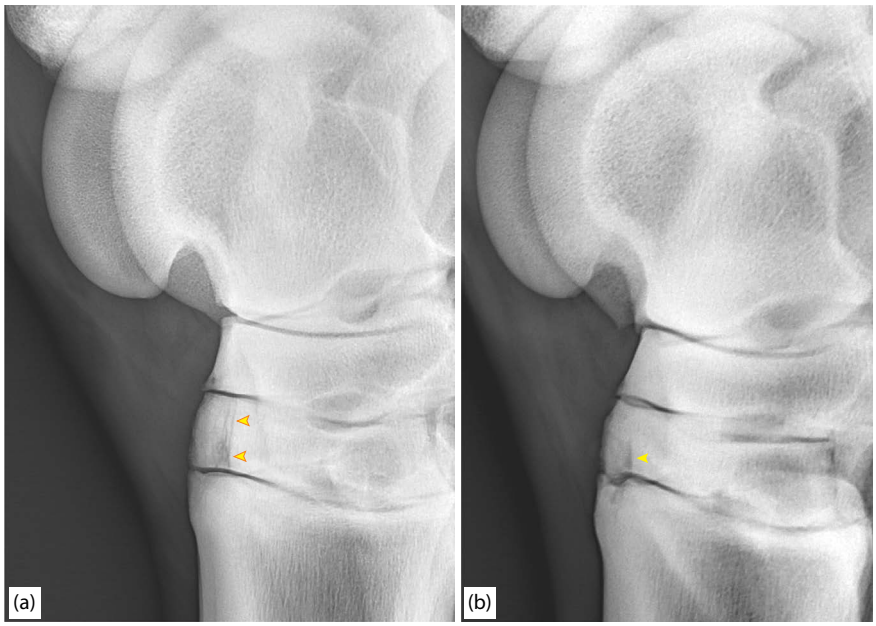


Fig. 6.173 **Frontal plane T3 fracture: (a) slab fracture (extending between TMT and DIT joints); (b) incomplete fracture in distal T3 (DMPlLO).**



Fig. 6.174 **'Wedge'-shaped T3 of medium severity (assessed on DMPlLO projection).**

History

- Acute-onset hindlimb lameness, usually noted after cooling off following fast work.
- Lameness may diminish rapidly over initial days if rested.
- Less commonly: mild-moderate hindlimb lameness, progressively worsening over days/weeks.
- May (rarely) be detected as incidental finding during radiographic survey of clinically normal horses with no prior history of lameness (Figure 6.176); in these cases presumed to have been sustained early in life.

Signs

- Moderate-severe unilateral hindlimb lameness.
- No visible abnormality of limb.
- Consistent, mild-marked pain response to palpation of front of lower row of hock bones.

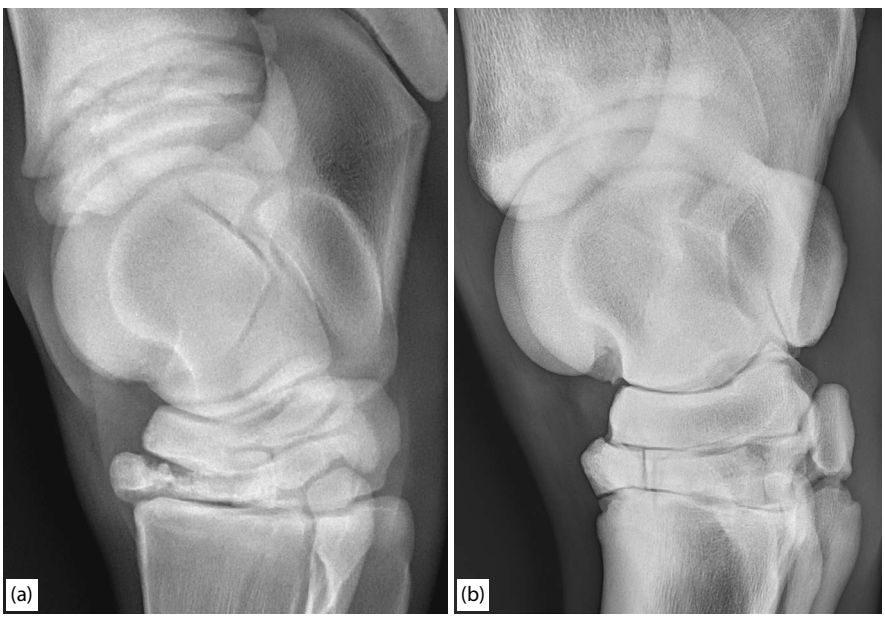


Fig. 6.175 **Developmental condition of neonatal incomplete ossification/tarsal bone collapse (a), leading to severe manifestations of adult wedge-shaped T3 (b) that can predispose to T3 fracture (latter also present in image b) (DMPlLO).**

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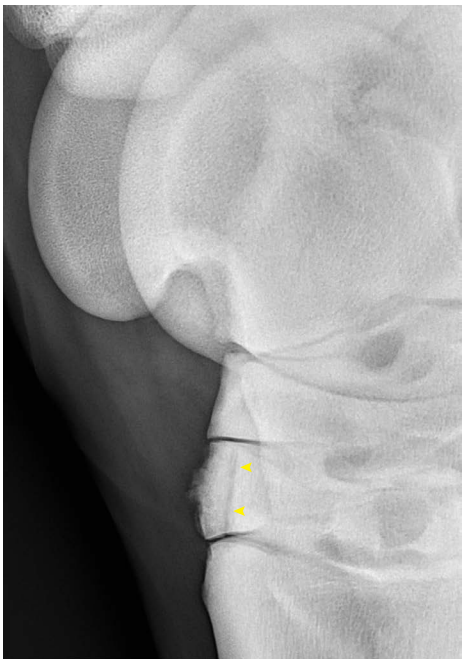


Fig. 6.176 T3 slab fracture detected as incidental finding during pre-purchase radiographic screening; no prior history of injury and remained clinically unimportant throughout career in this case.

- Occasionally subtle soft tissue thickening/heat at same site.
- Rarely: incidental radiological finding with no clinical abnormality.

Diagnosis
- Generally easily diagnosed with radiography: fracture line usually only visible on DMPlLO projection; multiple projections may be required.
- If not visible at initial examination, repeat radiography 7–14 days post-injury.
- Bone scan: sometimes required if radiography inconclusive; intense focal IRU.
- CT may assist surgical planning/management.

Management
- Most cases respond well to conservative management: stable rest (4–8 weeks)/walking (4–6 weeks).
- Athletic outcome (return to racing rates) similar for both conservative management and surgical fixation (lag screw); however while surgery not obligatory for good outcome, features that may lead to recommendation of surgery as treatment of choice include gross fragment displacement, marked 'wedging', and incomplete fractures (latter may heal incompletely or recur after conservative management).
- When detected at radiographic screening of clinically normal juvenile/yearling Thoroughbreds: no need for confinement/intervention; continued pasture turnout possible.

Prognosis
- Good prognosis for return to full use with both conservative and surgical management.
- Guarded prognosis for return to full use in horses with pre-existing marked collapse/wedging of distal tarsal bones.
- May display mild/moderate hock lameness (may be bilateral, and sometimes transient) on return to cantering: respond favourably to intra-articular medication.
- Reinjury is rare, although fracture of T3 in opposite hock may occasionally occur later in career.
- In juvenile/yearling Thoroughbreds, good radiological healing appears to occur in most horses without any need for intervention.

Central tarsal bone fracture
Fracture of the central tarsal bone may occur in the frontal or sagittal plane, and may be complete or incomplete. Unilateral.

Cause
- Presumed to result from compressive forces during loading as for T3 fracture (p. 208).

Risk factors
- Rare condition.
- Affects all ages; usually in horses in fast exercise phase of training.
- Risk factors unknown.

History
- Acute-onset hindlimb lameness, usually noted after cooling off following fast work.

Signs
- Moderate-severe unilateral lameness.
- Generally no visible abnormality of limb.
- Consistent, mild-marked pain response to palpation of front of hock.
- +/– tibiotarsal joint effusion.

Diagnosis
- Radiography: fracture line best visualized on DPl, DPl oblique or LM projections (Figure 6.177a,b).
- If not visible at initial examination, repeat radiography at 7–14 days post-injury.

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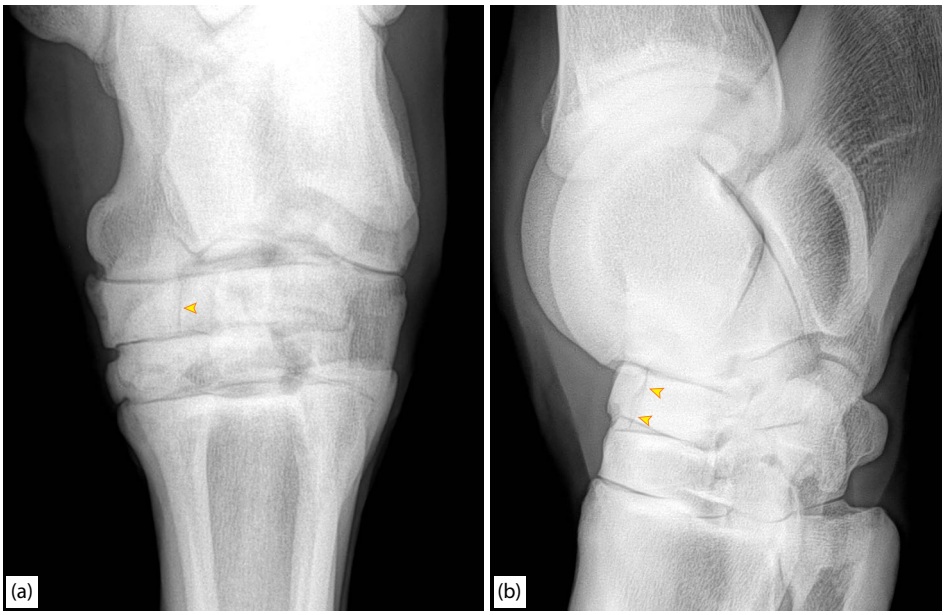


Fig. 6.177 **Central tarsal bone fracture: (a) sagittal (DPl) and (b) frontal (LM) plane injuries.**

- Bone scan: sometimes required if radiography inconclusive; intense focal IRU (Figure 6.178).
- CT: favoured imaging modality to assess fracture configuration for surgical planning.

Management

- Surgical fixation offers best chance of return to racing soundness.
- Conservative management: stable rest (6–8 weeks)/walking (4–6 weeks) an acceptable but less successful option.



Fig. 6.178 **Central tarsal bone fracture: Lateral scintigram in acute phase of injury with associated IRU (arrowhead).**

Prognosis

- Surgery: good prognosis for return to racing (approximately 70%).
- Conservative management: guarded-poor prognosis for return to full use (approximately 30%).

Dorsoproximal articular stress fracture of MT3

Stress fracture of the dorsoproximal MT3 (cannon) is a sporadic injury. Fracture configuration is typically oblique and in the frontal plane with articular (TMT joint) involvement. Usually incomplete, although minimally displaced complete fractures can occur. Unilateral or bilateral.

Cause

- Stress injury.
- Peroneus tertius/cranial tibial tendon insertions at top of cannon may contribute to cyclical loading at site.

Risk factors

- Rare condition.
- Typically in horses in cantering/fast exercise phase of training.
- Risk factors unknown.

History

- Acute-onset hindlimb lameness is most typical, but chronic/low-grade poor action may also occur.

Signs

- Typically a unilateral, moderate-marked lameness.

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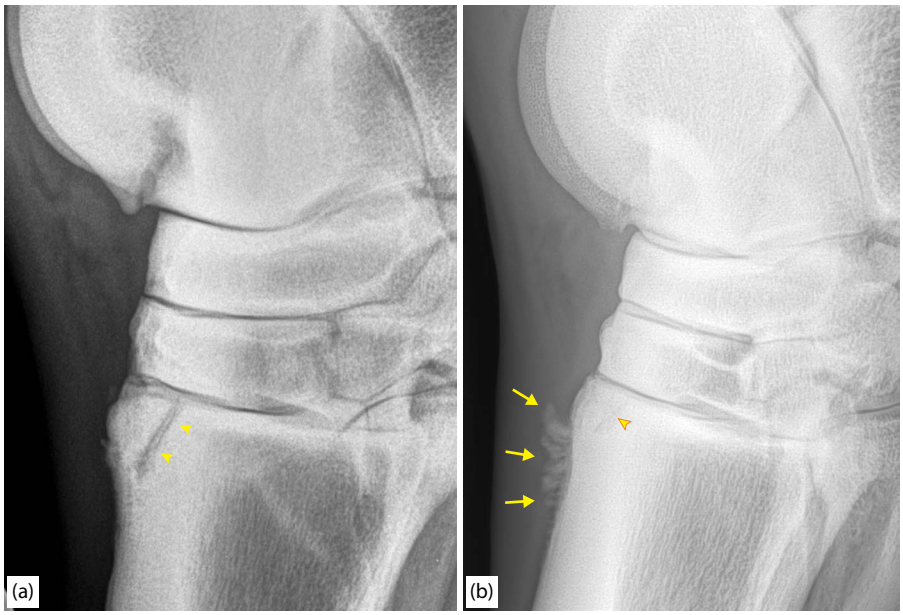


Fig. 6.179 **Dorsoproximal articular stress fracture of MT3: (a) clear oblique fracture line (arrowheads) communicating with TMT joint; (b) less distinct radiolucency at same site (arrowhead) but with associated marked periosteal proliferative change distally on MT3 (arrows); latter feature is characteristic of injury even in absence of radiologically visible fracture line.**

- Less commonly: bilateral hindlimb lameness/poor action that may resemble that associated with other pathology (e.g., tibial stress fracture)
- +/– pain response to palpation of front of high cannon/low hock region.
- +/– prominence of soft tissue/bone at site.

Diagnosis

- Radiography is definitive: oblique fracture line propagating from TMT joint to dorsolateral cortical margin of upper cannon bone +/– periosteal reactivity in region of distal end of fracture (Figure 6.179a,b).
- Bone scan not required for diagnosis but sometimes undertaken as initial imaging modality in horses with bilateral lameness: marked focal IRU at affected site(s) (Figure 6.180).

Management

- Conservative management: stable rest (4–6 weeks)/walking (4–6 weeks).
- Surgery (implant/osteostixis) can be considered for horses with chronic/recurrent injury.

Prognosis

- Excellent prognosis for return to full use.
- Uncommonly, poor radiological +/– clinical resolution of injury; return to training is generally possible regardless of non-healing fracture site and residual mild/moderate lameness.

Lateral malleolar fracture

The lateral malleolus forms the lower outer aspect of the tibia and serves as an attachment point for the long and short collateral ligaments of the hock. Fracture typically results in a displaced articular (tibiotarsal joint) fragment. Unilateral.

Cause

- Acute traumatic injury.
- Collateral attachments and origin as a separate centre of ossification (fuses with tibia in first year) may predispose it to avulsion fracture.



Fig. 6.180 **Dorsoproximal articular stress fracture of MT3: Marked IRU on lateral scintigram.**

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Risk factors
- Rare condition.
- More common in jumping disciplines (hurdling/steeplechase) than in flat racing.

History
- Acute-onset lameness: usually sustained during a fall, while being cast or from direct trauma such as a kick.

Signs
- Unilateral hindlimb lameness, generally of moderate severity.
- Typically some effusion of tibiotarsal joint.
- Usually with painful soft-tissue thickening over outside of hock in acute phase.

Diagnosis
- Clinical findings are non-specific.
- Radiography: definitive. Best observed on a DPl or D5^{0}MPlLO projection (Figure 6.181a,b).
- Ultrasonography: lateral malleolar fracture always involves some disruption to the short +/– long collateral ligaments. Ultrasound assessment not essential but may assist surgical planning.

Management
- Surgery (arthroscopic removal) is treatment of choice.
- May also respond favourably to conservative management: stable rest (4–6 weeks)/walking (4–6 weeks).

Prognosis
- Surgery: good-excellent prognosis for return to full use.
- Conservative management: fair-good prognosis for return to full use.

Lateral collateral ligament desmopathy

The short and long lateral collateral ligament(s) of the hock arise from the lateral malleolus of the tibia and run nearly perpendicular to each other to insert on the outside of the talus and calcaneus, respectively. They provide ML and torsional stability to the hock and may occasionally be the primary source of lameness (unrelated to lateral malleolar fracture, p. 212). Injury is most commonly associated with the short collateral ligament and predominantly involves the distal insertional site of this ligament on the talus. Lameness is typically unilateral although imaging abnormalities may be bilateral. Relationship with other/concurrent articular pathologies in hock may occur but is poorly understood at present.

Cause
- Not known whether injury arises from acute or repetitive strain.

Risk factors
- Rare condition.
- Typically occurs in horses in full training.
- Risk factors unknown.

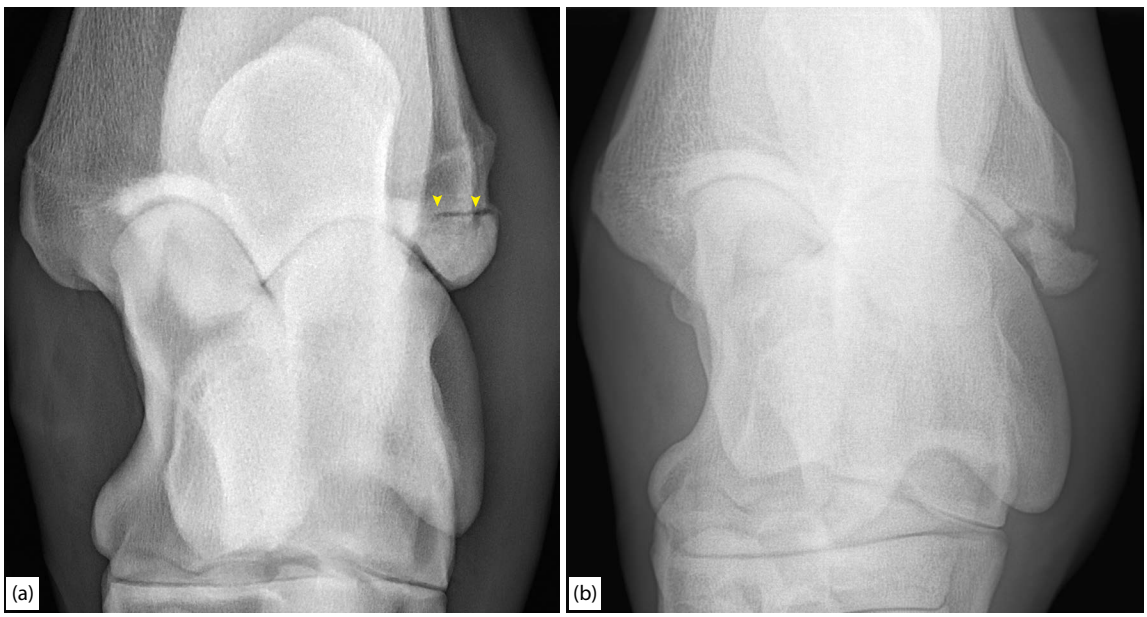


Fig. 6.181 **Lateral malleolar fracture. Typical appearance of non-displaced (a) and displaced (b) fracture fragments (DPl).**

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History
- Acute- or insidious-onset hindlimb lameness.
- Radiological evidence of previous/settled injury may occasionally be encountered as an incidental finding in horses without lameness.

Signs
- Unilateral hindlimb lameness, generally of moderate severity and worsening with workload.
- Lameness typically exacerbated following flexion test.
- Typically without obvious palpable abnormality of hock, although some cases may have focal painful swelling over injury site.
- +/– effusion/haemarthrosis of tibiotarsal joint (uncommon).

Diagnosis
- Clinical findings are non-specific.
- May be a diagnostic challenge: radiological signs may be subtle or easily missed without multiple lesion-oriented projections, and lack of clinical localising signs may result in underutilisation of ultrasonography in the first instance. Diagnosis frequently occurs through bone scan, with subsequent detection of ultrasound/radiological features once injury site has been determined.
- Diagnostic blocking: response to intra-articular blocking of tibiotarsal joint is inconsistent.
- Radiography: DPl and multiple lesion-oriented oblique projections most useful. Irregular bone reaction (enthesopathy) of the lateral talus +/or calcaneus is typical; degree of maturation of bone changes at time of diagnosis varies between cases (Figure 6.182a–c).
- Ultrasonography: moderate diffuse disrupted fibre pattern, and evidence of disruption/irregularity to bone interface (Figure 6.183).
- Bone scan: focal IRU of moderate-marked intensity, typically at distal insertional site (lateral talus) (Figure 6.184a–d). IRU of similar pattern has been reported with subchondral bone injury of dorsodistolateral calcaneus, and differentiation of these pathologies may require multi-planar imaging (CT/PET).

Management
- Conservative management: initial stable rest until free from lameness, then controlled exercise (walking). Lengthy (months) rehabilitation periods associated with best outcomes.
- Adjunctive therapies (intra-lesional medications, ESWT, etc.) may be prescribed but no good evidence that they improve athletic outcome.
- Intra-articular medication generally ineffective at limiting lameness.
- Continued training in the face of lameness may be possible in some cases providing clinical deterioration does not occur; may be preferable to lengthy rehabilitation with uncertain outcome (or retirement/sale).

Prognosis
- Guarded prognosis for return to full soundness: approximately 50% of horses may develop recurrent lameness upon return to full training following rehabilitation period.
- Short periods of rest are generally unrewarding and should not be considered.
- Some horses develop OA of tibiotarsal joint, presumably secondary to joint instability or inflammation.

Capped hock
Distension of acquired bursa over point of hock resulting from non-septic inflammation. Variable severity; unilateral or bilateral.

Cause
- Cause generally remains unknown but frequently arises from direct trauma (e.g., during transport, or from kicking walls).
- Can be exercise induced.

Risk factors
- All ages/stages of training but most common in horses in early training (yearling/2 YO).
- Risk factors not known.

History
- Acute or chronic in onset.
- When acute, typically follows incident such as being cast in stable.

Signs
- Unilateral or bilateral subcutaneous enlargement over point of hock (Figure 6.185).
- Severity varies from mild-marked distension.
- Lameness is usually not a feature, even with marked distension.
- Usually no pain on palpation.

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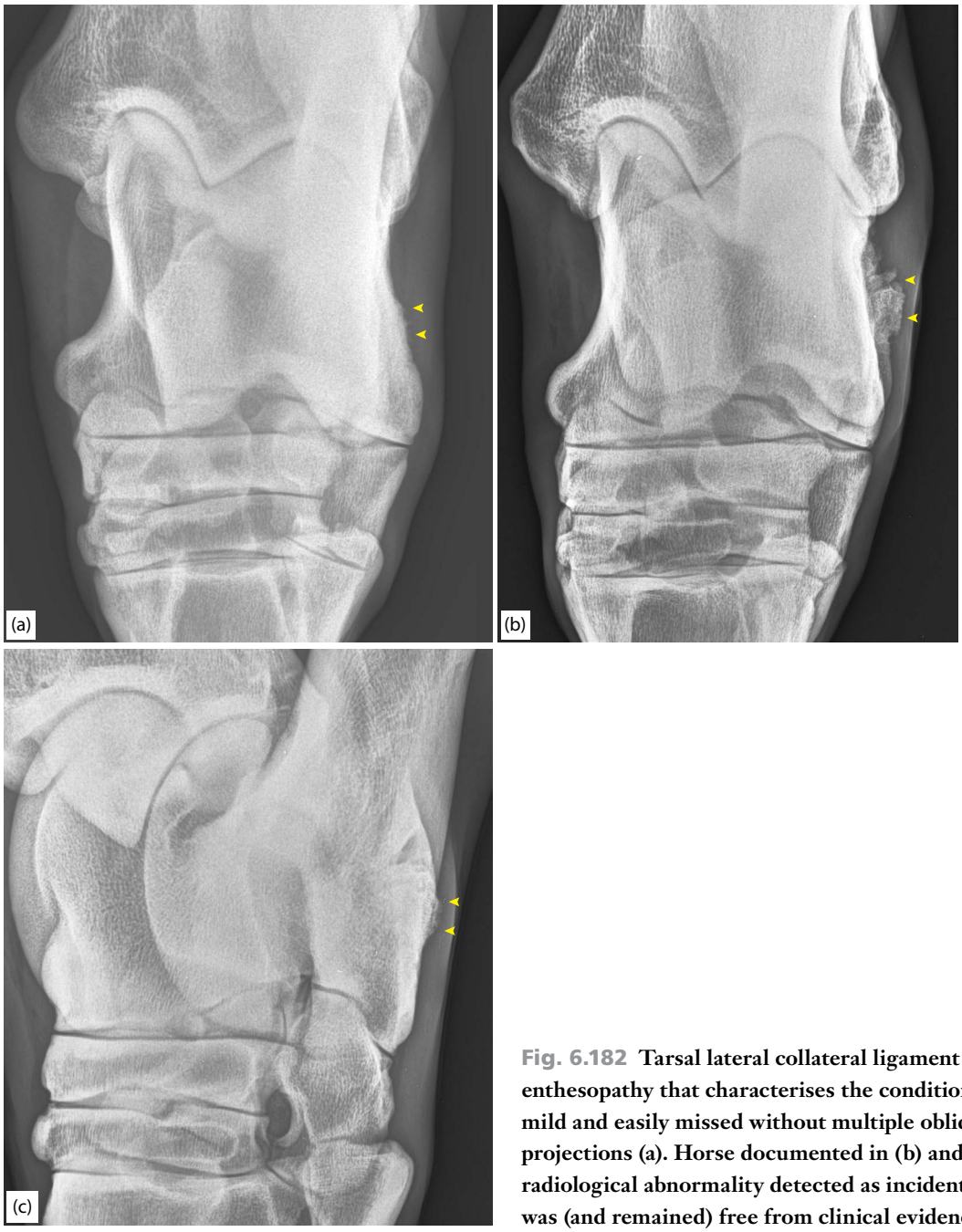


Fig. 6.182 Tarsal lateral collateral ligament injury: Proliferative enthesopathy that characterises the condition is typically subtle/mild and easily missed without multiple oblique DPl radiographic projections (a). Horse documented in (b) and (c) had marked radiological abnormality detected as incidental finding at this site but was (and remained) free from clinical evidence of injury.

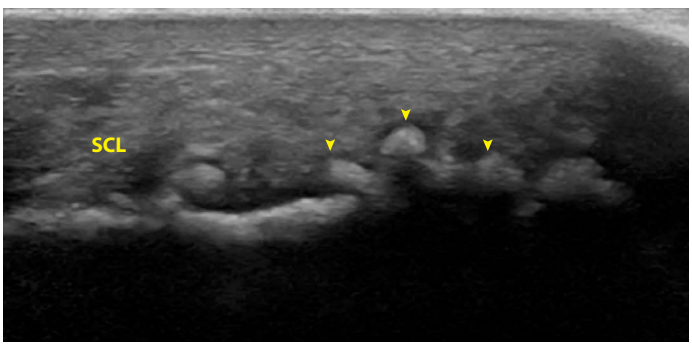


Fig. 6.183 Tarsal lateral collateral ligament injury: Longitudinal ultrasonogram (proximal to left) showing disruption/irregularity of bone margin at distal insertion of ligament (arrowheads); SCL: Short collateral ligament.

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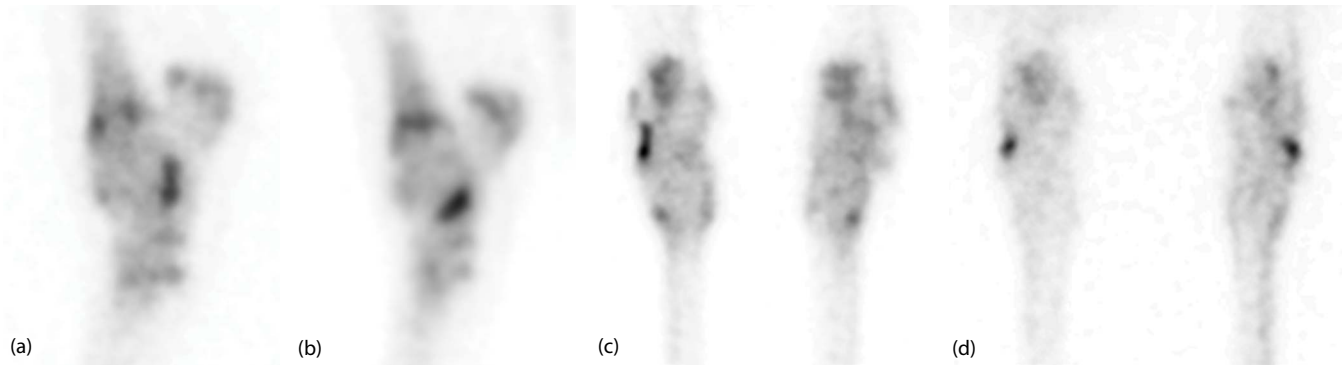


Fig. 6.184 **Tarsal lateral collateral ligament injury: Patterns of IRU associated with injury on lateral (a, b) and plantar (c, d) scintigrams; note bilateral IRU in image (d).**

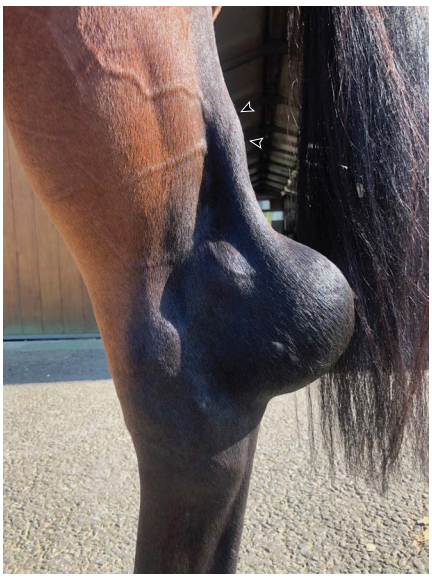


Fig. 6.185 **Permanently capped hock of marked severity: Note also secondary proximal distension of gastrocnemius and intertendinous bursae (arrowheads). Although cosmetically unsightly the condition had no impact on athletic function/performance in this case.**

Diagnosis
- Clinical findings definitive.

Management
- Treatment generally not required.
- If lame with marked distension/palpable pain: further investigation (imaging/synoviocentesis) to rule out bursal infection or tendon injury.
- Short period of rest +/– topical anti-inflammatory therapy may be beneficial.
- Intra-thecal medication of bursa (corticosteroid) warranted in some circumstances if cosmetic improvement desired; however, frequently results in only short-lived improvement.

Prognosis
- Rarely interferes with training; cosmetic blemish only.

Thoroughpin
'Thoroughpin' is a general term used to describe non-septic effusion of the tarsal synovial sheath. Unilateral or bilateral.

Cause
- As with effusion of the carpal synovial sheath, thoroughpin can arise from a number of causes including synovitis or injury to either the lining of the sheath or the structures (DDFT) that run through it.
- Simple synovitis (without overt injury) is most common.

Risk factors
- Rare condition.
- Any age/stage of training.
- Risk factors unknown.

History
- Typically chronic onset (over days/weeks).

Signs
- Well-defined fluid-filled swelling above hock, between Achilles tendon bundle and back of tibia (usually most prominent on outside of leg) +/– distension of sheath immediately below hock around flexor tendon bundle.
- Distinguish from other fluid swellings that can occur in same region: 'bog spavin'/'false' thoroughpin/calcaneal bursa.

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- Not usually associated with lameness.
- Presence of lameness may indicate injury to either the associated bones of the hock or DDFT (or infection) and should prompt further investigation.

Diagnosis
- Clinical findings usually definitive.
- Investigation only warranted if lame (rare).
- Radiography: DMPlLO and skyline projection of calcaneus useful to determine presence of bone involvement.
- Ultrasonography: may be useful to assess synovial lining, DDFT and bone margins of tarsal sheath.
- Synoviocentesis if infection suspected.

Management
- In the absence of lameness: no treatment or rest usually required.
- Short phase of reduced exercise/topical/systemic anti-inflammatory therapy may be beneficial to overall cosmetic outcome if thoroughpin has developed acutely.
- Marked effusion: intra-thecal medication (corticosteroid).
- Injury to structures within the sheath (DDFT, tarsal bones) is rare: surgery (tenoscopy) and rest offer best chance of resolution.

Prognosis
- Generally excellent: rarely interferes with training.
- May resolve spontaneously.
- Chronic distension of sheath may persist for medium term; usually considered cosmetic blemish only.

False thoroughpin
Discrete, fluid-filled swelling between Achilles tendon bundle and back of tibia. Severity varies between individuals. Result of herniated outpouching (synoviocoele) of tarsal sheath that probably arises from a defect in the sheath wall. Distension of tarsal sheath itself is not usually present. Usually unilateral.

Cause
- Cause unknown.

Risk factors
- Uncommon.
- Any age/stage of training.

History
- Onset is usually over few days/weeks.

Signs
- Fluid-filled swelling above and forward of point of hock and Achilles tendon bundle; usually most prominent on outside of leg (Figure 6.186a,b).
- Distinguished from tarsal sheath effusion ('thoroughpin') by not being palpable through the tarsal canal into upper cannon region.
- Not usually associated with lameness or palpable pain, although may occasionally physically impair action if markedly distended.



Fig. 6.186 **False thoroughpin: Lateral (a) and caudal (b) views; note distension of the acquired bursa on lateral aspect of hock.**

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Diagnosis
- Clinical findings definitive.
- Diagnostic imaging not warranted.

Management
- Unless large, no treatment or rest necessary.
- If large and considered to be affecting action (unusual), intra-thecal medication (corticosteroid); however, improvement is typically incomplete, recurrence common in short/medium term due to continued production/egress of fluid.

Prognosis
- Prognosis for athletic function is excellent, although continued/recurrent effusion is typical. Rarely interferes with training and generally of cosmetic importance only.

Lateral extensor tenosynovitis/ganglion

Effusion +/– synovial herniation ('ganglion') of the lateral extensor tendon sheath on the outside of the upper hock/lower tibia. Severity varies. Typically unilateral.

Cause
- Cause unknown.

Risk factors
- Uncommon condition.
- Any age/stage of training.

History
- Onset is usually acute.
- Initially develops as oedematous thickening over lateral aspect of hock +/– mild lameness; once acute inflammation subsides (days), discrete fluid-filled swelling remains.

Signs
- Fluid-filled swelling/pouch of variable size on lateral aspect of upper hock/lower tibia; may extend up along tendon sheath over lower tibia.
- +/– initial palpable focal thickening of extensor tendon mid-hock.
- Usually without lameness/palpable pain after acute phase.

Diagnosis
- Clinical findings definitive.
- Diagnostic imaging not usually warranted: ultrasonography may reveal tenosynovitis/synovial thickening/dissecting fluid in lateral tarsal soft tissues; contrast radiography may assist differentiation from articular pathology (Figure 6.187).



Fig. 6.187 Lateral tarsal synovial 'ganglion': Positive contrast radiograph of severe case showing extent of bursa (arrowheads) on lateral aspect of hock. This case resolved fully with conservative management.

Management
- Acute (initial days): topical and/or systemic anti-inflammatory therapy.
- Alteration of exercise not usually required.
- Intra-thecal medication (corticosteroid) may reduce size of swelling although frequently unrewarding due to continued production/egress of synovial fluid.

Prognosis
- Excellent. Interruption to training is unusual.
- May retain a non-painful swelling in medium/long-term: cosmetic blemish only.

Curb

'Curb' is thickening/bowing of the soft tissues at the back of the hock and describes a clinical syndrome rather than a specific injury, as several structures can potentially be affected. Caused by enlargement/inflammation of one or more of the following: periligamentous/peritendinous tissue, long plantar ligament, SDFT and, less commonly, the DDFT. Should be distinguished from

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simple prominence of the head of the outside splint bone seen in some individuals. Most commonly unilateral but may be bilateral.

Cause
- Maladaptive response (or acute strain) of ligaments/tendons at back of hock to training/loading.

Risk factors
- Common condition.
- Any stage of training but most common in 2 YOs during early cantering phase.
- Horses with sickle/weak-hocked conformation at greater risk.

History
- Acute (most common) or chronic in onset.

Signs
- Soft-tissue thickening/bowing at back of lower hock/upper cannon (Figure 6.188).
- +/– pain response to palpation.
- Not always associated with lameness.
- Broad correlation between severity of clinical findings and presence of lameness: mild thickening less likely to be associated with lameness than extensive, oedematous and painful curb.

Diagnosis
- Clinical findings definitive.

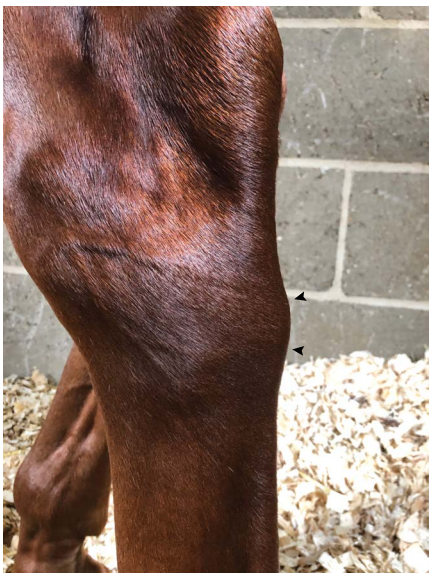


Fig. 6.188 Curb: Typical appearance of convex/prominent profile of caudal lower hock when viewed from side.

- Diagnostic imaging rarely necessary; if desired, ultrasonography is modality of choice.
- Ultrasonography: used to determine which soft-tissue structures are involved/extent of injury.

Management
- Management dictated by severity of clinical signs and is generally symptomatic.
- Mild thickening plus no lameness: no reduction in exercise required.
- Lameness: stable rest or walk until sound and free from palpable pain.
- Moderate-marked thickening plus moderate lameness: several weeks out of ridden exercise usually necessary.
- Topical +/– systemic anti-inflammatory therapy +/– local infiltration with corticosteroid in acute phase may assist recovery if combined with rest.
- Use of anti-inflammatory measures to facilitate continuation of training when rest is more appropriate may result in worsening of injury.
- Limited anecdotal support for counter-irritation for recurrent/troublesome cases non-responsive to rest.

Prognosis
- Excellent prognosis for return to full use regardless of severity.
- Mild cases: no/minimal interruption to training.
- Moderate cases: duration of rehabilitation dependent on initial severity but unlikely to exceed several weeks.
- Once ‘set’, recurrence is rare, although residual curb ‘profile’ is generally a feature.
- Rare cases of SDFT injury with fibre tearing (Figure 6.189) may require lengthy convalescence.

Slipped tendon (luxation of SDFT)

Lateral (very rarely medial) displacement of the fibrocartilaginous cap of the SDFT from its normal position on the point of the hock. Displacement may be partial or full, and in acute phase may slip off and on calcaneus with flexion of the leg. Most commonly unilateral but bilateral injury can occur (rare).

Cause
- Disruption of the calcaneal insertions of the SDFT +/– tearing of the tendon’s fibrocartilaginous cap results in loss of stability of tendon on point of hock.
- Not known whether injury is preceded by degenerative change in affected calcaneal insertion(s).

Risk factors
- Rare condition.

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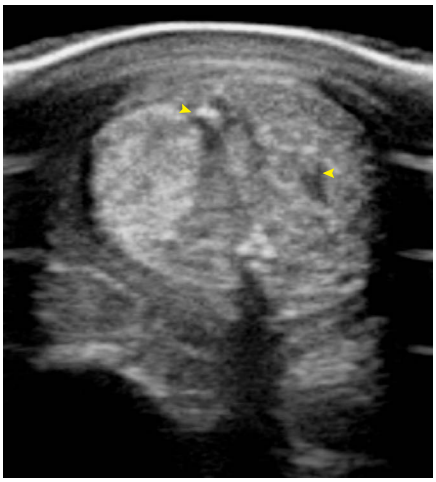


Fig. 6.189 Curb: Transverse ultrasonogram showing mixed echogenicity consistent with SDFT damage and secondary dystrophic calcification in atypical case demonstrating severe/persistent lameness associated with the injury (arrowheads).

- Any age/stage of training; more common in older horses.
- Risk factors unknown.

History
- Typically arises as acute injury at exercise.
- Occasionally preceded by some inflammation at site (distension of calcaneal bursa may be a warning sign).

Signs
- Moderate-severe hindlimb lameness with unusual gait.
- Acute phase: may show considerable distress; sedation may be needed to facilitate examination.
- SDFT usually observed over lateral aspect of hock, but in acute phase may be obscured by secondary inflammation at site.
- SDFT may return to normal position during weight-bearing or with manual assistance; palpation of point of hock and tendinous structures best performed with limb in flexion.
- Calcaneal bursal effusion.

Diagnosis
- Clinical findings definitive.

Management
- Some horses appear to panic when luxation initially occurs and sedation in acute phase may be necessary.
- Goal of treatment is to permit SDFT to settle permanently into new position on side of hock; however, expectation should be that most horses retain an unstable luxation long term.
- Conservative management: stable rest (8–16 weeks) followed by walking. Advisable to apply full-limb immobilizing bandage during initial phase (first 4–8 weeks).
- Systemic anti-inflammatory medication in acute phase.
- Intermittent (unstable) displacement: tenoscopy of calcaneal bursa to permit resection of any torn fibrocartilage cap may offer improved prognosis for return to athletic function.
- Surgical repair to reposition SDFT on point of hock is not favoured; rate of successful return to full use (and normal gait) better than for conservative management but significant risk of serious/potentially fatal complications in immediate post-surgical period.

Prognosis
- Guarded to fair prognosis for return to full use with conservative management.
- Lameness resolves with rest but horses typically retain a gait abnormality due to new position of SDFT; in some cases this may prove performance-limiting for racing.
- Prognosis for non-racing athletic pursuits excellent.

Stringhalt
Acute-onset neuromuscular gait defect characterized by involuntary exaggerated hyperflexion of one or both hocks. May resolve spontaneously or remain as chronic condition.

Cause
- 'Sporadic' form of condition usually affects one leg and can follow injury to front of hock or upper cannon.
- Neurotoxic form ('Australian' stringhalt): related to exposure to certain toxic pasture plants of the flatweed/dandelion group (esp. *Hypochaeris radicata*), although likely interaction of multiple host and environmental risk factors; suspect plants are not inherently a danger to grazing horses at all times/locations. Usually affects both hindlimbs. Neurotoxicity causes a generalized nerve dysfunction (distal axonopathy) with resulting neurogenic muscle atrophy. Seen both in individuals and as 'outbreaks'.
- Inciting cause in individual cases frequently unknown.

Risk factors
- Rare condition.

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- Any age/stage of training.
- Recent/current grazing exposure to causative plants: drought-affected, poor-quality pasture a recognized risk factor.

History
- Acute onset; no association with exercise.
- Onset of signs may be delayed and condition can occur some weeks after removal from affected pasture.
- Signs may be worse in cold weather.

Signs
- Characteristic, intermittent, exaggerated adducted flexion of hock(s); sometimes to the point of striking belly.
- Generally observed when backing up or at walk/trot and not associated with lameness.
- Severity may vary between individuals: from mild (noticeable only when backed/turned), moderate (present when walking/trotting especially when starting/stopping movement) to severe (hindlimb(s) hyperflexed for prolonged periods +/– recumbency).
- Clinical examination of limb usually unremarkable.
- Chronic cases may display muscle atrophy of affected limb(s) (extensor muscles primarily affected).
- Other signs of peripheral neuropathy (forelimb gait abnormality, laryngeal hemiplegia) may also be manifested in animals affected by neurotoxic form.

Diagnosis
- Clinical findings definitive.
- Radiography/ultrasonography of front of upper cannon/hock region may be warranted to determine underlying cause if sporadic form suspected.

Management
- Removal from pasture if toxicity suspected.
- Numerous medical therapies have been used for this condition but few have any solid scientific grounding.
- Best documented therapy is long-term use of muscle relaxants (specifically phenytoin).
- Also some anecdotal support for use of dantrolene sodium (systemic administration); botulinum toxin (Botox A) infiltration into long/lateral digital extensor +/– vastus lateralis muscles.
- Surgery (removal of lateral digital extensor tendon/muscle unit) can be considered for cases non-responsive to conservative/medical management.

Prognosis
- Continuation of training often possible.
- Many cases resolve spontaneously following removal from presumed source of toxin, although may take months/years; not possible to predict course of condition and some remain affected long term. Mild cases can recover more quickly.
- Very small proportion of affected horses never fully recover.

THE UPPER HINDLIMB

Applied anatomy

Tibia
The tibia articulates proximally with the femur at the stifle joint and distally with the trochlea of the talus at the hock. The fibula is a vestigial structure and articulates at its head with the lateral condyle of the tibia. The upper tibia is cloaked in muscle bellies on three sides, with only the medial aspect relatively exposed. The popliteus muscle inserts on the caudal surface of the proximal tibia and acts to flex the stifle and rotate the leg inward. The muscle bellies of the deep digital flexor arise in part from the roughened bone of the mid-caudal tibia, as well as the lateral condyle of the tibia. The deep head of this muscle lies on the caudal surface of the bone.

Stifle
The stifle is the largest joint in the horse and is the articulation between the tibia and the femur (Figure 6.190). The condyles of the femur do not conform in shape to the tibial plateau; two crescent-shaped menisci made of fibrocartilage provide congruency between the two bones and function to cushion the articular cartilage from compression forces of weight-bearing. The abaxial borders of the menisci are attached to the stifle joint capsule; the cranial ligament of each meniscus is anchored to the top of the tibia at the medial and lateral intercondylar eminences, respectively. There are two cruciate ligaments within the stifle: the cranial (CrCL) and caudal (CdCL). Although lying outside the joint space, they provide stability, with the CrCL under tension when the leg is in extension and the CdCL in tension when the leg is in extreme flexion.

The patella is a large, rhomboid-shaped bone in the insertional tendon of the quadriceps femoris muscle. It articulates with the large medial and smaller lateral trochlear ridges of the femur. From its base, three patellar ligaments converge distally to attachments on the tibial crest. A large fat pad separates the patellar ligaments from the femoropatellar joint (FPJ) capsule.

The reciprocal ('stay') apparatus is the means by which the horse can remain standing with little muscular effort.

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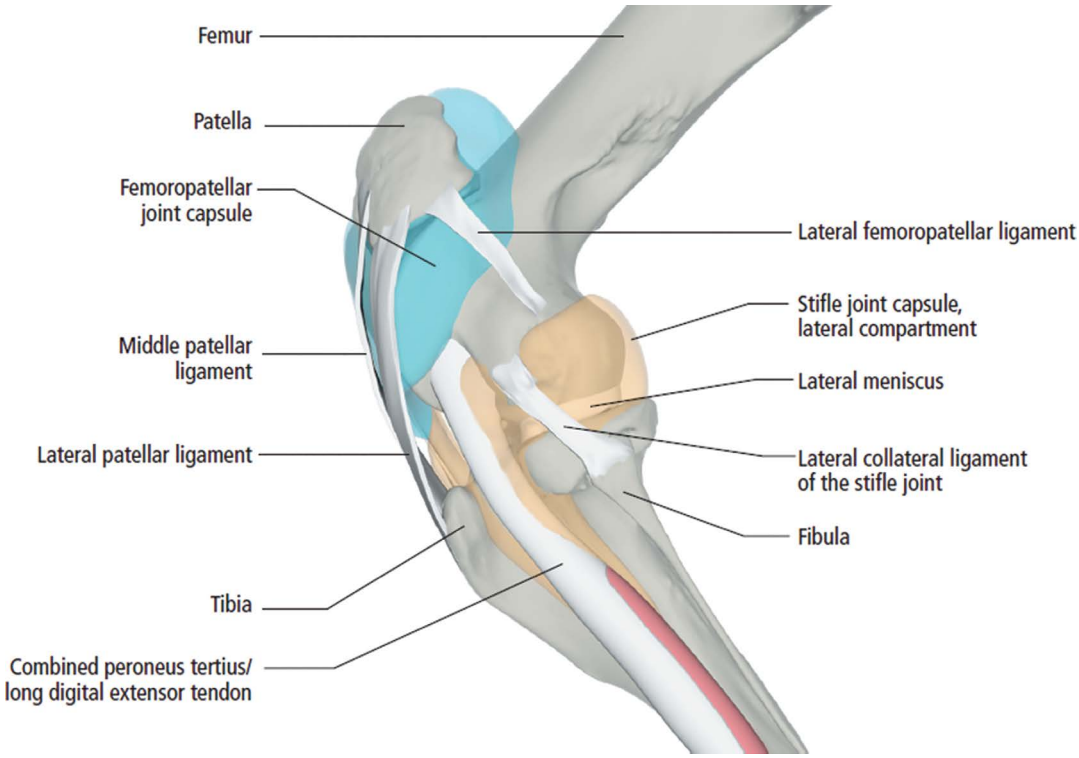


Fig. 6.190 **Major structures of the stifle (lateral view).**

This is achieved by a series of interrelated musculotendinous units, which ensure that the hock and stifle flex and extend in unison at all times, and by a functional peculiarity in which contraction of the quadriceps femoris muscle group hooks the patella over the medial trochlear ridge of the femur by way of the parapatellar fibrocartilage of the medial patellar ligament.

The stifle has three synovial spaces: the FPJ and the medial (MFTJ) and lateral (LFTJ) femorotibial joints. The FPJ at the front of the stifle is the articulation of the patella and the two trochlear ridges. The femorotibial joint spaces are distinct from each other and are found between each meniscus and its corresponding femoral condyle; the LFTJ is tightly constrained, whereas the MFTJ has a sometimes quite large pouch on the medial aspect of the limb.

Synovial communications

- FPJ and MFTJ: communicate directly in the majority (approximately 60–80%) of stifles.
- LFTJ and each of the other joints: communication is rare.
- Diffusion of local anaesthetic between the compartments occurs in a high proportion of stifles but diffusion cannot be relied on for drug delivery.
- Proximity of the caudal stifle joint capsule to nerves supplying the distal limb may result in inadvertent desensitisation of distal structures following diagnostic intra-articular analgesia.

Examination

Examination of the tibia is best undertaken with the limb raised. The medial surface of the bone is readily palpated, as it is largely free of muscle. Particular attention should be paid to the caudomedial aspect of the bone just above the hock; loss of the normal sharp definition of this site, focal thickening or palpable pain are strongly indicative of stress modelling/injury. Response to percussion or torsion of the tibia may also provide clinical information, but should be interpreted with caution and compared with the opposite limb.

Palpation of the stifle is undertaken with the limb weight-bearing. Apparent effusion of any joint compartment should be judged against the opposite limb and attention paid to stance, as limb position can influence perception of intra-articular pressure. Of the three compartments only FPJ effusion may be visible and is usually observed best with the leg in profile. MFTJ effusion is readily palpable on the inside of the groin just above the tibial plateau, whereas LFTJ effusion is rare

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and palpable only as a small pouch of fluid on the lateral aspect of the stifle.

Tibial stress fracture

Important and common racehorse injury. Three main predilection sites for injury exist: proximal (proximal caudolateral cortex), mid (mid-diaphysis, lateral cortex) and distal third (caudodistal cortex), with the latter being most common. Lameness when it develops is typically unilateral; however, pathology is commonly (up to one-third of cases) bilateral. Wide spectrum of severity but diagnosis is usually at incomplete fracture stage; catastrophic injuries very rare.

Cause

- Forces acting on the tibia are predominantly craniocaudal bending (with compression strain at back of tibia) with some superimposed torsion; torsional forces greatest in distal tibia.
- Cumulative microdamage from repetitive strains; mild or bilateral lameness may go unnoticed for considerable period before clinical lameness develops.
- Muscle fatigue may play role in overloading of tibia.

Risk factors

- The most common hindlimb stress fracture in many training centres.
- Unraced 2 and 3 YOs in cantering/early fast work stage of training at greatest risk.
- Typically in horses going through their first 'fast work' training cycle.
- Fillies may be at increased risk.
- Uphill training may increase risk: greater peak forces in hindlimb and increased stride frequency.
- Tibial morphology and relationship to BWT/type/conformation are risk factors in human athletes but have not been investigated in horses.

History

- Usually an acute-onset lameness.
- Commonly first detected on day following fast exercise.
- Less commonly as insidious mild lameness, worsening over days/weeks; also recent poor hindlimb action/sudden loss of performance at canter/faster paces.
- Very rarely, a profound unilateral acute lameness at/immediately after exercise, sometimes with rider reporting audible crack.

Signs

- Typically a unilateral hindlimb lameness of variable (most commonly moderate-marked) severity; usually a 'weight-bearing' lameness with low arc of foot flight and catching of toe.
- Bilateral lameness less common but characterized by close/plaiting hindlimb action at trot +/– poor hindlimb impulsion at canter.
- Usually without palpable abnormality of limb.
- Lameness usually resolves rapidly with short rest period but recurs on return to ridden exercise.
- Occasionally a painful bony thickening of medial aspect of lower tibia may be present.
- +/– pain response to percussion of exposed medial aspect of tibial shaft or forced torsion of tibia with the leg raised; however, manipulative tests have poor sensitivity and responses should be compared with opposite limb.
- Displaced or catastrophic injury: severe lameness/collapse; palpation of medial aspect of tibia may reveal pitting oedema/subcutaneous haemorrhage/instability.

Diagnosis

- Clinical findings and history strongly indicative.
- Imaging usually undertaken in the first instance (before diagnostic analgesia).
- Radiography (LM, CdCr, CdMCrLO projections of distal/midshaft/proximal tibia have highest yield) has only moderate sensitivity but very useful as initial imaging modality. If initial findings are ambiguous and strong clinical suspicion still rests on tibia, repeat examination at 7–14 days is recommended; development of periosteal proliferative change confirms diagnosis.
- Radiological signs may range from subtle to marked: multiple projections, good quality exposures and comparative images of opposite limb may be required.
- Distal tibia: ranges from subtle loss of corticomedullary definition through to caudal cortical thickening/periosteal reaction on LM projection. Area of increased/irregular radiodensity +/– linear lucency may be present on CdCr projection (Figure 6.191a–f).
- Mid tibia: range of findings from poorly defined increased radiodensity through mid-diaphysis or cortical thickening to short cortical fracture line +/or periosteal callus (Figure 6.192a–d). Subtle/focal immature periosteal callus may be the only finding at initial examination (and easily missed without multiple good quality exposures).
- Proximal tibia: reactive bone/callus on proximolateral aspect of tibia (below articulation with fibula) on CdCr (+/– LM) projection. Wide spectrum of radiological

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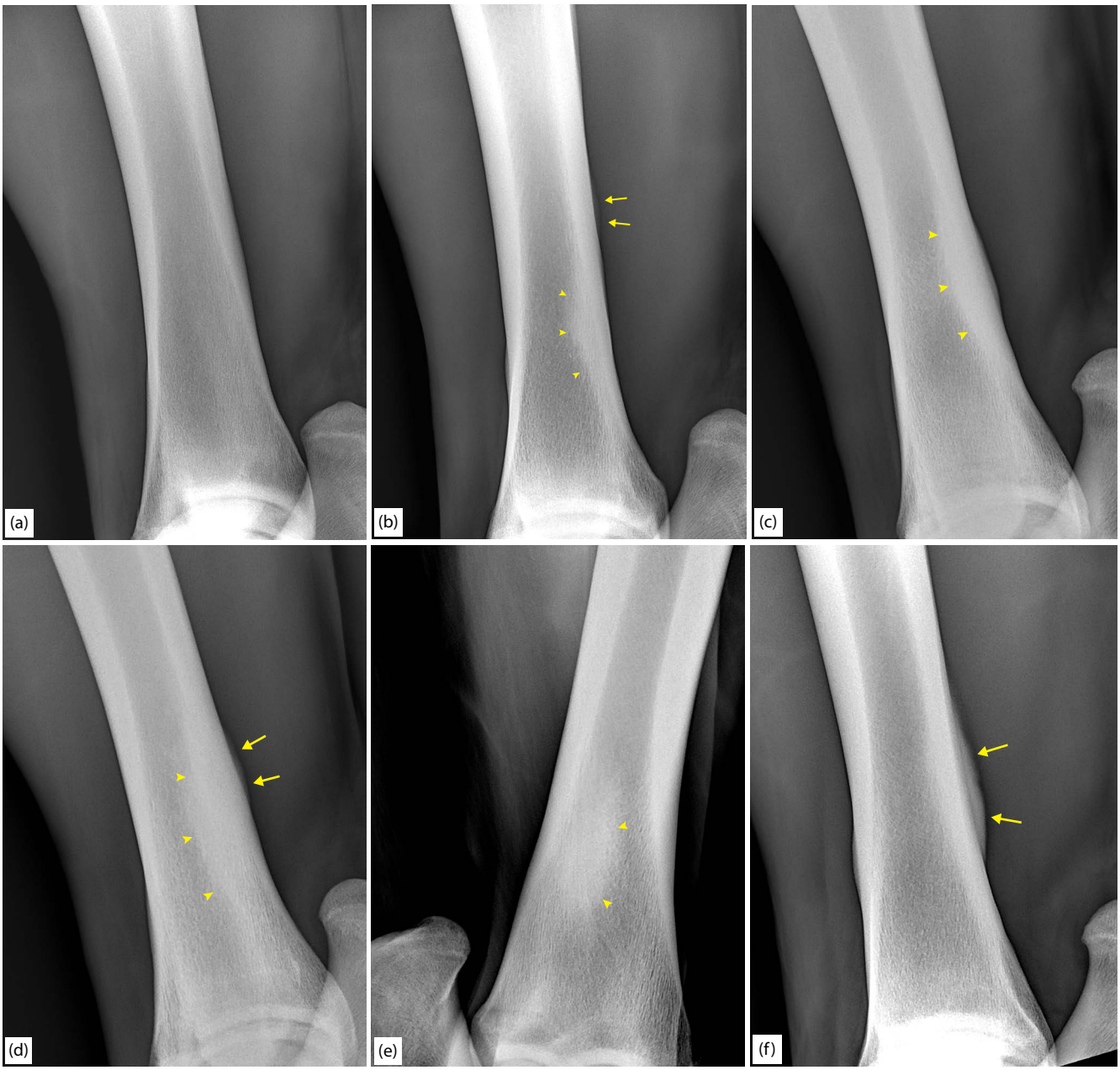


Fig. 6.191 **Distal tibial stress fracture: (a) appearance of 'normal' distal tibia on LM radiograph, with progressive narrowing of caudal cortical profile distally, clarity of trabecular pattern and well defined corticomedullary junction; (b) zone of increased radiodensity adjacent caudodistal cortex (arrowheads) typical of many tibial stress injuries, with additional immature callus higher on the caudal tibia (arrows) indicating extensive stress activity in this case (LM); (c) obvious thickening (arrowheads) of caudodistal cortex (LM); (d) extensive zone of increased endocortical radiodensity (arrowheads) with loss of corticomedullary definition, also with immature periosteal reaction (arrows) (LM); (e) patch of increased radiodensity in distal tibia on CdMCrLO projection; (f) abundant periosteal callus (LM).**

severity may be encountered at time of initial imaging (from subtle to profound) (Figure 6.192e–h). As with other tibial stress injury sites, initial findings may be ambiguous (subtle and easily missed periosteal callus: Figure 6.192i) but rapid change in initial weeks as callus matures (Figure 6.192j,k). Should not be confused with normal variants (muscular ridges/vascular impression) lower on diaphysis.

- Severe mid-shaft injuries (rare) with suspected cortical fracture frequently unremarkable on

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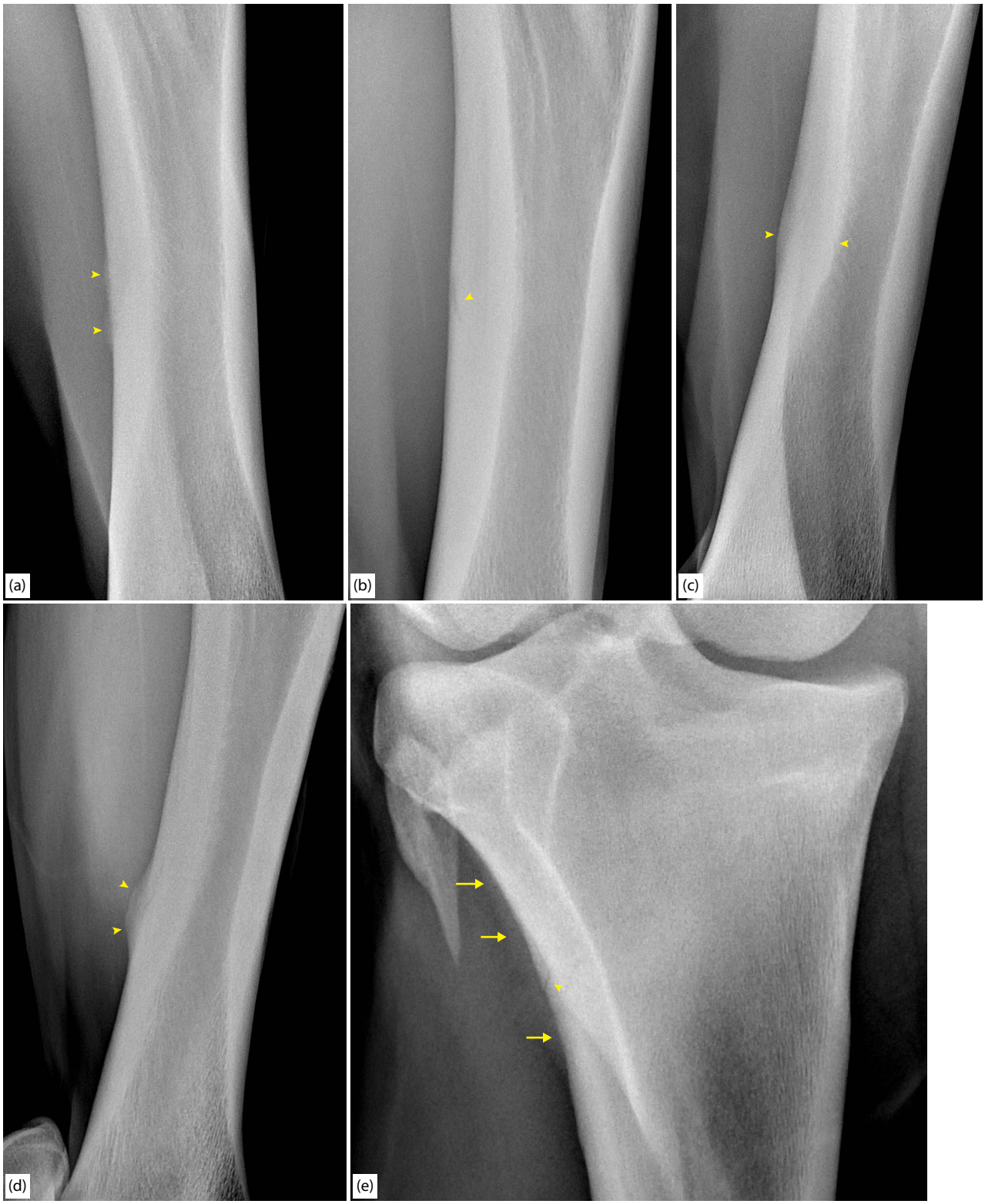


Fig. 6.192 **Tibial stress fracture: Radiological findings definitive for injury in mid (a–d) and proximal (e–i) predilection sites. Characteristic features of mid-shaft injuries include periosteal callus of varying maturity (a), linear lucency (b), cortical thickening (c), or combinations of both (d) on CdCr (a, b) and oblique CdCr (c, d) radiographs. CdCr projections most useful for proximal injuries: (e) short fracture line visible (arrowhead) with subtle but extensive immature callus (arrows) on proximolateral tibia;** *(Continued)*

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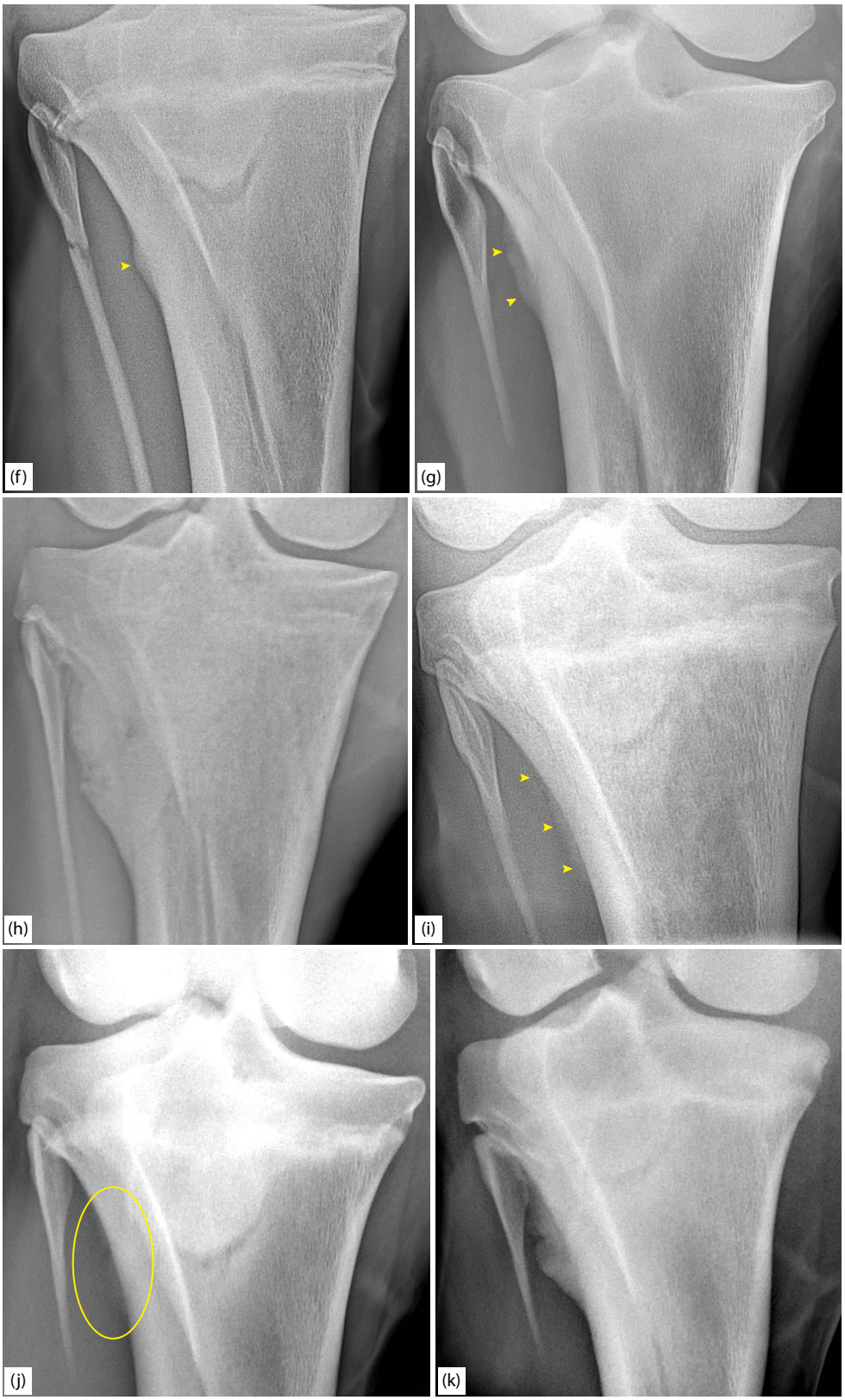


Fig. 6.192 *(Continued)* **latter finding requires good exposures for early recognition but becomes more obvious as callus matures (f–i). Early periosteal change (circled) in (j) difficult to discern at initial screening but 2 weeks later had become profound callus (k).**

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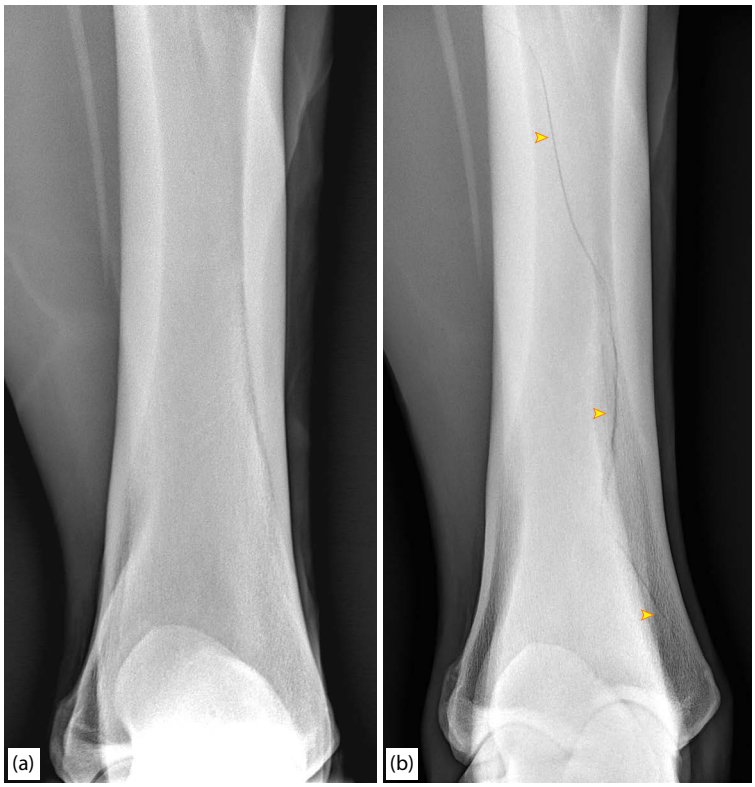


Fig. 6.193 **Tibial stress fracture: Initial (a) and delayed (at day 12 post-injury) CdCr radiographs of case with acute onset severe post-exercise lameness. Extensive initial radiographic survey of tibia unremarkable; however, the horse was cross-tied due to high clinical index of suspicion over tibial injury, confirmed at follow-up (b). Successful return to training is possible following injuries of this kind in horses that survive initial post-injury phase.**

initial radiography or may have only indistinct increase in medullary opacity, with development of marked radiological findings over subsequent weeks (Figure 6.193a,b).
- Bone scan: warranted if radiography inconclusive; good sensitivity and distinctive patterns of IRU (Figure 6.194a–i). Mid-shaft and distal lesions may be indistinct/subtle on lateral views due to attenuation and caudal/medial views should therefore be obtained; patterns of normal radiomarker uptake in the proximal and distal tibial growth plates may also confound diagnosis.
- Ultrasonography: may permit detection of periosteal callus but rarely employed.

Management
- Rehabilitation programme determined by initial clinical and radiological severity (see Chapter 5). Stable rest not always necessary.
- Typically require 6–10 weeks out of cantering exercise.
- Mild initial lameness: stable rest until sound then 3–4 weeks walk 3–4 weeks trot.
- Mid-shaft injuries appear to either heal more slowly/less satisfactorily or have greater propensity to reinjure than other sites, and fast-tracked rehabilitation of these cases is not recommended, even if initial lameness resolves rapidly.
- Radiographic monitoring not necessary for majority of cases.
- Cases of severe injury at high risk for displacement (rare) (such as in Figure 6.193): tying up or slinging (4–6 weeks) may be warranted; such management also carries risk of complications including pleuropneumonia. Radiographic monitoring to guide management.
- Complete fracture (Figure 6.195a–c): euthanasia.

Prognosis
- Majority of tibial injuries (stress 'reactions' to incomplete fracture) have excellent prognosis for return to full athletic soundness.
- Reinjury (same or opposite limb) is rare but when encountered is most frequently seen with mid-shaft injuries.

Stifle: Subchondral bone cyst ('osseous cyst-like lesion'/OCL)

Osteochondral lesion of variable size predominantly found in mid-weightbearing surface of the medial femoral condyle (MFC). Typically develops early in life and in the majority of cases remains clinically inactive throughout training. Very rarely found at other sites in the stifle

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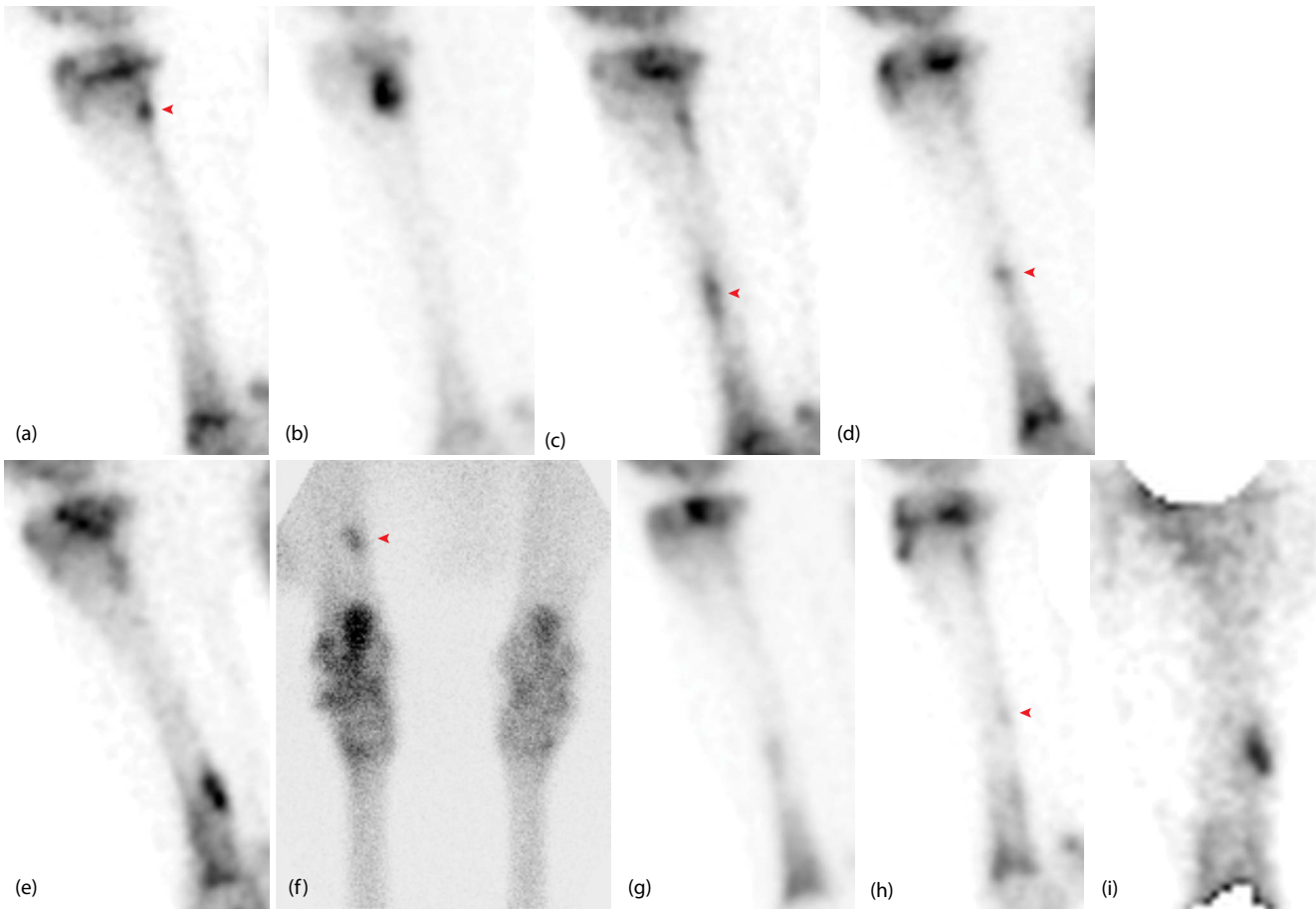


Fig. 6.194 Tibial stress fracture. Lateral scintigrams of range of IRU patterns seen with proximal (a, b), mid (c, d) and distal (e) injuries. Caudal views (f) are important for detection of mid-shaft injuries that may be poorly defined on lateral views due to attenuation (g); case illustrated in (h) and (i) has near-normal appearance on lateral scintigram (arrowhead) but marked focal IRU on caudal view.

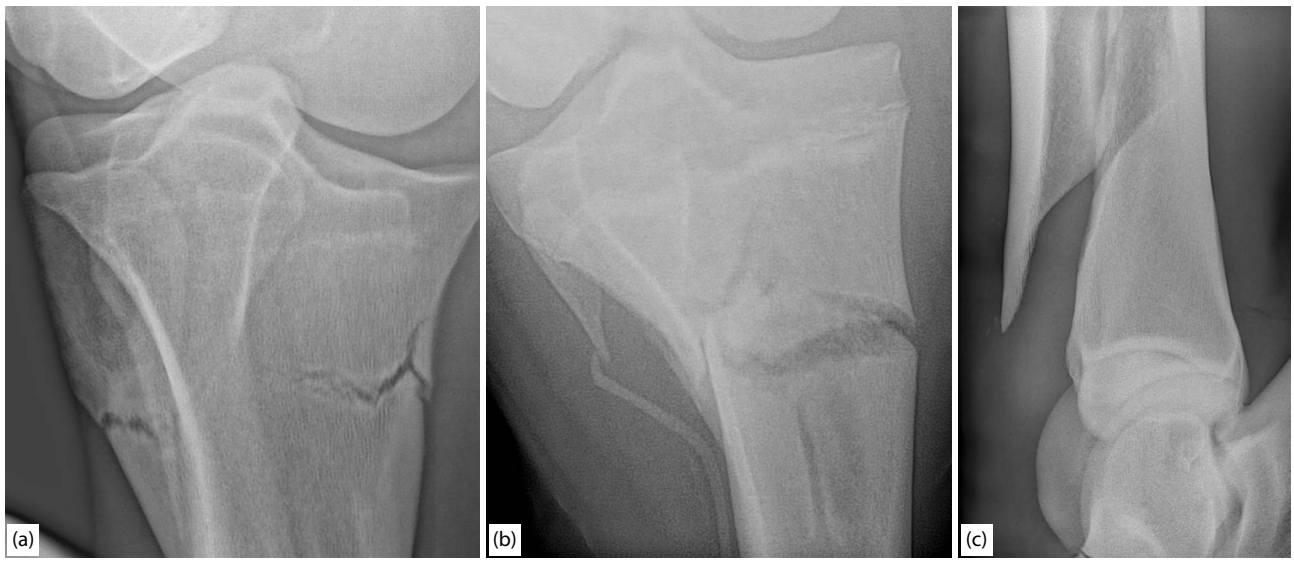


Fig. 6.195 Tibial stress fracture. Examples of complete non-displaced proximal (a) and displaced proximal (b) and mid-shaft (c) fractures; all were catastrophic.

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(proximal medial tibia or lateral femoral condyle). Bilateral lesions occur in approximately 20% cases.

Shallow subchondral lesion ('dimple') of MFC thought to be a related condition (possibly representing milder/resolving defects on the spectrum of 'cyst' pathology) and may arise from prior subchondral injury or delayed/incomplete ossification; of uncertain significance in most horses. If associated with lameness, there is usually an articular cartilage defect.

Cause

- Previously thought to arise from defect of endochondral ossification (Osteochondrosis (Overview), p. 267), however, likely that microtrauma (impact injury/supraphysiologic loading to zone of biomechanically weak developing cartilage on condyle) may have a role in development in many cases.

Risk factors

- Uncommon; prevalence of stifle cysts in sales yearlings approximately 2–6%.
- Greater prevalence of (non-cystic) subchondral lucencies of MFC (reported in 6–16% sales yearlings).
- Found in any age of horse, but most commonly first detected through radiographic screening at yearling stage, or clinically at 2-YO stage when exercise increases.

History

- Most frequently an incidental finding at yearling pre-sales or pre-purchase radiographic examination.
- If associated with lameness (uncommon), usually first arises during early training (trotting/cantering phase).

Signs

- Most cases are clinically silent.
- If lame: typically an acute, unilateral lameness of moderate severity.
- +/– effusion of MFTJ; limb frequently without clinical abnormality.

Diagnosis

- Radiography: CdCr projection (+/– CdLCrMO projections). Variable appearance of lesion between individuals and over time: size (small notch to large cavity), shape (semi-circular to circular), definition (poorly defined to well defined), articular communication (broad necked to narrow necked), location (central, medial or lateral in condyle) and presence/absence of sclerotic periphery (Figure 6.196a–i). Evidence of joint narrowing is an uncommon feature that requires caution in interpretation; typically no concurrent radiological evidence of OA in the young horse.
- Non-cystic subchondral irregularities characterized by flattening of MFC apex or wide/shallow subchondral radiolucency on CdCr and CdLCrMO radiographic projections (Figure 6.197a–e), but may be subtle (loss of cortical definition).
- Underexposed radiographs may partially/completely obscure lesion.
- Presence of even mild radiological abnormality often signifies presence of cartilage damage.
- Diagnostic blocking (if lame): partial or complete response to blocking of the MFTJ.
- Ultrasonography: may be useful to investigate significance of shallow radiological defect (assessment of integrity of articular cartilage).
- Bone scan: usually unremarkable in clinically silent cases; focal IRU of variable intensity in clinically active cases.

Management

- Management dependent on clinical activity/severity and stage of training.
- Interventions may be surgical (current favoured techniques include placement of transcondylar lag screw or bioabsorbable implant; also arthroscopic debridement of cystic cavity, chondrocyte implantation), or medical (intra-lesional injection in effort to control of inflammatory mediators arising from cystic lining).
- Surgery frequently warranted in yearlings to improve radiological appearance of lesion (for future sale) or minimise the risk of later interruption to training career.
- In a horse in training, if subclinical: no treatment required.
- In a horse in training, if lame, options are:
  - Intra-articular medication to facilitate continued training in short to medium term (generally only worthwhile if mild/non-progressive lameness). Response to initial medication determines medium-term management.
  - Surgery, followed by rest (range 3–8 months out of ridden work, dependent on technique and clinical progress). Surgical techniques are evolving and choice may be influenced by local expertise, although current preferred treatment for most cases is transcondylar screw/implant.

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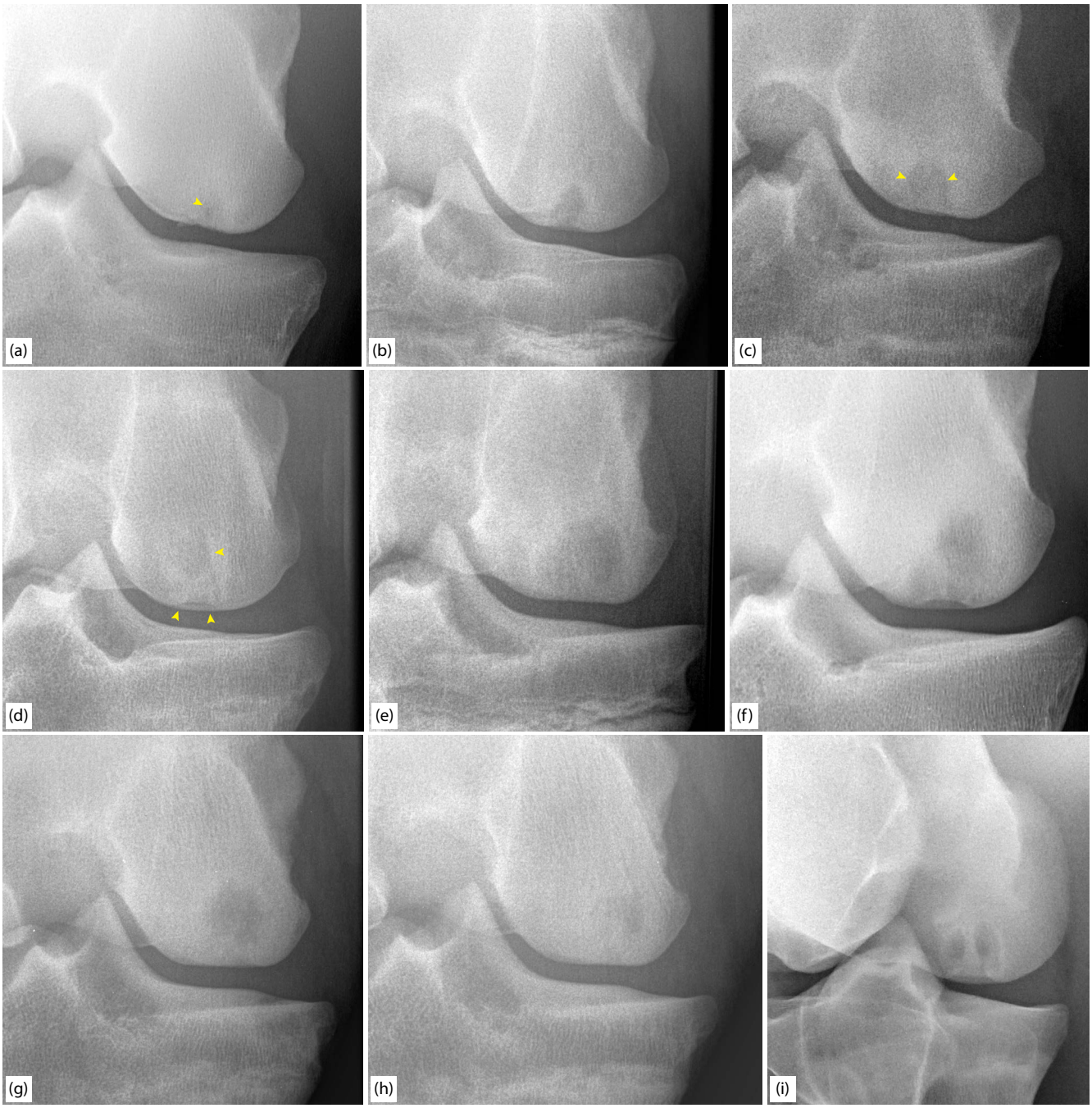


Fig. 6.196 **Stifle subchondral bone cyst. Range of radiological (CdCr) findings in medial femoral condyle: (a) small, indistinct radiolucency (arrowhead) typical of 'resolved' lesion; atypical articular fragment/irregularity also noted at condylar margin; (b–d) cysts of varying size/shape/degrees of peripheral radiodensity; (e, f) large cysts with apparent broad articular involvement (and of presumed greater clinical importance); (g, h) cysts with more regular appearance to subchondral bone and apparent isolation from joint (consistent with 'maturing' lesions); (i) atypical multilocular cyst (CdLCrMO).**

- Ultrasound-guided intra-lesional injection (corticosteroid, or autologous biological product), followed by rest (3–4 months out of ridden work): limited efficacy.
- Arthroscopic-guided intra-lesional injection (corticosteroid, or autologous biological product), followed by rest (may permit inspection of joint): limited efficacy.

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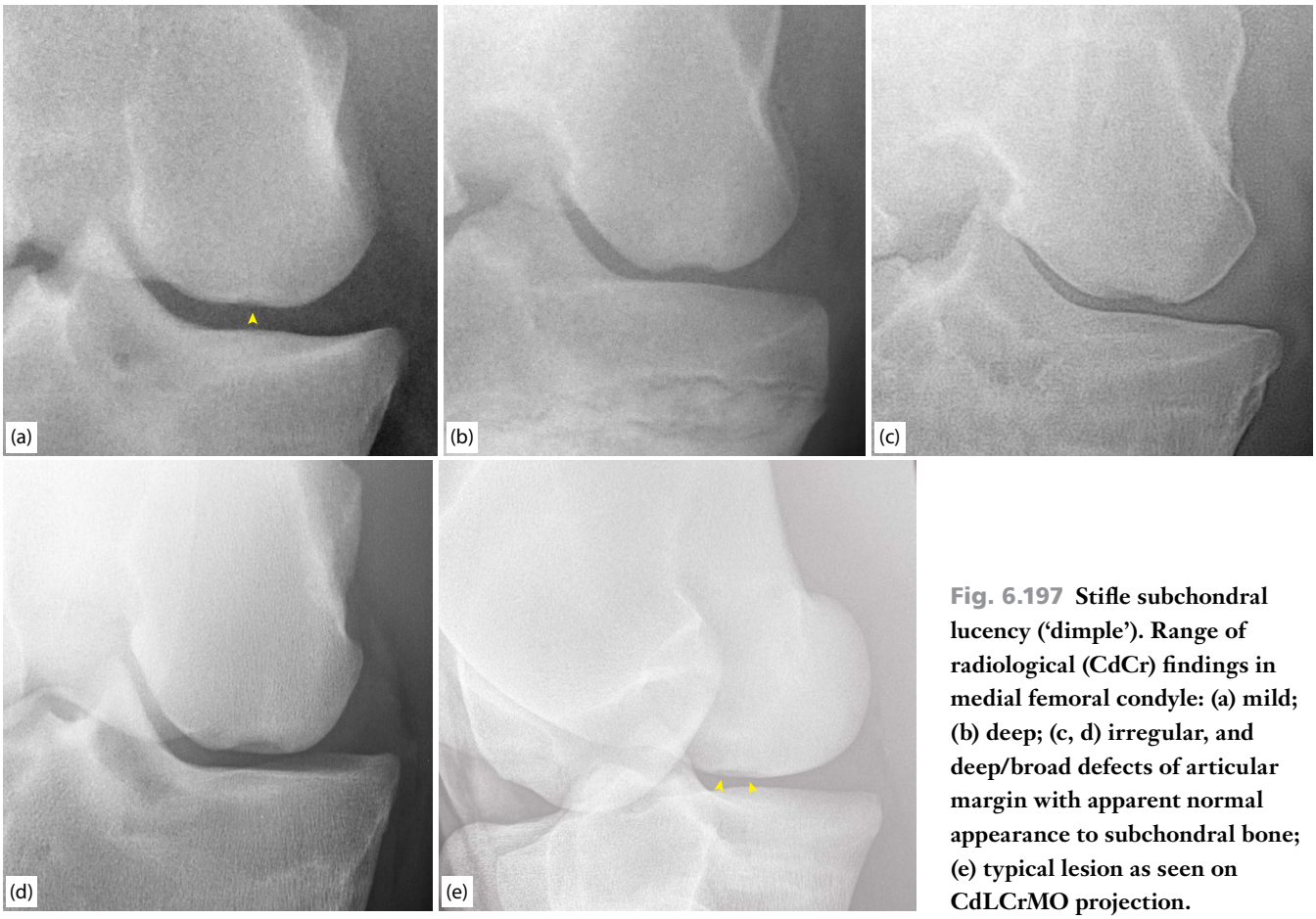


Fig. 6.197 **Stifle subchondral lucency ('dimple'). Range of radiological (CdCr) findings in medial femoral condyle: (a) mild; (b) deep; (c, d) irregular, and deep/broad defects of articular margin with apparent normal appearance to subchondral bone; (e) typical lesion as seen on CdLCrMO projection.**

- Surgical intervention is generally not recommended for shallow (non-cystic) subchondral lucencies ('dimples') as may result in worsening of lesion; surgical reattachment of cartilage has resulted in return to soundness in some individuals with extensive lesions.

Prognosis

- Many cases never develop lameness and train without incident.
- If significant lameness develops, interruption to 2 YO season can be expected regardless of management. Clinically affected horses have fewer starts at 2 and 3 YO than unaffected horses.
- Accurate determination of risk of lameness developing is not possible before horse has entered training (i.e., at yearling stage) due to unpredictability in behaviour of lesions, although action/stride length, presence/absence of joint effusion and some radiological features may permit refinement (although still highly subjective) of risk assessment in juveniles.
- When detected as an incidental (clinically silent) finding in a horse that has trained/raced ≥1 season, risk of developing future related lameness is considered very low. Highly unusual for a clinically silent long-standing stifle cyst in a mature racehorse to become clinically active later in career.
- Clinical cases: good prognosis for return to full use (approximately 65–80%) with both surgery and ultrasound-guided intra-lesional injections + rest. Surgery is favoured over rest/conservative management alone. Of surgical options, transcondylar screw/implant currently appears to have highest success rate.
- Prognosis appears not to be related to depth/configuration of lesion, but may be worse for lesions with large/wide involvement of joint surface, narrowing of joint space (and presumed concurrent meniscal damage) or those with concurrent subchondral defect in the proximal medial tibia (rare).
- Poorer success rate (for return to soundness) for older horses (≥3 YO), bilateral lesions or if radiological signs of OA at time of diagnosis.

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- Most non-cystic subchondral irregularities do not deteriorate clinically/radiologically with maturity; limited longitudinal evidence suggests approximately 70% of shallow subchondral lucencies in the MFC of juvenile horses either remain unchanged or diminish in size, with <5% developing into OCL.

Stifle: Osteochondritis dissecans

Within the stifle, the most common site for OCD lesions is the lateral trochlear ridge of the femur. Rarely, the medial trochlear ridge or patella may be involved. Bilateral lesions occur in approximately 30% of cases.

Cause

- See Osteochondrosis (Overview) (p. 267).
- Dynamic, multifactorial condition: lack of exercise/fast growth rate/nutritional/genetic factors in windows of susceptibility can cause abnormal cartilage development at certain articular predilection sites.

Risk factors

- Uncommon condition; prevalence in sales yearlings approximately 3–6%.

History

- May be detected as subclinical lesion at routine yearling radiography.
- Clinical entity of FPJ effusion +/– lameness at yearling/early 2-YO stage when exercise commences.

Signs

- Variable clinical signs.
- Often associated with considerable FPJ effusion.
- +/– lameness (mild-moderate severity).

Diagnosis

- Radiography: CdPrL-CrDiMO projection best for lateral trochlear ridge of femur (most common site of pathology).
- Radiological signs of OCD: flattening/irregular contour or changes in subchondral density of trochlear ridge; radiopaque fragments may be present in adjacent affected sites (Figure 6.198a–c).
- Ultrasound: greater sensitivity for lesion detection than radiography but currently not a routine component of yearling pre-sales or pre-purchase screening. Useful when OCD lesions are suspected but not evident on radiography.

Management

- Management dependent on clinical/radiological severity and stage of training at detection.
- If detected as incidental finding when already in training and no lameness, no treatment necessary.
- If lame, surgery (arthroscopic removal or reattachment of defective cartilage) is only effective treatment.

Prognosis

- Prognosis for racing soundness generally correlated with size of lesions.
- Unaffected by location of lesion or whether unilateral/bilateral.
- Horses with smaller lesions (total length <6 cm) undergoing surgery prior to their 2 YO season likely to have similar trainability to normal horses, although some evidence that career performance outcomes of horses operated on as yearlings may be lower than horses without stifle OCD.
- Horses with more extensive lesions (total length >6 cm) are less likely to race regardless of management.
- When detected as an incidental (clinically silent) finding in a horse that has trained/raced ≥1 season, considered a negligible risk for future-associated lameness (or interruption to training).

Stifle: Locking patella

Condition in which the patella fails to release correctly from its 'sleeping' position on the lower femur, thereby preventing the stifle from flexing. May be transient or require intervention to release.

Cause

- The 'stay apparatus' of the horse enables it to stand at rest with little muscular effort through hooking of the patella (parapatellar cartilage and medial patellar ligament) over the prominent medial trochlear ridge of the femur.
- Quadriceps femoris muscle helps to raise patella on and off the ridge.
- Failure to release the patella may result in stifle being 'locked' in extension.
- Certain movements (turning sharply) may precipitate.

Risk factors

- Common condition.
- All ages/stages of training.
- Main risk factors are loss of muscle tone and straight hindlimb conformation.

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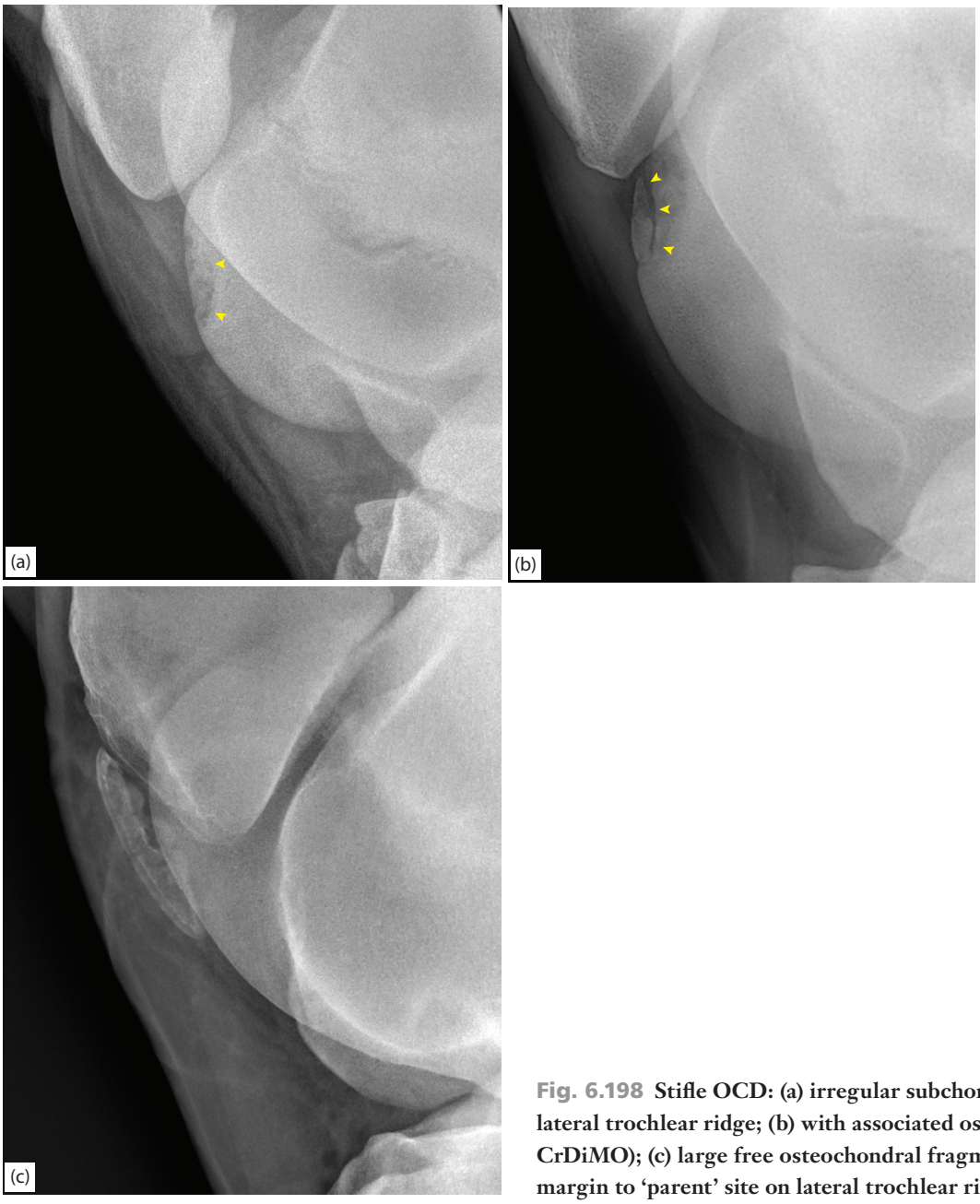


Fig. 6.198 **Stifle OCD: (a) irregular subchondral radiodensity of lateral trochlear ridge; (b) with associated osteochondral flap (CdPrL-CrDiMO); (c) large free osteochondral fragment, with irregular margin to 'parent' site on lateral trochlear ridge (PrL-DiMO).**

History

- May be encountered as sporadic or recurrent event.
- Commonly during phase of reduced exercise/stable rest.

Signs

- Affected hindleg held straight and rigid, often with toe drawn slightly underneath the body (Figure 6.199).
- Reluctance to walk forward and, when forced to do so, leg will drag/knuckle over; when turned will pivot on locked leg.

Diagnosis

- Clinical findings definitive.
- Easily differentiated from injury: inability to flex limb.

Management

- Most cases readily unlocked through firm pressure from heel of hand placed above and lateral to the stifle (in the quadriceps muscle mass) directed down/forward in the direction of the opposite front leg. May be assisted by drawing lower leg forward with rope.

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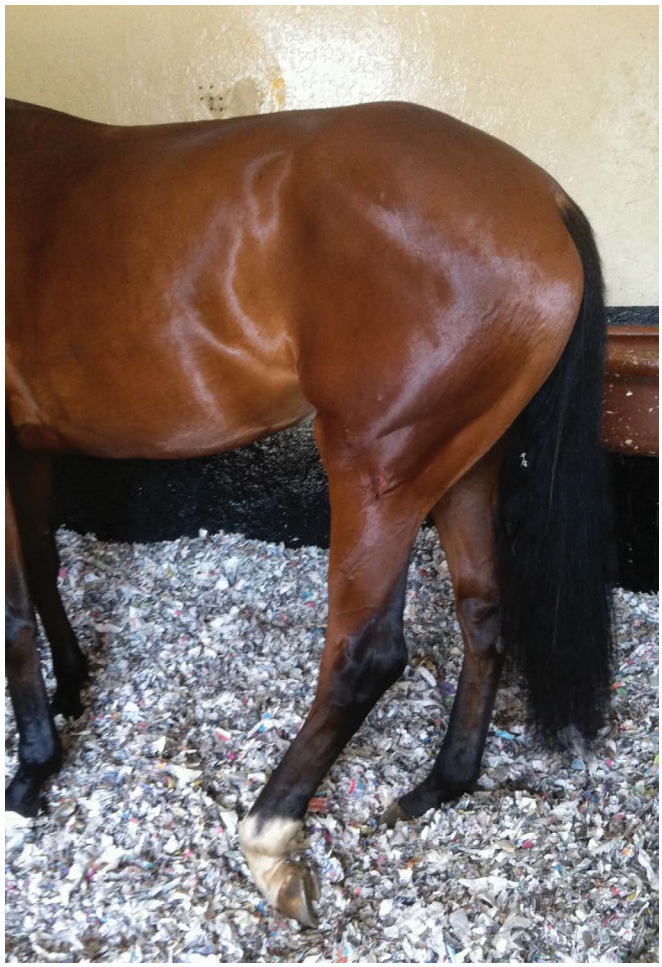


Fig. 6.199 **Typical presentation of locked patella.**

- Management of recurrent cases dependent on individual circumstances.
- Many resolve with strengthening/conditioning of the quadriceps (return to exercise/hill work/trotting on heavy/deep surface).
- Can often be managed in short term with twice-daily administration of acepromazine +/– shoeing hind feet with raised lateral heels/wedges; particularly useful for horses that still have to remain resting for unrelated reasons.
- Surgery rarely needed and generally considered a treatment of last resort for severe non-responsive cases; but is highly effective in resolving the condition in these cases.
- Surgical options: splitting or sectioning (desmotomy) of medial patellar ligament; long-term complications of desmotomy include patellar instability/fragmentation.

Prognosis

- Good. Rarely interferes with training.

Stifle: Trauma

Soft-tissue injuries of the stifle are typically acute and follow a fall or direct trauma. Damage is usually superficial with short-lived bruising/infection. Very rarely, supporting structures (menisci, cruciate, patellar or collateral ligaments) of stifle may be involved.

Cause

- The patellar and collateral ligaments are poorly protected by overlying soft tissue and vulnerable to direct trauma, particularly kicks.
- The menisci move over the tibia with flexion/extension of stifle and may become torn during hyperextension of joint.
- Cruciate ligament tears arise from rotation or hyperextension of the stifle; injury to the CrCL is more common than to the CdCL.

Signs

- Following kick or fall: soft-tissue swelling +/– skin abrasion or laceration to front or side of stifle (Figure 6.200).



Fig. 6.200 **Typical full-thickness skin abrasion to front of stifle after a fall/road injury.**

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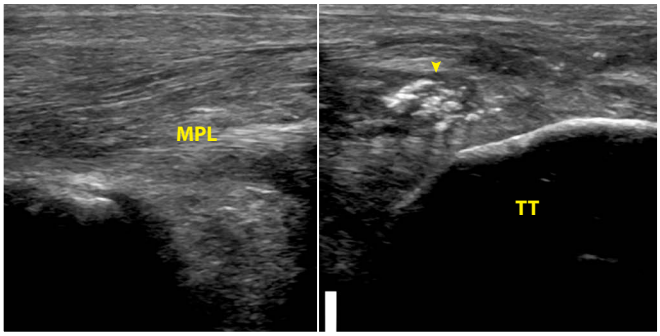


Fig. 6.201 Longitudinal ultrasonogram (proximal to left) of traumatic injury to medial patellar ligament (MPL), with osseous fragmentation (arrowhead) noted close to tibial tuberosity (TT) insertion.

- Acute, unilateral hindlimb lameness; moderate to marked severity in initial stages with reduced cranial phase to gait.
- Effusion of FPJ +/– MFTJ is common with injuries to supporting structures.
- Positive response to flexion test in most cases.

Diagnosis
- Acute stifle lameness is not specific for injury type.
- Diagnostic investigation generally only required if lameness persists beyond acute phase.
- Diagnostic blocking: response to blocking of stifle compartments varies with injury type.
- Ultrasound: good sensitivity for identifying meniscal/patellar/collateral ligament injury (Figure 6.201). Difficult to image cruciate ligaments satisfactorily.
- Radiography: indicators of injury are infrequent and not always specific. Meniscal injury: approximately one-third of horses have reactive new bone on the medial intercondylar eminence of the tibia. Cruciate injury: concurrent fragmentation of medial intercondylar eminence of tibia.
- CT: imaging modality of choice for diagnostically challenging cases; however, necessitates general anaesthesia.
- Arthroscopy may be used to diagnose meniscal injuries. Cruciate ligaments are largely extra-synovial and minor injuries difficult to detect.

Management
- Acute phase (no synovial infection): antibiotic/anti-inflammatory therapy and walking exercise until lameness subsides.
- Meniscal/cruciate/patellar/collateral ligament injury: conservative management, guided by clinical progress. Stable rest (4–8 weeks)/walking (4–8 weeks).
- If poor response to initial rest, arthroscopy for further diagnosis/treatment.

Prognosis
- Simple wounds with no involvement of deeper structures: rarely interfere with training beyond a few days.
- Damage to supporting structures (rare): prognosis for return to racing dependent on severity of injury.
- Mild/moderate injury to menisci/cruciate ligaments: fair prognosis for racing soundness with extended period of rest.
- Marked tears/severe lameness/presence of radiological change associated with poor prognosis regardless of management.
- Patellar/collateral ligament injuries: good prognosis.

Stifle: Tibial tuberosity fracture
Injury to front of stifle in which tibial tuberosity (top of tibia; point of attachment of the patellar ligaments) separates from the parent bone. Fracture fragment typically displaced cranially/proximally due to pull of quadriceps muscle. Usually non-articular.

Cause
- Arises from direct trauma (kick/jumping) or avulsion of quadriceps/biceps femoris muscle attachment following fall/slip.

Risk factors
- Rare condition.
- Any age/stage of training.

History
- Acute-onset lameness.

Signs
- Marked unilateral lameness.
- Local inflammation/pain on palpation of front of stifle.

Diagnosis
- Radiography: LM projection most useful (Figure 6.202a); differentiate from proximal tibial physis (open in 2 and 3 YOs) (Figure 6.202b).
- Ultrasonography: permits assessment of concurrent damage to soft-tissue structures (patellar ligaments/menisci).

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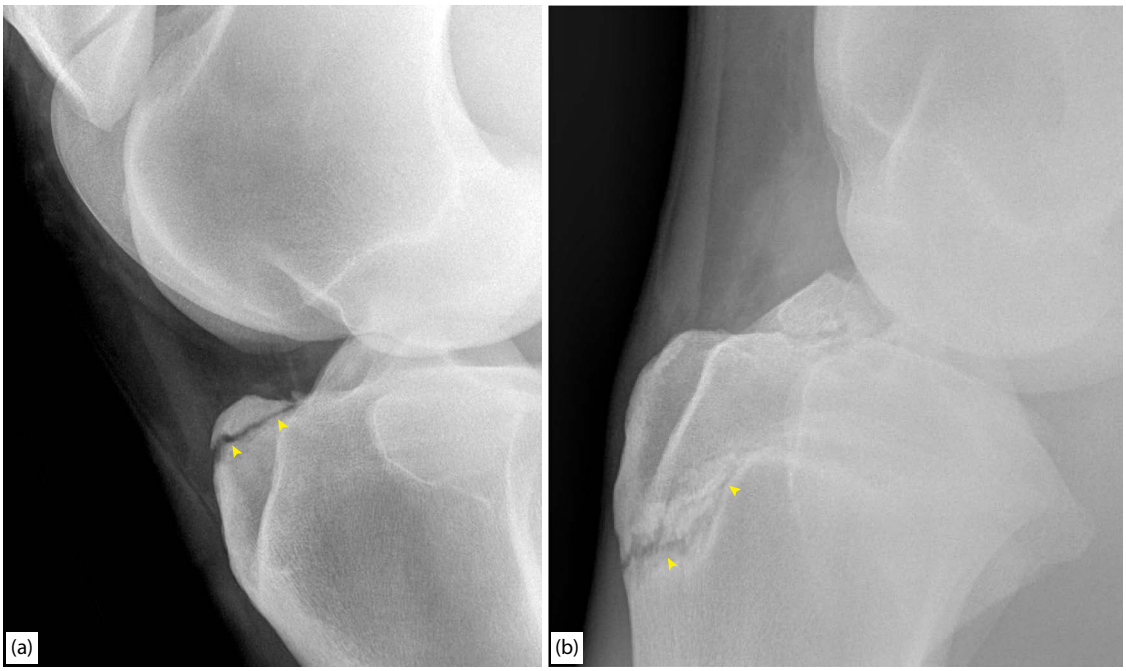


Fig. 6.202 **(a) Tibial tuberosity fracture (arrowheads); (b) normal appearance to proximal tibial physis (arrowheads).**

Management

- Non-articular fractures: conservative management: stable rest (8–12 weeks)/walking (4–8 weeks). Surgical reduction offers little advantage in most cases.
- NSAID medication during acute phase.

Prognosis

- Non-articular fractures: good prognosis for return to racing with conservative management (approximately 80%) regardless of fragment size/displacement; average time to return to racetrack 6–7 months.
- Concurrent soft-tissue damage to supporting structures of stifle can have a negative impact on prognosis.

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THE PELVIS

Applied anatomy

The pelvis comprises several fused bones (ilium, pubis and ischium) and articulates with the axial skeleton and both hindlimbs (Figure 7.1). The ilium is the largest bone on each side of the pelvis: the broad, dorsally concave wing of the ilium is largely covered by the gluteal muscles and only lies close to the skin laterally at the tuber coxa ('point of hip') and axially at the tuber sacrale ('jumper's bump'). The wing is continued caudally by the iliac shaft, which runs back to form part of the hip socket (acetabulum), where it terminates. The ischium forms the back of the pelvic girdle and is palpable on both sides of the tail base as the tuber ischium, to which the 'hamstring' caudal thigh muscles attach (biceps femoris, semitendinosus and semimembranosus). The pubis is the smallest of the pelvic bones and forms part of the acetabulum and ventral pelvis, fusing with its counterpart at the midline; it also forms the back border of the large obturator foramen on each side through which nerves and blood vessels pass. Important muscles include the large gluteal muscles that connect the ilium to the femur and act to abduct the limb and flex/extend the hip joint, and the biceps femoris muscle, which is complex in form and acts to extend the limb in propulsion. The pelvis is not a rigid structure and mild deformation occurs in response to limb loading and sacroiliac joint (SIJ) movement.

The SIJs are the articulations between the iliac wings of the pelvis and the wings of the sacrum on each side.

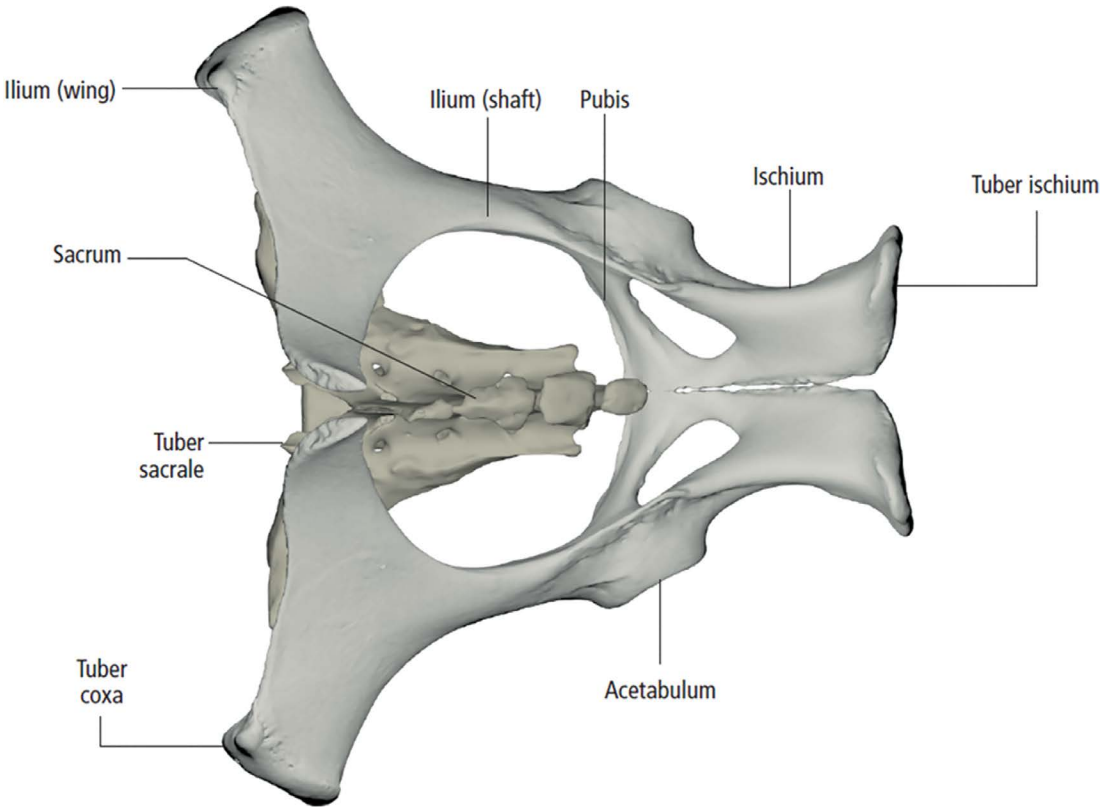


Fig. 7.1 **Major structures of the pelvis (dorsal view).**

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They have an important role in transmitting the forces generated by the hindlimbs up through the rest of the axial skeleton. Each SIJ is a broad, flat attachment that houses very little joint fluid; movements are small and are predominantly axial rolling and shear actions (rather than compression) in response to limb loading. The joints are supported by several pairs of strong ligaments on both their dorsal and ventral surfaces and are in close proximity to important neurovascular structures (sciatic and cranial gluteal nerves, cranial gluteal blood vessels).

The hip joint is a ball-and-socket articulation between the head of the femur and the acetabulum of the pelvis (formed from the convergence of the ilium, ischium and pubis). Although its configuration permits some rotation, the main movements are extension/flexion. The cavity of the acetabulum is extended by a fibrocartilaginous rim that, along with the strong accessory femoral and short round ligaments, keeps the head of femur in place. The joint is large but not accessible to palpation because of the overlying musculature.

There are some differences in pelvic morphology between the sexes: the pelvic outlet and obturator foramina of the female horse are larger, and the iliac shaft longer, than those of the male. Although these differences have been incompletely investigated, they may contribute to apparent predilection to certain pelvic injuries (such as those involving the ventral pelvis/acetabulum).

Examination

The pelvis should be assessed for both skeletal and muscular symmetry, with the horse positioned squarely on a level surface. The large muscle bellies of the quarters cloak the pelvis in such a way that only the bony promontories (tuber sacrale, tuber coxae and tuber ischia) close to the skin are directly palpable. Pelvic skeletal asymmetry is usually readily distinguished from muscular atrophy. Asymmetry in the region of the tuber sacrale is often an incidental finding and may arise from either relative enlargement of the dorsal sacroiliac ligament or gluteal muscle atrophy rather than skeletal deformation per se; however, asymmetry of the tuber coxae almost invariably signals current or previous injury. The tuber ischia either side of the tail base, and their associated 'hamstring' caudal thigh muscles, should also be examined for visible or palpable abnormality.

Assessment of pelvic/lumbar back pain is inherently subjective as it relies on manual palpation. Direct or translated movement of the pelvis through depression of the tuber sacrale/coxae may elicit a pain response. Lumbar epaxial and/or gluteal muscle spasm may be an indication of underlying skeletal pain or may be present in isolation. Manipulation of the hindlimb (flexion/adduction/abduction) rarely causes pain in cases of pelvic fracture, but if undertaken with care in cases of suspected pelvic instability it may provide useful audible or palpable evidence of crepitus. Horses with SIJ pain may resent the contralateral hindlimb being flexed/abducted. Rectal examination of the internal borders of the pelvis may also be appropriate to assist diagnosis of pelvic fractures, and firm digital palpation of internal soft-tissue structures (lumbosacral disc, iliopsoas muscles) can be informative.

Iliac stress fracture

The iliac stress fracture is the most common type of stress injury affecting the pelvis. The injury may develop on one or both sides of the pelvis, at single or multiple sites and with spectrum of pathology (and severity at time of detection) ranging from subtle/subclinical to catastrophic. The most common predilection site for cortical disruption is the medial part of the iliac wing, overlying the SIJ; however, stress-induced increased bone turnover frequently affects an area considerably beyond where any overt injury may be found. Fracture configuration can be complex and complete characterisation of location/direction of overt cracks generally only possible postmortem (Figure 7.2a,b); in practice injuries are categorized broadly as affecting predominantly the wing, shaft (or both) and as being non-displaced or displaced. In these simplified terms the most commonly encountered injury propagates forward from the caudal margin of the wing as a single or forking crack (Figure 7.3a,b). Iliac stress fractures less commonly extend to or originate in the shaft of ilium; these are considered to be more serious as close proximity to the iliac artery can result in catastrophic internal bleeding if displacement occurs.

Causes

- Stress/fatigue fracture.
- Main stresses are tensile forces on caudal iliac border and bending at SIJ.

Risk factors

- Uncommon condition.
- Develops during cantering/fast exercise phase of training.
- All ages but more common in horses ≥3 YO.
- Other risk factors unknown.

History

- Acute-onset hindlimb lameness that often develops during or immediately after exercise (either on track or upon return to yard).

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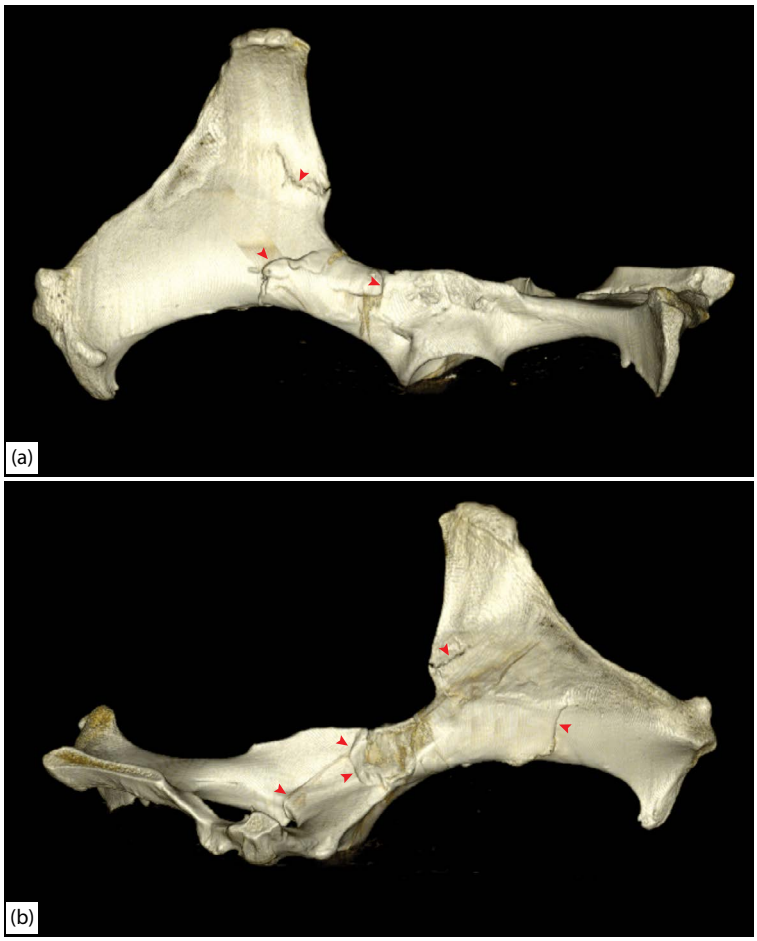


Fig. 7.2 Iliac stress fracture: Dorsolateral (a) and medial (b) postmortem CT images of left hemipelvis (right hemipelvis cropped) of a catastrophic pelvic injury, showing complexity of pathology. Fracture lines (arrowheads) are present at caudal border of iliac wing (incomplete), and spiralling through iliac shaft and into ventral wing (complete).

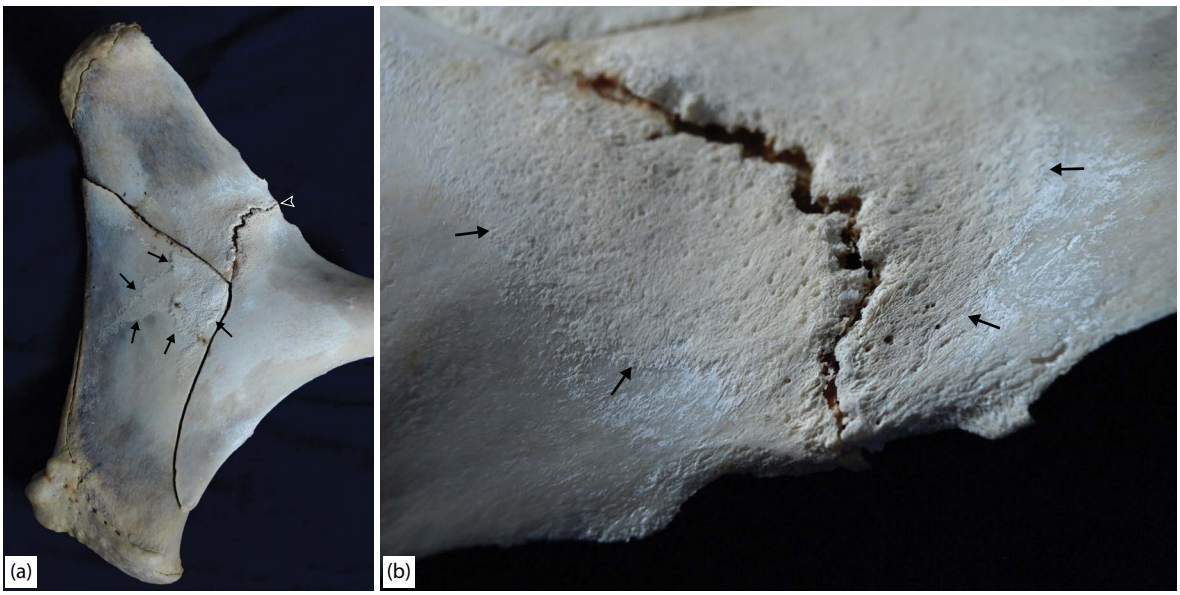


Fig. 7.3 Iliac stress fracture: Postmortem photographs of iliac wing stress fracture showing (a) site of origin of injury at caudal margin of left wing (arrowhead) from which forking, displaced crack has propagated; large area of periosteal callus on dorsal aspect of wing (arrows) indicating pre-existing stress pathology prior to catastrophic injury. (b) Close-up of caudal margin of wing showing broad lytic appearance to fracture line and roughened/porous periosteal callus surrounding it (arrows). Discontinuity at fracture line is the most readily identified ultrasonographic feature.

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- Some cases (particularly if pathology is mild or bilateral) present with poor performance/poor hindlimb action/reluctance to train over several days or weeks, rather than overt lameness; may be mistaken for muscle soreness/exertional rhabdomyolysis (p. 265).
- Occasionally catastrophic (non-weightbearing or recumbent) injury encountered on track.
- An early sign, sometimes reported by yard staff before overt lameness at exercise develops, is unilateral lameness/shortness of stride only noted when horse is moved/pushed across stable.

Signs

- Vary considerably; determined by fracture configuration and pathological stage at which injury tips over from subclinical to clinical.
- Lameness ranges from none/subtle to severe.
- Bilateral injury: plaiting, tight, short-striding hindlimb gait (at walk or trot) is typical.
- Persistence of lameness at trot may help to distinguish from exertional rhabdomyolysis (latter often trot freely/without lameness despite marked hindlimb stiffness at walk).
- If mild lameness, there may be few or no localizing signs.
- If marked lameness, reluctance to move or weight-bear on affected leg. Frequently accompanied by muscular spasm ('guarding') over gluteals or flank when approached or palpated +/– pain on depression of tuber sacrale on affected side.
- Displaced iliac wing fracture: asymmetry of pelvis (tuber sacrale +/– tuber coxae) may develop within 1–2 weeks of injury; lower on affected side. May be accompanied by gluteal muscle atrophy of variable severity.
- Displaced iliac wing +/– shaft fracture: crepitus +/– pelvic instability may be noted with external or internal examination. Marked asymmetry of pelvis may develop within 1–2 weeks of injury. Catastrophic internal bleeding may occur at any time during initial weeks following injury: collapse/shock/death, as can recumbency with difficulty/inability to rise. Acute worsening of clinical signs (+/– development of dependent haemorrhage in upper thigh/groin region) at any point during rehabilitation period is a poor prognostic indicator and generally signals fracture displacement incompatible with recovery.

Diagnosis

- Clinical findings strongly indicative (but not specific for location of injury).
- Blood analysis (muscle enzymes: AST/CK) may be useful to differentiate from exertional rhabdomyolysis ('setfast', p. 265), although elevation of muscle enzymes does not preclude concurrent presence of stress fracture and clinical judgement should take precedence over laboratory findings.
- Ultrasound (transcutaneous): used as initial screening aid (Figure 7.4a–h). Discontinuity/callus in upper face of iliac wing easily detected; however, absence of abnormality does not necessarily rule out presence of injury. Anechoic fluid accumulation adjacent to bone at site(s) of concern may provide contributory evidence of possible injury. Non-displaced iliac shaft injuries (Figure 7.4i,j) more difficult to detect, as injury site frequently on axial side of shaft and out of ultrasonographic 'reach'. Both sides of pelvis should be imaged.
- Iliac shaft injuries: internal haematoma +/– pelvic instability/crepitus may be palpable on rectal examination.
- Bone scan: definitive diagnostic modality; however, patterns and degree of increased radiopharmaceutical uptake (IRU) are highly variable (Figure 7.5a–l) and pathology may sometimes be missed, due either to attenuation of gamma radiation by the large mass of gluteal muscle or presence of radiomarker activity in bladder/urinary tract or musculature.
- Even after comprehensive imaging it is usually the case that full extent/configuration of fracture pathology remains undetermined.

Management

- See Table 7.1.
- Rehabilitation programme determined by initial clinical and imaging severity, and risk of further displacement (see Chapter 5). Inadequacies of diagnostic imaging and high-risk site mean advice should generally err on side of caution.
- Severely lame horses (at training/racetrack): avoid lengthy travel; hospitalization also often best avoided as may result in 'stranding' for initial high-risk phase (weeks). Risk assessment in field and transportation to local stable is preferable.
- Subtle/subclinical injuries detected through screening bone scan may require little time (few weeks) out of cantering; can commence walking exercise

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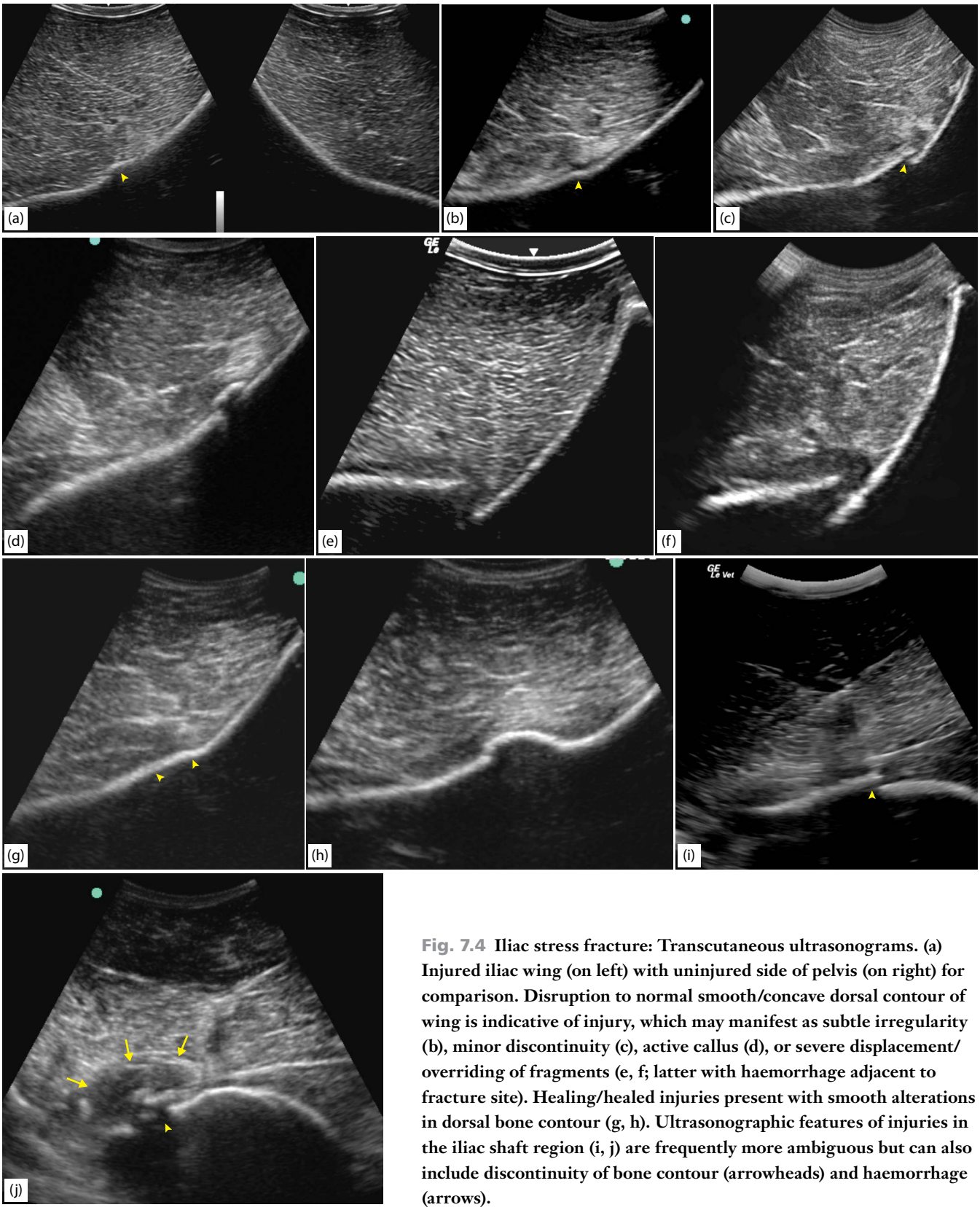


Fig. 7.4 **Iliac stress fracture: Transcutaneous ultrasonograms. (a) Injured iliac wing (on left) with uninjured side of pelvis (on right) for comparison. Disruption to normal smooth/concave dorsal contour of wing is indicative of injury, which may manifest as subtle irregularity (b), minor discontinuity (c), active callus (d), or severe displacement/overriding of fragments (e, f; latter with haemorrhage adjacent to fracture site). Healing/healed injuries present with smooth alterations in dorsal bone contour (g, h). Ultrasonographic features of injuries in the iliac shaft region (i, j) are frequently more ambiguous but can also include discontinuity of bone contour (arrowheads) and haemorrhage (arrows).**

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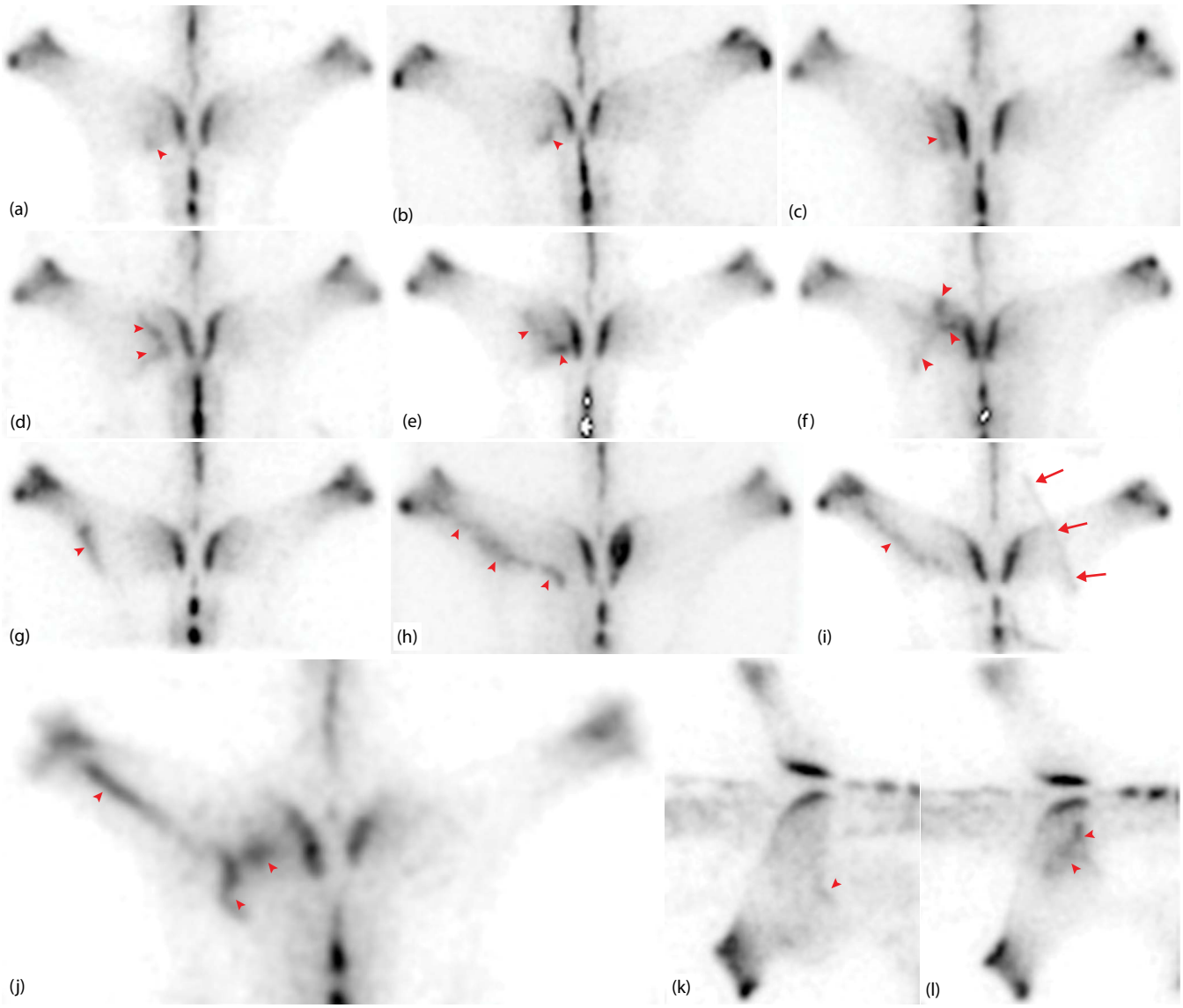


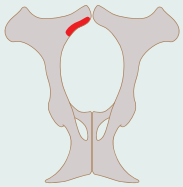
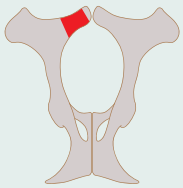
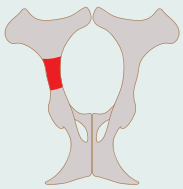
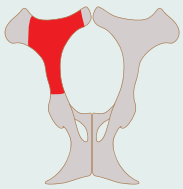
Fig. 7.5 Iliac stress fracture: Examples of diverse IRU patterns (arrowheads) that may be associated with injury on dorsal and dorsolateral scintigrams. IRU may be subtle (a) and good image quality and oblique views (k, l) assist detection in these cases. Band of muscular uptake in (i) (arrows) is incidental, clinically unimportant and readily distinguished from iliac IRU in this case, although marked muscular uptake may sometimes impair assessment of pelvis.

immediately. Continued bone scan activity beyond clinical resolution is common and does little to inform rehabilitation decisions.

- Marked lameness +/or ultrasonographic evidence (or strong clinical suspicion) of possible displacement (Figure 7.4e,f): tie up for 4–10 weeks (determined by severity and location of injury) with ultrasonographic monitoring to guide management. Measures to limit risk of supporting limb laminitis and pleuropneumonia (p. 93, p. 309).
- The most severely displaced injuries (Figure 7.6) require lengthier periods of stable rest +/– cross-tying to stabilize the injury site before paddock turnout can be considered, even if being retired from racing.
- For all but the most severe (unstable) cases, management is guided by initial clinical severity, suspected extent/displacement of injury based on ultrasonographic assessment and clinical progression (comfort/development of pelvic asymmetry) over initial 1–2 weeks. When advising on likely rehabilitation/

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Table 7.1 **Management of iliac stress fractures**

	INJURY	MANAGEMENT	LIKELY RETURN TO CANTERING	LIKELY RETURN TO RACETRACK
	Iliac wing: partial, non-displaced/ minimally displaced	Stable rest until sound then walking. 3–4+ weeks walk/3–4 weeks trot. Ultrasound review prior to commencing trot.	2–3 months	4–6 months
 	Iliac wing: full sagittal, minimally displaced Iliac shaft (+/– wing): non-/ minimally displaced	Stable rest +/– cross-tie (depending on clinical progress in initial 7–10 days). Ultrasound review at major step-up stages. When callus judged to be mature commence 4–6 weeks walk.	4-5 months	>6 months
	Iliac shaft (+/– wing): displaced, moderate-severe clinical/imaging features	Cross-tie (4–10+ weeks) then further stable rest depending on ultrasound/ clinical progress	8–10+ months	>12 months (retirement if marked residual asymmetry)

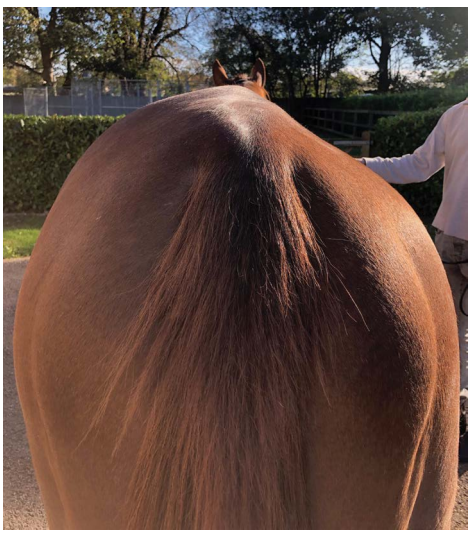


Fig. 7.6 Severe permanent alteration to pelvic symmetry resulting from displaced fracture of right ilium; injuries of this severity may heal satisfactorily for paddock/breeding soundness but not racing use.

prognosis these features are more important than location (iliac wing, shaft or both). Generally possible to give more precise rehabilitation guidance in post-acute phase once clinical progress has been assessed.

- Ultrasonographic monitoring particularly useful for iliac wing injuries to determine when fracture line/ discontinuity has been sufficiently bridged by mature callus (Figure 7.7a,b) to increase plane of exercise (first from stable rest to walking stage, then walking to ridden exercise stage).
- Catastrophic displacement with arterial disruption is a rare event but may be sudden, cannot be halted and invariably results in death.

Prognosis

- Non-displaced/minimally displaced wing or shaft fractures: prognosis for racing is good.
- For anything other than non-displaced or minimally displaced injuries, best to reserve judgement about likely rehabilitation length and prognosis for future

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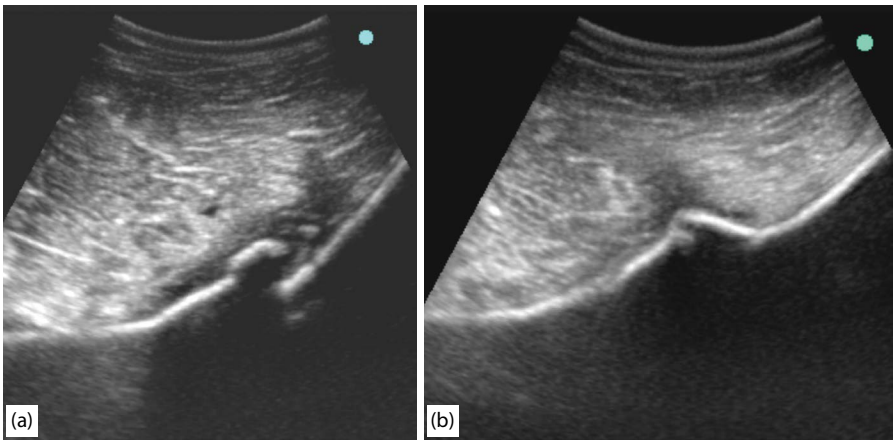


Fig. 7.7 **Iliac wing stress fracture: (a) Acute ultrasonographic appearance with disrupted dorsal bone contour and immature callus, and (b) follow-up scan appearance with sufficient bridging callus to recommend that walking phase of rehabilitation commences.**

racing until ascertaining presence/severity of asymmetry and comfort level over initial 2–3 weeks.
- Displaced wing fractures: retain some permanent pelvic asymmetry but action usually unaffected; prognosis for racing is good.
- Displaced shaft/wing fractures: risk of catastrophic deterioration/haemorrhage in initial days/weeks after injury in small number of cases. Beyond this period: guarded to fair prognosis for racing depending on degree of resulting pelvic asymmetry; markedly displaced injuries generally retired.
- Reinjury (same or contralateral sides) on return to full work/racing may occur, but uncommon and currently no criteria available for identifying at-risk animals.
- Prognosis for breeding: pelvic asymmetry following displaced fracture does not usually preclude breeding career for fillies; however, in cases of marked deformation per-rectum assessment by reproductive specialist may be warranted.
- The most problematic cases to give prognostic guidance on are those with marked asymmetry and long-term non-union or mal-union. Some will continue to periodically have difficulty rising from recumbency for months after injury; some ultimately require euthanasia while others may successfully enter a breeding career.

Pubic stress fracture

Rare injury affecting ventral pelvis. Unilateral, or may involve the cranial pubic symphysis. Typically non-displaced at time of injury detection.

Causes

- Presumed from history/presentation/signalment to be a stress/fatigue fracture.
- Biomechanical factors involved in development unknown.

Risk factors

- Rare condition.
- Risk factors unknown.

History

- Unilateral hindlimb lameness.
- May present with initial history of unilateral lameness/shortness of stride when horse is moved across/around stable.

Signs

- Lameness ranges from subtle to moderate.
- Sometimes with inconsistent gait characteristics such as lamer at walk than trot, initial few steps very lame, +/– 'tracking off' line at trot

Diagnosis

- May be a diagnostic challenge due to non-specific presenting signs and subtle/elusive diagnostic imaging features.
- Bone scan: definitive diagnostic modality. IRU in pubis (Figure 7.8a–e) of variable intensity. Pathology may readily be missed due to radiomarker activity in urinary tract and superimposition of other structures (caudal vertebrae); consequently, important to obtain oblique views or repeat scan if urinary activity obscures site of interest.
- Ultrasound (transrectal): usually employed following injury detection by bone scan to ascertain degree of cortical disruption.

Management

- Rehabilitation programme determined by initial clinical and imaging severity, and risk of displacement (see Chapter 5).

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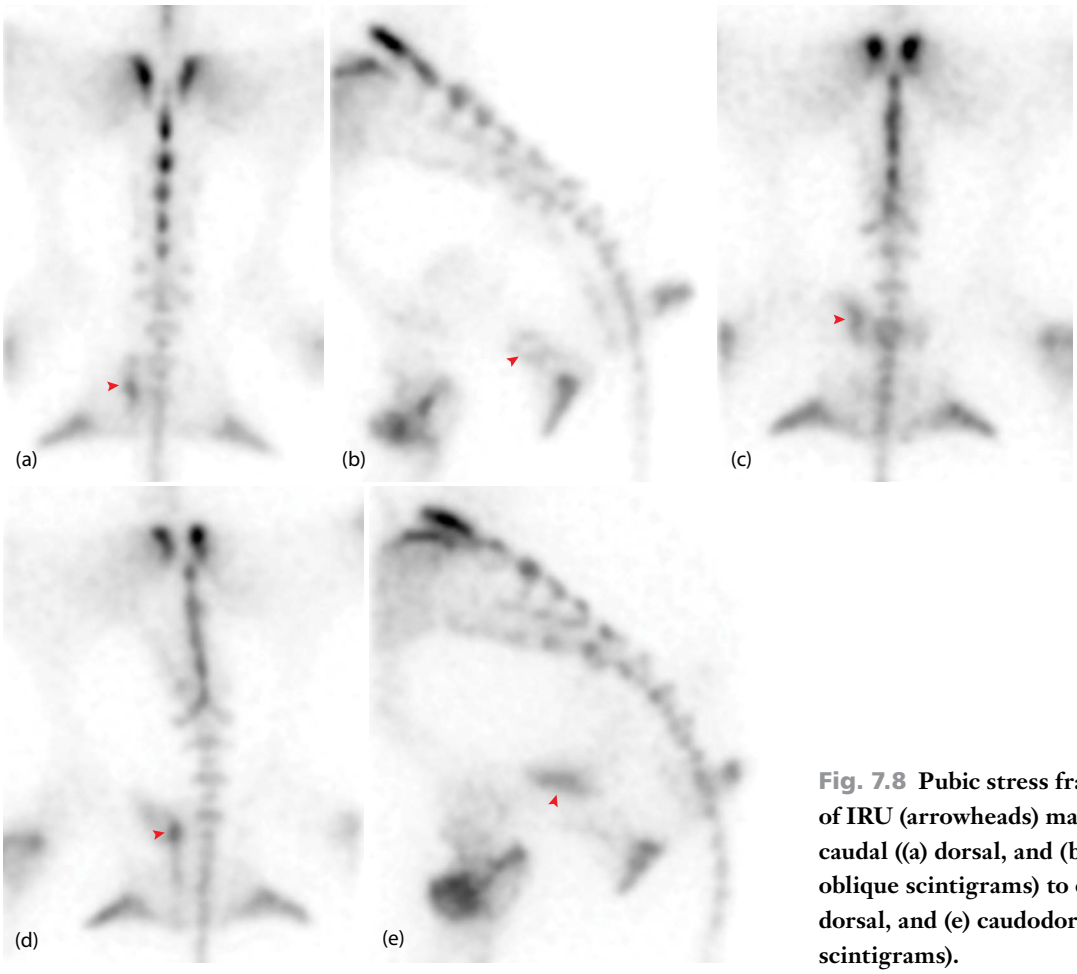


Fig. 7.8 **Pubic stress fracture: Position of IRU (arrowheads) may range from caudal ((a) dorsal, and (b) caudodorsal oblique scintigrams) to cranial ((c, d) dorsal, and (e) caudodorsal oblique scintigrams).**

- Ultrasonography may be used in some cases to monitor healing.
- Moderate lameness and non-displaced fractures: stable rest (4–6 weeks)/walking (4–8 weeks) usually sufficient.
- Subtle/mild lameness and non-displaced fracture: 3–4 weeks walking/3–4 weeks trotting usually sufficient.

Prognosis

- Non-displaced fractures: prognosis for racing is excellent.

Tuber coxa fracture

Commonly known as 'knocked down hip'; involves the lateral bony prominence of ilium, which is largely unprotected by overlying muscle. Fracture may either be complete (transverse or oblique) with caudolateral displacement of the tuber coxa, or partial with caudoventral or craniodistraction of fragments. Unilateral.

Cause

- May occur either as result of direct trauma (such as catching point of hip on doorway) or as muscular avulsion or stress fracture at exercise.

Risk factors

- Uncommon condition.
- Any age/stage of training.
- Risk factors unknown.

History

- Acute-onset lameness; initially noted either in stable or following exercise.

Signs

- Unilateral hindlimb lameness of moderate-marked severity.
- May be more severe at walk than at trot.
- May display tracking off gait.

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- Palpable and visible asymmetry of tuber coxae when examined from rear: affected side is lower or may appear absent with loss of normal bony landmarks +/– palpable bony fragment in paralumbar fossa.
- Haematoma +/– crepitus may be noted below point of tuber coxa.
- Pain on palpation/'guarding'/muscle spasm of flank in acute phase.

Diagnosis
- Clinical findings usually definitive.
- Ultrasonography (transcutaneous): not always definitive due to normal irregular bony margin of tuber coxae.
- Radiography (standing dorsomedial-ventrolateral 50^0 oblique projection): possible but rarely necessary or practical.
- Bone scan: rarely necessary but if performed, asymmetry of tuber coxae readily seen on dorsal and lateral images. Bone activity increased for many months following injury.

Management
- Conservative management.
- Partial fractures: stable rest (4–6 weeks)/walking (4–6 weeks).
- Complete fractures: stable rest (6–8 weeks)/walking (4–8 weeks).

Prognosis
- Good prognosis for return to full athletic soundness.
- Permanent pelvic asymmetry is inevitable; some abnormality of gait (particularly at walk) may result but unlikely to affect faster paces.
- Rarely, skin penetration by the fractured parent bone, or sequestration and fistulation may result: requires surgical resection (with resulting good prognosis for full resolution) (Figure 7.9).

Tuber ischium fracture

Injury to the bone promontory at the back of the pelvis from which the caudal thigh muscles originate. Usually unilateral.

Causes
- Usually direct trauma or avulsion injury following a backward fall/slip.
- Occasionally as a spontaneous avulsion injury sustained at exercise.



Fig. 7.9 Cosmetically obvious (but clinically insignificant) residual defect from prior sequestrated tuber coxa fracture.

Risk factors
- Rare condition.
- Any age/stage of training.

History
- Causative incident not always observed and lameness may be reported to have arisen at exercise.
- Often presented for examination as chronic low-grade lameness rather than during acute phase.

Signs
- Mild-moderate unilateral hindlimb lameness; reduced cranial phase of stride +/– reluctant to weight-bear on heels.
- Flattened appearance to affected tuber ischium.
- Acute phase: pain on palpation +/– visible dimpling of muscle/focal sweating over affected site. Associated hamstring muscles may be palpably tight.
- Chronic injuries: visible asymmetry of tuber ischia +/– atrophy of caudal thigh muscles (semimembranosus/semitendinosus). Rarely, a hamstring 'strain' will result in swelling/pain in these muscles in the subacute phase and can be detected ultrasonographically.

Diagnosis
- Clinical findings usually definitive.
- Ultrasonography: obvious 'step' in tuber ischium usually visible (Figure 7.10), although bone margin has irregular appearance anyway in normal horses.
- Bone scan: may be required for diagnosis in cases with chronic/ambiguous presentation (Figure 7.11a,b).

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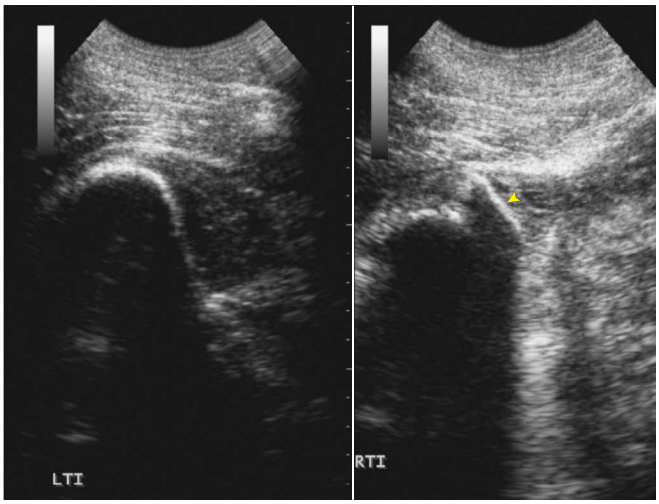


Fig. 7.10 **Tuber ischium fracture: Normal tuber ischium (on left) and fractured (arrowhead) tuber ischium with fragment displacement (transcutaneous ultrasonograms).**

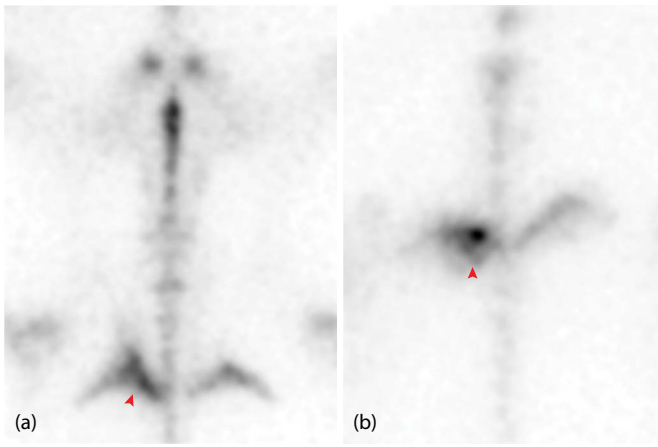


Fig. 7.11 **Tuber ischium fracture: Caudodorsal oblique (a) and caudal (b) scintigrams showing focal IRU (arrowheads) associated with injury of left tuber ischium.**

Management
- Conservative management: stable rest (4–6 weeks)/walking (4–6 weeks).
- Insufficient initial rest may result in chronic/recurrent lameness

Prognosis
- Excellent prognosis for return to racing soundness.
- Affected side remains flattened but action unaffected.
- Atypical (rare) cases occasionally encountered with propagation of fracture cranially and secondary sequestration: guarded prognosis for full resolution.
- Very rarely scarring of caudal thigh musculature (fibrotic myopathy) may be a long-term complication causing some permanent restriction of gait.

Fractures of the ventral pelvis/acetabulum

Fracture of one or more of the pelvic bones that form (or are in the region of) the hip socket (acetabulum) is typically an injury that occurs in the stable and is unrelated to exercise, although it can also be associated with iliac shaft stress fracture. Severity varies and instability of hip joint may result (Figure 7.12a–c). Typically unilateral although complex injuries sometimes involve both sides of the pelvis.

Causes
- Usually a traumatic injury arising from slip or fall (generally in the stable).
- Occasionally due to caudal propagation of iliac shaft stress fracture (p. 238).

Risk factors
- Rare condition.
- Any age/stage of training.
- Almost invariably occurs in fillies; very rarely in colts/geldings. Reason for this unknown but presumed to be associated with differences in pelvic dimensions between the sexes.
- Fractures through the growth plate may occur in juveniles.
- Stabling risk factors have been incompletely investigated; however, likely that flooring, stable size and proximity to other yard activities (resulting in excitement) are important contributors.

History
- Usually found with acute severe lameness in stable.
- May be evidence of a fall or having been cast.

Signs
- Severe acute hindlimb lameness is typical.
- Reluctance to move; if able to walk, markedly shortened caudal phase of gait may confound initial localisation of injury.
- In contrast to many other injuries, may display a preference (when stationary) to weight-bear fully on the affected leg (regardless of severity of injury).
- Guarding response +/– pelvic crepitus to manipulation/movement of affected limb.
- +/– pelvic asymmetry, visible prominence of hip region due to swelling/haemorrhage in acute phase.
- +/– swelling of upper thigh (due to haemorrhage) in cases of displaced fracture.

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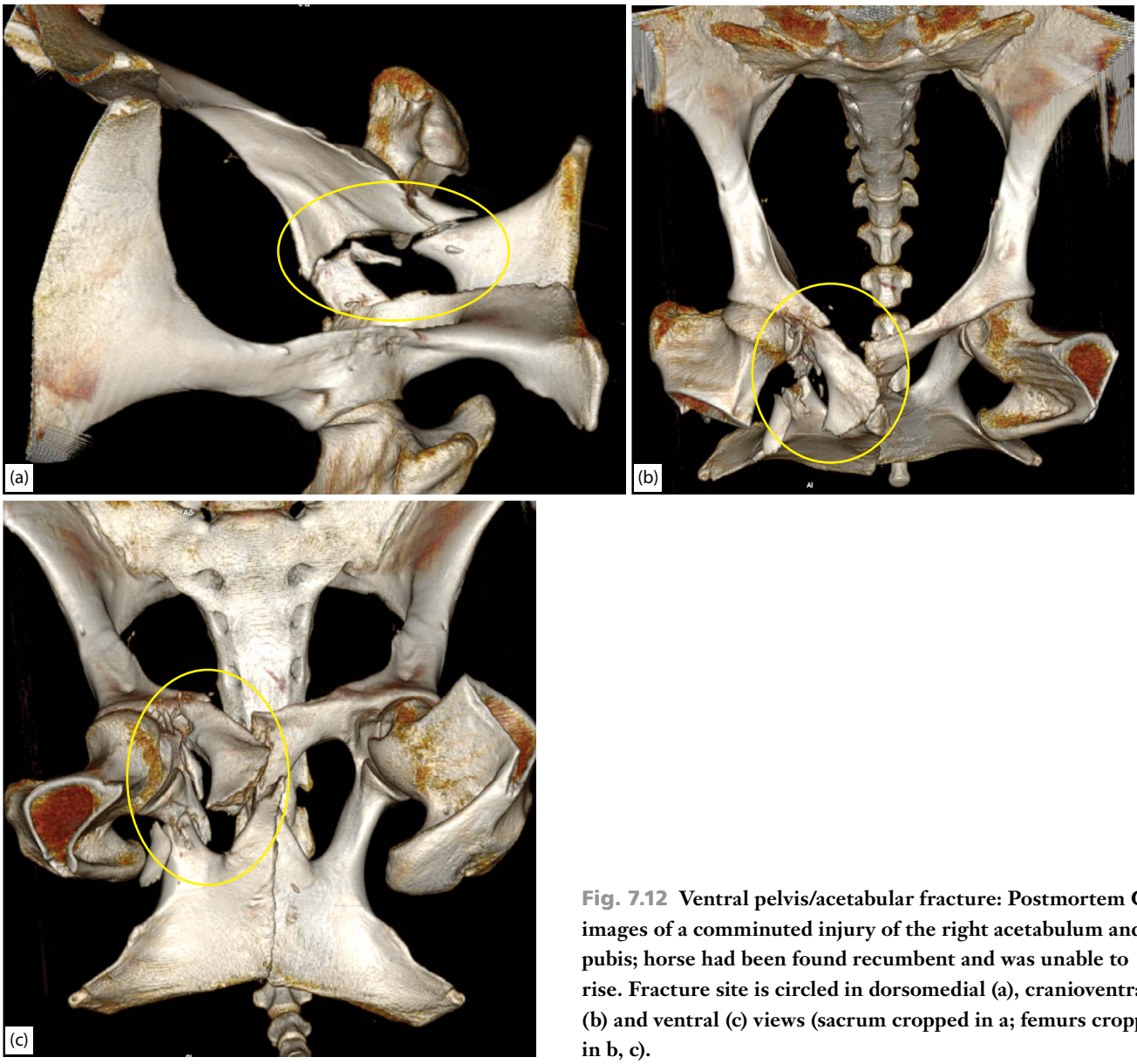


Fig. 7.12 Ventral pelvis/acetabular fracture: Postmortem CT images of a comminuted injury of the right acetabulum and pubis; horse had been found recumbent and was unable to rise. Fracture site is circled in dorsomedial (a), cranioventral (b) and ventral (c) views (sacrum cropped in a; femurs cropped in b, c).

- Less commonly, may be found recumbent (and unable to rise) in stable.

Diagnosis

- Rectal examination: internal haematoma may be palpable on affected side.
- Ultrasonography (transcutaneous and transrectal): obvious 'step' in contour (Figure 7.13) of acetabular bone; may be overriding of fragments +/– focal haematoma.
- Full assessment of extent of injury difficult due to frequently complex fracture configuration. Ultrasonography generally underestimates extent/severity of injury and clinical appraisal should also guide management.
- Bone scan examinations (Figure 7.14a,b) have greater sensitivity for detecting acetabular fracture than ultrasound, but rarely practical (as transport inadvisable in acute phase). Standing radiography is usually definitive in cases of displaced fractures in lightly muscled or juvenile horses (poor sensitivity for non-displaced/minimally displaced fractures) but also rarely practical (as not feasible with ambulatory radiographic equipment).

Management

- Management dependent on clinical/ultrasonographic severity, and desire to commit to lengthy rehabilitation with uncertain outcome.
- Has previously been commonplace for euthanasia to be recommended; however, there is now greater

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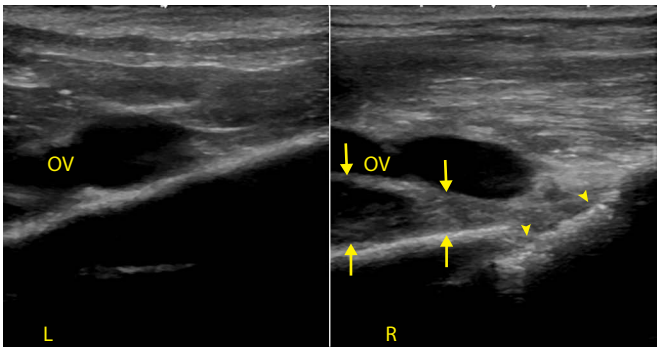


Fig. 7.13 **Ventral pelvis/acetabular fracture: Transrectal ultrasonogram of normal (left) and injured (right) sides; disruption of bone contour of the internal/medial aspect of the acetabulum is noted (arrowheads), with oedema/haemorrhage (arrows) also present between the bone margin and obturator vessels (OV).**

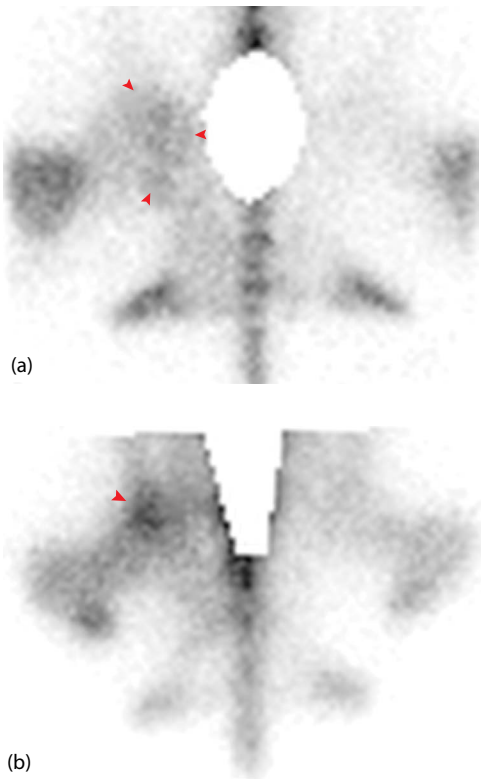


Fig. 7.14 **Acetabular fracture: Dorsal scintigrams of caudal pelvis, with mild diffuse (a) and marked focal (b) IRU associated with injuries involving left acetabulum.**

awareness that return to use (racing or breeding) is possible for many cases.
- Preferable to tie-up (pp. 60-61) for initial period (6+ weeks); however, potential risks of recumbency need to be weighed against those of tying up and should be managed on an individual basis. Ultrasonographic monitoring to assess stability and guide management.
- Analgesics/deep bedding to minimize risk of supporting limb laminitis (p. 93), although many will weight-bear satisfactorily when stationary. Pleuropneumonia is a risk (p. 309).
- Persistence (or recurrence) of severe lameness/pelvic instability beyond initial phase, or severe clinical deterioration/pain (Figure 7.15) may warrant euthanasia.

MANAGING SUSPECTED FRACTURES OF THE VENTRAL PELVIS/HIP

- Clinical history and initial appraisal usually sufficient to suspect fracture of ventral pelvis/hip joint.
- If standing: cross-tie and administer non-steroidal anti-inflammatory medication. Do not transport.
- If recumbent: attempt to obtain diagnosis and assisted attempt(s) to rise; euthanise if unsuccessful.
- Presumptive diagnosis often (but not invariably) possible with ultrasonographic examination (transcutaneous + transrectal); scan can be performed electively (not necessarily an emergency procedure, unless recumbent).
- Prognosis (for life/athletic/breeding future) may not become clear until several weeks have elapsed.

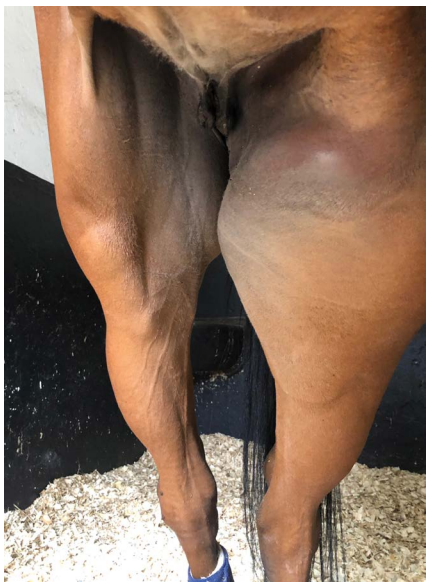


Fig. 7.15 **Ventral pelvis/acetabular fracture: Catastrophic displacement of ventral pelvic injury despite being cross-tied; acute, marked swelling of medial left thigh/upper hindlimb resulting from internal haemorrhage (and coinciding with clinical deterioration).**

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Prognosis
- Moderate risk of catastrophic displacement or supporting limb laminitis in initial weeks.
- Progress in initial weeks is strongest prognostic indicator: if survival beyond period of pelvic instability, fair prognosis for paddock soundness.
- Prognosis for athletic soundness: guarded-poor depending on degree of resulting pelvic asymmetry. Return to training/racing is possible in some individuals (reported in up to 30% horses surviving beyond diagnosis), but currently difficult to categorise cases for likely outcome based on initial imaging/clinical features. Injuries resulting in marked pelvic asymmetry +/– significant disruption to the acetabulum are considered to have the poorest prognosis for athletic soundness.
- Prognosis for breeding soundness (for horses that survive the healing period) dependent on degree of resulting derangement of internal pelvic architecture, but generally considered to be good. Internal assessment of birth canal (once healed) by reproductive specialist useful for planning but not definitive: dystocia is rare in mares that have previously sustained ventral pelvic/acetabular fractures.

Third trochanter (of femur) fracture

Fracture of the bony prominence on the lateral upper aspect of the femur is typically an injury that occurs in the stable and generally considered to be unrelated to exercise. Fracture usually involves displacement/fragmentation of third trochanter although rarely non-displaced fracture can occur. Clinical severity varies, often marked initial lameness. Unilateral.

Causes
- Usually a traumatic injury arising from slip or fall.
- Third trochanter is the bony prominence which serves as the primary site of insertion of the tendon of the superficial gluteal muscle; a slip of hindlimb resulting in extension/lateral rotation of hip could cause avulsion fracture.

Risk factors
- Rare condition.
- Any age/stage of training.

History
- Usually found with acute severe lameness in stable.
- May be evidence of a fall or having been cast.

Signs
- Hindlimb lameness: usually marked in acute phase, moderate post-acute phase.
- Acute phase: reluctance to move/protract limb, although usually very comfortable standing on the affected leg (in common with acetabular fractures, p. 247).
- Clinical localisation can be challenging, although localised swelling +/– pain response to palpation of injury site in upper hindlimb may be present.
- Ipsilateral gluteal or thigh muscle atrophy may develop following injury.

Diagnosis
- Important to rule out other (particularly pelvic) conditions that might have similar presentation.
- Ultrasonography (transcutaneous) is field modality of choice: fragmentation (usually cranial displacement) or irregular bone margin, +/– anechoic fluid accumulation overlying third trochanter (Figure 7.16).
- Bone scan: moderate to intense focal IRU of affected site (Figure 7.17). Mild IRU of third trochanter may be an incidental finding and should be interpreted with caution.
- Standing radiography ($Cr50^{0}L30^{0}CdMO$ projection) is usually definitive in cases of displaced fractures in lightly muscled horses but use is uncommon.

Management
- Management protocols have not been validated/refined due to uncommon occurrence of this injury, but are guided by experience and clinical progress.

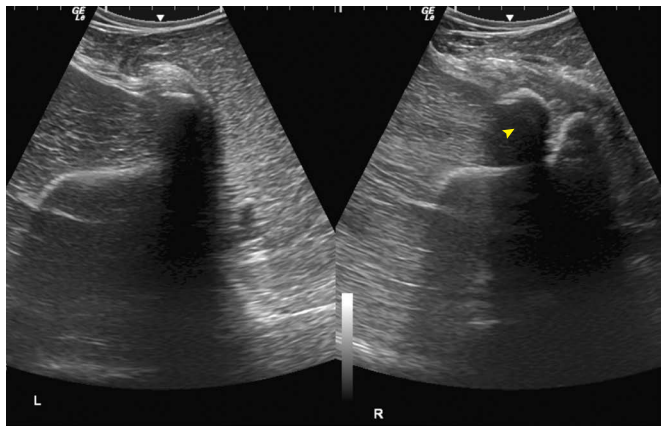


Fig. 7.16 Third trochanter fracture: Transcutaneous ultrasonograms of normal (left) and injured (right) proximal femur, with displaced fragment from third trochanter (arrowhead) indicated.

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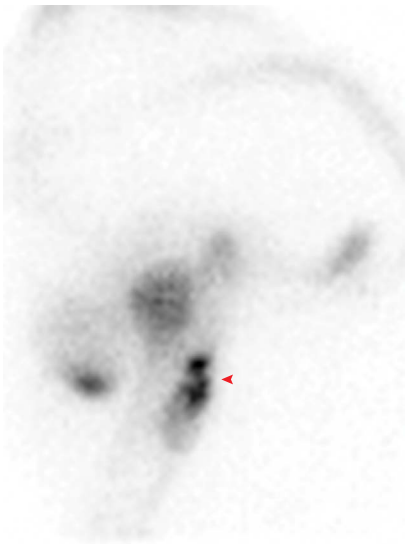


Fig. 7.17 Third trochanter fracture: Lateral scintigram of left hip (cranial to left), with marked and irregular/multifocal IRU in region of third trochanter noted (arrowhead). The focus of radiomarker activity cranial to the femur is bladder/urine.

- Initial 3–4 weeks stable rest, followed by 4–6 weeks controlled walking generally sufficient for resolution. Lameness usually improves significantly over initial 2 weeks following injury.
- Analgesics in acute phase although will usually weight-bear satisfactorily when stationary.
- Injury is considered to heal by fibrous union; hence, ultrasonographic monitoring is of little use to guide management. Bone scan activity can persist beyond clinical resolution and is also of little use for rehabilitation management.

Prognosis

- Good to excellent prognosis for athletic soundness.

Sacral/caudal vertebral fracture

Fractures involving the sacrum are rare and usually the result of direct trauma. They may be associated with (or mistaken for) other fractures of the pelvic girdle, including the hip. Fracture is usually at the S2/S3 site with variable displacement; this site is the last of the sacral vertebral junctions to fuse and may be predisposed to injury. Specific fracture configuration is often difficult to determine, but is characterized by callus with concurrent/secondary reactive bone change occurring at the sacral facet joints. Acute or chronic (through callus impingement) damage to nerve roots in the proximity of the fracture site may lead to associated neurological signs (neurogenic muscle atrophy, urinary +/or faecal incontinence).

Fractures of the caudal vertebrae (particularly the tail base) also result from direct trauma and generally cause short-lived pain/inflammation and loss of tail tone.

Cause

- Generally considered to be traumatic injury arising from fall (stress/fatigue fractures of sacrum may occur).

Risk factors

- Rare condition.
- Any age/stage of training.

History

- Sacral fracture: loss of hindlimb action +/– rapid-onset focal/generalized muscle loss over pelvis/sacrum.
- Recent history of fall/slip.

Signs

- Sacral fracture: marked 'hollow' focal/generalized muscle atrophy over caudal pelvis/sacrum (unilateral or bilateral) (Figure 7.18).
- Urinary/faecal retention and/or incontinence may occur depending on if neural damage is a feature.
- Marked pain on palpation +/– muscle spasm/'guarding' in sacral/tail base region.

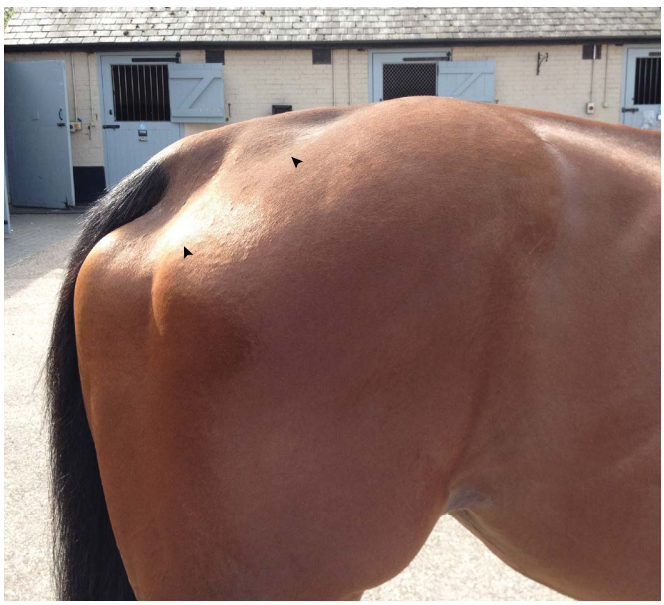


Fig. 7.18 Sacral fracture. Note the focal neurogenic muscle atrophy over caudal gluteal region (arrowheads).

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- +/– diminished tail tone/anal reflex.
- +/– hindlimb lameness; reduction/loss of caudal phase of stride with injuries involving hip.
- Tail base fractures characterized by localized pain (often severe) and loss of tail tone; +/– urine/faecal scalding as unable to raise tail.

Diagnosis

- Clinical findings strongly indicative but sacral fractures also may resemble bilateral pelvic (iliac wing) stress fracture.
- Ultrasonography (transrectal and percutaneous): detection of displacement/bone callus at fracture site.
- Bone scan: may be useful to characterize injury and involvement of other pelvic structures.

Management

- Anti-inflammatory medication in acute phase.
- Conservative management: 4–6 weeks stable rest with return to light exercise guided by ultrasonographic monitoring.
- May require intensive supportive care if incontinent. If urine retention: catheterization or regular manual decompression of bladder +/– evacuation of faeces per rectum until neurological improvement.
- Persistence of neuromuscular signs beyond acute phase may justify ultrasound-guided perineural anti-inflammatory medication (corticosteroid).

Prognosis

- Displaced sacral fractures: varies with severity but generally poor prognosis for full neuromuscular normality (commonly retain some muscular atrophy).
- Guarded-fair prognosis for return to full use.

Sacroiliac and lumbosacral pain

Despite their importance to locomotion, conditions of the sacroiliac/lumbosacral region are poorly understood and difficult to investigate. Neural pain may be implicated in many cases as well as a range of osseous pathologies (some degenerative and others congenital/developmental). Manual assessment is highly subjective and localisation of source of suspected pain is challenging. Diagnostic imaging is limited by access and specificity, and diagnostic blocking may also lack sensitivity and specificity. Postmortem surveys suggest that a high proportion of 'normal' racehorses probably have degenerative changes of both SIJs; however, it is generally considered that true sacroiliac 'pain' is more common in other disciplines (showjumping, dressage) than in flat racing.

The lumbosacral junction is the articulation of the lumbar spine with the pelvis (iliac wings) and sacrum (Figure 7.19). Degenerative arthropathy may affect the lumbosacral and intertransverse joints; severity varies and pathology may not be symmetric.

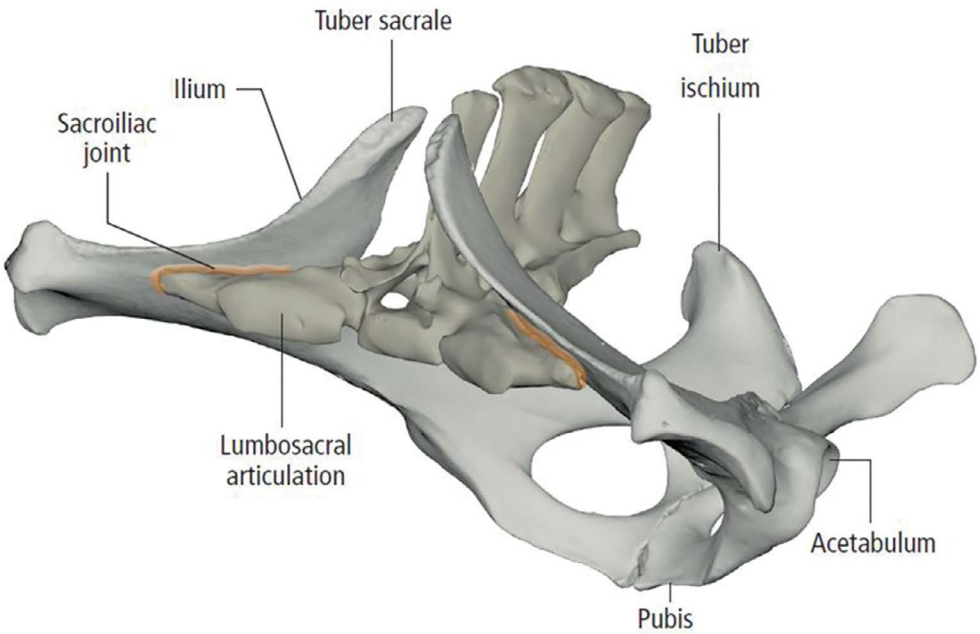


Fig. 7.19 **Lumbosacral anatomy.**

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Causes
- Generally chronic/degenerative conditions.
- Underlying mechanisms of injury poorly understood.

Risk factors
- Uncommon; true prevalence unknown; probably under-diagnosed as a cause of poor performance/lameness.
- All ages/stages of training.
- Risk factors unknown.

History
- Presenting signs are typically mild and chronic.
- Include poor hindlimb impulsion, back stiffness and poor muscling over thoracolumbar spine.
- May display resistance behaviour (bucking/kicking/unwillingness to jump off at start of canter).

Signs
- +/– mild bilateral hindlimb lameness with toe drag (unilateral lameness not usually a feature).
- The tubera sacrale ('jumpers bumps') are usually level; variable response to manual pressure on these structures.
- Reluctance to flex and extend the lumbosacral region.
- May resent opposite leg being picked up.

Diagnosis
- Not possible to definitively diagnose sacroiliac or lumbosacral pain from clinical examination alone.
- Clinical signs non-specific and similar to many hindlimb conditions (e.g., plantar osteochondral disease of fetlocks/tibial stress fracture/proximal suspensory ligament desmopathy).
- Should be considered a diagnosis of exclusion: comprehensive nerve blocking +/– imaging (bone scan and ultrasonography) to rule out other potential problems.
- Ultrasonography: callus on ventral margin of SIJ or lumbosacral joints (transrectal scan) or disruption/enlargement of dorsal and/or ventral sacroiliac ligaments (transcutaneous scan) may contribute to diagnosis.
- Bone scan: generally non-specific; bone scan appearance of normal horses varies considerably with age, muscling and symmetry of hindquarters, and pathology on underside of lumbosacral junction may not be discernible due to attenuation by intervening tissues.
- Trial medication of sacroiliac and intertransverse joints (combination of three approaches: cranial/cranial midline/caudal) may assist diagnosis.
- Diagnostic blocking associated with small risk of temporary ataxia/recumbency through inadvertent blockade of major nerves in the region (sciatic and cranial gluteal) and (rarely) clostridial myositis (frequently fatal).

Management
- In absence of viable intra-articular techniques, treatment usually consists of periarticular deposition of anti-inflammatory (corticosteroid) +/– sclerosing drugs (prolotherapy).
- Efficacy of periarticular medication has not been substantiated.
- Prolonged drug detection times are a feature of deposition of corticosteroid in ligamentous tissue, and limits applicability of medication in horses in training.
- Horses should be kept in work to maintain muscular fitness.

Prognosis
- Prognosis for continued training hinges on severity of clinical signs and response to medication.
- Rarely curtails racing career.

THE NECK AND BACK

Applied anatomy

The spinal column serves both to protect the spinal cord and to be the axis upon which the limbs act to generate motion. The column consists of vertebrae, intrinsic musculature and longitudinal and intervertebral ligaments. Although the vertebrae vary in form throughout the neck and back, their basic structure consists of a cylindrical vertebral body surmounted by a vertebral arch through which the spinal cord passes, and vertebral processes that act as attachment points for muscles and ligaments. Each vertebra articulates with its neighbours through 'ball-and-socket' fibrocartilaginous joints (with intervertebral discs) at the vertebral body, and at dorsolateral cranial and caudal synovial (facet) joints on each side of the vertebral arch (Figure 7.20). The orientation of the synovial articular facets changes along the spine, reflecting different ranges of motion of particular spinal segments. Stability and movement of the spinal column comes from longitudinal and intervertebral muscle units and from longitudinal ligaments that traverse the dorsal spinous summits (nuchal and supraspinous ligaments), the vertebral canal (dorsal longitudinal ligament) and the vertebral bodies (ventral longitudinal ligament). Movement between adjacent vertebrae is limited but overall range of movement is sometimes considerable. Segmental movements are complex and vary with location and gait.

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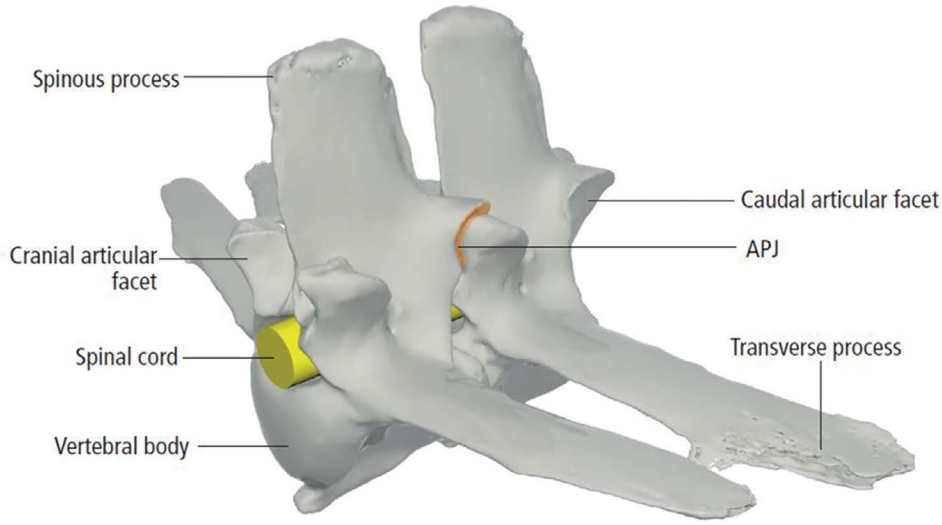


Fig. 7.20 **Lumbar vertebral anatomy.**

Neck

There are seven vertebrae in the neck. The first two vertebrae are specialized in form: C1 (atlas) articulates with the skull and has broad wings that are palpable behind the poll; and C2 (axis) has a large dorsal spinous process (DSP) that serves as an attachment point for the nuchal ligament. The nuchal ligament extends from the skull to the withers. Movement at the neck–skull articulation is primarily flexion/extension, whereas that at C1/C2 is rotation; through the remainder of the neck lateral bending predominates. The muscles of the neck lie largely lateral and ventral to the spinal column and have a role in locomotion.

Back

The back is made up of 18 thoracic and six lumbar vertebrae, although anatomical variation in the lumbosacral region (in number of vertebrae, presence of transitional vertebrae and location of divergent DSPs) is common. The contribution of this anatomical variation to the risk of developing back pathology is unknown. The thoracic vertebrae have large DSPs (most prominent in the withers: highest at T4–T6) and each articulates with a pair of ribs, whereas the lumbar vertebrae are notable for their relatively wide transverse processes. The DSPs are oriented caudally through the thoracic spine to the 'anticlinal' vertebra (T16), beyond which they proceed to angle cranially. The spinal column continues into the fused sacrum, which by articulating with the pelvis at the SIJs links the trunk to the hindlimbs. The junction of the lumbar spine and sacrum is an important site for mobility of the back and comprises the lumbosacral joint (between L6 and S1) and the paired lumbar sacral facet and intertransverse joints.

The supraspinous ligament continues on from the nuchal ligament of the neck and attaches to the dorsal spinous summits to limit flexion of the back. The interspinous ligament joins the caudal aspect of a dorsal spine to the cranial margin of its neighbour. Epaxial muscles lying above and hypaxial muscles below the transverse processes facilitate lateral flexion and also provide dynamic stability to the back. The major epaxial muscle is the longissimus dorsi, with the multifidus muscles also playing a large role in stability. A thick thoracolumbar fascia serves as the origin of several muscles important to hindlimb impulsion and hip flexion, and as in humans is likely to have significant load-bearing and sensory roles.

The vertebral segments are each capable of dorsal flexion/extension, lateral bending and axial rotation to various degrees. Movement (dorsoventral) is greatest at the lumbosacral articulation, while lateral bending and axial rotation are greatest in the mid-back (around T11). The thoracolumbar vertebral column and its musculature maintains a relatively rigid structure with less than 10% flexion possible at each intervertebral joint; as speed increases muscular tension ensures further stabilisation of the spine. Conformation may significantly affect biomechanics of the spinal column, with longer backs being associated with greater lateral bending in the lumbar region.

Examination

Examination of the neck includes observation of muscular symmetry and palpation to detect obvious deformation or guarding of the vertebral column. Lateral and dorsoventral flexibility are tested actively and passively

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for resentment or restriction of movement. Local skin reflexes and jugular vein patency are also assessed.

Examination of the back begins with assessment of symmetry of the musculature, spine and pelvis. There is great variability between horses in their response to palpation/manipulation of the back and findings should be interpreted in the context of clinical presentation. Note should be made of asymmetry or atrophy of epaxial musculature as well as areas of visible tack contact. Palpation of epaxial muscles and dorsal spinous summits may permit localization of pain or muscular spasm/fasciculation, but is inherently subjective. Stimulation of movement can also allow for assessment of the limits of flexion, extension and lateral bending present in the thoracolumbar and lumbosacral regions. Generally, restricted motion may indicate skeletal pathology and hypermobility/aggression may indicate superficial muscle 'soreness' or simply skin sensitivity. Back pain typically results in reduced dynamic back flexibility and stride length due to guarding; assessment of gait in hand and ridden (to determine any influence of weight of rider) may be useful. While hindlimb lameness may cause alterations to back movement, when lameness and back pain co-exist it is reasonable to assume that the primary cause of the lameness is to be found in the limb (as even marked back pain does not typically cause lameness).

Investigation of suspected back pain is complicated by lack of specificity of signs (horses may present with features of back pain or poor performance for a multitude of reasons, including behavioural) and the knowledge that many horses with advanced back pathology continue to perform at elite level. There is currently only incomplete understanding of the detailed neuroanatomy of the thoracic and lumbar vertebral column of the horse, and this may be a significant factor in the variability in application and response to diagnostic analgesic and therapeutic interventions in these regions. Given the limitations of diagnostic analgesia when applied to structures of the topline, diagnosis of back pain is frequently highly subjective and rarely definitive.

Cervical vertebral (neck) fracture

Fractures of the neck may involve the vertebral body or arch (most common in mid-neck region C3–C6) or the facet joints (most common in lower neck C5–C7) of the cervical vertebrae. The spinal cord is not always affected. Spinal cord involvement in the acute phase can be due to direct bone impingement or local haemorrhage/oedema. In some cases with no acute involvement of the spinal cord, subsequent encroachment of inflammatory pannus or bony callus during the healing phase may result in delayed onset of neurological signs.

Cause

- Usually the result of a traumatic incident such as rearing over, diving under the starting stalls, pulling back or fall at exercise.

Risk factors

- Any age/stage of training.

Signs

- Determined by configuration/location/severity of fracture and involvement of spinal cord.
- Range from mild neck stiffness to marked neurological deficits or recumbency.
- Neck may be held abnormally low +/– stiffness/guarding/swelling/patches of sweating.
- +/– ataxia: varies in severity and may be transient or persistent.
- Assessment of voluntary or passive ability to flex neck to each side (avoid if concern over unstable fracture) may reveal pain or restriction.
- +/– segmental sweating (typically unilateral).

Diagnosis

- Radiography: latero-lateral (LL) projection (Figure 7.21a) most useful for displaced vertebral body fractures; oblique projections essential for full assessment of lateral processes/facet joints (Figure 7.21b,c). Fracture line not always evident. Projections from left and right sides essential for comparison.
- CT useful for assessment/prognosis in some cases.

Management

- 'Treat the patient not the radiograph' is particularly relevant, as imaging abnormalities may sometimes be marked yet result in good clinical outcome.
- Fractures with severe or deteriorating neurological signs or recumbency: may warrant euthanasia (surgical stabilization/internal fixation possible but rarely undertaken).
- Cases with no or few neurological signs: conservative management. Duration of stable rest dependent on clinical/radiological progress; typically 6–8 weeks.
- No return to ridden exercise while neurological signs persist.
- Anti-inflammatory medication in acute phase, although local swelling/muscle guarding limits movement and may be protective.
- +/– use of a cradle to 'splint' the neck.
- Hay/feed/water provided at height to avoid lowering of neck; avoid tying up.

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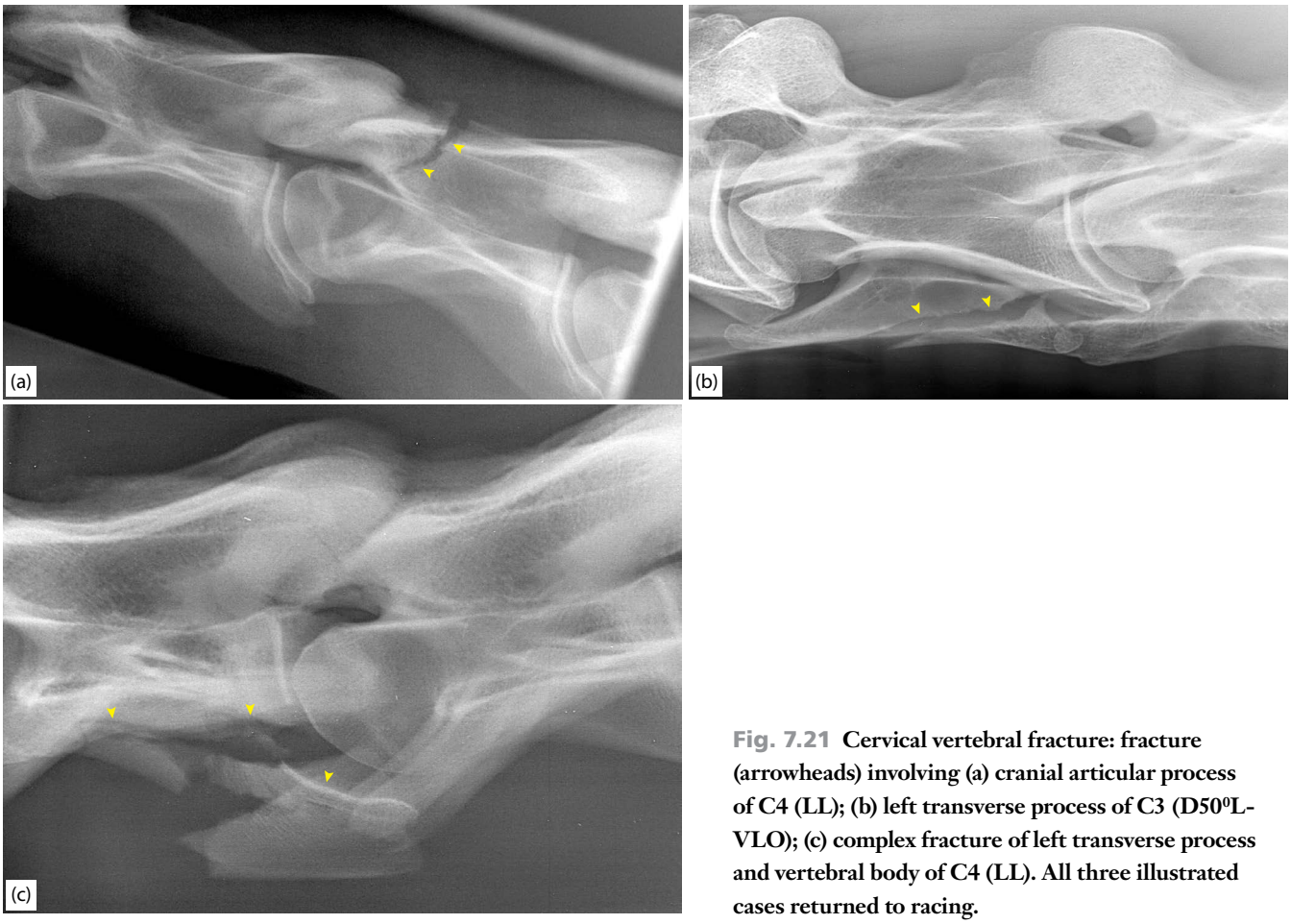


Fig. 7.21 **Cervical vertebral fracture: fracture (arrowheads) involving (a) cranial articular process of C4 (LL); (b) left transverse process of C3 (D50^{0}L-VLO); (c) complex fracture of left transverse process and vertebral body of C4 (LL). All three illustrated cases returned to racing.**

Prognosis

- Depends on initial clinical and radiological severity and any resulting neurological deficit caused by compression of spinal cord.
- Horses with no or minimal neurological signs: good prognosis for racing: return to ridden exercise usually possible within 3–6 months.

Dorsal spinous process impingement ('kissing spines')

Close apposition, impingement or overriding of DSPs of thoracic or lumbar vertebrae is widely known as 'kissing spines'. May affect one or multiple sites: most commonly in saddle region (T13–18). Pathology may also involve interspinous ligaments and associated bone interface. Very common but in flat racehorses rarely clinically active or cause interruption to training and only occasionally associated with overt evidence of back pain.

Cause

- May reflect a combination of factors including conformation, age and heritability.

Risk factors

- Very common: most (70–90%) 'normal' racehorses (free from signs of back pain) have at least one site of 'kissing spines' in the higher grades of radiographic severity.
- Factors determining severity and significance of lesions are unknown.

History

- Frequently first detected during imaging of back (radiography/bone scan) as part of investigation into suspected orthopaedic injury; in these circumstances likely to be an incidental finding.
- When clinically active: may have history of being cold-backed, displaying poor action under tack, uncoupled canter or lack of hindlimb impulsion.

Signs

- Clinical findings usually non-specific and cannot be readily distinguished from other forms of pelvic/thoracolumbar pain (or some hindlimb injuries).
- +/– palpable focal back pain or epaxial muscle spasm (non-specific).

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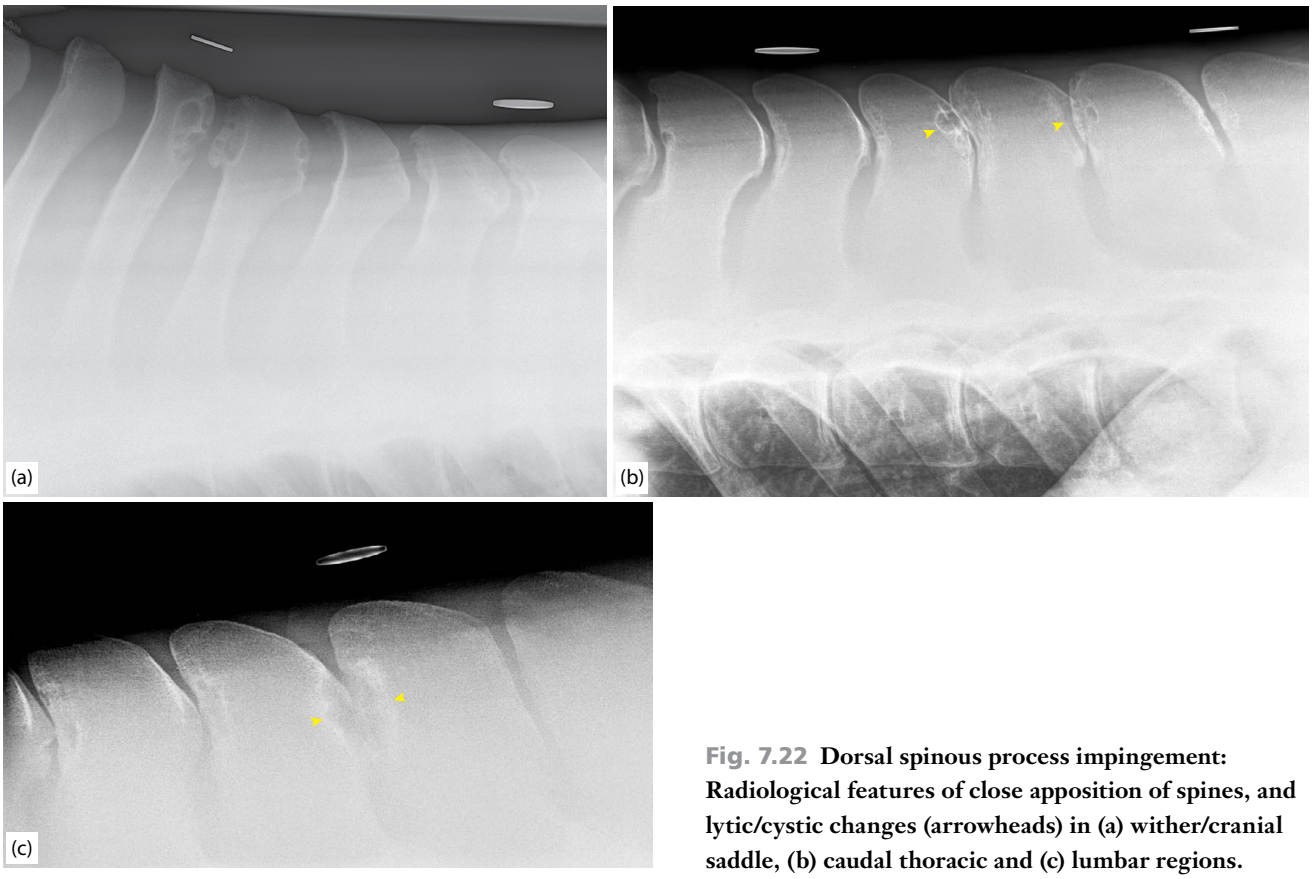


Fig. 7.22 **Dorsal spinous process impingement: Radiological features of close apposition of spines, and lytic/cystic changes (arrowheads) in (a) wither/cranial saddle, (b) caudal thoracic and (c) lumbar regions.**

- Not typically associated with lameness.
- 'Cold-backed' behaviour (usually seen when initially mounted by rider) appears to be poorly correlated to 'kissing spines' or other thoracolumbar pathology.

Diagnosis

- Clinical examination not definitive in isolation: requires radiographic/ultrasonographic imaging.
- Radiography: lateral projections of entire thoracolumbar region (placement of markers to assist future blocking/medication). Radiological severity ranges from narrowing of interspinal space +/– densification of adjacent bone margins to marked bony modelling/cystic change (Figure 7.22a–c).
- Determining significance is problematic as there is very poor correlation between radiological severity and clinical signs. Exclusion of other potential injuries +/– regional analgesia/medication of suspected sites is important.
- Ultrasonography: assessment of thoracolumbar articulations, DSPs and supraspinous ligament readily undertaken in ambulatory setting and useful to identify possible sources of back pain. Asymmetry/ atrophy of multifidus muscles may be associated with sites of bony pathology.
- Bone scan: presence and intensity of radiomarker uptake at sites of DSP impingement is variable (Figure 7.23).

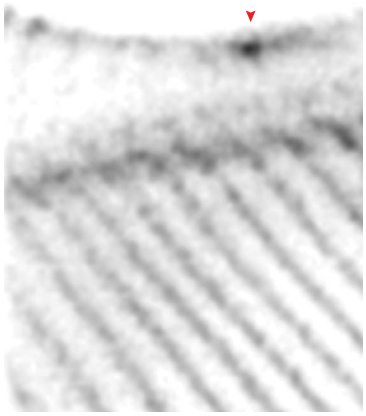


Fig. 7.23 **Dorsal spinous process impingement: Lateral scintigram with focal IRU (arrowhead) at single impinging site in thoracic spine noted.**

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- Defining relevance of radiological/bone scan findings may require diagnostic local anaesthesia and/or trial medication, as imaging abnormalities are common in 'normal' horses. Response to diagnostic blocking/medication may vary between horses, due in part to poor current understanding of neuroanatomy of the region and lack of standardisation/validation of technique.
- Genuine thoracolumbar back pain more likely with greater severity/extent of radiological signs of impingement, presence of concurrent arthritis of facet joints and/or increased bone scan activity.

Management

- Most horses with kissing spines (even of higher grades of severity) exhibit no evidence of back pain and require no treatment. Strong evidence of clinical importance should be present before treatment/management is recommended.
- Additionally, due to relatively short career and lack of necessity for racehorses to work in an outline, kissing spines rarely require/receive significant intervention (unlike in sport horses).
- Local infiltration of local anaesthetic +/– medication(s) (e.g., corticosteroid) on both sides of impingement sites can be used as presumptive treatment or diagnostic measure, but important to recognise that local anaesthetic may have effects on gait/back biomechanics even in normal horses and is therefore not necessarily specific for pain at site of interest.
- Prolonged drug detection times occur with deposition of corticosteroid in ligamentous tissue and limit usefulness of medication in horses in training.
- Physical therapies such as ESWT, manipulative/massage techniques and hot packs may assist relief of associated muscle spasm (Chapter 5, p. 55).
- In rare cases of presumptive diagnosis + poor response to previously described treatments + continued interference with training: surgery can be considered. Surgical techniques include desmotomy of interspinous ligament at affected sites, or removal of part (cranial wedge) or total affected portions of DSPs. Success of surgery in resolution of clinical signs is unpredictable.

Prognosis

- Highly unusual for kissing spines to interfere significantly with training or negatively impact on racing performance in flat racehorses: existing (limited) evidence suggests no correlation between radiological severity and long-term racing performance.
- Response to medication/surgery is broadly a function of strength of initial diagnosis.
- Corticosteroid infiltration +/– physical therapies carry a fair prognosis for improvement in signs and continued training. Repeat medication usually necessary.
- Surgery: fair to good prognosis for return to full use.

PRE-PURCHASE ASSESSMENT AND 'KISSING' SPINES

- Most racehorses have 'kissing'/impinging DSPs.
- In most cases they are considered unimportant.
- Attributing clinical significance to kissing spines should largely be through exclusion of other possible problems: typically a lengthy diagnostic undertaking.
- Not possible to make a determination on likelihood of current/future back pain based simply on detection or imaging appearance of back.
- For these reasons, radiographic screening of the back as part of pre-purchase assessment is not recommended.

Thoracolumbar vertebral stress injury

Stress injuries of the spinal column are common in the lower back of racehorses; however, most go undetected because of indistinct/mild clinical signs that generally do not interfere with training. Prevalence of specific pathologies remains under investigated. The spectrum of injury includes arthropathy of the articulating process joints (APJs or 'facet joints') and stress fracture involving the vertebral bodies or articulations, with these generally considered clinically indistinguishable. It is common for several sites in the back to be involved; stress fractures are often unilateral and involve the lumbar vertebrae (particularly at the lumbosacral junction) but are also found in mid- and caudal thoracic vertebrae. Modelling or fracture typically occurs at the cranial aspect of the vertebral lamina, between the cranial articular and DSPs, with extension to the adjacent APJ also observed (Figure 7.24). Occasional progression to catastrophic complete fracture of vertebral column (most commonly at L5/L6 junction) and in these cases there is usually postmortem evidence of maladaptive response to bone stress (vertebral endplate sclerosis) or intervertebral instability (ventral spondylosis).

As with some other abnormalities of the back, it is likely that detection of arthritic facet joints is not always clinically significant and should be interpreted in the context of a full diagnostic workup.

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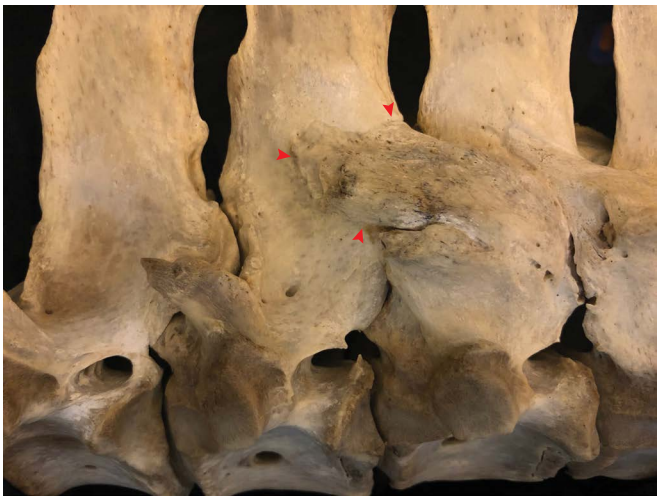


Fig. 7.24 Vertebral facet arthropathy: Profound long-standing articular modelling (arrowheads) resulting in fusion of adjacent caudal thoracic vertebrae (cranial is to left). Another (contralateral) site of vertebral articular fusion was present in this horse, which had displayed chronic back stiffness in training but raced successfully.

Causes
- Stress/fatigue injury.
- Some degeneration of facet joints probably occurs with age/training but progression poorly understood.

Risk factors
- Prevalence in racehorse population not known but appears to increase with age.
- Risk factors unknown but conformation, training and concurrent orthopaedic disease likely to play role in development.

History
- May present with history of suspected back pain, loss of action (shortened stride/tracking off line), loss of performance or reluctance to train.
- May be encountered when imaging the back, in isolation or along with other abnormalities.
- May be subclinical.

Signs
- As with other potential sources of back pain, clinical findings not specific or definitive.
- When clinically active: may display back stiffness, restricted gait at walk/trot, tracking off line at trot, mild hindlimb lameness, reduced thoracolumbar epaxial muscle bulk and focal muscle spasm.
- Rarely, encountered as catastrophic injury during racing: manifests as a fall, or sudden loss of hindlimb support and subsequent failure to rise.

Diagnosis
- Bone scan: focal IRU (mild to marked intensity depending on activity and soft-tissue attenuation) associated with one or more vertebral lamina/body/APJ (Figure 7.25a–d).
- Ultrasonography (transcutaneous): modelling/callus formation on dorsal aspect of transverse process (Figure 7.26), or modelling/enlargement of facet joints with loss of definition of the joint margins and variable amount of callus formation depending on aetiology (stress injury or degenerative arthropathy). Mild 'rounding' of the joint contour may be the only sign in the latter. Essential to compare left with right and with neighbouring joints.
- Radiography: frequently unrewarding due to superimposition of ribs and depth of tissues in lower back (high-quality radiographs required).

Management
- Dependent on severity of clinical/imaging findings.
- Suspected stress fracture (without neurological signs): rest (stable rest/walking for 8–12 weeks) +/– anti-inflammatory medication.
- Complete fracture: euthanasia.
- Facet joint arthropathy (mild signs/subclinical injury): continuation of training +/– ultrasound-guided intra-articular medication (corticosteroid) usually feasible with awareness that any deterioration may warrant rest period.

Prognosis
- Stress fracture: in all cases other than (rare) catastrophic/complete fracture (with peracute paraplegia) prognosis is good; usually settle with rest and recurrence rare. Complete fracture: hopeless prognosis.
- Facet joint arthropathy: guarded. Recurrence of clinical signs is common; may cause persistent poor performance

Rib fracture

Rib fractures are an uncommon and sporadic cause of poor performance or lameness. Usually involves a single rib but can be multiple; fractures may be complete or incomplete. The 1st rib is frequently affected, with injuries occurring at/near the costochondral junction or higher in the shaft. Fractures also occur further back in the ribcage (of these, 18th rib is most common).

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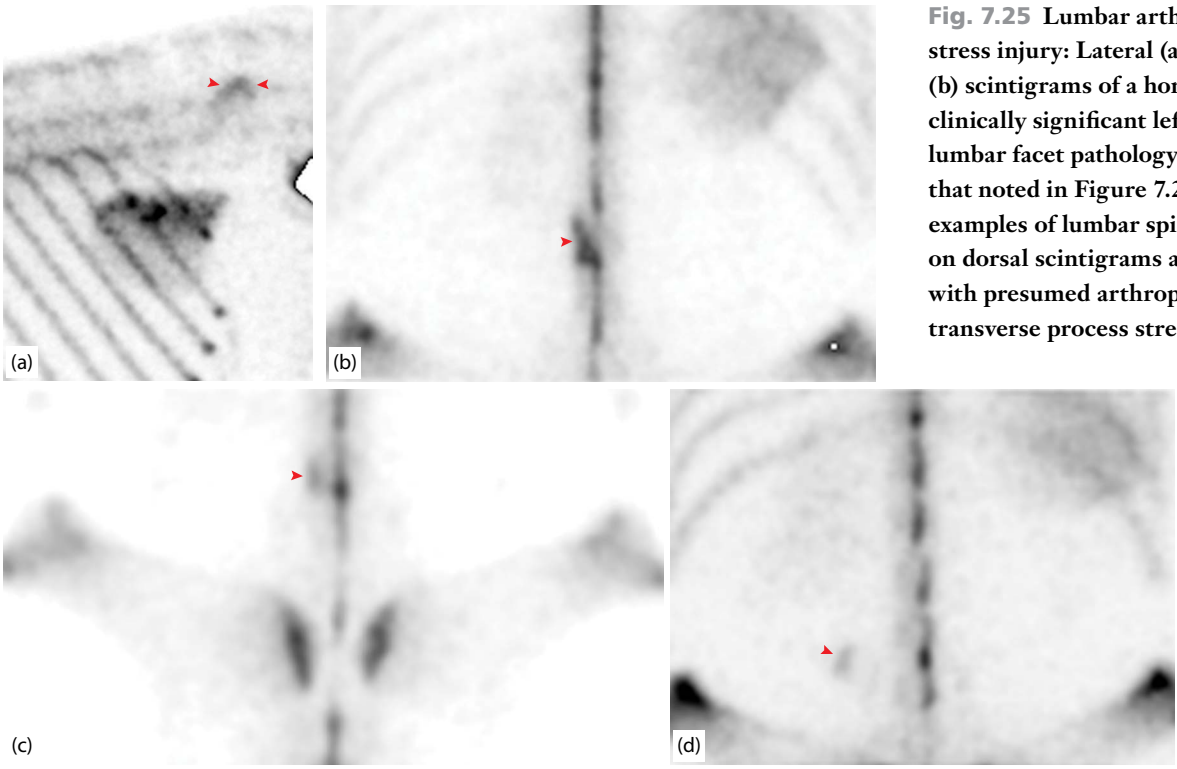


Fig. 7.25 **Lumbar arthropathy/stress injury: Lateral (a) and dorsal (b) scintigrams of a horse with clinically significant left-sided lumbar facet pathology similar to that noted in Figure 7.24; further examples of lumbar spinal IRU on dorsal scintigrams associated with presumed arthropathy (c) and transverse process stress injury (d).**

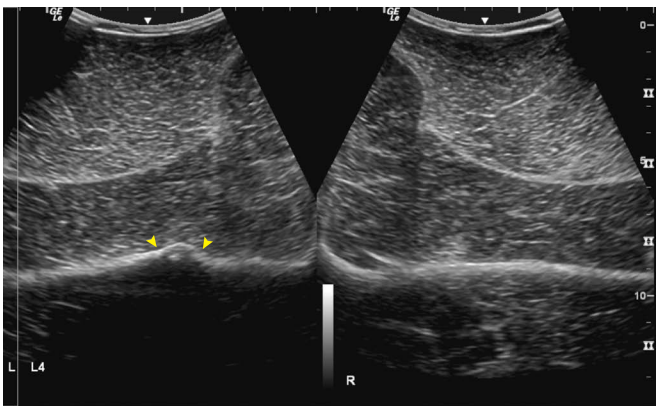


Fig. 7.26 **Lumbar transverse process stress fracture: Transcutaneous transverse ultrasonograms of injured (left) and normal (right) transverse process of L4, with cortical disruption/callus on dorsal aspect of bone noted.**

Cause

- Fractures involving the exposed mid- and caudal ribs of the trunk are usually attributed to direct trauma (e.g., following a fall).
- First rib is well protected by muscles of upper forelimb/chest; the underlying mechanism of injury at this site is not known but likely to be repetitive (stress fracture) or acute overload (single incident at exercise) by muscular forces acting on ribcage.

Risk factors

- Rare condition.
- Usually in horses in fast exercise/racing phase of training.
- Underlying risk factors poorly understood; gait characteristics or incidents at high speed (interference) may contribute.

History

- First rib fractures: typically present as insidious forelimb lameness that worsens over several days, but may be acute/severe in onset. May follow incident during fast exercise/racing.
- Mid- to caudal rib fractures: history of poor performance/poor action under tack/anxiety when girth tightened/reluctance to train. Delay between incident and onset of clinical signs may obscure exact origin of injury.

Signs

- First rib fractures: unilateral forelimb lameness of moderate severity, with abduction of limb and reduced cranial phase of gait at walk. Forced extension of limb may be resented. Frequently with guarding/spasm of muscles of shoulder girdle (especially triceps) on affected side. Neck lateroflexion may be resented/restricted.

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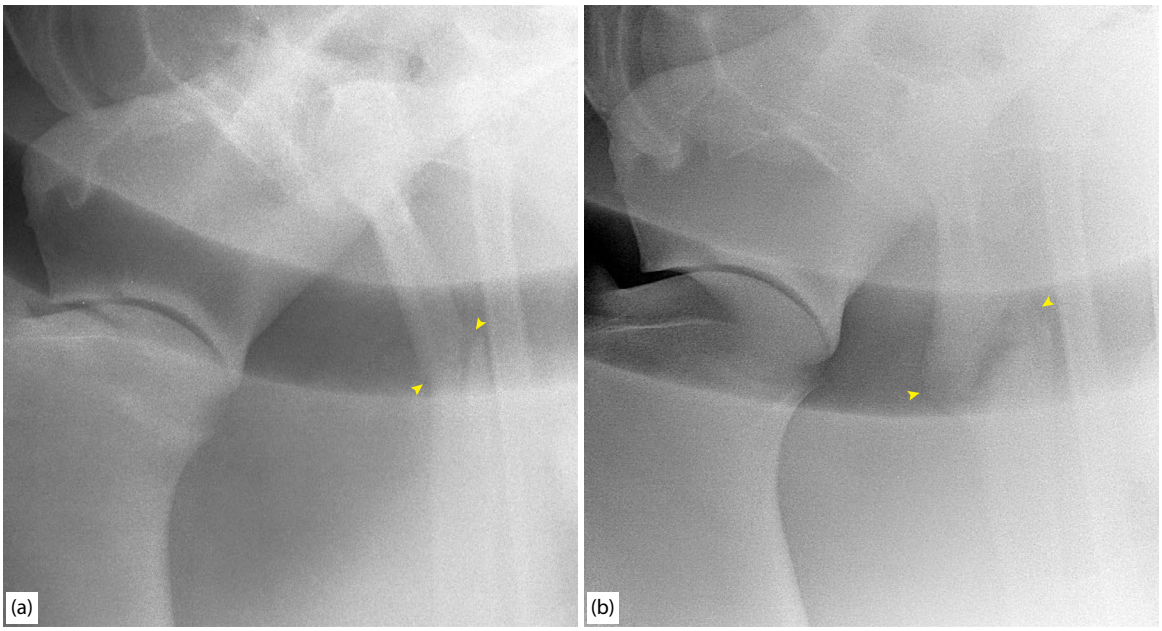


Fig. 7.27 **Rib fracture: Radiological (ML) appearance of displaced 1st rib fracture at acute stage (a) and 4-month follow-up (b) showing abundant callus/non-union that is typical of the injury.**

- Mid- to caudal rib fractures: +/– localized pain on palpation but frequently without obvious sign of injury; bone callus occasionally palpable/visible.

Diagnosis

- Radiography: cranial ribs difficult to image but complete 1st rib fractures usually visible on mediolateral shoulder projection (Figure 7.27a, b).
- Ultrasonography: useful to confirm fracture (discontinuity of bone margin) when palpable abnormality of mid- to caudal ribs is present (Figure 7.28). Sensitivity for fractures in mid- to caudal thorax is superior than radiography.
- Bone scan: most useful imaging modality (Figure 7.29a–c), although not all cases show increased activity in acute phase. IRU from cranial rib fractures may be masked by upper forelimb, and non-standard views may be required (Figure 7.29d).

Management

- Conservative management: stable rest (4–8 weeks)/walking (4–6 weeks).
- Radiographic monitoring useful to guide management although partial or non-union is a common outcome and should not preclude return to training if clinical progress is satisfactory.

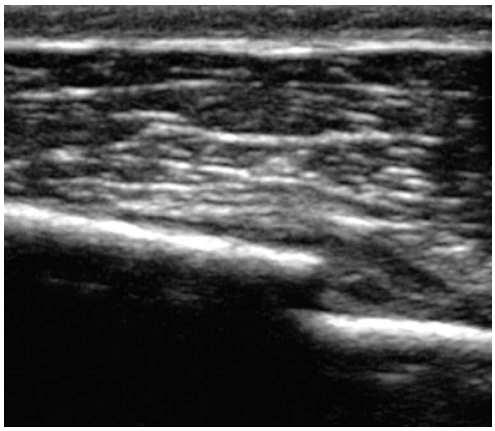


Fig. 7.28 **Rib fracture: Longitudinal ultrasonogram of caudal thoracic rib fracture showing discontinuity of bone margin.**

Prognosis

- Good prognosis for return to full use.
- Non-union with secondary nerve palsy may follow 1st rib fractures in people but has not been reported in horse.

Fractured withers

The prominence of the vertebral DSPs of the withers makes them susceptible to blunt injury. Fracture typically involves the dorsal third of several (≥3) adjacent DSPs but can be solitary, most commonly from T5–T7 (highest

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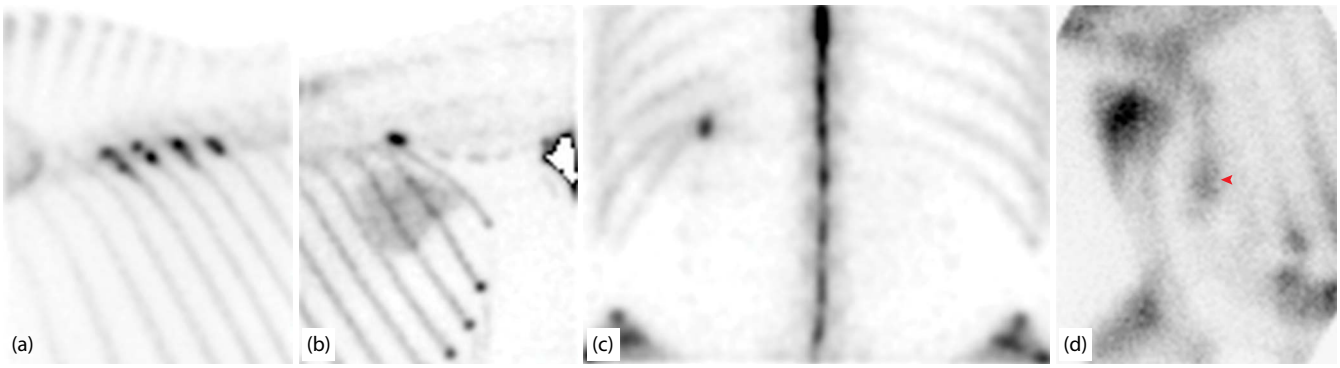


Fig. 7.29 Rib fracture. Examples of scintigrams of rib injuries at different sites: Multiple proximal mid-thoracic (a); displaced proximal caudal rib from lateral (b) and dorsal (c) views; lateral (with ipsilateral leg protracted) scintigram of 1st rib (d).

point of the withers). Complete fracture with marked displacement +/– vertical impaction of fracture fragments is typical. Rarely, open fractures may occur.

Cause
- Injury arises from direct trauma: fall/rearing over.

Risk factors
- Any age/stage of training.

History
- Invariably associated with recent history of falling/rearing over.
- Injury may not be immediately apparent.
- Stiffness of gait and/or resentment of saddle may prompt examination.

Signs
- Stiff/shuffling forelimb gait at walk.
- Withers usually flattened in profile with shoulder blades the highest point.
- Dorsal spinous summits may be palpably out of line or absent.
- 'Guarding' of withers, hypersensitivity of local skin and pain on palpation are characteristic (may resemble infection/abscess).

Diagnosis
- Clinical findings usually definitive.
- Radiography: (LL projection) not necessary for diagnosis but sometimes useful to determine severity (Figure 7.30). Fracture fragments should not be confused with separate centres of ossification associated with summits of T2–T7/8.

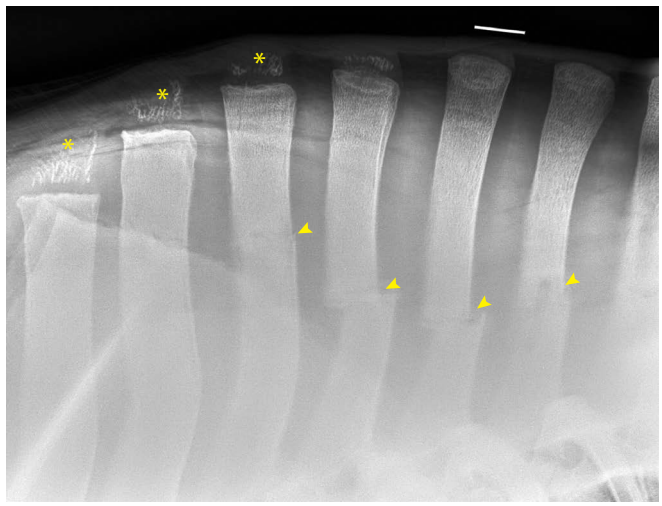


Fig. 7.30 Fractured withers: Complete fracture (arrowheads) of several consecutive thoracic DSPs; separate centres of ossification (stars) are also present but clinically irrelevant.

Management
- Anti-inflammatory medication during acute phase.
- Conservative management: varies between individuals but 2–4 weeks stable rest/6–8 weeks walking is typical. Shorter rehabilitation periods possible when initial clinical severity is mild.
- Management guided by resolution of palpable pain/lameness.
- Open fractures treated with debridement and antibiotic therapy.

Prognosis
- Excellent prognosis for return to full racing function.
- Return to fast ridden exercise/racing typically within 4–5 months.

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- Permanent flattening or residual asymmetry of withers can be expected; non-union/fibrous union of fractures is usual outcome.
- Resulting deformity/asymmetry of withers may necessitate use of fitted saddle or pad (to minimize saddle rubs) in small proportion of cases.
- Once site has settled, has no impact on soundness or trainability.

Injection abscess/reaction

Intramuscular (IM) injections occasionally result in septic abscessation of affected side of neck. Non-septic inflammation can also occur (most commonly following vaccination) and is typically transient.

Risk factors

- Use of non-sterile medications or needle/syringe most common cause of infection.
- No apparent link with injection site disinfection.
- Some horses appear susceptible to vaccine-specific non-septic adverse reactions.

History

- Neck stiffness that develops in days following IM injection.

Signs

- Stiffness/thickening/hypersensitivity/guarding of neck.
- Reluctance to lower head to eat.
- +/– subcutaneous oedema at injection site.
- Infection: affected site in neck progressively larger/more painful over several days and may 'point' into abscess.

Diagnosis

- Ultrasonography: in the case of infection, IM fluid cavity develops over course of several days; readily identified as anechoic pocket and measured depth useful to assist treatment.

Management

- Non-septic inflammatory (adverse drug) reaction: anti-inflammatory medication usually results in remission of signs in 1–2 days.
- Infection/abscessation: anti-inflammatory medication to manage clinical signs until abscess ready to lance; followed by open drainage or catheter lavage (sterile saline) + systemic antibiotic medication.
- If infection suspected but little ultrasonographic evidence of fluid accumulation, systemic broad-spectrum antibiotic medication may be warranted in early stages to prevent abscess formation.

Prognosis

- Excellent prognosis but may interfere with training for short period.

Poll injuries

Injuries to the poll are sustained by rearing over or throwing head up and catching doorway/ceiling. Most injuries are simple lacerations immediately behind the poll (nuchal crest). Full-thickness wounds may be deep and fragmentation of nuchal crest may occur.

Tension/torsion/rotation injury due to pulling back from chain/tether may cause avulsion of the semispinalis capitis tendon +/– nuchal ligament injury. Less commonly, momentum of fall and hyperextension of neck may cause muscle avulsion injury to the base of the cranium (rectus capitis/longus capitis muscles). This is a more severe injury, with resulting guttural pouch/retropharyngeal haemorrhage, neurological signs and high mortality.

Cause

- Direct or dynamic trauma to poll region.

Risk factor

- Any age/stage of training.

Signs

- Simple poll wound: may resent examination. Small transverse laceration (Figure 7.31); anatomical dead space ensures wound usually deep. Reluctance to raise neck/head immediately after injury. Neurological signs not a feature. May develop head shyness due to local pain.
- Skull base fracture: profuse bilateral epistaxis +/– neurological deficits/collapse +/– respiratory distress.

Diagnosis

- Uncomplicated poll injuries require no diagnostic imaging.
- Suspected skull base injuries: radiography (LL projection for detection of avulsion fragment) +/– endoscopy of guttural pouches. CT is imaging modality of choice but has practical limitations for acutely ataxic horse.

Management

- Simple poll injuries: wound management (clip/lavage) +/– antibiotic/anti-inflammatory medication.

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Fig. 7.31 Poll injury: Typical full-thickness skin wound associated with direct trauma.

- Failure of poll wound to heal may indicate nuchal crest fragmentation/sequestrum: surgical removal or prolonged antibiotic medication.
- If neurological signs or suspected skull base fracture, hospitalization. Care with handling to minimize risk to personnel.

Prognosis

- Simple poll injuries: rarely interfere with training beyond acute phase (may preclude bridle use for several days).
- Skull base fractures: risk of mortality or serious neurological complications is high.

Saddle sores

Bruising, rubs, dermatitis or infection secondary to pressure from tack may occur at any site from the withers to the mid-back; usually found on midline, although sores/seromas may develop on either side of the withers.

Causes

- Peak pressures on back are greatest at back-of-saddle region when trotting and at withers during cantering and galloping.
- Treeless saddles tend to put pressure on mid-saddle region, while full-tree saddles impact further back.

Risk factors

- Poor fitting or unhygienic tack.
- New rider.
- Prominence/shape of topline may predispose to tack pressure: horses with high withers +/– light thoracic epaxial muscling appear to be more prone.

Signs

- Focal dermatitis, subcutaneous bursa (seroma) +/– surrounding oedema in saddle region (Figure 7.32a,b).
- +/– pain on palpation.
- +/– open wound/broken skin/exudative dermatitis/subcutaneous abscess (Figure 7.32c,d).

Diagnosis

- Clinical findings definitive.
- Non-infected bruising/seroma: relatively painless on palpation.
- Infection associated with presence of broken skin +/– intense pain +/– lymphatic 'runners' (Figure 7.32e).

Management

- Management determined by severity/location/presence of infection/presence of broken skin.
- Infection/broken skin: topical +/– systemic antibiotic medication (typically 3–5 days).
- Bruised/inflamed without infection: topical emollient or anti-inflammatory ointment.
- Avoid further pressure at site from tack in short term; rest from ridden exercise (typically few days) may permit most rapid resolution.
- Fitted saddle pad, or pad with cut-out hole over site may be useful (latter more suitable for open wounds, but prone to slipping).
- Focal seroma: if large/fluctuant consider intra-lesional corticosteroid, although does not resolve underlying/predisposing cause.
- Muscular bruising: daily application of hot packs useful.

Prognosis

- Interference with training usually minimal (days).
- Recurrence uncommon, although some horses appear predisposed or may develop semi-permanent seroma.

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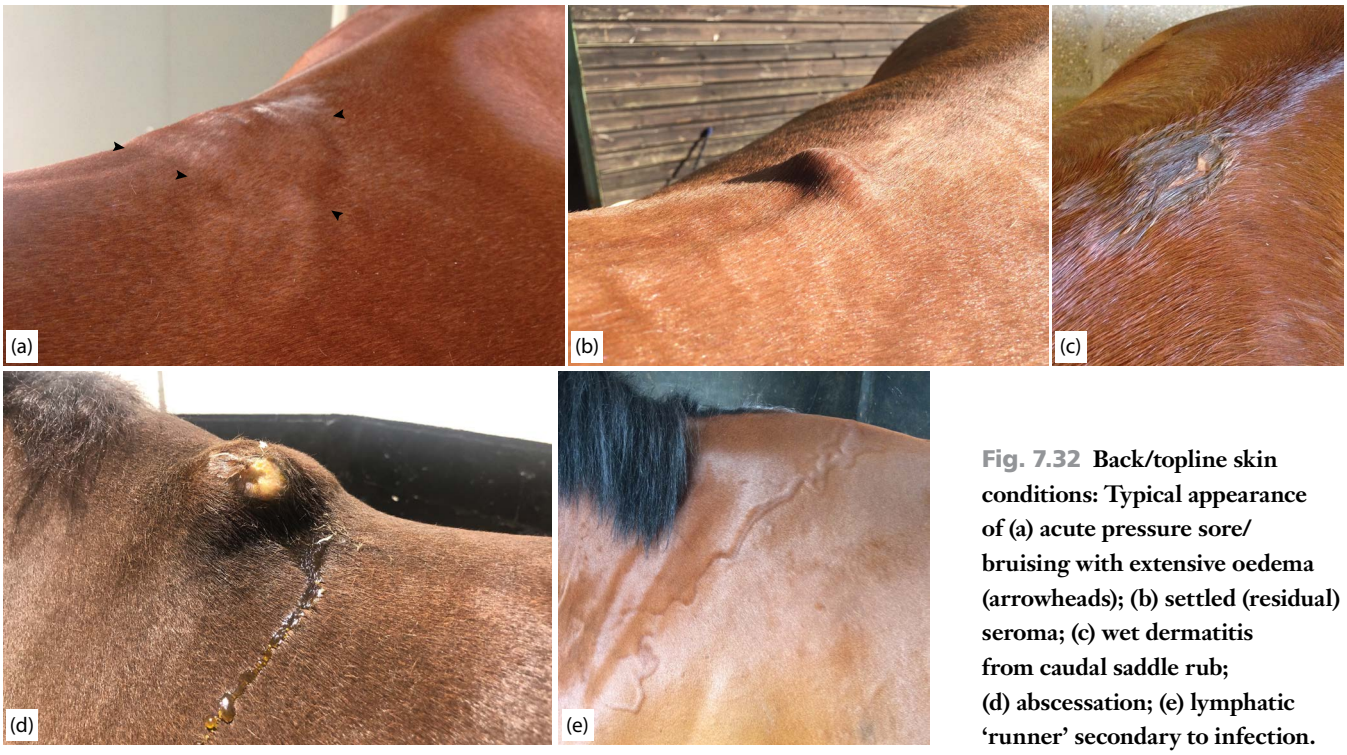


Fig. 7.32 **Back/topline skin conditions: Typical appearance of (a) acute pressure sore/bruising with extensive oedema (arrowheads); (b) settled (residual) seroma; (c) wet dermatitis from caudal saddle rub; (d) abscessation; (e) lymphatic 'runner' secondary to infection.**

OTHER MUSCULOSKELETAL CONDITIONS

Exertional rhabdomyolysis ('setfast'/'tying-up')

Episodic muscle disorder characterised by stiffness, muscle cramping and discomfort during or following exercise. Predominantly affects the hindlimbs but occasionally may involve muscles of the forelimb or flank.

Causes

- Most individuals affected by recurrent episodes have an underlying intrinsic abnormality of skeletal muscle related to contraction/relaxation (disruption of normal calcium regulation/transport in muscle cell) or muscle cell energy metabolism. Further exposure to risk factors relating to exercise, diet and temperament may then trigger clinical episode in susceptible horses.
- Moderately heritable: likely to be genetically complex and mechanism of heritability has not been fully defined. Affected genes appear to be those involved in energy metabolism and intracellular calcium regulation of skeletal muscle. Possible that selective breeding for race performance has led to trait becoming commonplace.
- Unrelated to raised lactic acid levels.
- When horses experience 'one-off' (rather than recurrent) episodes, these are likely to be idiopathic and related primarily to management factors, rather than underlying intrinsic/genetic muscle disorder.

Risk factors

- Common; affects approximately 5% of horses in training.
- Susceptibility to condition may be inherited, but likelihood, frequency and severity of episodes varies between individuals due to influence of other factors such as sex, temperament, exercise, fitness and diet.
- Fillies, 2 YOs and horses with nervous/excitable temperament at greater risk.
- Most episodes follow simple management errors that expose susceptible horses to a risk factor (e.g., over supply of energy ration relative to expenditure/rapid step up in exercise/stress).
- Recurrently affected horses likely to be those with greater genetic expression of muscle abnormality and for whom threshold for clinical disease is lower.
- No direct link with oestrus in fillies but possibly a factor in some individuals.
- Male horses appear more susceptible to the rarer forelimb expression of the condition.
- Diet under fortified with potassium is a recognised risk factor: typically seen with inadequate forage intake.

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History
- Episode occurs during/after training.
- Horse usually trains without incident but gait stiffens while walking in.
- Can follow training at any pace (walk/trot/canter/gallop).
- Typically occurs when exercise is being stepped up following a period of relative inactivity (i.e., start of week, or following rest period for lameness/illness/weather).
- Episodes during racing are rare: horse may shorten in final stages of race or develop stiffness post-race.

Signs
- Typical presentation is restricted/stiff hindlimb gait (bilateral) at walk or reluctance to move, but trot freely and without lameness.
- Tightness/cramping of gluteal +/– caudal thigh musculature +/– muscle fasciculations a variable feature and not always obvious.
- Moderately/severely affected horses may sweat.
- May mimic colic (scraping floor/flank-watching); however, recumbency is rare and horses typically stand over pile of scraped-up bedding (rather than moving around stable) (Figure 7.33).
- Rarely may affect forelimb(s): restricted protraction of limb (sometimes profound) at walk, although lameness typically absent at trot. Mild-moderate lameness occasionally at both walk and trot but not typical.

Diagnosis
- Clinical presentation usually definitive.



Fig. 7.33 Exertional rhabdomyolysis: Typical post-exercise behaviour of scraping up bedding without displaying other signs associated with colic.

- Blood analysis: elevations of muscle-derived enzymes CK and AST confirm diagnosis, severity and timing of episode.
- CK: 'acute' enzyme and peaks at 4–6 hours after episode, dropping to normal levels by 3–4 days.
- AST: peaks later at 1–2 days, and takes longer to return to baseline depending on severity of episode.
- CK higher than AST usually means episode occurred on day of sampling.
- Subclinical episodes occur and most likely to be associated with acute CK levels of 1,000–2,000 IU/l.
- Muscle biopsy of little practical use for either diagnosis or management.

Management: Acute care
- Most episodes simply require management of acute pain: single dose of NSAID (+/– acepromazine if distressed). Forced walking not recommended.
- Severe episodes (very rare) with recumbency: intensive support with IV isotonic fluids and diuretics to limit renal damage from myoglobinuria.
- Rest period following episode should be avoided in all but severe cases.
- No necessity to allow muscle enzyme levels to normalize before return to exercise: rest from ridden exercise simply prolongs risk period for further episodes.
- Resumption of normal training on day(s) following episode aided by prophylactic use of skeletal muscle relaxant dantrolene sodium (see below).
- Episode of mild-moderate severity does not preclude racing in short term; horses may perform satisfactorily with muscle enzymes (CK and/or AST) above normal reference ranges.
- 'Fitness to race' best determined by clinical severity of episode rather than absolute blood muscle enzyme levels.

Prevention
- Avoidance of 'no exercise' days (affected horse should do some exercise every day of the week: level of exercise required determined on individual basis).
- Use of horse-walker exercise +/– daily turnout in addition to regular ridden work.
- May benefit from extended 'warm-up' (walk/trot) prior to regular exercise.
- Prophylactic use of dantrolene sodium for susceptible horses on 'high-risk' days such as following period of relative inactivity
- Maintenance on low-dose dantrolene sodium throughout training may be necessary for severely

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affected horses; successful maintenance level varies between individuals and determined by trial and error.
- Oral dantrolene sodium highly effective as prophylactic: it interferes with excitation-contraction coupling of muscle cells by blocking release of calcium. Given 45 minutes to 4 hours pre-exercise (blood levels peak at approximately 4 hours): initial treatment 1.5–2 mg/kg; tapering of dose over subsequent days as normal exercise resumed, with increased dose on subsequent higher risk days. Outline treatment schedule shown in Table 7.2. Period for which prophylaxis required, and tapering/short-term maintenance dose varies between individuals and determined by clinical response/relapses; few horses require treatment beyond initial days/weeks.
- +/– prophylactic use of low-dose acepromazine in excitable horses.
- Dietary management: partial replacement of carbohydrate with high oil/fat +/– digestible fibre (sugar beet pulp, soya hulls) may be beneficial, although wholesale replacement of starch in diet is usually incompatible with maintenance of satisfactory body condition in full work. Utilising dietary changes alone (i.e., without exercise/dantrolene sodium) to manage the condition is usually unrewarding.
- High-fat (15–20% of daily digestible energy intake) diet well tolerated and may act by reducing excitability.
- Assessment of diet to ensure adequate potassium intake: 1.5–2% bodyweight (BWT)/day good-quality forage advisable.
- Dietary changes should follow (rather than precede) any anticipated increase in workload.
- Some empirical evidence to support use of branched-chain amino acid +/or beta-alanine supplementation.
- Little/no benefit of supplementation with sodium bicarbonate or vitamin E/selenium.

Prognosis
- Majority of cases easily managed with greater attention to feeding and exercise.
- Additional prophylactic use of dantrolene sodium +/– acepromazine permits training of more troublesome cases.
- Effect on racing performance thought to be minimal in most cases.
- Many fillies affected at 2 YO show reduced susceptibility later in career.
- Very small minority of affected horses fail to respond satisfactorily when managed as previously mentioned and retirement considered if racing performance repeatedly affected.

Osteochondrosis (overview)
Osteochondrosis is a collective term for developmental defects of cartilage/subchondral bone: includes subchondral bone cysts ('osseous cyst-like lesions') and osteochondritis dissecans (OCD). The majority of subchondral bone cysts and OCD lesions found in the young racehorse are present from an early age (generally from foal). Defective differentiation of articular cartilage can lead to cartilage or cartilage/bone flaps (OCD), which may become detached, or cystic lesions in subchondral bone. Lesions can also develop following trauma to cartilage or subchondral bone that are pathologically distinct from osteochondrosis but manifest in similar ways.

Table 7.2 **Exertional rhabdomyolysis: Example prophylactic dantrolene sodium dosing regimen**

	WEEK 1	WEEK 2†	WEEK 3†
Monday	600–800 mg	300–400 mg	200 mg
Tuesday	400 mg	200 mg	100 mg
Wednesday	200 mg	100 mg	-§
Thursday	200 mg	100 mg	-
Friday	200 mg	100 mg	-
Saturday	200 mg (or 400 mg*)	100mg (or 200 mg*)	-
Sunday	(200 mg if exercised)	-	-

* Higher dose used if greater exercise intensity intended.

† Taper over successive weeks dependent on clinical progress.

§ Total cessation (or intermittent continuation) of prophylaxis dependent on clinical/biochemistry evidence of relapse(s).

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Osteochondrosis is a dynamic condition and particularly in the case of OCD, lesions may develop then heal early in life with little external sign of joint disease. Lesions that remain after 12 months of age are generally thought to be permanent/semi-permanent. Subchondral bone cysts typically remain visible radiologically long term, although size and appearance can also vary over time: progressive enlargement when it occurs is the result of inflammation within the cyst cavity. Lameness, if present, may result from synovitis or, in the case of subchondral bone cysts, increased intracystic or intraosseous pressures.

Both OCD and subchondral bone cysts have certain predilection sites within joints, with cysts in particular often being found in association with high load-bearing joint surfaces. Predilection sites are:

- OCD: most common sites are in fetlock (sagittal ridge), hock (distal intermediate ridge and medial malleolus) and stifle (lateral trochlear ridge).
- Subchondral bone cyst: most common sites are stifle (medial femoral condyle) and carpus (ulnar carpal bone) but also encountered sporadically at many other sites including fetlock, pastern, foot, elbow, shoulder and other carpal bones.

Causes

- Multifactorial: nutritional, exercise and conformation factors can act in windows of susceptibility (foal/yearling) to cause development of focal areas of abnormal cartilage at predilection sites.
- Dam (age, parity) and stud farm management factors (maternal nutrition during gestation) may have a role in prevalence of some OCD lesions; however, further investigation of specific influences required.
- Heritability: genetic influence varies between lesion sites, but overall heritability in the Thoroughbred considered to be low.
- Microtrauma may play a role in the development of some bone cysts (e.g., stifle/medial femoral condyle); aetiology differs from osteochondrosis, but similarities of presentation and outcome permit them to be considered together.

Risk factors

- Growth rate and body size: high-energy (not high-protein) diets, fast growth rates and nutritional imbalances or deficiencies of some trace elements are risk factors. Feeds with high glycaemic index may be implicated.
- Mechanical loading of joints: restricted exercise at early age increases risk of osteochondrosis (normal paddock exercise as foal important for appropriate cartilage development).

History

- Frequently first detected at routine yearling radiography.
- May be incidental finding or clinically active (joint effusion +/– lameness).

Signs

- Clinical findings vary widely with lesion type and location.
- Many lesions are subclinical.
- Generally considered that subchondral bone cysts beneath high load-bearing joint surfaces are more likely to display lameness.

Diagnosis

- Radiography: most OCD and subchondral bone cyst lesions are visible radiologically. Small lesions may not be obvious and secondary arthritic change or alternative imaging may be required for diagnosis.
- Ultrasonography: useful for detection of radiologically silent lesions in hock and stifle, or for better characterisation of extent/depth of radiologically evident lesions.
- MRI/CT: useful if radiological findings are equivocal. Imaging modalities of choice for detection and assessment of lesions in DIPJ and PIPJ.
- Bone scan: generally poor sensitivity. Of little use for most OCD lesions, and activity of subchondral bone cysts is highly variable.

Management

- Management dependent on lesion type, location, appearance and clinical relevance.
- Many lesions cause no/few problems in training.
- Primary goal of any intervention is maintenance of acceptable degree of soundness in training. Intervention to achieve improved radiological appearance solely for perceived effect on sale value (rather than for athletic soundness) is commonplace but likely to be subject to increasing ethical scrutiny in future.
- OCD: surgical debridement (if required) best undertaken at yearling stage for minimal interruption to training.

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- Subchondral bone cysts: treatments aim to limit growth or diminish size of lesion and promote healing of associated cartilage defect to improve integrity of joint surface. Intra-lesional medication, surgical implant (transcortical screw or bioabsorbable implant) or surgical debridement +/– bone or cell grafting are possible treatment paths. Bisphosphonate therapy in developmental phase may have some merit, although evidence remains anecdotal and use in juveniles subject to regulatory sanction in many jurisdictions.

Prognosis

- OCD: fetlock and hock lesions rarely interfere with training. Stifle/elbow/shoulder lesions: vary with location and appearance.
- Subchondral bone cysts: risk of developing lameness is generally low (regardless of location) but currently largely unquantifiable, and when clinically active may be career threatening (see also Chapter 18). Aside from stifle lesions little objective information available on prognosis for racing or likelihood of remaining sound in training.

Peritarsal/inguinal infection (lymphangitis/cellulitis)

Acute and painful infection of hindlimb. Typically affects front of hock and/or inner thigh/groin region. Generally of unknown origin but presumed to seed from site in lower limb with proximal progression through lymphatic system. Associated with acute severe lameness that can be mistaken initially for injury or synovial infection.

Causes

- Infection arises in lower leg, often from small/long-standing skin lesion.
- Spread of infection presumed to occur through lymphatic system, with initial localized inflammation commonly at front of hock or inner thigh/groin.
- Severity of pain/lameness in cases of peritarsal infection likely to be due to inflammation in anatomical region tightly constrained by retinacula.
- Causative bacteria typically opportunistic pathogens, most commonly *Staphylococcus aureus*.

Risk factors

- Uncommon condition.
- Any age/stage of training.

History

- Acute onset and progression (hours).
- Usually found lame in stable; no association with exercise.

Signs

- Severe unilateral hindlimb lameness.
- Lameness improves with forced walking (not the case if orthopaedic injury or synovial sepsis are responsible).
- +/– mild fever in acute phase.
- Profound pain response to palpation of front of hock or groin: may cause marked flexion/abduction of leg.
- +/– raised lymphatic vessel or ridge/plaque of oedematous filling on dorsal hock or inner thigh (Figure 7.34a,b).



Fig. 7.34 Peritarsal/inguinal infection: (a) swollen dorsomedial aspect of hock; (b) raised oedematous thickening on medial aspect of thigh (with vascular distension).

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- +/– secondary effusion of tibiotarsal joint: may resemble synovial infection.
- Typically no generalized filling/cellulitis of leg in peracute phase but may progressively develop within 1–2 days, particularly if initial treatment has been delayed/inadequate/ineffective.

Diagnosis
- Clinical findings usually definitive.
- Blood analysis: rarely needed, however, inflammatory response permits differentiation from orthopaedic injury.
- Can usually be differentiated from synovial sepsis (tibiotarsal joint) clinically through presence of peri-articular inflammation and dependent oedema in lower limb. Synoviocentesis should be undertaken only when strong clinical suspicion exists that presentation is not consistent with peritarsal infection, and with caution that procedure carries a risk of inoculating infection into joint when none exists.

Management
- Antibiotic and anti-inflammatory medication.
- Forced exercise is essential to recovery and limits filling of leg: delay in commencing exercise is associated with risk of substantial lymphatic compromise and longer course of disease/poorer outcome.
- Level of exercise may be limited by lameness severity: in acute phase (initial 1–3 days) hand-walking/horse-walker exercise several times daily; as soon as comfortable at walk should commence trotting exercise. Return to cantering usually possible within 2–5 days.

Prognosis
- Excellent.
- Early diagnosis and prompt, aggressive management usually results in minimal interruption to training.
- Rarely (with delayed or inappropriate management): semi-permanent thickening of limb (disrupted lymphatic drainage), focal abscessation or extension of infection to associated splint bones/synovial structures (hospitalization).

Enostosis-like lesions ('bone islands')
Unusual condition in which focal proliferation of bone occurs within the medullary cavity of one or more long bones, with characteristic location in the vicinity of nutrient vessel foramen. Lesions occur most commonly in the tibia, radius and cannon, and less frequently in other long bones including the femur and humerus. Forelimbs and hindlimbs affected. Many cases have concurrent lesions in 1–3 bones. Lameness is a variable feature and in many cases the lesions are clinically silent.

Causes
- Cause unknown.
- Location of intramedullary lesions in close proximity to nutrient foramen suggests possible link with raised intramedullary pressure or vascular occlusion.

Risk factors
- Rare condition.
- Risk factors unknown.

History
- In many cases lesions are unrelated to lameness and detected as incidental finding during radiographic/scintigraphic screening.
- Lameness when present is often intermittent/transient and of unusual character; may occur at/after exercise and diminish within minutes/hours.

Signs
- Affected limb palpably normal.
- May be clinically silent.
- +/– unilateral and frequently inconsistent lameness of moderate severity.

Diagnosis
- Diagnostic blocking: if located in cannon, blocking pattern may be similar to that for suspensory ligament desmopathy.
- Radiography: characteristic focal smooth-margined endosteal radiopacity in mid-diaphysis, generally opposite/adjacent nutrient foramen (Figure 7.35a–c). Multiple lesions common. Radiological definition more difficult for lesions in proximal limb (humerus/femur).
- Bone scan: focal IRU at lesion site (Figure 7.36a,b); may sometimes be difficult to distinguish from stress fracture. Possible correlation between IRU intensity and lameness severity.

Management
- Clinically silent lesions: no treatment necessary.
- Clinically active lesions: lameness generally resolves with rest (removal from ridden exercise for 6–8 weeks). Strong anecdotal support for bisphosphonate therapy in clinical cases; return to training dependent on abolition of lameness but typically 1–3 weeks.

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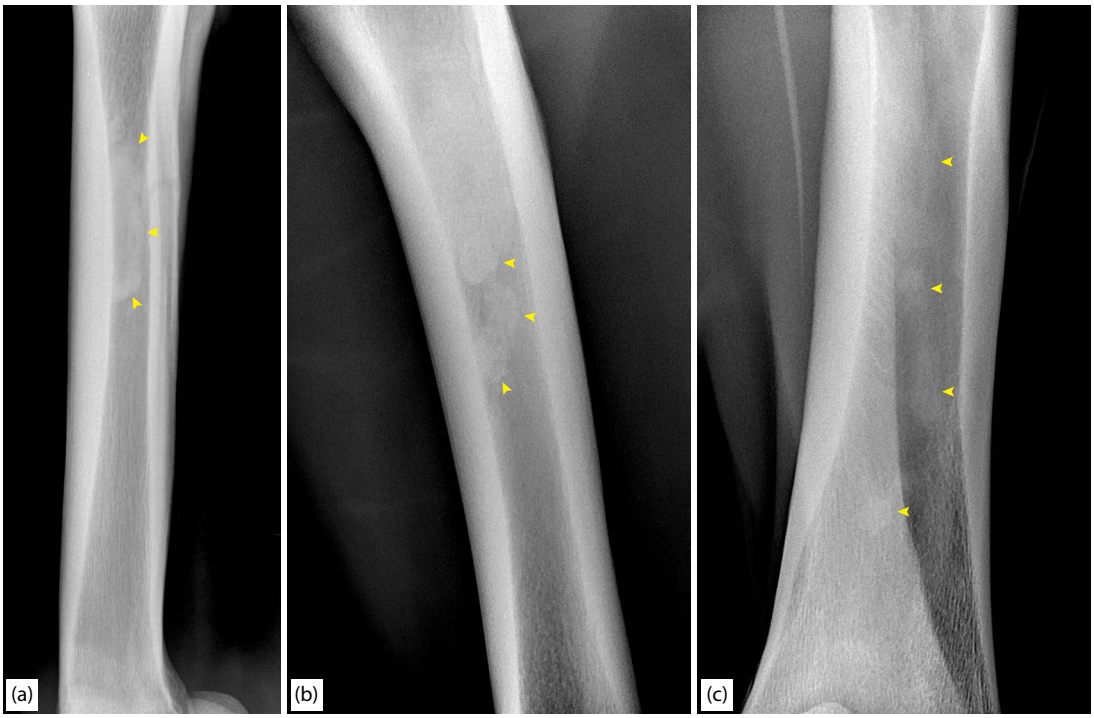


Fig. 7.35 **Enostosis-like lesion: Typical appearance of medullary/endocortical radiopacity (arrowheads) opposite nutrient foramen in mid-shaft of metacarpus (a); multifocal intra-medullary radiopacities on LM (b) and CdCr (c) projections of tibia.**

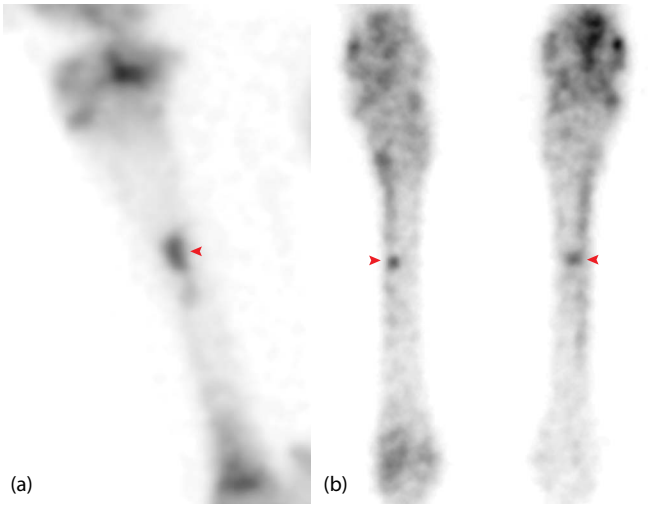


Fig. 7.36 **Enostosis-like lesion: Scintigrams of mid-diaphyseal lesions in tibia (a) and hind cannons (b) (latter bilateral).**

Prognosis

- Excellent prognosis for return to full use.
- Radiological/bone scan appearance returns to normal with time/rest.
- Recurrence (at same or different site) may occur but is rare. Very rarely, persistence of condition may interfere with racing career.

Haematoma

Haematomas usually develop at the back of the thigh but also over the pectoral muscles of chest, and are presumed to arise from tearing of a muscle belly or its fascial lining. Subsequent fluid accumulation forms a subcutaneous pouch; severity varies between individuals.

Causes

- Cause rarely determined.
- Most frequently arises from exercise or following a slip or fall.
- Less commonly: direct trauma (kick).

Risk factors

- Uncommon condition.
- Any age/stage of training.

History

- Acute onset, although may enlarge over initial few days.

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Fig. 7.37 Hindlimb haematoma: (a) ventral accumulation of fluid (arrowheads) typical of initial phase (note sweat patch above); (b) fully distended haematoma on lateral aspect of thigh.

Signs
- Unilateral fluid-filled, fluctuant swelling typically at back or side of thigh (Figure 7.37a,b).
- Size variable but frequently large.
- Generally no lameness.
- Usually painless to palpate (mild inflammation of haematoma edges in acute phase).

Diagnosis
- Clinical findings definitive.

Management
- Reduced exercise level (removal from cantering) for initial few days recommended to minimize final extent of swelling.
- Most small- to moderate-sized haematomas resolve completely with no intervention required.
- Moderate-large haematomas: may be lanced. Small (2–5 cm) vertical incision at lowest extent of swelling; wound management over subsequent 1–2 weeks to facilitate drainage +/– local or systemic antibiotic medication. No rest required.

Prognosis
- Excellent.
- Any interruption to training is usually brief.
- Heals with minimal or no cosmetic blemish.

Idiopathic recurrent haemarthrosis

Rare condition characterized by recurrent joint filling and lameness secondary to articular haemorrhage. Antebrachiocarpal and tibiotarsal joints are most commonly affected.

Cause
- Unknown.

Risk factors
- Rare condition.
- All ages/stages of training but more frequent in horses in fast exercise.
- Risk factors unknown.

History
- Acute initial onset.
- Intermittent recurrence despite rest/medication.

Signs
- Moderate-marked unilateral joint effusion.
- +/– restricted or painful flexion of affected joint.
- Lameness usually a feature but severity varies.

Diagnosis
- Typically a diagnosis of elimination of other (more common) pathology.
- Frank haemorrhage within joint revealed by synoviocentesis.
- Radiography/scintigraphy/MRI/CT should be undertaken to rule out presence of fracture or osteochondral injury.

Management
- Marked improvement in joint filling/lameness usually results following initial synoviocentesis and systemic anti-inflammatory medication.

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- Short rest +/– intra-articular medication (corticosteroid) usually undertaken initially.
- Recurrent cases: surgery (diagnostic arthroscopy) in attempt to determine underlying lesion and permit synovectomy, followed by prolonged rehabilitation (2–6 months out of ridden exercise).
- Radiosynovectomy (yttrium-90) has also been described but efficacy uncertain.

Prognosis

- Guarded prognosis for return to full use.
- Unpredictable course of condition; recurrence is common (approximately 50% cases) regardless of management.
- Retirement may be necessary in severely affected individuals that are non-responsive to treatment.

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OTHER BODY SYSTEMS

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UPPER RESPIRATORY CONDITIONS

UPPER AIRWAY OBSTRUCTIONS

Applied anatomy

Nasal passages

The nostrils are large, mobile and dilated by muscular action during exercise. The left and right nasal passages are separated by the nasal septum and partitioned further on each side by scrolls of thin bone lined by vascular mucosa (dorsal and ventral nasal conchae). The air passages around these conchae communicate and converge in the pharynx. The lowermost passage (ventral nasal meatus) is the largest and the one commonly used during routine endoscopy and nasogastric intubation.

Larynx

The larynx (Figure 8.1) is made up of several cartilages (epiglottis, cricoid, thyroid and paired arytenoids) linked by joints and moved by intrinsic and extrinsic muscles. It functions to both protect the airway from aspiration of food during swallowing and facilitate unobstructed passage of air into trachea and lungs during breathing. The key muscle relevant to laryngeal function during exercise is the cricoarytenoideus dorsalis (CAD), which opens (abducts) the arytenoid cartilages and is the major dilating muscle of the laryngeal opening. The epiglottis is a prominent triangular-shaped cartilage; size and relative stiffness/flaccidity can vary between individuals. Its only muscular attachment is the hypoepiglotticus, which serves to tense the epiglottis down onto the soft palate. On each side of the epiglottis, a fold of mucosa confluent with the corniculate process of the arytenoid cartilage forms the aryepiglottic fold. Within the opening of the larynx, connecting the arytenoids to the thyroid cartilage, are the vocal folds, which border blind-ended lateral ventricles.

Pharynx

Between the rigid nasal passages and the semi-rigid trachea, the airway traverses the muscular tube of the pharynx. The pharynx is the intersection between the respiratory and digestive tracts and must perform diverse functions. To accommodate swallowing it must constrict

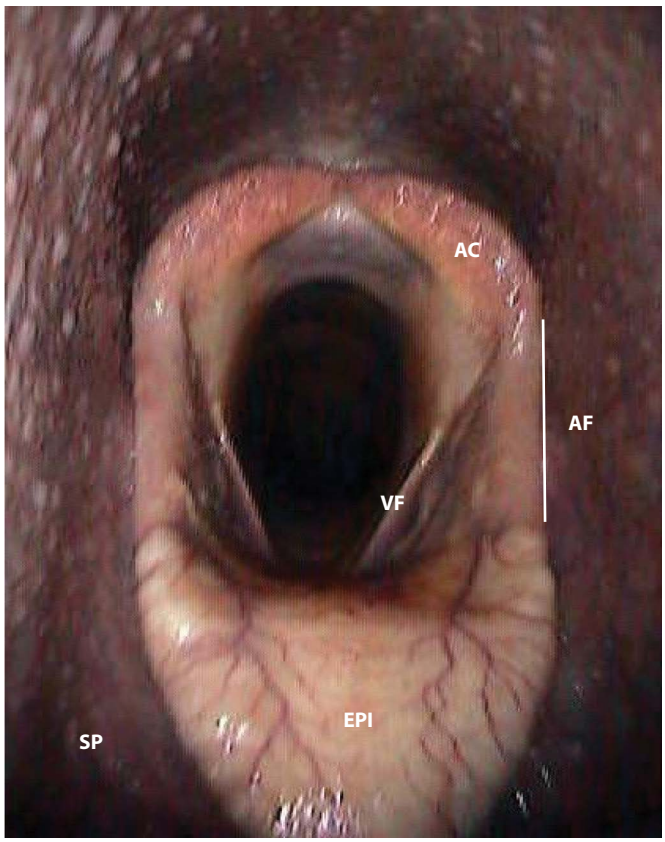


Fig. 8.1 Endoscopic view of the nasopharynx/larynx with key features marked: Epiglottis (EPI); arytenoid cartilage (AC); vocal fold (VF); aryepiglottic fold (AF); soft palate (SP).

for the food bolus to move back towards the oesophagus, yet to permit free flow of air during exercise it must remain dilated in the face of the strongly negative pressures of inspiration.

During exercise the soft palate (which separates the oral from the nasal portions of the pharynx) must remain anchored below the epiglottis, and the larynx firmly seated in the airtight 'buttonhole' of the pharyngeal ostium, otherwise respiratory obstruction will ensue. Correct soft palate and pharyngeal function is dependent on many factors including the relative position of the larynx and hyoid apparatus and coordinated action of the

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intrinsic and extrinsic muscles. Hyoid apparatus movements are determined by contributions of the tongue muscles as well as the strap muscles of the neck (sternohyoideus and sternothyroideus), while tensing of the soft palate itself is the function of the intrinsic palatal muscles (levator veli palatini, tensor veli palatini, palatinus and palatopharyngeus).

Guttural pouches

The paired guttural pouches are located between the pharynx and base of the skull and are accessed through a slit-like opening on each side of the pharynx. The pouches contact each other on the midline but do not communicate. Each pouch is partially divided into a large medial and smaller lateral compartment by the stylohyoid bone. Important neurovascular structures traverse the inside of each pouch including the internal and external carotid arteries and several cranial nerves; the function of the guttural pouch is uncertain, but one role is likely to be thermoregulation of the cranial vasculature.

Overview

Oxygen utilization by the racehorse during exercise is considerable and respiratory function is one of the key limiting factors to performance. Energy is expended in overcoming airway resistance, and even small obstructions have a disproportionately large impact on resistance and the work of breathing, leading to fatigue and effects on performance. The majority of airway resistance in the normal horse derives from the upper airway (nostrils, nasal passages and throat).

The horse is anatomically and functionally adapted to breathing solely through the nostrils whether at rest or exercise, and unlike other species (humans, dogs) cannot switch to mouth breathing when the respiratory demands of exercise increase. Major swings in intraluminal pressure of the airways occur between expiration and inspiration to facilitate movement of large volumes of air between nostril and lung. At strenuous exercise (and peak airflow) the negative pressures of inspiration have the potential to cause airway collapse. Pressures and turbulence are greatest at the floor of the front of the nasopharynx, and within the larynx. While this has little effect on the rigid/semi-rigid parts of the respiratory tract (nasal passages and trachea), the nostrils, pharynx and larynx must rely on muscular activity to remain dilated and have the greatest potential to weaken and impede airflow.

It is failure to maintain full function in the face of peak physiological demand (rather than structural abnormality) that characterizes the most important upper airway obstructions of racehorses. The manifestations of neuromuscular disorders, such as recurrent laryngeal neuropathy (RLN) and palatal displacement, vary considerably between rest and high-intensity exercise and this has ramifications for both diagnosis and treatment. Dynamic airway collapse is also frequently complex in nature, with more than one anatomical structure involved. These dysfunctions can occur concurrently or at different stages of exercise, or may emerge following surgical correction of other respiratory obstructions.

Even though racehorses (unlike most other equine disciplines) operate at the limits of respiratory capacity, the true effect of any upper airway obstruction on athletic performance is not completely understood. A general presumption has been that airway obstructions are more likely to have a detrimental effect over longer racing distances; however, even over sprint distances energy production is primarily aerobic. Significance of any given pathology may differ between horses and interpretation of findings may be influenced by age, racing form and stage of career, as well as severity of the condition. It is preferable that treatment decisions take into account individual circumstances rather than being prescriptive, and they should be based on appropriate diagnostic information.

Assessment of upper airway function

Upper airway assessment is performed during the investigation of respiratory noise or poor performance and during pre-purchase examinations. Traditionally, assessment has been based on the presence and character of respiratory noise at exercise ('wind testing') and resting endoscopy. While these modalities continue to have roles, direct visualization of upper airway function during high-speed exercise (overground/treadmill endoscopy) is now the method of choice as most conditions are dynamic (and frequently complex) in nature.

Respiratory noise

Respiratory noise at exercise may be the first sign of impaired airway function and although not diagnostic in isolation, it may be used to guide further investigations. It should be noted that the positive predictive value of the characteristic noise patterns for the major dynamic airway obstructive conditions in isolation is low: attempting to associate noise patterns with resting endoscopic findings results in incorrect conclusions at least 50% time.

In the normal horse at canter and gallop, expiratory sounds have approximately 2.5 times the amplitude of

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inspiratory sounds; when inspiratory noise becomes prominent the implication therefore is that functional abnormality and likely dynamic airway obstruction are present. Noises may vary in character depending on pathology, exercise intensity, head-carriage and whether exercised on a lunge or in straight lines. Relevant history that assists interpretation and may guide further investigation includes:

- Whether noise is reported by trainer or jockey/rider.
- Length of time that noise has been noted.
- Intensity of exercise (training canter/training gallop/race) and point during exercise (throughout/under pressure/pulling up) at which horse usually makes noise.
- Previous use (and effect) of headgear such as crossed noseband, tongue-tie.

Wind testing should be arranged to occur under conditions in which the horse is most likely to make any reported noise. Abnormal noises are categorized as inspiratory, expiratory or pan-respiratory; at canter/gallop, exhalation occurs during peak thoracic loading and inhalation begins when the leading forelimb leaves the ground. Inspiratory 'whistle' or 'roar' has moderate specificity for RLN (or vocal cord collapse), whereas expiratory 'gargle' suggests palatal displacement. However, sensitivity of respiratory noise for the presence of pathology is low. Characteristics typical of the main obstructive pathologies are described in Table 8.1. 'High blowing' expiratory noise is common at slow canter paces and usually due to vibration/flutter of the nostril. Respiratory noise (particularly inspiratory noise) frequently varies considerably in character between 'uncoupled' and 'coupled' (one breath per stride) breathing and reserving assessment until horse commences coupled breathing is recommended.

Breathing pattern

At canter and gallop paces breathing is directly linked to stride ('locomotor-respiratory coupling'), with expiration occurring when the forelimbs hit the ground and the horse taking one breath per complete stride. This is thought to confer energetic and mechanical advantages. Changes in breathing pattern may occur in association with dynamic upper respiratory tract obstructions, and 'breath holding' is a commonly reported presenting complaint. Horses with normal upper airway function may swallow intermittently during exercise (which suspends breathing for one stride); however, the frequency of swallowing should decrease as speed increases. Frequent swallowing at higher speeds may indicate the presence of soft palate dysfunction. Occasionally, transient 'decoupling' of the locomotor-respiratory link occurs with a single large breath taken over two strides: unless excessive, this is considered to be a normal 'readjustment' feature and is not associated with upper airway dysfunction.

Resting endoscopy

Resting endoscopy allows assessment of upper airway anatomy and resting laryngeal function. Sedative drugs may negatively influence laryngeal movements and should be avoided; if a nose twitch is used for restraint while passing the endoscope, it should be released for assessment of the throat.

The pharynx and larynx are examined for anatomical flaws and evidence of previous surgery. Laryngeal function is assessed through observation of natural movements of the laryngeal cartilages and by provoking opening (abduction) of the arytenoid cartilages by swallowing and/or blocking of nostrils (Figure 8.2a–c). Laryngeal function is graded according to symmetry at rest, synchrony of movement and ability to achieve/maintain full symmetric

Table 8.1 **Respiratory noise characteristics and dynamic URT obstructions**

NOISE	PHASE OF RESPIRATION	POSSIBLE DYNAMIC OBSTRUCTION TYPE/S
'High blowing'	Expiratory (and usually at slow canter pace)	Nostril flutter
Buzzing/vibrating	Inspiratory and expiratory	Alar fold collapse; epiglottic entrapment
Gurgling	Expiratory	Dorsal displacement of soft palate
Whistling/roaring	Inspiratory	Dynamic laryngeal (vocal fold +/– arytenoid) collapse; medial deviation of aryepiglottic fold(s)

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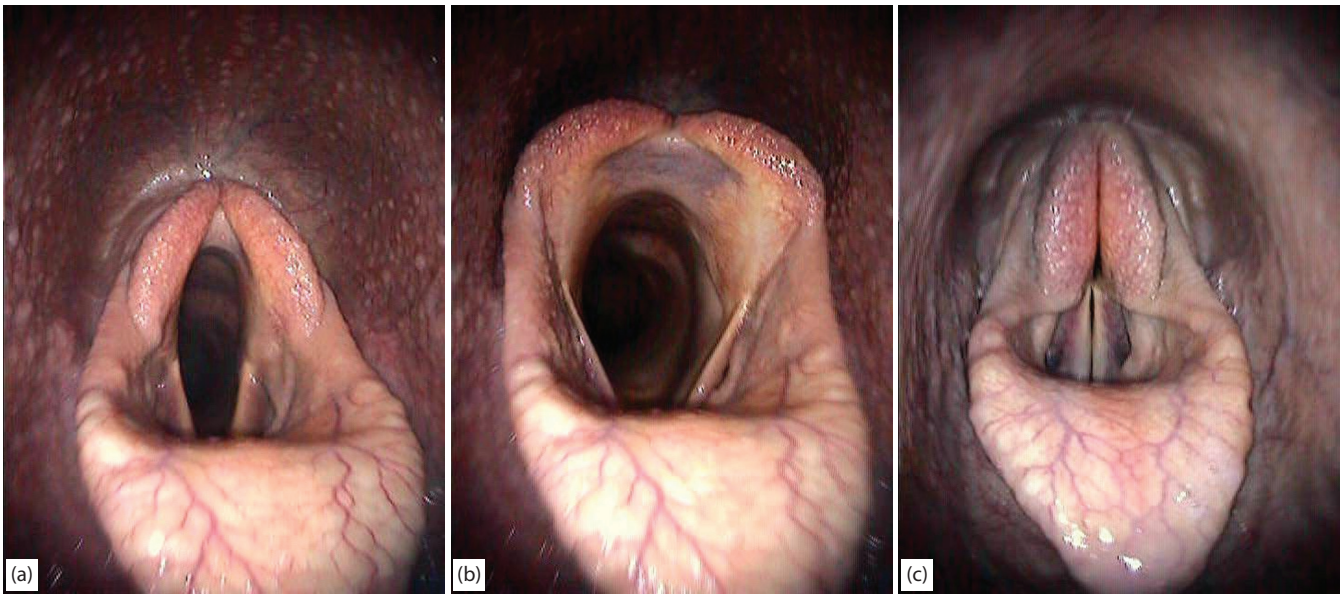


Fig. 8.2 Normal range of laryngeal movements: (a) at rest, (b) full abduction of arytenoid cartilages, (c) full adduction/closure (reflex closure or during swallowing).

abduction (Table 8.2) (Figure 8.3a,b). Asynchronous movement of the arytenoid cartilages may include tremor, hesitation or flutter.

Overground and treadmill endoscopy

Overground or treadmill endoscopy is the gold standard for assessment of airway function given that most obstructive conditions are dynamic in nature (Figure 8.4). Laryngeal function at exercise is graded according to degree of collapse of the affected arytenoid cartilage (Table 8.3), which in cases of severe dysfunction can result in a markedly occluded airway (Figure 8.5). Exercise tests should replicate as close as is practicable the circumstances in which it is suspected that the obstruction occurs: testing

Table 8.2 **Endoscopic grading of resting laryngeal function**

	HAVEMEYER	LANE
Resting symmetry and full arytenoid abduction can be achieved and maintained; all movements (adductory and abductory) synchronised	1	1
Full arytenoid abduction can be achieved and maintained with some asymmetry/asynchrony at times: 1. Transient asynchrony 2. Asymmetry much of time at rest but occasions (swallowing/nasal occlusion) when full symmetrical abduction is achieved and maintained	2 (2.1) (2.2)	2 3
Full abduction can be achieved but not maintained; asymmetry/asynchrony of cartilage movements much of time	3.1	
Full abduction cannot be achieved; obvious resting asymmetry	3.2	4
Full abduction cannot be achieved; marked resting asymmetry with little arytenoid movement	3.3	
Complete immobility/paralysis of arytenoid cartilage	4	5

Adapted from Dixon PM, Robinson NE, Wade JF (Eds) (2003). *Proceedings of a workshop on equine recurrent laryngeal neuropathy, Havemeyer Foundation Monograph Series No. 11*. R&W Publications, Newmarket.

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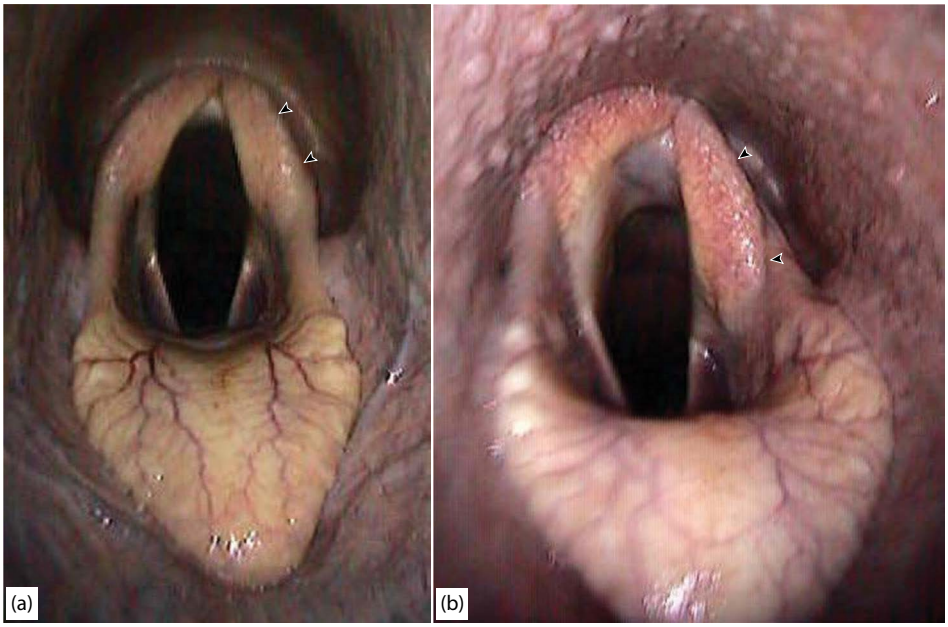


Fig. 8.3 **Recurrent laryngeal neuropathy: Resting endoscopic appearance of (a) moderate resting asymmetry with left arytenoid cartilage (arrowheads) closer to midline than opposite cartilage; (b) marked asymmetry with left arytenoid cartilage (arrowheads) slumped to midline while opposite cartilage is fully abducted.**

over shorter distances or slower speeds may not elicit the dysfunction. At submaximal exercise, head-carriage (poll flexion) can worsen dynamic respiratory obstructions by raising airway pressures and provoking laryngeal or pharyngeal collapse. Understanding of the variability in dynamic airway obstructions (presence/absence, and degree) that may occur between different exercise tests should inform interpretation of results. Concurrent information on speed and heart rate may assist interpretation of findings. The capacity to analyse recordings frame by frame or in slow motion is essential, as at full gallop the respiration rate may exceed 150 breaths/min.

Ultrasonography

Transcutaneous ultrasonography permits imaging of some intrinsic muscles important to laryngeal function, and assessment of cartilaginous landmarks (useful to determine whether congenital abnormalities are present). Neurogenic atrophy results in increased echogenicity of the cricoarytenoideus lateralis (CAL) muscle (Figure 8.6) due to replacement of muscle tissue by fibrosis and fatty tissue and, combined with assessment of left-right symmetry, may assist interpretation of resting endoscopy findings. Transoesophageal ultrasonography is a superior diagnostic aid performed under sedation that permits objective measurement of CAD muscular atrophy and subjective assessment of echogenicity; although a specialised procedure, it has an important role in selection of suitable candidates for surgery.

RESTING ENDOSCOPIC LARYNGEAL ASSESSMENT: WHY MAY VETS DISAGREE?

- When comparing resting laryngeal function grade for the same horse, disagreement between vets (in terms of exact grade) is common. Greatest variability occurs in the 'intermediate' grades (Havemeyer 2.2 and 3.1) which rely on whether full and sustained arytenoid abduction is observed.
- Factors responsible for this variation include those relating to horse (level of stimulation, tiredness, variation over time, use of sedation) and observer (experience, conscious and unconscious biases).
- Any observation is at best only a short 'snapshot' of a dynamic structure with complex neuromuscular activity: laryngeal movements (symmetry and synchronicity of abduction/adduction of arytenoid cartilages) can show variation even during the course of a single examination.
- Grading systems have inherent limitations that do not permit categorization of all 'shades of grey'.
- Observer subjectivity is important: even when all horse factors are controlled (i.e., in video clips) there is significant variation both between vets and between repeat gradings by the same vet.
- Differences in conditions of sale between countries +/– sales companies (particularly inclusion or otherwise of wind-testing) influence pre- and post-sale interpretation of laryngeal function by vets and purchasing clients.
- Regardless of that previously mentioned, most vets will agree broadly whether or not a throat 'meets conditions of sale'.

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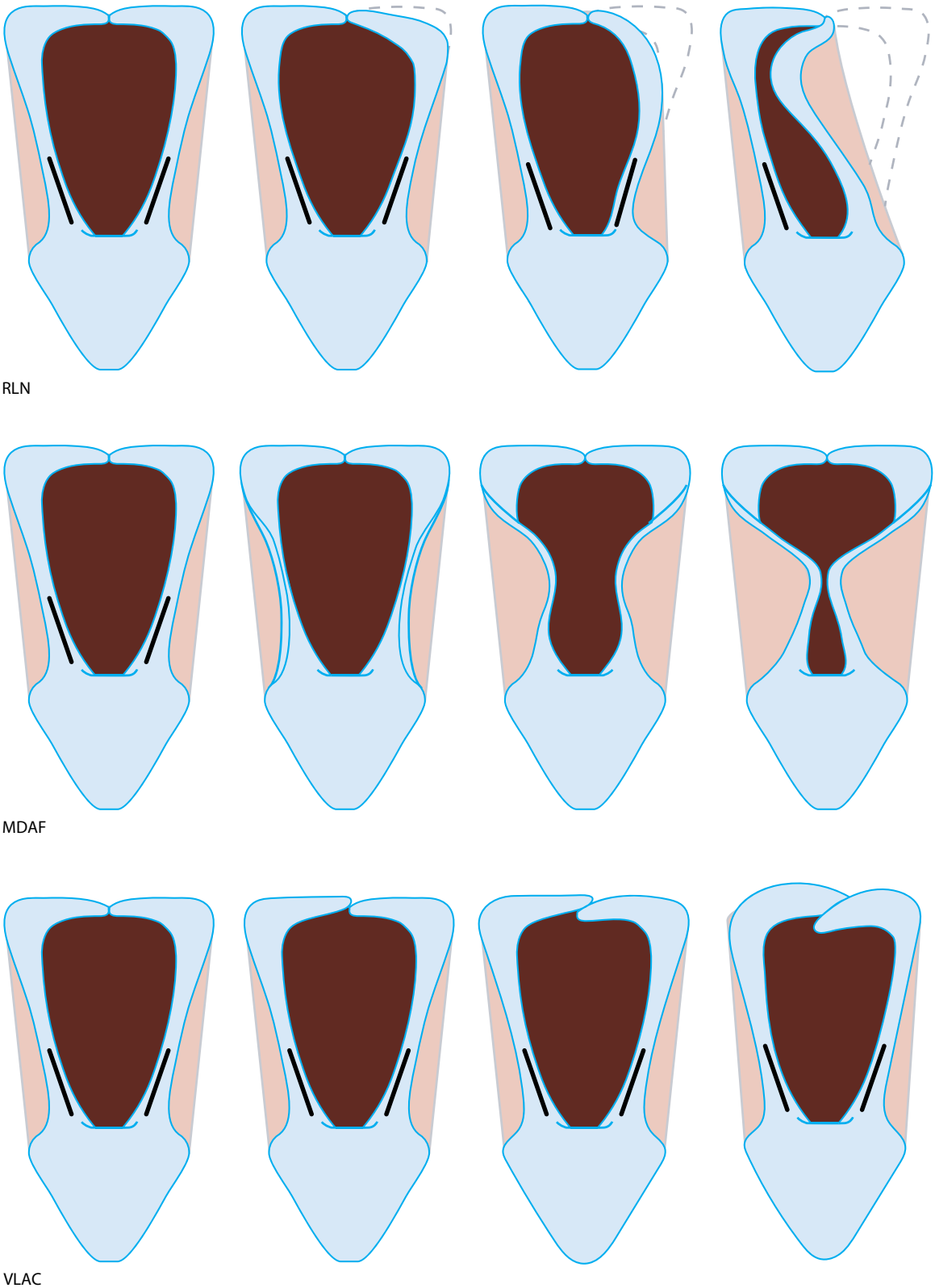


Fig. 8.4 **Dynamic obstructive conditions of larynx (and range of severity): Recurrent laryngeal neuropathy (RLN); medial deviation of aryepiglottic fold(s) (MDAF); arytenoid apex collapse (VLAC).**

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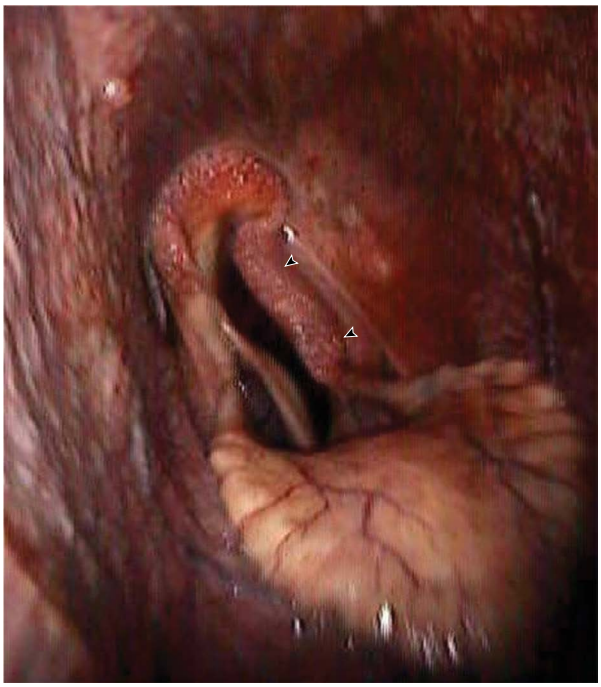


Fig. 8.5 **Severe (grade D) dynamic collapse of left arytenoid cartilage at exercise, with markedly occluded airway.**

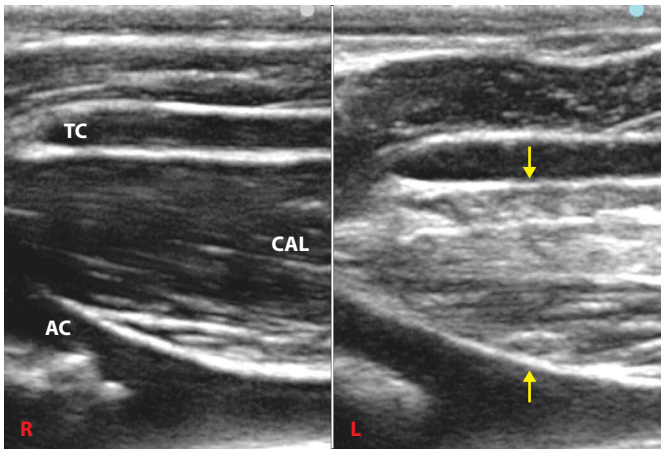


Fig. 8.6 **Transcutaneous transverse ultrasonograms of right (R) and left (L) sides of larynx with relative hyperechogenicity of the left-sided CAL muscle (AC: arytenoid cartilage; TC: thyroid cartilage; dorsal is to left).**

Table 8.3 **Endoscopic grading of laryngeal arytenoid function at exercise (Figure 8.4)**

A	Full abduction of arytenoid cartilages during inspiration.
B	Partial abduction of affected arytenoid cartilage (between full abduction and the resting position).
C	Abduction less than the resting position.
D	Abduction less than the resting position, including collapse into the contralateral half of the rima glottidis during inspiration.

Adapted from Rakestraw PC, Hackett RP, Ducharme NG *et al.* (1991) Arytenoid cartilage movement in resting and exercising horses. *Vet Surg* 20(2):122–127.

Recurrent laryngeal neuropathy ('roaring'/ laryngeal hemiplegia)

RLN is an important cause of dynamic respiratory obstruction. The arytenoid cartilages of the larynx are highly mobile and during exercise are normally maintained fully open by the action of intrinsic muscles. Neuromuscular dysfunction can lead to failure to achieve/maintain full abduction, particularly in the presence of negative pressures acting during inspiration at exercise. Collapse of the cartilage and/or associated vocal fold into the airway during breathing is responsible for the characteristic inspiratory noise (whistle/roar) and has the potential to affect performance. It is most commonly left-sided; right-sided weakness is rare. Range of severity encountered from mild to complete paralysis and the condition is frequently progressive, although rate of deterioration is highly variable.

Due to high level of vigilance early in life (particularly at yearling public auction), many animals significantly affected by the condition are selected against; consequently the number of horses exhibiting or developing career-limiting RLN in the training population is relatively small compared with other dynamic airway obstructions such as palatal displacement. Importance of the condition and issues around accuracy and predictive value of diagnostic tests therefore lies chiefly in the selection and pre-purchase assessment of horses, with management of clinically affected individuals challenging but less commonplace.

Causes

- Disease of the recurrent laryngeal nerves that innervate important paired intrinsic muscles of larynx.
- Progressive 'dieback' of affected nerves causes first loss of adductor, then abductor function: wastage of CAD muscle is of greatest importance.
- Cause(s) uncertain; however, length (longest motor nerve in body) and complicated path of left nerve may be implicated.
- Nerve lies close to jugular vein; some cases may arise secondary to perivascular injection of irritant drugs.

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Risk factors

- Approximately 2–3% of yearlings and 3–4% of horses in training have true left-sided laryngeal paralysis detectable with resting endoscopy.
- May be encountered at any age/stage of training.
- More common in colts than fillies.
- Tall horses at greater risk.
- Genetic factors: estimated that heritability is relatively low, although some bloodlines appear to be particularly affected. Mode of inheritance is complex and multiple genes probably responsible. Affected sires are more likely than unaffected sires to produce affected progeny.

History

- May be an incidental finding at routine or pre-purchase endoscopy.
- Characteristic inspiratory noise (whistle/roar) may be heard during cantering/galloping.
- +/– poor performance.

Diagnosis

- Resting +/or overground/treadmill endoscopy to visualize range of laryngeal movements.
- Resting endoscopy: practical screening aid with high sensitivity and specificity for determining the presence of dynamic laryngeal collapse at exercise (however, dynamic function is best assessed using overground/treadmill endoscopy).
- Resting laryngeal function grading is based on quality of arytenoid cartilage movements (Table 8.2); however, considerable subjectivity around what constitutes asynchrony, asymmetry and premature weakening (and how to stimulate/assess each of these parameters). Ordinal categorization of any biological condition with a wide spectrum of individual variation carries inherent inaccuracies; none of the grading systems in use for resting function reliably correlate to pathological status in all animals/situations, and scientific literature pertaining to RLN should be interpreted in the knowledge that grading may differ between researchers.
- Intermediate grades (Havemeyer 2.2/3.1; Lane G3) of resting function incorporate determination of whether arytenoid cartilages achieve and/or maintain full abduction, and require observation of larynx in both relaxed and stimulated states. Interpretations based on stimulated swallowing alone (rather than prolonged nasal occlusion) may not permit full assessment of sustained abduction and could account for inter-observer variability. Another important source of inter-observer variability in pre-purchase situations is conscious/unconscious bias when assigning grade to throats with 'weak' intermediate function.
- Overground/treadmill endoscopy is diagnostic aid of choice to assess dynamic laryngeal function.
- Sedative drugs (acepromazine/detomidine/xylazine/romifidine) can cause or exacerbate resting laryngeal weakness, although evidence is mixed.
- Palpation of larynx: may reveal prominence of muscular process of left arytenoid cartilage (due to loss of overlying muscle) with advanced disease.
- Transcutaneous ultrasound: good sensitivity and specificity for RLN and useful adjunct to endoscopy in 'borderline' cases, but poor prognostic value as a screening modality for future development of laryngeal dysfunction.
- Transoesophageal ultrasound: increasingly used to assess suitability of surgical candidates for nerve graft surgery. Permits objective measurement of CAD size/atrophy. Good correlation between degree of left-sided atrophy and endoscopic (both resting and dynamic) grading of laryngeal function.

Relationship between resting and exercising function

- Moderate correlation between resting and exercising function.
- Most (>80%) horses that fail to achieve full abduction at rest (nasal occlusion/swallowing: Havemeyer G3.2/Lane G4) display some degree of dynamic collapse at exercise.
- Most (approximately 90%) horses with good (symmetric) resting grades show good function at exercise.
- Exceptions to both rules occur: small proportion (3–9%) of resting G1 and 2 throats display dynamic collapse at exercise, and some G4 throats achieve full abduction at exercise (Figure 8.7). Reason for marked disparity between resting and dynamic function in these cases is not always clear.
- Horses with 'intermediate' resting laryngeal function grades (some asymmetry but capable of achieving full abduction) can be considered 'normal' but are more likely to display dynamic collapse at exercise than 'better' graded throats.

Effect on performance

- Dynamic laryngeal function is only one factor influencing athletic ability: direct effect on performance in any individual is difficult to determine.

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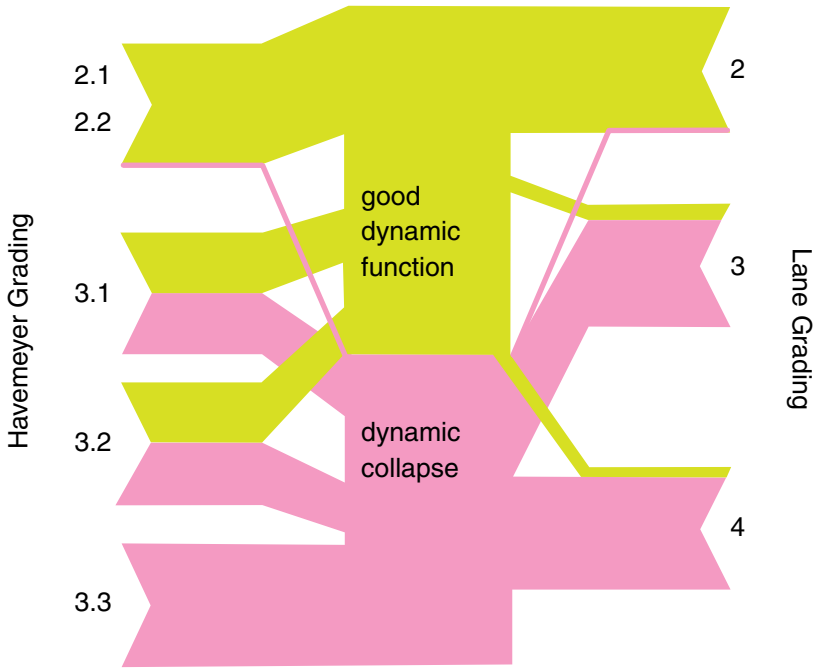


Fig. 8.7 **Illustration of correlations between resting laryngeal function score (Havemeyer grade on left, Lane grade on right) and likelihood of dynamic dysfunction at exercise. A small proportion of horses with 'good' resting grades have dynamic collapse, and a small proportion of horses with 'weak' resting grades have good dynamic function.**

- Horses with serious dynamic collapse are ineffective athletes, but horses with intermediate resting +/or dynamic grades can have satisfactory (and occasionally elite) careers.
- Horses with weak resting grades (i.e., unable to achieve or sustain full abduction: Havemeyer >2.2/Lane > G3) are significantly less likely to be successful athletes.
- Deterioration in resting laryngeal function can occur over time: speed and degree of progression cannot be predicted and may affect all grades.

Management

Treatment decisions should be based on appropriate assessment of laryngeal function (preferably overground endoscopy) and informed by the trainer's opinion regarding relative importance to individual circumstances. In many cases treatment may not be considered worthwhile for a variety of factors (cost, lost training time, athletic potential of horse) and horses allowed to complete their careers or be sold without intervention. Surgical procedures are directed at reducing inspiratory noise and/or preventing dynamic laryngeal collapse. Age, stage of career, severity of laryngeal dysfunction, race form and athletic potential/value influence choice of procedure. Surgical options include:

- Ventriculectomy ('Hobday'): ablation of lining of ventricle(s); reduces severity of vocal fold collapse and respiratory noise. Likely to have little therapeutic value. Typically 3–4 weeks rest required post-surgery.
- Ventriculectomy and vocal fold resection: ablation of ventricle and resection of vocal fold (usually unilateral) (Figure 8.8). Considered more effective than ventriculectomy (significantly reduces respiratory noise, reduces airway obstruction and improves inspiratory upper airway pressures), and



Fig. 8.8 **Prior ventriculectomy/vocal fold resection: Absence of left vocal fold (circled).**

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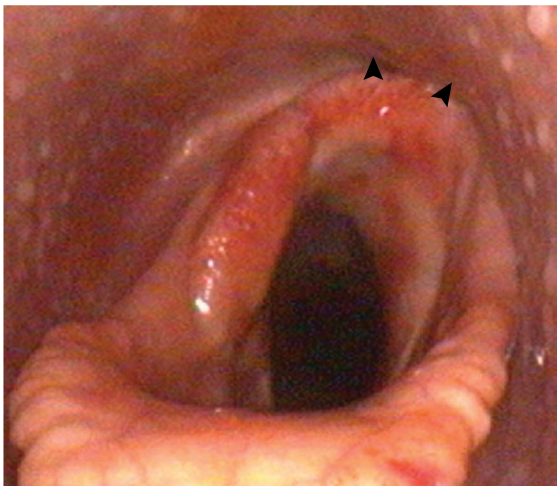


Fig. 8.9 Prior prosthetic laryngoplasty ('tie-back'): Full abduction of left arytenoid cartilage (arrowheads) with right arytenoid cartilage at rest.

frequently the treatment of choice for milder grades of RLN. Typically 3–4 weeks rest required post-surgery.

- Prosthetic laryngoplasty ('tie-back'): placement of sutures to 'pin back' left arytenoid cartilage and emulate action of CAD muscle (Figure 8.9); goal is to stabilize arytenoid cartilage and prevent dynamic collapse. For moderately severe cases. Commonly combined with ventriculectomy and vocal fold resection. High (80–90+%) proportion of horses return to racing, but improved performance reported in <50% horses, and is generally considered a procedure with a poor success rate in flat racehorses. Reduces upper airway obstruction, but post-operative complications include implant failure, infection, dysphagia or dynamic collapse of other structures. Gradual loss of degree of arytenoid abduction is a common feature. Treated horses more prone to coughing (food inhalation, particularly with rostrocaudal mismatch between arytenoid position), inflammatory airway disease and significant exercise-induced pulmonary haemorrhage (EIPH) (p. 305) including epistaxis through remainder of career. Typically 6 weeks rest post-surgery.
- Partial arytenoidectomy: removes some of the respiratory obstruction but return to function not equal to tie-back, and generally considered a salvage procedure. Similar risk of food inhalation to tie-back. Typically 6 weeks rest post-surgery.
- Re-innervation of laryngeal (CAD) muscle: cervical and/or spinal accessory nerves are harvested and directly implanted into the CAD muscle. Exercise commences 3–4 weeks post-surgery, back to full work 10–16 weeks; active rehabilitation important as exercise stimulates nerves. Full/adequate muscle function expected between 3–9 months post-surgery (dependent on severity at time of surgery). Has limited/variable effect on resting endoscopic appearance of larynx; rather, it normalises 'stimulated' function at exercise. Also eliminates complications (coughing, EIPH, food inhalation) associated with laryngoplasty. Best undertaken in juvenile (yearling) horses, and is treatment of choice for more severe grades of RLN in this age-group. Transoesophageal ultrasound: >10% CAD atrophy indicates presence of RLN; best surgical candidates appear to be those with between 10% and 40% CAD atrophy. Horses with >40% CAD atrophy typically have dynamic laryngeal collapse (grade C) and time required for satisfactory function to return generally makes re-innervation alone undesirable in these latter cases. Can be combined however with carefully positioned prosthesis in more severe cases (dynamic neuroprosthesis [DNP]), to provide some abduction of affected arytenoid and allow earlier return to training, with fewer complications than with regular laryngoplasty.
- General recommendations for mature racehorses: those with Grade B exercising function may be best treated by ventriculectomy and vocal fold resection; horses with Grade C or D exercising function consider laryngoplasty (or CAD re-innervation/DNP + ventriculectomy +/– aryepiglottic fold resection).

Prognosis

- Disease is chronic and generally progressive; however, deterioration is not always linear or predictable.
- Up to 15% of horses show deterioration in laryngeal function over time; it is also possible to encounter horses that 'improve' in resting grade over time.
- Efficacy of surgery in restoring/improving athletic potential in any individual is difficult to quantify and varies with procedure used.
- In general, horses diagnosed whilst in training with poor +/– deteriorating laryngeal function resulting in serious dynamic collapse may be difficult to rehabilitate to good athletic form regardless of surgical intervention.
- In general, horses diagnosed with RLN early in life (prior to commencing training) have greater opportunities for career success: some (regardless

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of intervention) will perform satisfactorily as 2 YOs (over shorter race distances) and others will undergo surgical treatments selected on the basis of long-term efficacy rather than as pragmatic mid-/late-career salvage procedures.

Palatal displacement/instability

During exercise the soft palate and palatopharyngeal arch form an airtight seal around the larynx. Displacement of the caudal free border of the soft palate from its regular position below the epiglottis (Figure 8.10) occurs during swallowing of food, but if it takes place during exercise this seal is disrupted, creating an obstruction to airflow. A loud expiratory noise ('gurgling'/'choking') typically results and may be accompanied by immediate interruption of athletic effort as the horse breaks stride. Displacement is transient but variable in duration between episodes, sometimes returning to normal position within the same stride and at other times lasting until cessation of exercise. Displacement is not always associated with an abnormal respiratory noise.

Dorsal displacement of the soft palate (DDSP) is an intermittent and unpredictable condition with considerable variation in severity both between individuals and over time. Episodes most commonly occur during peak exertion; it may also be precipitated by change in speed, effort and neck flexion on pulling up at the end of exercise.

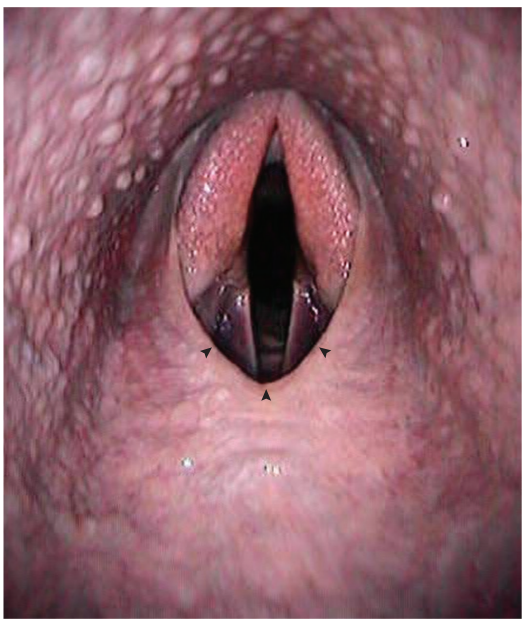


Fig. 8.10 Dorsal displacement of soft palate: Caudal free margin of palate (arrowheads) temporarily dislocated above the epiglottis.

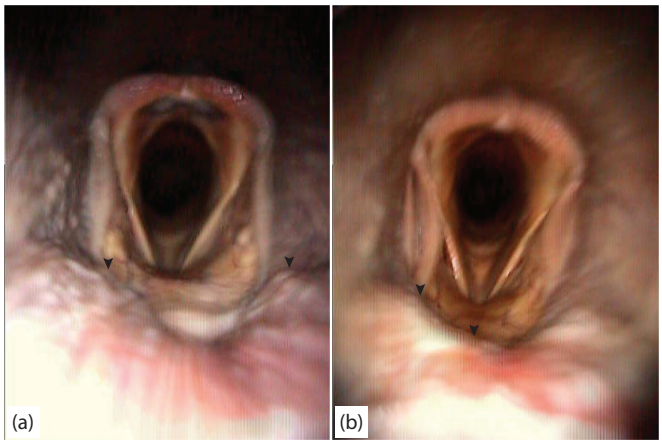


Fig. 8.11 Palatal instability: Mild (a) and marked (b) billowing of soft palate at exercise.

Palatal instability (PI) (progressive 'billowing' of caudal or rostral parts of the soft palate, with flattening of the ventral surface of the epiglottis against the upper surface of the soft palate) is considered part of the syndrome and often precedes DDSP at exercise (Figure 8.11a,b) but is also encountered as a stand-alone condition. PI, and subsequent DDSP, appear to occur secondary to caudal retraction of the larynx. There is evidence of a link between DDSP and speed/maximum heart rate, leading to speculation that anticipation (of DDSP) might occur and cause some affected horses to put in sub-maximal effort to avoid displacement of the palate.

DDSP can be a serious impediment to athletic success and is of particular concern as current diagnostic aids do not permit detection of the condition at rest (thereby precluding pre-purchase assessment at public auction).

Causes

- Neuromuscular dysfunction: multifactorial, and multiple pathways to displacement/instability.
- Failure of coordinated control of intrinsic and extrinsic musculature to maintain palatal tension and counteract negative pressures of inspiration. May arise from fatigue of key muscles (including paired thyrohyoid muscles) during exercise.
- Structures potentially involved: extrinsic muscles controlling positioning of tongue/hyoid apparatus; intrinsic 'tensing' muscles of soft palate; pharyngeal branch of vagus nerve, hypoglossal nerve.
- Potential role of respiratory inflammation/infection (particularly in young horses) due to close proximity of neural structures to lymphoid tissue in retropharyngeal region.

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Risk factors

- Prevalence unknown but is the most common dynamic respiratory obstruction encountered in racehorses.
- Any age/stage of training but typically more common in 2 YOs.
- Risk factors poorly understood.
- Unlikely to be heritable.
- Role of epiglottis size/function uncertain.

History

- 'Performance limiting' DDSP: typical history of sudden cessation of athletic effort during race/gallop in association with loud/expiratory ('gurgling') noise; or persistent/recurrent DDSP during training exercise that does not improve with maturity/fitness and cannot be mitigated by use of preventative tack.
- Up to 30% of affected horses do not make a noise: in these cases poor performance may be the primary presenting complaint.

Diagnosis

- Overground/treadmill endoscopy is sole means of definitive diagnosis.
- Even with endoscopy, can be challenging to diagnose: exercise test conditions should replicate as closely as possible those in which horse usually displays problem (typically at fast gallop, and potentially over race distances, speeds and pressures).
- Resting endoscopy not useful in diagnosis of DDSP/PI; palatal displacement occurs during most resting examinations and is not predictive for future development of palatal problems.
- If overground/treadmill endoscopy unavailable, abolition of respiratory noise by application of tongue-tie or Cornell collar may be indicative (but not diagnostic).

Management

- 2 YOs: many cases of suspected DDSP resolve with maturity/improved fitness; investigation/treatment best reserved for horses displaying exercise intolerance.
- Recommended that conservative measures are tried initially: tongue-tie (anchors tongue to mandible, moving larynx and hyoid apparatus forward); crossed noseband (prevents opening of mouth); Cornell collar (positions larynx in similar fashion to laryngeal tie-forward surgery, see later).
- Surgical intervention if conservative measures unsuccessful.
- Surgical options include one (or combination of several) of the following:
  - Laryngeal tie-forward: sutures placed to emulate function of thyrohyoid muscles; positions larynx further forward and higher in throat. Typically 3–4 weeks rest from ridden exercise post-surgery. Current treatment of choice.
  - Thermocautery: firing of nasal or oral surfaces of soft palate, with aim of inducing scarring/stiffening of palate. Efficacy (whether used in isolation or in combination with other surgical procedures) is limited/poor.
  - Tension palatoplasty: surgical procedures intended to 'tighten' the mid- and caudal soft palate.
  - Myectomy/tenectomy: removal or cutting of insertional tendon(s) of strap muscles of neck; thought to limit caudal retraction of larynx.
  - Staphylectomy: resection of the free border of palate.
  - Intra-palatal infiltration of collagen-stiffening polymer. Efficacy untested.
- Potential future therapies may include 'inspiratory muscle training' and muscular pacemakers to strengthen stabilising muscles of upper airway.

Prognosis

- Approximately 50% of affected horses respond favourably to conservative measures such as use of tongue-tie: empirically a better success rate with conservative measures for younger horses than older horses.
- Reported improvements in performance with current surgical techniques occur in approximately 50–70% horses; however, true efficacy of different procedures and for different types (age, racing class and distance) of horse remains poorly researched.
- Currently considered that most appropriate surgical procedure for horses is the laryngeal 'tie-forward', with a 70% reduction in post-operative DDSP seen on repeat overground endoscopy reported.
- Post-surgical racing performance of hurdlers/steeplechasers following laryngeal 'tie-forward' and thermocautery (+/– resection of aryepiglottic folds) has been shown to be statistically decreased compared with unaffected controls.
- Small proportion of affected horses cannot be managed successfully and are retired.

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Medial deviation of the aryepiglottic fold(s) (MDAF)

The aryepiglottic folds are membranous structures on each side of the throat that are normally tensed between the epiglottis and arytenoid cartilages. Dynamic collapse of the aryepiglottic folds can occur during exercise and may be associated with an inspiratory noise. Intermittent and sometimes transient condition. Usually bilateral, but may be unilateral. Severity of respiratory obstruction varies: many horses display very mild (and presumably clinically insignificant) medial deviation of the aryepiglottic fold(s) (MDAF), with moderate/severe deviation less common. Can occur in isolation, or in combination with other upper respiratory tract conditions such as PI, DDSP and RLN; the greater the severity of MDAF, the more likely it is that other upper airway problems will be present.

Causes

- No muscular tissue in fold so may represent stretching of tissue or altered position/function of arytenoid/epiglottis/soft palate.
- PI (with associated dorsal epiglottal excursions) and/or the position of adjacent arytenoid cartilage resulting in loss of tension of the fold has been implicated as a cause, as has increased negative pressure adjacent to right aryepiglottic fold as a result of a sub-maximally positioned left arytenoid cartilage (in cases of RLN).

Risk factors

- Any age/stage of training.
- Relatively common condition, particularly in association with other dynamic obstructions. Primary diagnosis of moderate-severe MDAF in <10% of racehorses investigated for dynamic respiratory obstructions.

History

- Inspiratory noise during cantering/galloping exercise.
- Noise frequently similar in character to that associated with RLN.

Diagnosis

- Overground/treadmill endoscopy is sole means of diagnosis.
- Severity graded as mild/moderate/marked depending on degree of axial deviation (Figures 8.4 and 8.12a–c) relative to position of vocal fold. Mild: aryepiglottic folds remain lateral to vocal cords; moderate: medial deviation to less than halfway between vocal fold and midline; marked:

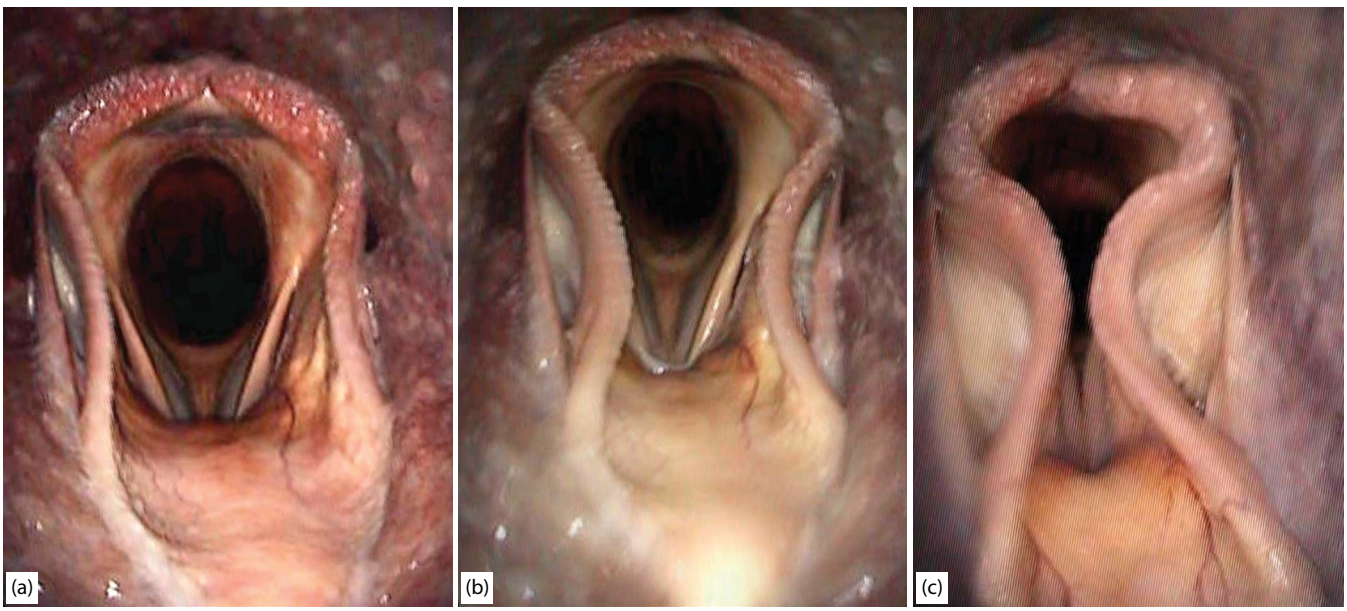


Fig. 8.12 **Medial deviation of the aryepiglottic folds: (a) mild; (b) moderate; (c) marked (latter with concurrent vocal fold collapse and VLAC).**

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aryepiglottic folds collapse to greater than halfway between vocal fold and midline.

Management
- Mild deviations rarely interfere with training.
- Treatment warranted if moderate-severe MDAF considered to be affecting performance or responsible for respiratory noise.
- Surgery: thermoablation (laser) of aryepiglottic fold(s). Typically 2–4 weeks out of ridden exercise post-surgery.
- Concurrent treatment of other respiratory problem/s.

Prognosis
- Spontaneous resolution in some horses without treatment.
- Surgery resolves MDAF in most cases but may not have any effect on other components of complex dynamic collapse (if present). Recurrence of MDAF following surgery may occur.

Epiglottic entrapment
Condition in which the epiglottis becomes temporarily or semi-permanently enveloped in a fold of subepiglottic tissue. Usually observed at resting endoscopy but also occurs less commonly as a purely dynamic condition at exercise only (Figure 8.13a,b). Not necessarily associated with poor performance and frequently subclinical.

Cause
- Underlying cause unknown.

Risk factors
- Any age/stage of training.
- Incidence reported as 0.06–1.3% of yearlings and horses in training.
- Some affected horses have small epiglottic dimensions; however, this is not a universal feature.

History
- Most make a harsh but non-specific inspiratory and expiratory respiratory noise at exercise.
- Many cases detected as incidental finding at routine endoscopy (no respiratory noise).

Diagnosis
- Resting endoscopy: epiglottis appears sheathed in close-fitting 'glove' of tissue; this tissue may be ulcerated/thickened (Figure 8.13c).
- Determining whether entrapment is intermittent or fixed is important for management.

Management
- While not generally associated with poor performance, surgical treatment is straightforward, has a low complication rate, appears to be beneficial (performance and respiratory noise) and is therefore recommended.
- Treatment is elective and can be planned to fit with training programme/racing engagements.
- Surgical release of entrapping tissue is treatment of choice and usually performed as a standing/sedated procedure. Requires little time out of training (1–3 weeks).

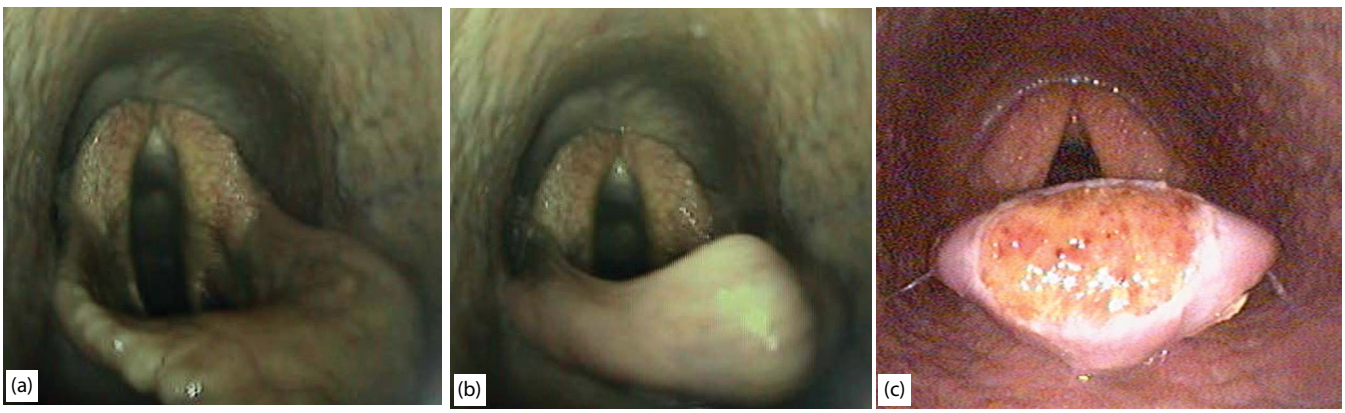


Fig. 8.13 Epiglottic entrapment: (a,b) intermittent, dynamic entrapment at exercise with normal (a) and entrapped (b) appearance, with subepiglottic tissue cloaking the epiglottis in the latter; (c) constant entrapment, with typical thickened/ulcerated appearance to mucosa.

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- If entrapment is intermittent, surgical resection of subepiglottic tissue may be required.
- Conservative management (rest and anti-inflammatory throat spray) also effective but less favoured.

Prognosis
- Uninterrupted training (no treatment) possible in horses without respiratory noise/poor performance.
- Excellent prognosis for return to full use following surgery.
- Approximately 5–10% cases recur.
- Rare complications of surgery include development of DDSP and scarring of epiglottis.

Epiglottic retroversion
Dynamic condition in which epiglottis 'flips' up and occludes airway during inspiration at exercise (Figure 8.14). Frequency increases with speed and may be exacerbated by neck flexion.

Causes
- Unknown but likely to be a neuromuscular disorder.
- May arise from damage to hyoglossal nerve, or to hyoepiglotticus or geniohyoideus muscles.

Risk factors
- Rare.



Fig. 8.14 Epiglottic retroversion at exercise: Vertical/caudal tipping of epiglottis (arrowheads) and occlusion of airway.

- Any age/stage of training.
- Recent upper airway surgery or inflammation may predispose.

History
- Loud harsh intermittent respiratory noise (inspiratory) and exercise intolerance.

Diagnosis
- Overground/treadmill endoscopy is sole means of diagnosis.
- No abnormality on resting endoscopy.

Management
- Rest +/– anti-inflammatory medication if post-surgical inflammation thought to be responsible.
- Surgery (epiglottopexy) may resolve condition and permit return to racing, but surgical techniques are evolving and success and complication rates are currently unknown.

Prognosis
- Limited information available; surgery appears to offer best prognosis for athletic recovery (otherwise poor with conservative management).

Arytenoid apex collapse (ventromedial luxation of apex of corniculate process of arytenoid cartilage, VLAC)
Normally the top of each arytenoid cartilage (apex of corniculate process) maintains close apposition with its partner throughout all phases of breathing and exercise. Subluxation/luxation of one apex from its normal position may occur such that the tip sits below (rarely, in front of) the opposite cartilage (Figure 8.15a–c), sometimes with slight ventral 'tilting' of the entire affected arytenoid cartilage. Usually occurs as a dynamic collapse; larynx usually appears normal at rest and luxation occurs suddenly at full inspiration (can often be stimulated by blocking nostrils during resting endoscopy). Most commonly left-sided, but right-sided and bilateral collapse also occur. Severity varies between individuals from subtle malalignment of the apices (with no appreciable effect on airway geometry) to marked overriding of the cartilages with associated deformation of the airway (Figure 8.4). May be associated with respiratory noise +/– poor performance. May be associated with other forms of upper airway obstruction (particularly RLN), but relationship with these currently unknown.

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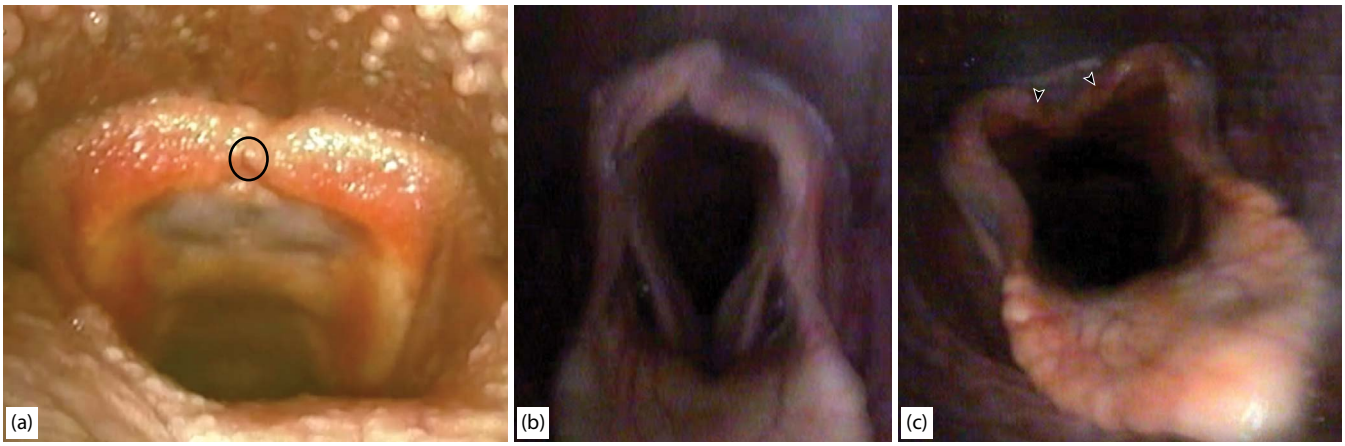


Fig. 8.15 **Arytenoid apex collapse: (a) very subtle ventral displacement of left corniculate process (circled) of low significance; (b) more significant left-sided collapse (+/– RLN) associated with dynamic respiratory obstruction; (c) bilateral (arrowheads) dynamic collapse.**

Cause
- Currently unknown, but widening/dysfunction of the ligament (transverse arytenoid ligament) which keeps the tips of the arytenoid cartilages aligned is presumed.

Risk factors
- Rare condition.
- Any age/stage of training.

History
- Typically detected as an incidental finding at routine or pre-purchase endoscopy: not always associated with respiratory noise or poor performance.
- Respiratory noise, if present, is usually inspiratory.
- May not always be evident on videoendoscopic clips presented at public auction, as sustained nasal blockade often required to stimulate dislocation of cartilages.

Diagnosis
- Endoscopy (resting or overground) is definitive. Larynx may have normal appearance during passive observation at rest but distinct subluxation of apex readily and repeatably prompted through (sustained) stimulation of full arytenoid abduction.

Management
- Currently no reported/validated treatment options.

Prognosis
- Little is currently known about the condition, including progression; may persist long term and is generally considered to be a permanent dysfunction (particularly if bilateral).
- Potential impact on performance dependent on severity and whether concurrently affected by other forms of dynamic upper airway collapse.
- Cases that have full/normal laryngeal function at exercise and in which the degree of subluxation does not appear to detract significantly from the dimensions of the fully open larynx (rima glottidis) appear unlikely to display or develop negative effects on career.
- May be confused with the early stages of RLN, and may warrant repeated examination and/or laryngeal ultrasonography to differentiate.

Nasopharyngeal collapse

Dynamic collapse of the dorsal or lateral walls of the pharynx, or 'circumferential' collapse of all the pharyngeal walls as well as the soft palate, can cause serious upper airway obstruction. Severity varies between individuals. Current knowledge about the condition is very limited. May occur concurrently with other types of dynamic upper airway obstruction.

Causes
- Cause is uncertain but likely to involve neuromuscular immaturity, dysfunction or fatigue of upper airway dilator and pharyngeal constrictor muscles +/– dysfunction of sensory reflexes involving laryngeal mucosa.
- Dorsal pharyngeal collapse may be caused by pressure differential between guttural pouches (above pharynx) and nasopharynx (Figure 8.16a,b).
- Head-carriage (neck flexion) may be a contributing factor.

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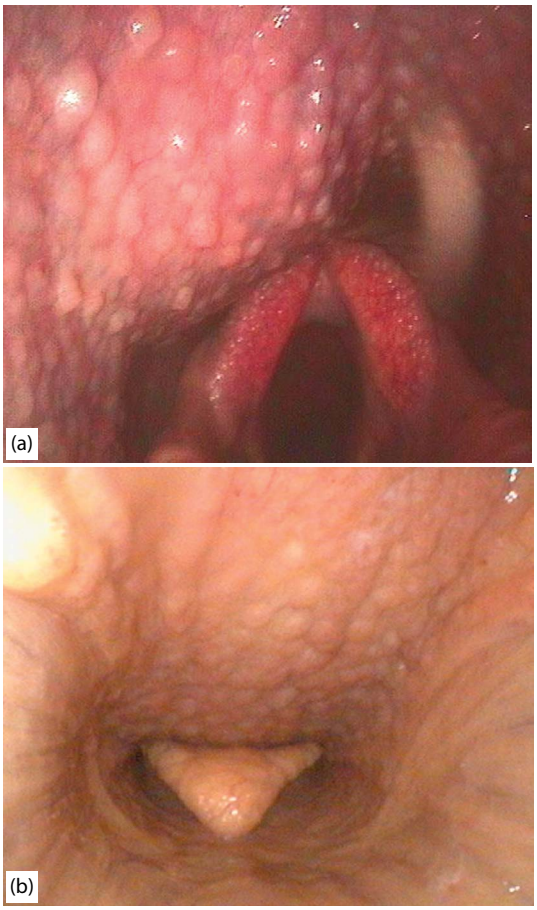


Fig. 8.16 **Dorsal pharyngeal collapse: (a) right-sided; (b) profound dorsal collapse caused by guttural pouch dysfunction.**

Risk factors
- Any age/stage of training.
- Risk factors unknown.

History
- Typically present with background of abnormal (inspiratory) respiratory noise +/– poor performance.

Diagnosis
- Endoscopic appearance: collapse of walls of pharynx, such that larynx becomes partially/totally obscured from view depending on severity.
- May be observed in some horses during resting endoscopy, but correlation with dynamic collapse at exercise is uncertain.
- Overground/treadmill endoscopy is essential to confirm diagnosis.

Management
- Currently no specific treatment available.
- Elimination of concurrent guttural pouch abnormalities, e.g., infection.
- Some cases may respond favourably to conservative management (rest +/– anti-inflammatory medication) but no validated guidelines exist.
- Some experimental evidence supporting use of 'inspiratory muscle training' to strengthen stabilising muscles of upper airway: increased respiratory load applied by daily use of mask at rest, over treatment period of weeks/months. Efficacy currently unknown.

Prognosis
- Information on prognosis not available but currently considered to be guarded.
- Likely that more severe pharyngeal collapse (as diagnosed at resting or exercising endoscopy) can persist long term and can be performance-limiting.

Alar fold collapse

The alar fold is a skin flap within each nasal cavity that divides the true nostril from the small upper blind-ended pocket known as the 'false' nostril. At its caudal margin it continues as the alar cartilage. The transversus nasi muscle acts to elevate the alar cartilage during deep inspiration, which closes the opening of the false nostril. Collapse of the alar fold occurs if air enters the false nostril, and causes partial obstruction of the nasal cavity. May be associated with poor performance.

Cause
- Unknown, but presumed to arise from either dysfunction of the elevating (transversus nasi) muscle or excessive size of the alar fold.

Risk factors
- Rare condition.
- Any age/stage of training.

History
- Respiratory noise: typically a continuous vibrating/buzzing noise throughout inspiration and expiration. Should be distinguished from 'high blowing' (common respiratory noise during expiration only caused by nasal flutter, present at slow canter pace).

Diagnosis
- Initial steps should be to exclude other forms of dynamic upper airway obstruction (overground endoscopy).

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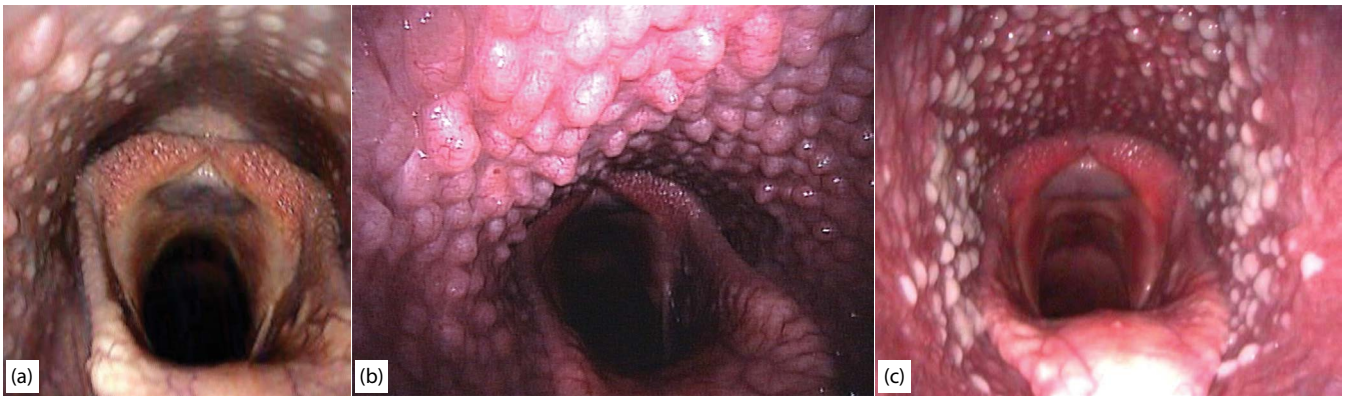


Fig. 8.17 Pharyngeal lymphoid hyperplasia: (a) mild; (b) marked; (c) inflamed appearance consistent with active upper respiratory infection.

- Placement of temporary mattress suture through each alar fold and over bridge of nose: elimination of noise is diagnostic.

Management
- Surgical resection of alar folds is treatment of choice.

Prognosis
- Surgery resolves condition.

Pharyngeal lymphoid hyperplasia (juvenile pharyngitis)
The mucosa lining the dorsal/lateral walls and floor of the nasopharynx is rich in lymphoid tissue and is the equivalent of the human tonsil. It plays an important role in immune defence, priming the rest of the body against bacterial and viral challenge. Enlargement of this lymphoid tissue is a normal finding in yearlings and 2 YOs. Severity varies between individuals. When pharyngeal lymphoid hyperplasia is marked or persistent, it may have an effect on upper airway function but generally has no impact on performance.

Cause
- Part of the natural priming of immune system and represents adaptation to microbes found in the training environment.

Risk factors
- Almost ubiquitous in young (yearling/2 YO) horses in training.

History
- Usually an incidental finding at routine endoscopy.
- May display harsh respiratory noise at exercise.

Diagnosis
- Resting endoscopy: enlarged lymphoid patches in walls/roof of nasopharynx (Figure 8.17a–c).

Management
- Treatment is not required in majority of horses (normal phase of immune system maturation).
- If associated with dynamic airway collapse/respiratory noise: treatment with topical/systemic anti-inflammatory (corticosteroid) +/– antibiotic medication may be justified, although efficacy undetermined.

Prognosis
- Excellent. Invariably resolves with maturity.

Arytenoid ulceration/chondritis
Ulceration/erosions occasionally occur on medial aspect of one or both arytenoid cartilages of the larynx. Most lesions involve only the mucosa and resolve spontaneously; however, a small proportion goes on to develop more advanced disease at the site including granulomas or (rarely) infection of cartilage (chondritis). Chondritis most likely to be left-sided but can be bilateral or right-sided.

Cause
- Underlying cause unknown in most cases.
- May be multifactorial: infection, trauma (from nasogastric intubation or endoscopy) or 'kissing' lesions from dynamic airway collapse.
- Infection of cartilage appears to be associated with opportunistic colonisation of traumatised tissue by common respiratory bacterial species (most commonly

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Streptococci). Multi-drug resistance of bacterial isolates is common.
- When associated with RLN, is assumed to be secondary; however, mechanical arytenoid dysfunction from enlargement/distortion of cartilage also possible.

Risk factors
- Any age/stage of training.
- Prevalence of ulcers/small granulomas in yearlings: approximately 0.6%.
- Prevalence of chondritis in yearlings: approximately 0.2%.
- Training on dirt/all-weather tracks may be associated with increased risk.

History
- Usually detected at routine endoscopic examination.
- Generally no outward sign of disease.
- No respiratory noise at exercise unless a concurrent dynamic respiratory obstruction is present.

Diagnosis
- Resting endoscopy: mild/prodromal disease seen as localized ulceration (Figure 8.18a) (+/– small granuloma) at rostral or medial margin of vocal process of one or both arytenoid cartilages. Chondritis appears as thickening/inflammation of affected cartilage (Figure 8.18b,c), often with superficial erosions; +/– reduced movement of cartilage.
- Ultrasonography: useful to determine extent of cartilage involvement and assist treatment planning.

Management
- Ulcers and small granulomas: most require no treatment and usually resolve spontaneously, although surgical removal of granulomas may be warranted.
- If secondary to dynamic collapse of arytenoid cartilage: consider treating underlying RLN.
- Arytenoid chondritis: rest (removal from ridden exercise) and medical treatment; systemic antibiotic and anti-inflammatory medication +/– anti-inflammatory topical throat spray. Choice of antibiotic influenced by clinical experience and antimicrobial stewardship guidelines; likelihood of mixed (gram-positive, gram-negative, anaerobic) bacterial involvement. Endoscopic monitoring guides return to ridden exercise.
- Rarely, large lesions or those that respond poorly to medical treatment may require surgical resection of affected tissue (partial/total arytenoidectomy). Considered a salvage procedure, and post-operative rehabilitation generally several weeks out of ridden exercise (until mucosa heals).

Prognosis
- Ulcers/granulomas: not associated with performance loss and heal without complication in most cases.
- Approximately 5% of horses with mucosal ulcers go on to develop chondritis.
- Occasionally, progression to arytenoid chondritis will occur requiring more aggressive treatment.
- Arytenoid chondritis: can disrupt training for weeks/months depending on severity and response to treatment; surgery is an uncommon but possible outcome. Prognosis for return to full use is fair to

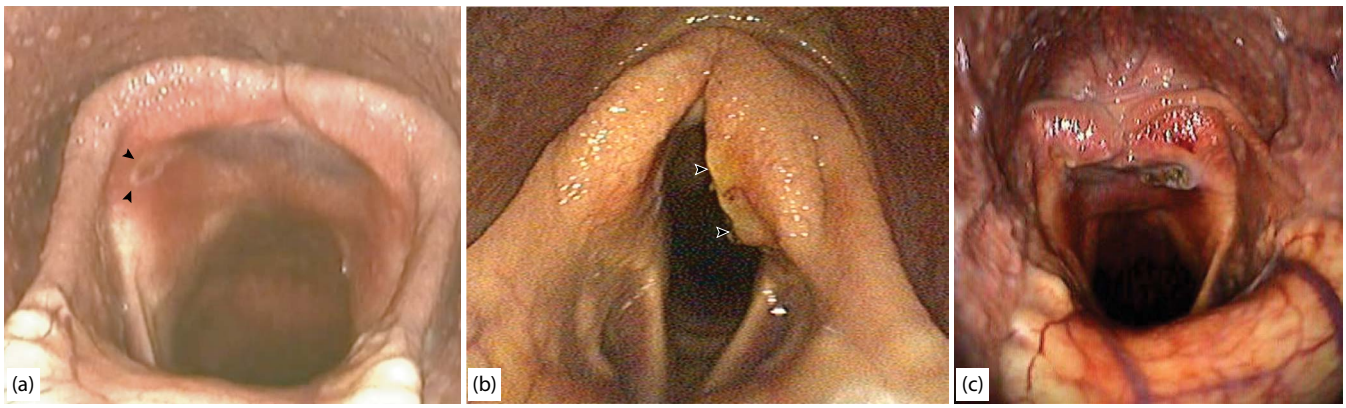


Fig. 8.18 Arytenoid chondritis: (a) mild ulceration medial to right arytenoid cartilage (most lesions of this type do not progress to chondritis); moderate (b) and marked (c) chondritis of left arytenoid cartilage.

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good; as with other upper airway conditions residual effect on racing performance (particularly following surgery) varies between individuals and remains uncertain until return to racetrack.

Laryngeal dysplasia (4th and 6th branchial arch defects; 4-BAD/6-BAD)

Syndrome of congenital defects of the cartilages and muscles of the larynx:

- 4th branchial arch defect (4-BAD) may involve the cricothyroid articulation, thyroid cartilage or muscles cricothyroideus and cricopharyngeus; individuals may be affected to varying degrees and defects may be present on one or both sides of the larynx. Also sometimes referred to as 'rostral displacement of palatopharyngeal arch' (RDPA) because absence of cricopharyngeal sphincter muscle may cause apex of corniculate processes of arytenoids to be obscured by the palatal pillars (Figure 8.19). Should be noted that this latter feature does not invariably indicate the presence of laryngeal dysplasia (may result from administration of sedation in normal horses).
- 6th branchial arch defect (6-BAD): muscular process of arytenoid cartilage is affected. Most affected individuals display varying degrees of laryngeal weakness: most commonly (65%) right-sided.

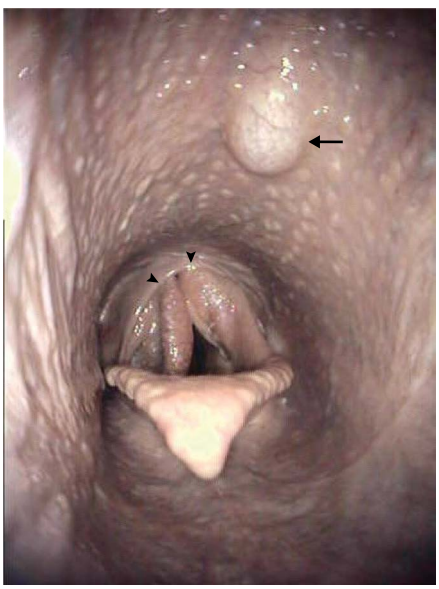


Fig. 8.19 Laryngeal dysplasia: Rostral displacement of palatopharyngeal arch (arrowheads) that may be seen with 4-BAD laryngeal dysplasia. Dorsal pharyngeal cyst (of no significance) also present (arrow).

Cause

- Incomplete development of embryological derivatives of the 4th (most commonly) or 6th branchial arches.

Risk factors

- Rare condition; prevalence approximately 0.2% in unselected yearlings.
- Risk factors unknown.
- Not considered to be heritable.

History

- Majority make respiratory noise at exercise.
- Many cases detected for first time at routine yearling endoscopy.
- Affected horses commonly 'swallow air' due to incomplete upper oesophageal sphincter and 'hiccups'/'belching' and intermittent colic may be features.

Diagnosis

- Palpation of larynx usually reveals a gap between thyroid and cricoid cartilages (should normally overlap).
- Resting endoscopy: inability to visualize corniculate process apices of arytenoid cartilages (due to a band of soft tissue) +/– right (+/– left)-sided laryngeal weakness strongly indicates laryngeal dysplasia.
- Other imaging modalities rarely needed; however:
  - Radiography: presence of column of air from pharynx into oesophagus is confirmatory (insufficiency of upper oesophageal sphincter).
  - Ultrasonography: lack of cricothyroid articulation on affected side; dorsal extension of thyroid cartilage; +/– muscular abnormalities.
  - CT and MRI: may be used to better define specific defects present in an individual.

Management

- Considered surgically irreparable at present; laser thermoplasty may be used to correct rostral displacement of the palatopharyngeal arch but cannot address underlying anatomical fault. Partial arytenoidectomy may be considered.
- Laryngeal prosthetic surgery generally unsuccessful due to abnormal cricoarytenoid articulation.

Prognosis

- Many affected horses do not enter training, as selected against at yearling stage.
- Of those that do: reasonable prognosis for racing, although few are considered effective athletes and

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strong empirical evidence that affected horses do not become elite performers.

Epiglottitis

Inflammation/infection of the epiglottic cartilage.

Causes

- Unknown.
- In some cases may be seen secondary to epiglottic entrapment (or its surgical management): possible effect of avascular necrosis/direct trauma.

Risk factors

- Rare condition.
- Any age/stage of training.
- Sometimes associated with other upper airway conditions (DDSP, epiglottic entrapment).

History

- Respiratory noise.
- Coughing.
- Exercise intolerance.

Diagnosis

- Resting endoscopy: inflammation/thickening/scarring of tip of epiglottis.

Management

- Acute phase: removal from ridden exercise plus anti-inflammatory (systemic +/– topical) and antibiotic therapy.
- Chronic phase (scarring/deformity): surgical resection may be warranted depending on severity.

Prognosis

- Most cases resolve with medical therapy.
- Some permanent deformation of epiglottis usually results.
- Occasional development of DDSP or epiglottic entrapment.

Pharyngeal cyst

Solitary cystic masses in the pharynx may be found at several sites but most commonly occur in subepiglottic position or in dorsal pharyngeal wall. Present from early in life (foal or yearling). Not invariably progressive.

Cause

- Developmental, arising from remnants of embryonic ductal tissue.

Risk factors

- Rare condition.
- Usually present from early age.

History

- Dorsal pharyngeal cysts typically an incidental finding at routine endoscopy.
- Subepiglottic cysts may cause dynamic respiratory dysfunction and can present with respiratory noise (+/– DDSP), poor performance and, less commonly, dysphagia.

Diagnosis

- Resting endoscopy: smooth, thin-walled cystic mass of variable size (Figure 8.19).
- Subepiglottic cysts may be concealed by soft palate; however, stimulation of swallowing usually results in visualization (Figure 8.20). Endoscopy from oropharynx and/or CT may be required in some cases.

Management

- Treatment of incidental or small dorsal pharyngeal cysts not necessary unless respiratory function affected.
- Surgical removal/laser ablation of subepiglottic cysts/masses readily performed as standing surgical procedure.

Prognosis

- Recurrence following ablation is rare.
- Interruption to training minimal.

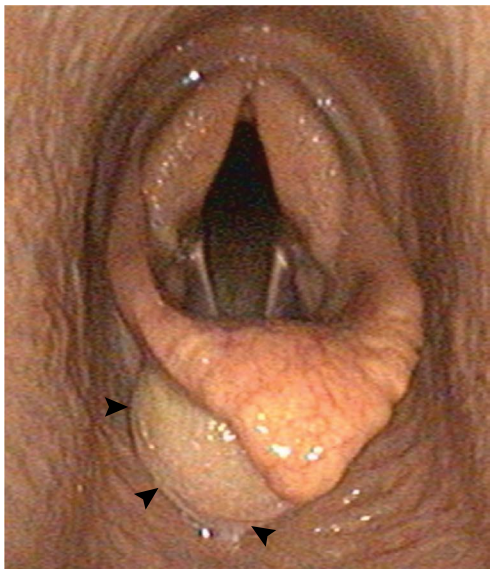


Fig. 8.20 **Subepiglottic cyst/mass.**

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LOWER RESPIRATORY CONDITIONS

LOWER AIRWAY DISEASE

Applied anatomy

Lower airways

The primary function of the respiratory tract is gas exchange; however, this only occurs in the terminal alveoli, with the remainder of the airways serving to deliver, humidify, warm and filter inspired air. The tracheobronchial tree is largely composed of cartilaginous tubes, with only the terminal airways being free of cartilage and surrounded by smooth muscle. The trachea and the bronchi that branch from it are lined with mucosa that both secretes mucus and mobilizes it in the direction of the larynx through the 'beating' action of epithelial cilia.

Mucus

Mucus is composed primarily of water, mucins and inflammatory cells and is largely produced by glands in the lining of the small airways. It plays an important role in maintaining airway health by trapping inhaled matter and bacteria and clearing it from the respiratory tract up the ciliary 'escalator'; it also contains immunologically active factors important to respiratory defence.

The amount of mucus present in the lower airways depends on both rate of production and rate of clearance (speed of removal by trachea is normally around 20 mm/min). Inflammation and infection can increase mucus secretion as well as inhibit mucus clearance (through damage to the cells lining the airway). The presence of inflammatory cells and debris increases the purulence of mucus and can change its viscoelasticity, also leading to reduced clearance.

Coughing

Coughing clears mucus and foreign material from the large (but not small) airways and is an important defence mechanism. The cough reflex can be stimulated by irritation of the lining of the larynx or lower trachea, and is mediated by nerve endings that are sensitive to both mechanical and chemical stimuli.

Assessment of lower airway disease

History

Obtaining accurate history from training staff is an important component of lower airway investigations. Diagnostic procedures/tests (discussed later) frequently do not provide certainty about the origin or likely progression of the problem, and understanding the background to the case can contribute significantly to appropriate management. Useful details include age of horse, stage of training, immediate and recent history of coughing (including number of coughs, whether before/during/after exercise, and whether increasing or diminishing over time), previous treatment, and level of recent/current respiratory disease in cohorts.

Coughing

Coughing during or after exercise is commonly used as a marker of disease by trainers; however, the horse has a relatively insensitive cough reflex and reliance on coughing for the detection of lower airway disease is inherently inaccurate. Many horses with increased tracheal mucus or blood do not cough at all. Regardless of this, coughing can contribute useful information that may assist respiratory investigations:

- Horses that cough several (≥4) times at or after exercise generally do have increased mucus; this can be used as threshold to select horses for endoscopy.
- Overall prevalence and severity of coughing in a yard can be a reasonable indicator of group health status.
- Uncharacteristic coughing (few coughs, often described as 'soft' in character) immediately after fast exercise may indicate exercise-induced pulmonary haemorrhage (EIPH).
- Very high (>15–20) and uncharacteristic cough frequency after exercise is typically associated with inhalation of track material ('kickback').

Physical examination

Nasal discharge is an unreliable indicator of lower airway disease. Bilateral purulent nasal discharge may

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accompany lower airway infection but can also arise from upper airway inflammation such as rhinitis. Haemorrhage from the nostril(s) (epistaxis) may originate in the nasal passages as well as the lungs and, when associated with EIPH, does not always correlate with severity of tracheal blood accumulation. Observation of breathing rate, respiratory effort and auscultation of the lung fields for increased or diminished respiratory sounds is indicated in cases of suspected pneumonia or respiratory distress in the systemically ill horse (but not generally for routine investigation of coughing).

Endoscopy

Resting tracheobronchial endoscopy is the most useful diagnostic technique for the detection of lower airway disease in the racehorse. It is preferably performed immediately following exercise to permit the most representative observation/sampling of lower airway secretions. Most horses can be examined with simple restraint and sedation is rarely necessary. Note is made of inflammation/discharge within the nasal passages and pharynx during passage of the endoscope. Grading systems for the quantification of increased tracheal mucus and blood (Table 9.1) are reproducible (low inter-observer variability) and are useful to assess/record severity of disease; however, they should not be considered comprehensive or free from subjective interpretation. Accumulations of mucus/blood are rarely uniform along the length of the trachea, and also vary with time elapsed and coughing stimulated by endoscopy: grading therefore should be considered only an approximation of quantity of mucus/blood rather than an absolute measurement of disease severity. Additional observations that may assist diagnosis/management include quality (viscoelasticity/colour) of the mucus and inflammation (vascularity/oedema) of the tracheal lining.

Endoscopic sampling

The most practical and rapid method of sampling lower airway secretions in racing practice is transendoscopic tracheal lavage ('tracheal wash'). Mucus and inflammatory cells accumulate in the natural 'sump' of the trachea at the thoracic inlet; instillation of 10–30 mL isotonic saline into the lower trachea through a catheter passed from the working channel of endoscope allows harvesting of this material. Sampling is not always representative and may depend on technique, mucus viscosity and coughing during the procedure. Highly viscous mucus (Figure 9.1) may fail to dislodge during lavage leading to retrieved sample having lower cellularity and mucus content than if mucus is less viscous. Coughing during procedure typically results in a retrieved sample with greater cellularity/mucus content than expected as well as contamination by pharyngeal/upper tracheal bacteria.



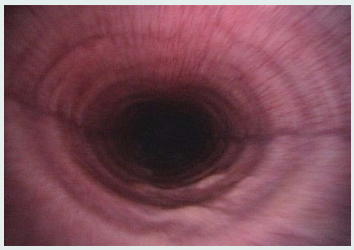
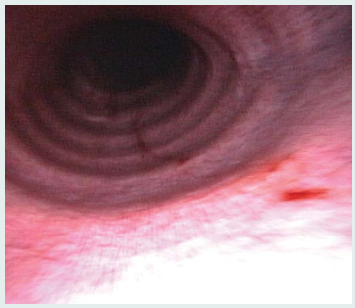
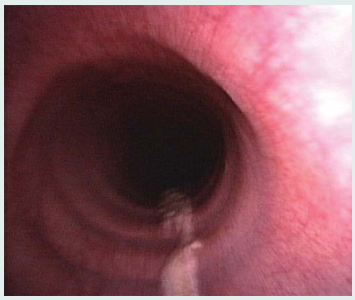
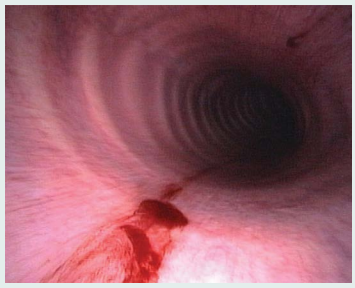
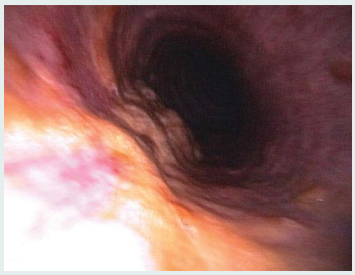
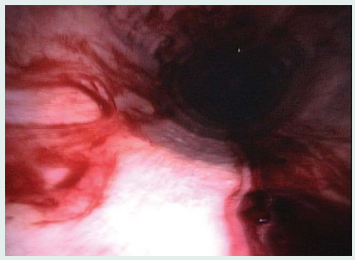
Fig. 9.1 Highly viscous/tenacious tracheal mucus may be difficult to dislodge during tracheal lavage, leading to poorly representative samples.

Tracheal lavage samples can be subjectively assessed for turbidity (Table 9.2), or submitted for laboratory analysis (cytology and bacteriology):

- Cellular content of sample can be affected by several factors; however, neutrophil proportion of >20% considered to represent lower airway inflammation.
- Cytology may permit some differentiation of infection from non-septic causes of airway disease based on neutrophil morphology: presence of 'degenerate' neutrophils in young racehorses is strongly correlated to the presence of potentially pathogenic bacteria, whereas absence of degenerate neutrophils can support a diagnosis of non-septic lower airway disease.
- Samples can also be used for bacteriological culture to assist treatment planning.
- Bacterial culture should be interpreted with caution: many affected horses have no significant bacterial growth; also the distal trachea of the normal horse is not sterile and passage of endoscope may cause contamination of retrieved sample with pharyngeal bacteria.
- Pure growth of single pathogenic organism, or quantitative culture (>10^3 cfu/mL), more likely to indicate true infection

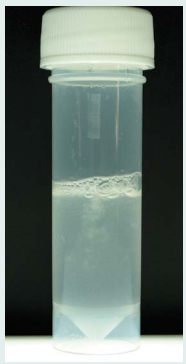
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Table 9.1 **Endoscopic grading of tracheal mucus**

TRACHEAL MUCUS		GRADE	TRACHEAL BLOOD	
No visible mucus		0		No visible blood
Singular small drops		1		One or more flecks of blood, or ≤2 short narrow (<10% trachea) streams of blood
Mild: multiple partly confluent drops		2		One long (narrow) stream of blood (>half the length of trachea) or >2 short streams occupying less than 1/3 of tracheal circumference
Moderate: thin continuous stream or several large accumulations		3		One long, or multiple streams of blood occupying more than one-third of tracheal circumference. No blood pooling at thoracic inlet
Marked: broad continuous stream		4		Multiple, coalescing streams of blood covering >90% of tracheal circumference. Blood pooling at thoracic inlet

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Table 9.2 **Grading of tracheal lavage gross turbidity**

GRADE		
0		Clear; occasional small spot of flocculent material
1		Clear with mild levels of flocculent material
2		Slightly cloudy/opaque
3		Turbid; moderate-marked flocculent material
4		Thick mucopurulent material, often difficult to aspirate

TRACHEAL ENDOSCOPY

- **Is laboratory analysis of tracheal lavage samples necessary for good decision-making?** In most routine circumstances, it is satisfactory to base immediate clinical decision-making on endoscopic findings + clinical history (of coughing, duration etc.) without laboratory analysis.
- **Does tracheal lavage analysis correlate well with disease status?** Tracheal lavage cytology is not always representative of lower airway pathology, and is also not a purely objective test: may be influenced by sample collection (e.g., technique, mucus viscosity, coughing) and storage/preparation, also cytological interpretation by pathologist.
- **Should a 'dirty' scope result stop a horse from running?** While there is a statistical association between lower airway disease and performance, translating this to practical advice about likelihood of underperformance is not straightforward; horses with significant tracheal mucus may win races. Wide array of horse and trainer factors may be as important as endoscopic appearance when deciding merits of running a 'dirty' horse.
- **How soon after exercise should a horse be scoped?** Ideally within 30–60 minutes. Mucociliary 'elevator' efficiently moves mucus/blood up from the lower airway such that delayed scoping limits accurate assessment of lower airway health. Scoping the day following a race is of little/no practical value in determining whether a horse bled or not (or the severity of any episode of EIPH).
- **Does it matter if a horse has eaten before it is scoped?** No; assessment of trachea is unaffected by whether a horse has had access to food before being scoped.

Other examinations

- Routine blood analysis is of no use in the detection of inflammatory airway disease or EIPH, but may have a role in the investigation of suspected respiratory infection in the systemically ill animal.
- Ultrasonography of the chest permits detection of fluid/gas in the pleural cavity and consolidation of lung, and is therefore an important aid in the investigation of suspected pleuropneumonia/pulmonary abscess.
- Radiography of the chest is similarly reserved for cases of suspected pleuropneumonia/pulmonary abscess or chronic/severe 'bleeders' (p. 305).

Endoscope disinfection

- Biosecurity to safeguard individual and herd health is important when conducting respiratory endoscopy.
- Sterilization/high-level disinfection between individual examinations not always feasible during routine racetrack rounds.

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- Protocol used is guided by assessment of horse-to-horse infection risk.
- Disinfection between horses in a single group or training yard is generally acceptable for routine endoscopy, whereas between training yards sterilization/high-level disinfection should be standard practice.
- Disinfection should be undertaken as soon as possible after endoscope use.
- Immersion is preferable to wipe/spray (all parts in contact) but not always possible in ambulatory practice.
- Pre-cleaning with enzymatic detergent to remove organic matter (mucus/blood): effective soak times relatively short (approximately 1 minute).
- Follow by disinfection with liquid chemical disinfectant +/– rinsing with sterile water: minimum effective concentration and soak/contact times differ between products and are specified by manufacturer; may be temperature dependent:
  - Glutaraldehyde (2%): broad efficacy but long contact times required and human health implications.
  - Ortho-phthalaldehyde: broad efficacy; 5 minutes soak time at 25°C.
  - Ethanol (70% v/v): fast-acting and good efficacy in absence of organic matter.
  - Quaternary ammonium compounds not recommended due to limited efficacy against many pathogens.
- Gas (ethylene oxide) sterilization or automated high-level disinfection of endoscope following high-risk examinations. Good practice to perform on regular basis in combination with leakage testing.

Lower airway infection/inflammation

Lower airway inflammation, characterized by increased tracheal mucus, coughing and poor performance, is commonly encountered in racehorses worldwide. It is a syndrome rather than a single disease, and factors such as bacterial and viral infection, air quality and individual immune status may contribute. Young immunologically naïve racehorses entering the training environment and exposed to respiratory challenge (mixing populations, housing and exercise) are susceptible to opportunistic pathogens and may develop lower airway infection/bronchitis. The condition is usually self-limiting (average duration 2 weeks); however, a small proportion of horses remain affected by persistent or recurrent disease (sometimes categorized as 'mild equine asthma') and appear particularly sensitized to inhaled particulate matter and further infections. Most horses typically experience several episodes during a training career, with unidentified bacterial or viral infections typically responsible. Possible link in some horses between lower airway infection, upper airway infection and dynamic upper airway obstructions.

Causes

- Multifactorial: causative factors include infection (bacterial and viral) and poor air quality.
- Bacterial infection generally more important in onset (and spread) than viral infection.
- Main bacterial species involved (*Streptococcus zooepidemicus*, *Streptococcus pneumoniae*, *Pasteurella* spp.) occur widely in normal racehorse populations, but particular strains may act as opportunistic pathogens.
- Other bacteria (e.g., *Bordetella* spp.) may be primary pathogens.
- Herpesvirus, influenza virus and equine rhinovirus may compromise respiratory defences and predispose to secondary bacterial infection.
- Exposure to airway irritants such as dust, fungi, endotoxins and noxious gases may exacerbate or prolong disease.
- Possible that training may have an immunosuppressive effect in some horses and thereby permit colonisation by opportunistic pathogenic bacteria.

Risk factors

- Any age/stage of training but most prevalent in 2 YOs.
- Risk of developing lower airway disease decreases with age and time in training (as immunity to common bacterial and viral pathogens develops).
- Influence of vaccination history (influenza/herpesvirus) and air hygiene on risk is unquantified.

Effects on performance

- Presence of mucus in airways can interfere with oxygen exchange and increase energy expenditure during breathing, resulting in reduced athletic capacity.
- Many horses train and race satisfactorily despite mild-moderate levels of tracheal mucus.
- Moderate-severe grades of tracheal mucus often linked to poor racetrack performance.
- Association with EIPH (p. 305) uncertain (Figure 9.2): common perception that continuation of high-speed training/racing in the face of significant lower airway inflammation may precipitate pulmonary haemorrhage, but likely to be true in only small proportion of cases. Conversely, known that episode of EIPH is followed by secondary (and transient) lower airway inflammation.

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Fig. 9.2 Concurrent presence of mucus and blood in trachea.

History

- Coughing at exercise is main presenting sign but is an insensitive indicator of disease and not always present (approximately 40% of affected horses cough).
- +/– poor racetrack performance.
- Increased tracheal mucus frequently an incidental finding at routine endoscopy.

Signs

- Most affected horses appear outwardly healthy.
- +/– coughing at/after exercise.
- Nasal discharge ('dirty nose') (Figure 9.3) +/– fever only in small proportion of cases.

Diagnosis

- Tracheal endoscopy (best performed immediately/within 30 minutes following exercise): excessive tracheal mucus defines the condition. Also permits assessment of features that may assist subjective differentiation of active infection from non-septic inflammation (e.g., inflamed upper/lower airway mucosa, quality of mucus, hyperreactivity to passing of endoscope).
- Grading of mucus accumulation in trachea (Table 9.1) correlates well with severity but does not provide information on duration or likely cause.
- Clinical history often a good guide to origin/progression of disease: age of horse/cough frequency and duration/previous medications/yard health.
- Transendoscopic tracheal lavage can permit sampling of respiratory secretions for subjective evaluation (turbidity/mucus viscosity/colour) or laboratory analysis.
- Bronchoalveolar lavage (BAL) considered to have greater diagnostic accuracy but less useful in clinical practice due to impracticalities (sedation/more invasive and lengthier procedure).
- Resting breathing rate and effort, auscultation of lung fields and blood analysis generally unremarkable.

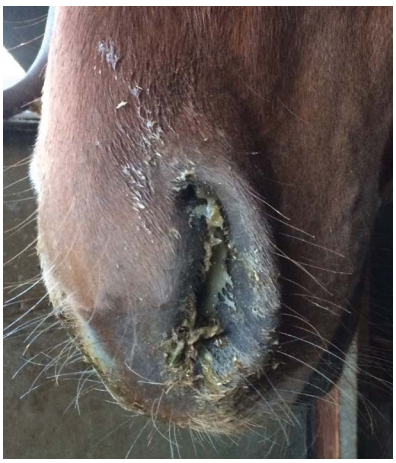


Fig. 9.3 Mucopurulent nasal discharge commonly seen with upper and lower respiratory disease in yearlings/2 YOs.

Laboratory aids

- Cytology and bacteriology of tracheal lavage sample may be used to assist management in troublesome individual cases or yard health planning.
- Laboratory findings should be interpreted with caution; consider primarily as an adjunct to clinical history and endoscopy.

Management

Individual

- Endoscopic findings and clinical history determine whether to treat as primarily an infectious or inflammatory condition.
- Management influenced by age/stage of racing season/proximity to race targets/economic constraints.
- Treatment may not be necessary or desired for young horses (yearling/early 2 YO) or those not currently in full exercise.
- Treatment usually reserved for horses in full training when poor performance or interruption to training/racing schedule is undesirable.
- If active infection suspected or confirmed: systemic antibiotic medication (to effect; typically 7–10 days). Choice of antibiotic guided by antimicrobial stewardship principles and efficacy against common respiratory pathogens.
- Exercise: guided by clinical severity; avoidance of fast exercise/racing during course of active disease usually recommended. Validated protocols do not exist; however, cantering may generally continue.
- Periodic endoscopic review useful to monitor resolution/progression.

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- Persistence of tracheal mucus after initial treatment: consider further anti-inflammatory +/– mucoactive therapy.
- 'Mucoactive' agents can modify clearance of mucus from lower airways: 'expectorants' (increase water volume of airway secretions so that more readily coughed up), 'mucolytics' (reduce mucus viscosity) and 'mucokinetics' (increase clearance rate by directly acting on ciliary cells). Commonly used mucoactive agents include clenbuterol, dembrexine and sodium/potassium iodides.
- Corticosteroids (systemic or inhaled), improved air hygiene (haylage or soaked/steamed hay, stable ventilation), mucolytic or expectorant drugs or treatments (e.g., nebulization with inhaled hypertonic saline) and inhaled bronchodilators may have a role in individual therapy, particularly in chronic/poorly responsive cases. Before commencing treatment with corticosteroids important to establish with reasonable certainty that no active infection is present.
- Decision-making regarding whether to permit an affected horse to race is influenced by severity of endoscopic findings, history of coughing/performance/treatment and trainer-influenced factors such as expected level of performance/aversion to risk of underperformance. Excessive tracheal mucus does not preclude satisfactory racetrack performance.

Group

- (See also Chapter 21).
- Generalized increase in coughing or poor racing performances may warrant wider investigation and/or treatment within yard.
- Assess incidence of disease in the group through endoscopy of a sample of horses (including those thought to be healthy).
- Bacterial opportunistic pathogens naturally circulate in racing yards; biosecurity measures intended to eradicate infection are unrealistic but minimizing shedding, spread and exposure may be beneficial.
- Treatment of choice is dependent on stage of season/level of disease in yard/suspected cause.
- Treatment of affected/at-risk group (e.g., 2 YOs) justifiable in limited circumstances if applied responsibly and in compliance with antimicrobial stewardship guidelines.
- Endoscopic monitoring of horses with upcoming race targets to identify subclinical cases, instigate timely treatment and lower risk of poor racetrack performances.

Prevention

- Inevitable that large proportion of horses will be affected at some point in their first year in training.
- Goal is to reduce susceptibility to disease (vaccination programme/biosecurity/air hygiene) and assist recovery (medication/exercise modification) so that total lost training days are minimized.
- Timing of influenza/herpesvirus vaccinations to account for high-risk periods.
- Preferable to introduce yearlings to yard only after end of main racing season (if there is to be close proximity to older horses).
- Quarantine protocol for new entrants during racing season.
- +/– use of immunostimulants (*Parapox ovis* virus/*Propionibacterium acnes*-derived products) in selected horses prior to high-risk periods: some evidence of improved non-specific immune response/reduced disease severity.

Prognosis

- Most episodes are self-limiting and regardless of management resolve within several weeks.
- Small proportion of horses display persistent disease due either to recurrent infections or to non-septic lower airway reactivity, and require enhanced attention to stabling, air hygiene and ongoing/repeat medical therapies. Most such cases resolve or improve (requiring less veterinary input) during subsequent racing season/s; progression to severe asthma ('heaves') does not appear to occur.

Exercise-induced pulmonary haemorrhage ('bleeding')

EIPH is a condition in which bleeding occurs into the small airways of the lung, typically during strenuous exercise. Caused by rupture of small capillaries in the dorsocaudal lung lobes. Severity varies between individuals and regardless of severity usually goes unnoticed; blood rarely appears at the nostrils (Figure 9.4a,b). Progression of condition is unpredictable and degree of bleeding on different occasions is not always consistent. In the majority of horses causes no significant problems but in a small proportion can lead to loss of performance when severe and persistent. Physiological adaptations of the Thoroughbred (high cardiac output, pulmonary arterial pressures and very large gas exchange capacity) may predispose to the condition.

Causes

- Stress failure of pulmonary capillary wall occurs when pressure across wall exceeds wall strength. Forces arise from disparity between very high blood pressure

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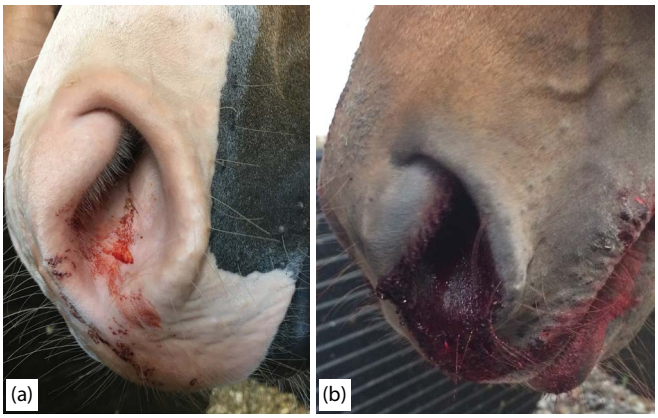


Fig. 9.4 **(a,b) Epistaxis typical of EIPH.**

in pulmonary arterial supply during fast exercise and subatmospheric pressure in alveolus during inspiration, leading to 'bursting' of blood through the very thin vessel/alveolar (blood-gas) tissue barrier (Figure 9.5).

- Chronic exercise-induced hypertension in dorsocaudal lung leads to remodelling (thickening of walls and reduced compliance) of the pulmonary veins, which in turn causes local increases in pulmonary capillary pressure.
- Subsequent haemorrhage causes small airway inflammation and has potential to lead to fibrosis in lung.
- Interface between normal and fibrosed lung tissue is mechanically more prone to further stress failure, such that repeated episodes of EIPH may result in further damage to lung.
- Involvement of progressively larger areas of lung tissue increases risk of more severe episodes.
- Impact of forelimbs striking the ground creates shear waves that are focused in dorsocaudal lung fields and may contribute to the condition.

Risk factors

- Risk factors summarised in Table 9.3.
- May be encountered in any horse in cantering/fast exercise.
- At microscopic level (presence of haemosiderophages in respiratory samples) all racehorses in fast exercise (and most in moderate exercise) have some evidence of bleeding ('all horses bleed'); however, at a practical level this is clinically insignificant in most cases.

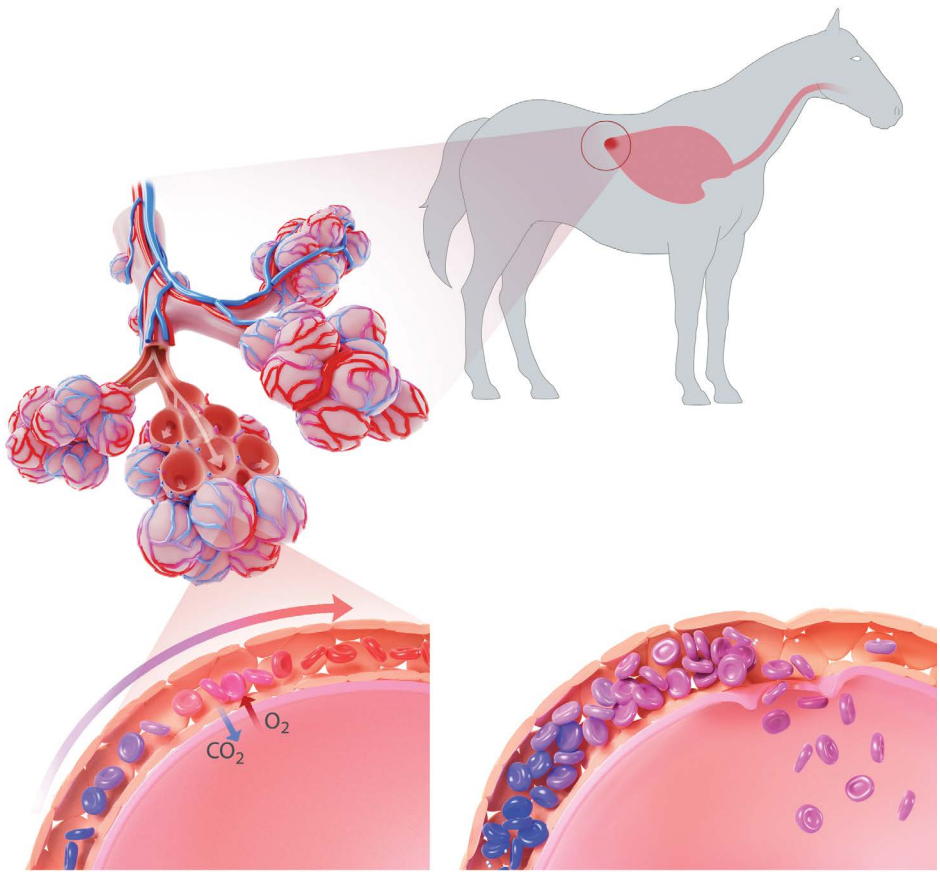


Fig. 9.5 **EIPH: Pressure equilibrium that permits exchange of gases and oxygenation of blood in the alveoli (left) is disrupted in EIPH, resulting in stress failure of the blood-gas tissue barrier (right) and entry of blood into the small airways.**

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Table 9.3 **Risk factors for EIPH +/– epistaxis**

Stage of career	Risk of EIPH increases with both years in training and total number of career starts.
Heritability	Whether EIPH is heritable remains unknown; high/universal prevalence in racehorse population makes heritability investigations problematic. Suspected genetic component to more severe forms of the condition (including epistaxis); however, quality of evidence to date is low.
Upper airway function	No direct link with upper airway obstructions (including RLN and DDSP), although previous laryngeal 'tie-back' surgery may predispose to EIPH and epistaxis.
Lower airway disease	No direct link with respiratory infection. EIPH causes transient non-septic lower airway inflammation (in response to blood in airways and damage to alveolar/capillary tissues); conversely only very low quality evidence to support premise that lower airway inflammation causes EIPH.
Season and temperature	Significantly increased risk of EIPH, and severity of EIPH, with colder ambient temperature. Prevalence of epistaxis in most countries is greatest in autumn/winter/spring.
Air quality	In the stable: no difference in risk between bedding on paper or straw. At the track: some anecdotal indication that poor air quality (dust/pollution) may increase the incidence of EIPH but good evidence lacking.
Race type/conditions	Greater risk of epistaxis with steeplechase/hurdle (approximately 3–5/1000 starts; 0.34–0.54%) races than with flat racing (approximately 1/1000 starts; 0.13%). Flat racing: epistaxis more prevalent over sprinting distances in UK (conflicting evidence between countries). Increased risk of epistaxis with faster (harder) ground. Greater risk of more severe EIPH with faster early race speed.

- During training exercise: <10% horses have visible tracheal blood at post-exercise endoscopy.
- Racing: approximately 50% horses have visible tracheal blood at post-race endoscopy; majority of these bleeds are minor.
- Between 5% and 15% of horses have grade 3 or 4 EIPH (Table 9.1) post-race and less than 0.5% of race starts are associated with blood from nose (epistaxis).
- Epistaxis affects approximately 2–3% of the racing population.
- Possible that factors which increase airway resistance (such as dynamic upper airway obstructions and lower airway inflammation) may affect pressure gradient across alveolar blood-gas barrier and heighten risk of vessel rupture; however, consistent statistical link with these conditions has not been established.
- Cold ambient temperature consistently linked with risk of serious EIPH; reasons underlying this poorly understood; however, exercising in cold temperatures has been associated with lower respiratory tract inflammation/bronchial constriction/pulmonary hypertension in other species.
- Increased risk with faster early race speeds (compared with incremental increase in speed) and anecdotal link with excitability/failure to settle in race in some individuals; possible relationship with pulmonary arterial pressure gradient.

History

- Frequently an incidental finding at routine tracheal endoscopy.
- May be associated with poor performance or coughing following exercise.

Signs

- Most episodes of EIPH are subclinical: no coughing/epistaxis/poor performance.
- More severe grades of EIPH may be accompanied by coughing after exercise.
- Epistaxis following exercise does not invariably imply EIPH: may also arise from nasal passages/sinuses.

Diagnosis

- Tracheal endoscopy is most practical method of determining severity of episode(s) (visual grading, Table 9.1) but does not permit assessment of severity of underlying pathology.
- Laboratory analysis of tracheal lavage sample: provides little additional information of practical use. Presence of red blood cells (or macrophages – haemosiderophages – containing their breakdown product haemosiderin) is specific and sensitive; however, all horses in fast work have microscopic evidence of EIPH (which may persist for many weeks beyond episode).

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- BAL is the most sensitive method for diagnosing EIPH; however practicalities limit usefulness in first opinion practice.
- Radiography: rarely employed but may be indicated in chronic/severe cases to confirm extent of pathology. Most horses with EIPH have few radiological changes; chronic 'bleeders' have bronchointerstitial pattern of density in dorsocaudal lung fields.

Effects on performance

- Bleeding into lung during exercise has potential to affect gas exchange and therefore performance, although evidence to support this is weak.
- Detrimental effect on performance dependent on severity of episode: generally considered that mild episodes have no/negligible impact on race outcome.
- Strong association between severe bleeding episodes (grade 4 +/– epistaxis) and poor performance. Severe EIPH episode in any given race linked to slower final 600 m/less likely to win or place than horses without evidence of EIPH.
- Despite this, many individual instances of horses performing well at elite level (and over all race distances) in spite of moderate-severe EIPH.

Management

- Background information important when attempting to determine significance of single episode of EIPH (previous endoscopy findings/exercise intensity/recent lower airway disease). Without sufficient information it is difficult to advise on management, and further endoscopic examinations (following fast exercise) are useful.
- Uncharacteristic or single severe episode: diagnostic investigation (blood sampling/tracheal endoscopy) to establish underlying respiratory/systemic health may be warranted. If no concerns about intercurrent disease, preferable to resume training and determine whether EIPH is persistent or transient rather than prescribe rest period if not required. When encountered at slow speed in young horses (2 YOs): generally transient episode of unknown origin, and of no long-term concern.
- If concerned about poor systemic/lower airway health contributing to EIPH episode, consider rest from high-speed exercise. However, as there are no validated recommendations for optimal period of rest, individual circumstances will guide whether/for how long horse is permitted light exercise. Lengthy spelling periods are rarely warranted and do not improve long-term prognosis.
- Recurrent 'bleeders': address any concurrent respiratory disease (lower airway inflammation/upper airway obstruction), minimize severity of bleeding at fast exercise during training (Table 9.4) and maximize recovery times between severe episodes.
- Prophylactic use of furosemide (for high-speed exercise training sessions) is rational, effective and recommended; however, whether such management results in overall mitigation of clinical deterioration (or improved career outcome) remains untested scientifically.
- Race day treatment/management restricted in most jurisdictions.
- Total or partial water deprivation for variable period pre-race is sometimes practiced but efficacy is untested and no validated recommendations (that account for wide range of individual factors such as environmental conditions, travel distance, race distance etc.) currently exist.
- Wide range of dietary/herbal supplements (including bioflavonoids, antioxidants, vitamin K) marketed for management of EIPH; purported to have diverse effects on capillary wall strength, airway inflammation and blood coagulation. None of these have validated efficacy and at present should not be considered as practically useful therapies for prevention or treatment.
- Wide range of pharmaceuticals (including conjugated oestrogens, aminocaproic acid, bronchodilators, anti-inflammatories, vasodilators) have been/are used for prophylaxis against EIPH; evidence of efficacy for all such interventions (other than furosemide) is absent or weak.
- Anecdotal evidence that controlling excitability and/or racing pace is effective preventative in some individuals.

Prognosis

- Not invariably progressive and performance of horses displaying mild forms of condition may be unaffected throughout career.
- Risk of suffering a repeat episode of serious EIPH with subsequent racing is not predictable: >40% of horses will display either no or very mild EIPH at next race after a serious (grade 4) bleed.
- Prognosis for horses suffering an episode of epistaxis is guarded: recurrence of epistaxis is common (>30%) and does not appear to be influenced by treatment or rest.
- More severely affected horses may display continued or slowly worsening EIPH over time and when associated with poor performance may justify retirement.
- Career longevity can be jeopardized by progressive nature of condition and penalties for epistaxis in some jurisdictions; however, only appears to be important

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Table 9.4 **Management aids for EIPH**

TREATMENT	ACTION	RESEARCH SUPPORT FOR EFFICACY
Furosemide	Diuretic with short half-life that increases urine production (starting 15–30 minutes after administration and lasting for 2–3 hours). Reduces pulmonary capillary blood pressure and additionally is a potent bronchodilator. Dosage: 0.5–1.0 mg/kg BWT IV 2–4 hours pre-exercise (restrict water from time of administration); no apparent difference in efficacy between administration 2 or 4 hours pre-exercise. Poor/variable absorption of oral formulation.	Strong. Reduces incidence and severity of EIPH (by at least one grade in >60% horses). Enhanced racetrack performance thought to be due at least in part to weight loss arising from diuresis.
Herbal diuretics	Herbal extracts used in traditional human medicine as purported diuretics include *Sambucus* spp., *Zea mays* and *Orthosiphon stamineus*	None/weak
FLAIR nasal strip	Acts to reduce upper airway resistance to inspiratory airflow caused by collapse of the highly moveable nasal valve region.	Uncertain. Clinical effect on severity of EIPH less than that seen with furosemide.
Haemostatic/ antifibrinolytic drugs	Drugs that have an effect on blood coagulation cascade: include aminocaproic acid, carbazochrome and tranexamic acid. Rationale for use is tenuous, as no indication that EIPH is linked to defective coagulation or fibrinolysis.	None/weak
Vasodilatory drugs	Some theoretical rationale for use of drugs that selectively address pulmonary hypertension.	None/weak
Bioflavonoids, vitamins C and K, herbal haemostatics (e.g., Yunnan Baiyao)	Proposed to improve integrity of capillary wall/improve blood coagulation, thereby potentially limiting EIPH.	None/weak
Omega-3 fatty acids	When supplemented over months may reduce lower airway inflammation.	Uncertain
Inhalation therapies (water, chelated silver, other)	Variety of potential actions including reduced lower airway inflammation +/– respiratory resistance.	None/weak

Research support definitions: **strong** (studies generally support effectiveness); **uncertain** (some positive findings are available, but confirming research needed); **none/weak** (little or no positive data available).

for horses with highest grade (grade 4) EIPH or epistaxis. Horses with mild to moderate grades (1–3) of severity do not have curtailed careers.
- No evidence that EIPH is associated with increased risk of sudden death during/resulting from racing.

Pleuropneumonia

Sporadic but serious condition in which infection of the lungs spreads to the pleural space. Potentially life-threatening. Typically encountered as isolated cases (non-contagious), but clusters of disease in racing yard/training centre occasionally occur and may follow respiratory disease in wider population.

Causes

- Breakdown in normal defence mechanisms of lung leading to colonization of lower airways by oropharyngeal bacteria.
- Aerobic bacteria (beta-*Streptococci*, *Pasteurella*, *Escherichia coli* and *Enterobacter* spp.) most commonly involved; however, mixed anaerobic infections (*Bacteroides*, *Clostridium* spp.) are responsible for up to a quarter of cases.

Risk factors

- Rare condition.
- Any age/stage of training.
- Recent (within past week) long-distance travel, exposure to viral respiratory disease or confinement with head elevated (during travel or due to injury) are recognized risk factors.
- Inability to lower head impairs normal postural drainage of respiratory secretions and leads to bacterial contamination of lower airways.

History

- Development of signs usually acute.
- Recent history of coughing and/or recurrent fever is typical.

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Signs

- Fever/depression/lethargy, sometimes profound.
- +/– coughing.
- Increased respiration and heart rates, with increased respiratory effort.
- Pleural pain may mimic low-grade colic or cause horse to appear stiff/laminitic.
- +/– sternal oedema.
- +/– nasal discharge.

Diagnosis

- Blood analysis: marked acute inflammatory profile.
- Auscultation of chest: increased lung sounds dorsal fields +/– absent/muffled lung sounds ventral fields.
- Ultrasonography: fluid in lung parenchyma or pleural space +/– consolidated lung tissue (Figure 9.6); gas echoes are indicative of anaerobic infection. Assessment of deep lung tissue not possible.
- Radiography: interstitial or alveolar pattern of opacification of lung tissue (Figure 9.7a) +/– evidence of pulmonary abscess formation (Figure 9.7b,c)
- Tracheal endoscopy: tracheal mucopus not invariably present.

Management

- Require intensive management and hospitalization recommended.

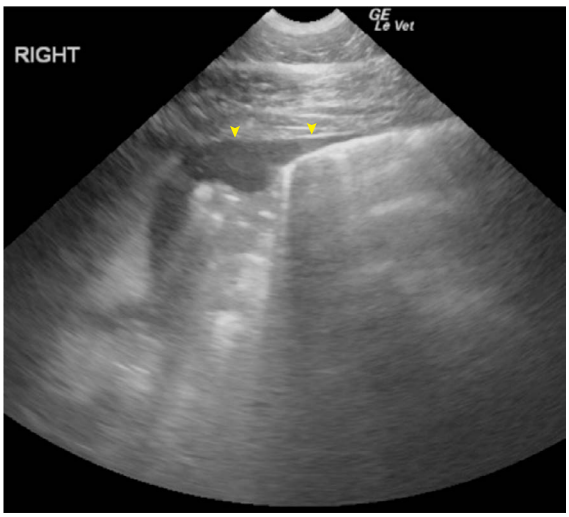


Fig. 9.6 **Pleuropneumonia: Transcutaneous ultrasonogram showing pleural fluid (arrowheads) and consolidated lung.**

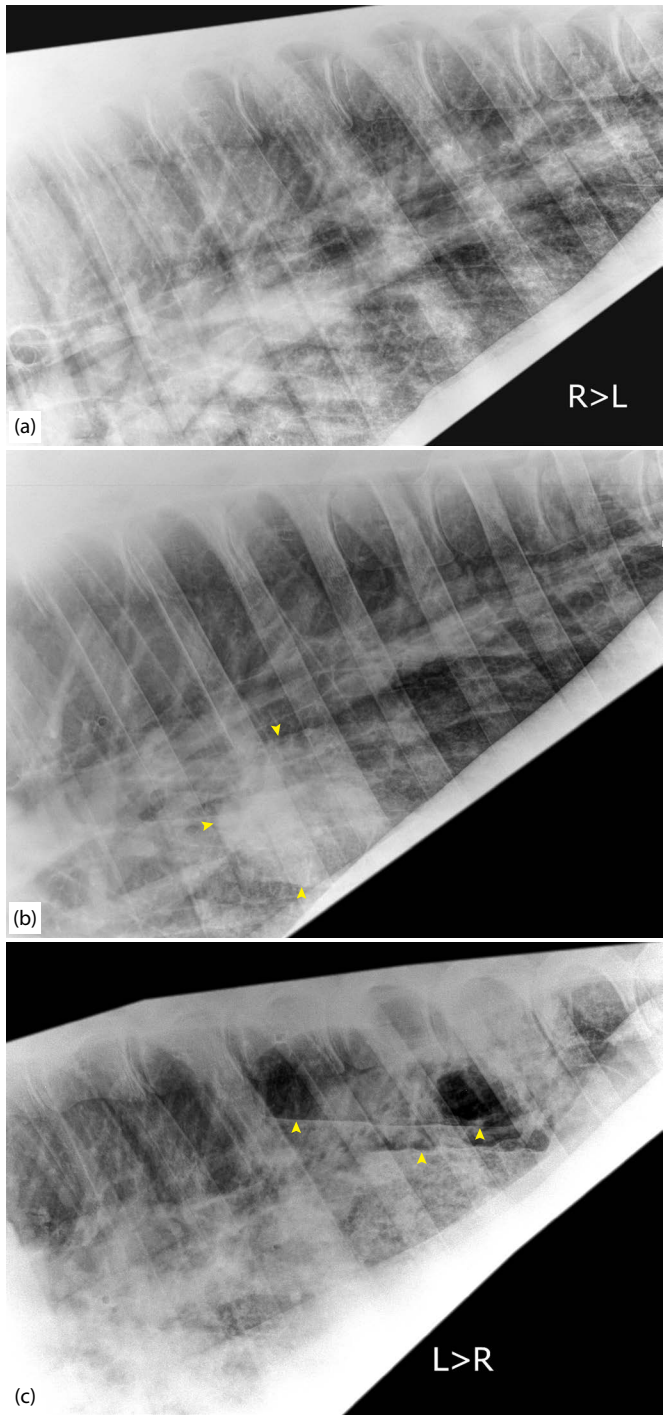


Fig. 9.7 **Pneumonia/pleuropneumonia: Radiological features may include diffuse 'interstitial' pattern of radiopacity throughout lung(s) (a); focal density (b) and fluid/gas interfaces (c) consistent with abscessation.**

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- Ultrasound-guided drainage of any substantial pockets of fluid (+/– indwelling drain) as required.
- Aggressive antibiotic and anti-inflammatory medication, guided by culture/sensitivity of sampled fluid. Duration of treatment 4–6 weeks.
- Supportive/intensive care (IV fluids/anti-endotoxic therapy) as required.
- Condition/progress monitored through repeat haematology/ultrasonography/radiography.

Prognosis

- Reasonable prognosis for survival (60–90%) with appropriate management.
- Anaerobic infections tend to be associated with poorer outcomes.
- Horses that survive have a good prognosis for return to racing (60%) regardless of duration of hospitalization.
- Complications (pulmonary abscess/thoracic mass/thrombophlebitis) associated with poorer prognosis for racing.

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HEART MURMURS

Heart murmurs are abnormal sounds, generated in or around the heart, that are detectable during auscultation of the chest. They arise from turbulence of blood flow and may be physiological or the result of structural or functional defects such as heart valve insufficiency. Both physiologic and clinically relevant murmurs are common. Murmurs are defined by their timing during the heart cycle (systolic or diastolic), their intensity and quality of sound and their location as determined by point of maximal intensity over the left or right chest walls (Table 10.1). Auscultation is reasonably specific and sensitive for detection of valve regurgitation. Significance varies greatly and is dependent on type of murmur, age of horse, stage of training and racing form. Loud murmurs or those of uncertain origin

Table 10.1 **Types of cardiac murmur**

CLASSIFICATION	DESCRIPTION	CAUSE	PREVALENCE	SIGNIFICANCE FOR RACING
Atrioventricular (AV) regurgitation	Systolic murmur, loudest on affected side of heart	Backflow through left or right AV valve	Incidence and intensity increase with training and age, largely due to adaptation of cardiovascular system to training (eccentric hypertrophy of heart muscle/ increased blood volume)	Valve leakage implies reduced cardiac efficiency, but generally no link between presence/severity of AV murmurs and racing performance
Tricuspid	Right-sided (heart base) holo- or pansystolic murmur	Backflow through right AV valve during systole	Common in large/mature trained racehorses (>50% of mature steeplechasers); up to 25% of trained 2 YOs	Usually low/none Echocardiography usually indicated only for loud murmurs +/– poor performance
Mitral	Left-sided holo- or pansystolic murmur, loudest over apex	Backflow through left AV valve during systole	Uncommon in yearlings but prevalence increases with training (up to 20% of racehorses in training)	May be associated with poor performance if loud +/– increased resting HR (particularly in untrained horse): requires echocardiography for prognosis
Aortic regurgitation	Diastolic murmur, audible from both sides of chest but usually loudest on right side	Backflow through aortic valve during diastole	Rare in yearlings/young racehorses (<2%) but prevalence increases with age	Potential to be associated with poor performance but determine on individual basis and aided by echocardiography (+/– ECG, to detect ventricular arrhythmias)
Systolic ejection	Early systolic murmur; intensity can increase with higher HRs such as during/after exercise	Turbulence in region of aortic or pulmonary valves	Very common in normal racehorses	Low/none
Ventricular septal defect	Right-sided (often audible left side) loud harsh pansystolic murmur	Congenital defect of ventricular wall permitting left-to-right shunt	Rare	Dependent on defect size but potentially highly significant, although can be found in effective athletes; echocardiography to determine severity/risk of poor performance

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require dynamic imaging of the heart (Doppler echocardiography) +/– electrocardiogram (ECG) exercise testing before a judgement on effect on performance can be made. Accurate assessment of severity and prognosis for deterioration is challenging and is a medical specialism. In a pre-purchase context an echocardiographic examination is advisable for any horse with a moderate to loud, mid- to late-systolic/holosystolic/pansystolic murmur or any holodiastolic decrescendo murmur. Echocardiography enables imaging of heart valves, quantification of severity and direction of any valvular backflow and measurement of cardiac dimensions. Negative prognostic factors include evidence of volume overload (heart chamber or great vessel enlargement/altered shape), structural valve lesions and secondary arrhythmias. Follow-up examinations (interval determined by clinical concern) may be useful to monitor cardiac function and refine prognosis.

ARRHYTHMIAS

The pacemaker of the heart, the sinoatrial (SA) node, is located in the right atrium and drives normal ('sinus') heart rhythm. It generates regular electrical signals that travel through the atria, down to the atrioventricular (AV) node and eventually to the ventricles. This sequential depolarization enables contraction of first the atria then the ventricles, with coordinated filling and ejection phases required for each effective heartbeat. Interruption to normal rhythm can occur at several points in the circuit and may be physiological or pathological, and isolated or secondary to other factors (such as structural heart disease, metabolic and medical disorders). The spectrum of cardiac arrhythmias (Table 10.2) ranges from clinically irrelevant to life-threatening; scientific understanding of the causes and relevance of different types of arrhythmias is limited but evolving.

Table 10.2 **Types of cardiac arrhythmia**

CLASSIFICATION	DESCRIPTION	CAUSE	PREVALENCE	SIGNIFICANCE FOR RACING
Second-degree AV block	Regular 'dropped beats' in 'regularly irregular' rhythm; usually abolished by exercise or excitement	Inherent blood pressure regulatory mechanism in horse: blocked conduction of impulses generated by the pacemaker	Entirely normal feature, occurs in most horses at rest	None
Ventricular premature complexes (VPCs)	Early extra beat, followed by long compensatory pause	Depolarizations arise from ventricular muscle	Relatively common during/after exercise in normal horses	In general are not associated with poor performance. May warrant further investigation if frequent (particularly at rest), or history of poor performance/collapse. Several VPCs in succession (ventricular tachycardia) may indicate underlying disease
Atrial premature complexes (APCs)	Extra early beat, followed by normal beat interval (resetting of SA node)	Depolarizations arise from outside the SA node	Relatively common in normal horses, particularly before/during/after exercise	In general are not associated with poor performance. May warrant further investigation if frequent (particularly at rest)
Atrial fibrillation (AF)	Chaotic ('irregularly irregular') heartbeat +/– high resting HR	Pacemaker impulses from disorganized waves of depolarisation in atrial muscle: loss of coordination between atrial and ventricular contraction. Sometimes caused by underlying heart disease but in most racehorses is purely an electrical disturbance	Rare as clinical entity, although paroxysmal AF is the most common arrhythmia in post-race period	High potential for significance, although clinical relevance in immediate post-race period needs further investigation. Cardiac output compromised (ineffective filling of ventricles); not usually evident at rest but can cause severe exercise intolerance

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Arrhythmias are common in the immediate post-exercise period in racehorses and when only present (transiently) in this period the significance to performance is considered doubtful. To some extent arrhythmias of all types are potentiated by both chronic changes to the heart that occur during athletic training and fluctuations in electrolyte and autonomic regulation that occur during strenuous exercise. Auscultation of the heart often permits detection (and differentiation) of arrhythmia, but ECG examination is typically required for definitive diagnosis. Undertaking ECG at exercise or continuously over a longer time period (24+ hours) may be necessary for detection of transient arrhythmias or to establish a relationship with poor performance. Risk assessment for the likelihood of a serious adverse event (poor performance, collapse or sudden death) developing secondary to arrhythmia in any individual is complex and a medical specialism.

Atrial fibrillation

Atrial fibrillation (AF) is the most common arrhythmia detected in poorly performing horses. In racehorses AF is usually transient or 'paroxysmal', arising without warning during exercise and causing a sudden loss of performance, pulling-up or even collapse. This may be accompanied by epistaxis (exercise-induced pulmonary haemorrhage [EIPH], p. 305). Return to normal sinus rhythm may occur within minutes or hours. Less commonly (but more typical in older horses), persistent AF may occur; in these cases (and those of recurrent paroxysmal AF) it is thought that underlying microstructural cardiac lesions, electrical remodelling or activity originating in the muscle of the pulmonary vein may be contributing factors.

Paroxysmal AF requires no treatment, as horses return to normal rhythm within a short time (<72 hours). If recurrent paroxysmal AF is suspected as a cause of poor performance, an exercising ECG should be performed.

Cases of sustained/persistent AF (>72 hours) should undergo prompt treatment if return to racing is desired; conversion to normal sinus rhythm may be achieved through medical therapy (oral quinidine sulphate) or transvenous electrical cardioversion; in both cases hospitalization is necessary. Both treatment paths have associated risks (including, rarely, of fatal outcome); comparative efficacy is uncertain but longer-term success rates currently thought to be similar. Prognosis following return to normal sinus rhythm is good, although longer-standing cases or those with underlying heart disease carry a more guarded prognosis. Recurrence not uncommon but timing is unpredictable and highly variable (up to years) and horses can perform successfully between episodes. Recurrence more common for persistent AF than paroxysmal AF. Rapid return to training (<1 week) following paroxysmal or short-duration persistent AF is possible, whereas a 4- to 6-week rest period is advisable for horses converted from long-standing AF.

Heritability of AF is considered to be low.

MISCELLANEOUS CONDITIONS

Jugular thrombosis/thrombophlebitis

Jugular thrombosis/thrombophlebitis is inflammation of the wall of the jugular vein, followed by formation of a blood clot (thrombus) and resulting in partial or total blockage of the vein. Most frequently occurs following catheterization for fluid or drug administration during hospitalization. It is usually unilateral and, once formed, the obstruction is permanent, although partial re-canalization may occur over time.

Causes

- Inflammatory cascade leads to clot formation within vein.
- Usually caused by irritation/inflammation of venous endothelium (perivascular or intravascular injection of irritant drugs/long-term catheterization).

Risk factors

- Hospitalized horses with endotoxaemia (e.g., diarrhoea/colic) at greatest risk: hypercoagulable state potentiates onset.
- Intravenous (IV) administration of large-volume non-sterile fluids or irritant (e.g., hyperosmolar) solutions associated with increased risk of development.

History

- Acute onset: during or immediately following period of jugular catheterization or repeated IV injections.

Signs

- Acute phase: swelling/heat/inflammation over affected jugular vein +/– fever.
- Subcutaneous veins on affected side of face and upper neck are prominent/congested due to impeded venous return (Figure 10.1). In acute phase may be accompanied by swelling of face/lips.
- Chronic phase: non-painful but affected part of vein palpably fibrous/'corded'.
- Patency can be assessed by occluding jugular vein at base of neck and observation of venous filling.
- If bilateral (rare): initial venous congestion may be profound and cause respiratory obstruction.

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Fig. 10.1 Jugular vein obstruction secondary to thrombus: Engorgement of superficial veins on affected side of face/head.

Diagnosis
- Clinical findings definitive.
- Ultrasonography (aided by manual occlusion of vein) may permit assessment of extent of clot (Figure 10.2a,b).

Management
- Acute phase: aggressive management to limit extent of clot formation. Systemic anti-inflammatory and antibiotic medication (avoiding IV administration) +/– topical dimethyl sulfoxide (DMSO) +/– hot packs. Thrombophlebitis usually sterile but potential for bacterial colonization merits use of antibiotics.
- Anticoagulant (heparin, aspirin) +/– local fibrinolytic therapy (streptokinase) may be useful in acute phase.
- Once established/chronic, medical therapy is of little use: no treatment required.
- Very rarely, unresolved septic thrombophlebitis may require surgical intervention (thrombectomy).

Prognosis
- Permanent partial/total blockage of one or both jugular veins is of little importance beyond the acute phase due to development of collateral/compensatory venous return.
- No effect on racing performance.
- Main long-term consequence is inconvenience for future IV administration of drugs.
- Very rarely, septic thrombophlebitis may seed infection/clots elsewhere in body such as lungs, heart valves and synovial spaces (latter resulting in closed synovial sepsis).

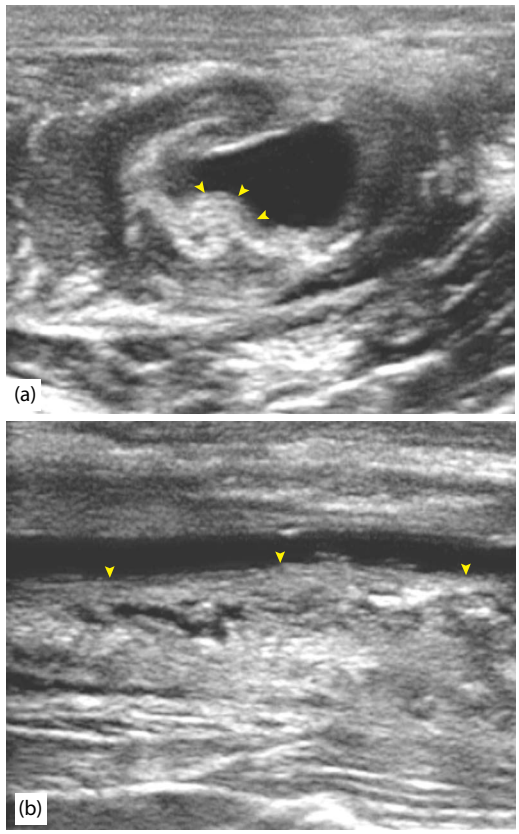


Fig. 10.2 Jugular vein thrombophlebitis: Transverse (a) and longitudinal (b) ultrasonograms showing an organizing intraluminal thrombus (arrowheads).

Aorto-iliac thrombosis

Chronic condition in which presence of a blood clot (thrombus) at the termination of the major arterial blood supply to the hindlimbs/abdomen may cause partial or total arterial obstruction and result in exercise-induced lameness and signs of discomfort. In the caudal lumbar region (at the level of L5/L6) the aorta gives off the two external iliac arteries (which course distally in the limbs to become the femoral arteries) then terminates as the two internal iliac arteries. Location and extent of thrombus varies between individuals but generally considered to be a slowly progressive disease.

Causes
- Several proposed (but not substantiated) theories: vessel wall damage from parasitic larval migration/exercise-related haemodynamic factors; lodgement of 'free' thrombi from elsewhere in cardiovascular system.

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- Resulting restricted blood flow to (predominantly) muscles becomes clinically evident during/after exercise (when metabolic demands of muscles/limbs are increased).

Risk factors
- Rare condition.
- Risk factors unknown.

History
- Usually present with acute or insidious history of intermittent post-exercise discomfort or hindlimb lameness.

Signs
- Signs are predominantly of 'intermittent claudication': limb/muscular pain arising from insufficient blood supply to muscles during exercise.
- Spectrum of signs depending on extent of arterial occlusion.
- Typically: progressive hindlimb lameness (unilateral or bilateral), which occurs immediately after exercise, is quickly (within 30–60 minutes) relieved by rest, and which is reproducible when exercise repeated. Lameness may be accompanied by signs of discomfort/colic/sweating/recumbency.
- Affected hindlimb may have reduced digital pulses/reduced saphenous vein fill/be palpably cooler than unaffected limb.
- Presentation may resemble exertional rhabdomyolysis.
- Mild signs may be non-specific (poor performance/tailing off/fatigue).

Diagnosis
- Clinical findings strongly indicative, but diagnosis may be challenging as low prevalence means potential involvement rarely considered by examining clinician.
- Transrectal (for terminal aorta) and transcutaneous (upper medial thigh, for femoral artery) ultrasonography usually definitive: presence of thrombus in lumen of affected artery (Figure 10.3).
- Bone scan (vascular phase) sometimes aids diagnosis in challenging cases.

Management
- As usually established/chronic by time of diagnosis, medical therapy is generally of limited use; however, pentoxifylline +/– aspirin frequently prescribed to prevent progression.
- Surgical intervention (thrombectomy) is treatment of choice for thrombi located in femoral artery.

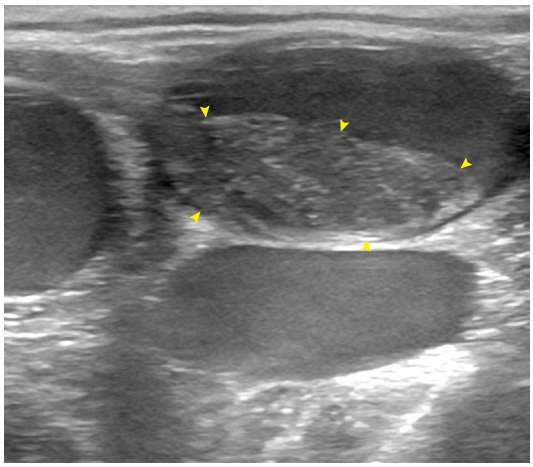


Fig. 10.3 Aorto-iliac thrombosis: Transrectal ultrasonogram showing intraluminal thrombus (arrowheads) partly obstructing internal iliac artery.

Prognosis
- Unlike jugular thrombosis [p. 315], collateral blood supply sufficient to eliminate clinical signs rarely develops.
- Prognosis for complete resolution of exercise-related signs is generally considered to be guarded to poor, although greater use of surgical interventions may improve outcomes in future.
- Prognosis influenced by location and extent (worse if bilateral) of lesions.
- Surgery (femoral artery): prognosis for return to full use reported as up to 65%.

Exercise-related sudden death (cardiopulmonary failure)

Acute collapse and death of a previously apparently healthy racehorse during or immediately after exercise is rare, but accounts for 10–20% of all racetrack fatalities. The cause of death may be cardiac or pulmonary failure, pulmonary haemorrhage or blood vessel rupture, but frequently remains undetermined even after postmortem examination ('autopsy-negative') due to challenges in establishing cause-effect relationship between commonly encountered findings. Fatal cardiac arrhythmia (sudden cardiac death) is probably responsible for around half of all acute exercise-related sudden death cases. Pulmonary haemorrhage is common in exercise-related sudden death and may arise secondary to cardiac events. Macroscopic and microscopic cardiac lesions are found in horses that do not experience sudden death, making interpretation of significance of these findings in sudden death cases difficult.

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Causes

- When postmortem examination fails to reveal a definitive cause of death, it is presumed (but not possible to prove) that a fatal cardiac arrhythmia is responsible.
- Arrhythmias are electroconductive disturbances that in general do not appear to be related to pre-existing structural damage to the heart muscle.
- Arrhythmias (ventricular premature contractions [VPCs], p. 314) are common in the immediate post-race period in normal horses; the circumstances that lead to fatal arrhythmias are unknown, but electrolyte or metabolic derangements that can occur during/after exercise postulated to potentiate the heart to risk of ventricular fibrillation (fatigue may contribute).
- Training causes chronic electrical and structural changes ('remodelling') in the heart and these adaptations are considered to be responsible for greater incidence of arrhythmias (both benign and fatal) in certain human athletic disciplines than general population. Likely that similar factors may contribute to risk of exercise-related sudden death in racehorses.
- Link between acute fatal pulmonary haemorrhage and EIPH (p. 305) uncertain in some cases but generally considered that the two conditions are unrelated.
- Aortic rupture is rare.

Risk factors

- Rare.
- Occurs in approximately 0.07–0.08/1000 starts in flat turf races.
- Greater incidence in jump racing (0.3–1/1000 starts).
- Risk factors include greater race distance and season (greater risk in summer).
- Fatal cardiac arrhythmias (sudden cardiac death) more likely to occur during training than racing, and therefore may not be strongly correlated with exercise intensity.
- Association of sudden unexplained death with pre-race administration of some medications, electrolytes or supplements is sometimes suspected (or subject of public speculation) but statistical evidence of correlations remains absent.

History

- Usually no history of pre-existing illness or exercise intolerance.

Signs

- Collapse and sudden death, most commonly during latter stages of a race but also in the immediate post-race period.

Diagnosis

- Postmortem examination does not furnish a definitive cause of death in approximately 50% cases. Histological sampling of the heart and toxicology testing (of multiple organs/tissues) are recommended when gross abnormalities are absent.
- Cardiac failure generally considered to arise from fatal arrhythmia; correlation with macroscopic or microscopic damage to heart found at postmortem difficult to establish.
- Pulmonary congestion/oedema/haemorrhage can be associated with both pulmonary and acute cardiac failure.
- Significance of pulmonary haemorrhage as a cause of sudden death in an individual is a subjective assessment based on severity.

Prevention

- At present the positive predictive value of available screening tests (ECG, cardiac biomarkers) for identification of at-risk horses is considered to be low, and therefore of little current usefulness.

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DENTISTRY

Routine rasping ('floating')

The equine dentition has evolved to accommodate long daily periods of grazing low-energy forage. Cheek teeth constantly erupt and are composed of three materials (enamel, dentine and cementum) that differ in strength/rates of wear. This ensures a rough occlusal surface to assist breakdown of fibrous food.

Stabled racehorses spend relatively little time chewing as their nutritional needs are met largely by concentrate diets. Altered chewing patterns and reduced wear may consequently predispose to the development of focal dental overgrowths. The upper (maxillary) arcades are broader and set further apart than the lower (mandibular) arcades (Figure 11.1a). Therefore, common sites for development of sharp enamel points are the outer edges of the upper and inner edges of the lower cheek teeth. These may impinge on adjacent cheeks/tongue causing ulceration (Figure 11.1b) and oral discomfort. The distribution of sharp edges differs between horses and over time; they may extend along an entire arcade or be present on only a few teeth. It is generally the case that 'routine' sharp focal overgrowths do not cause inappetence, but they may be associated with head-carriage problems at exercise. Conversely, shedding of deciduous cheek tooth dentition (premolar 'caps') is more likely to be associated with temporary inappetence than oral discomfort during exercise.

Routine rasping ('floating') is the process of removing sharp enamel edges using manual rasps or motorized blades/burrs. Most horses will accept the procedure without sedation. The occlusal (grinding) surface is not rasped as this may cause oral pain or affect ability to chew. Routine dental examinations also afford the opportunity to detect/monitor other oral pathology and remove loose premolar 'caps' (see next section). It is advisable to undertake routine dentistry/inspections at 6-monthly intervals in young horses in training.

Premolar 'caps'

- First three cheek teeth in each arcade (12 teeth in total) have deciduous precursors (Figure 11.2).
- Shed naturally in chronological order as underlying permanent tooth erupts and through attrition of the remaining deciduous crown (Figure 11.3a,b).
- Timing of shedding of these 'caps' varies between individuals.

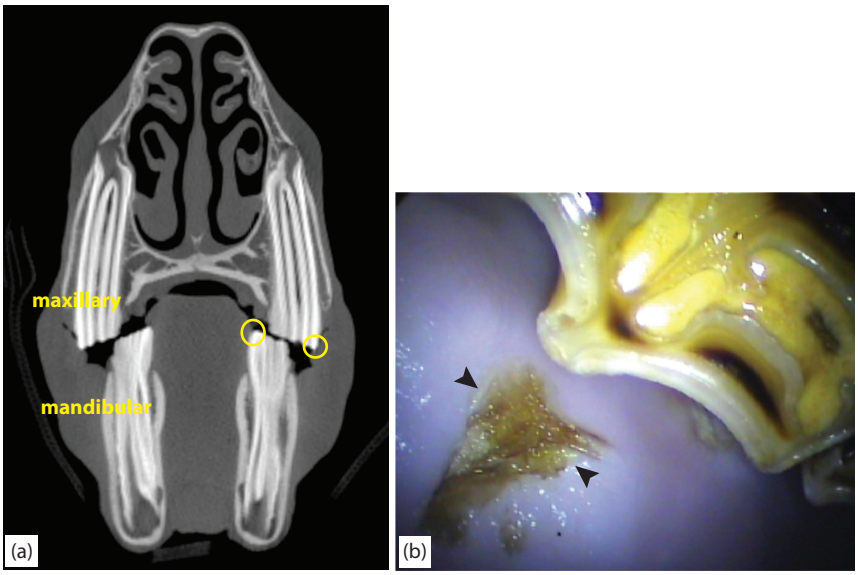


Fig. 11.1 **(a) Transverse CT image showing relationship between maxillary and mandibular arcades, and common sites for development of sharp enamel points (circled); (b) typical chronic abrasion on inside of cheek, adjacent enamel point.**

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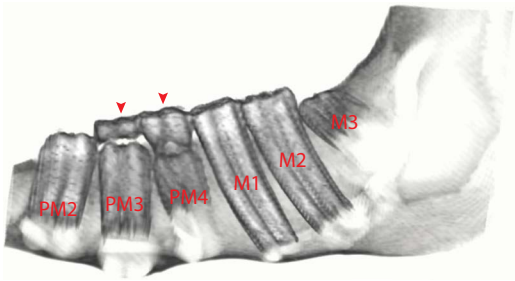


Fig. 11.2 **Mandibular dentition of a 2.5-YO horse. Deciduous premolar 'cap' has already been shed from first cheek tooth (PM2), is separating from second cheek tooth (PM3) and is still firmly in wear on third cheek tooth (PM4). Molars 1 and 2 (M1, M2) are in wear and the third molar (M3) is erupting.**

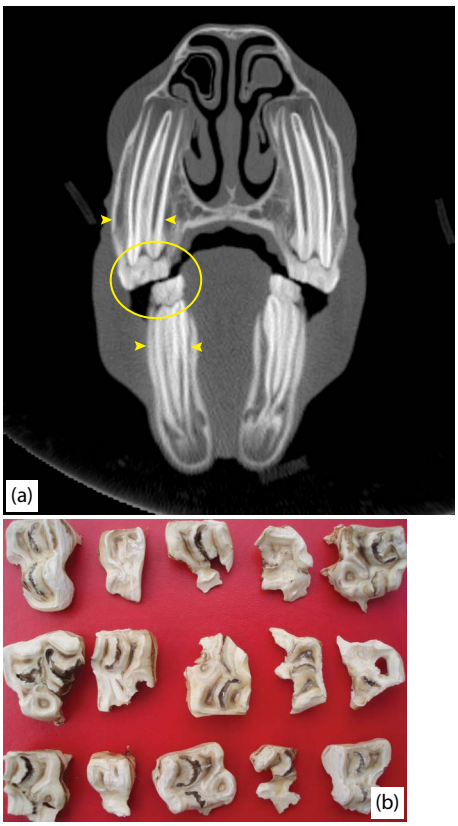


Fig. 11.3 **(a) Permanent premolar dentition (arrowheads) erupting below maxillary and mandibular 'caps' (circled); (b) examples of 'caps' showing range of attrition at point of shedding.**

- Usually shed in 3–5 month cycles from late 2 YO until 4 YO.
- Shed entire or as fragments.
- Many horses show transient reduction in feed intake during cap shedding. Loose/broken caps also occasionally lacerate adjacent cheek/tongue and can cause more severe inappetence and excessive salivation.
- Removal of loose caps readily performed in unsedated horse; however, forced premature extraction is inadvisable as may damage underlying permanent tooth.

'Wolf' teeth

- Small evolutionary remnant tooth found adjacent to first cheek tooth; not present in all individuals.
- Usually only in upper arcades (Figure 11.4a); unilateral or bilateral and shape/size variable.
- May remain unerupted ('blind') beneath gum.
- Never associated with inappetence.
- Link with bitting problems often assumed but difficult to prove and likely to be uncommon. Majority of horses with wolf teeth demonstrate no head-carriage/bitting problems.
- Most likely to cause bitting problems if 'blind', large/sharp or prominent following shedding of first premolar 'cap' (Figure 11.4b).
- Many horses naturally shed wolf teeth after loss of first cheek tooth 'cap'.

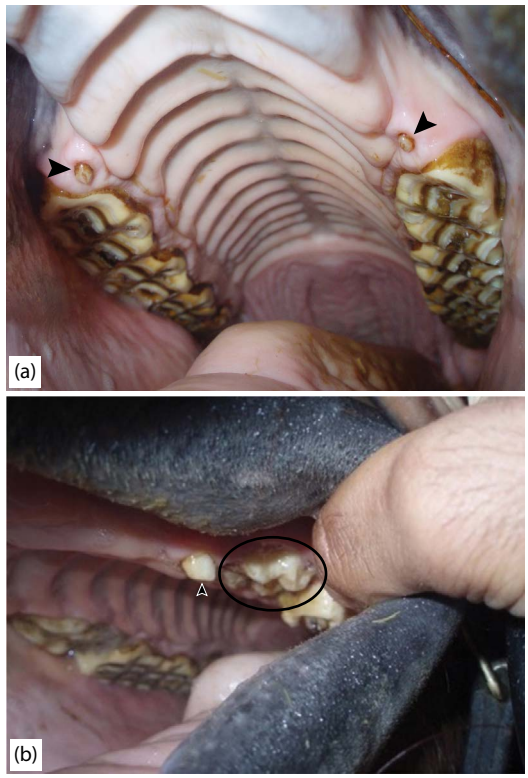


Fig. 11.4 **'Wolf' teeth: (a) typical location of maxillary wolf teeth; (b) relative prominence of wolf tooth (arrowhead) following loss of first maxillary cheek tooth 'cap' until permanent tooth (circled) comes into wear.**

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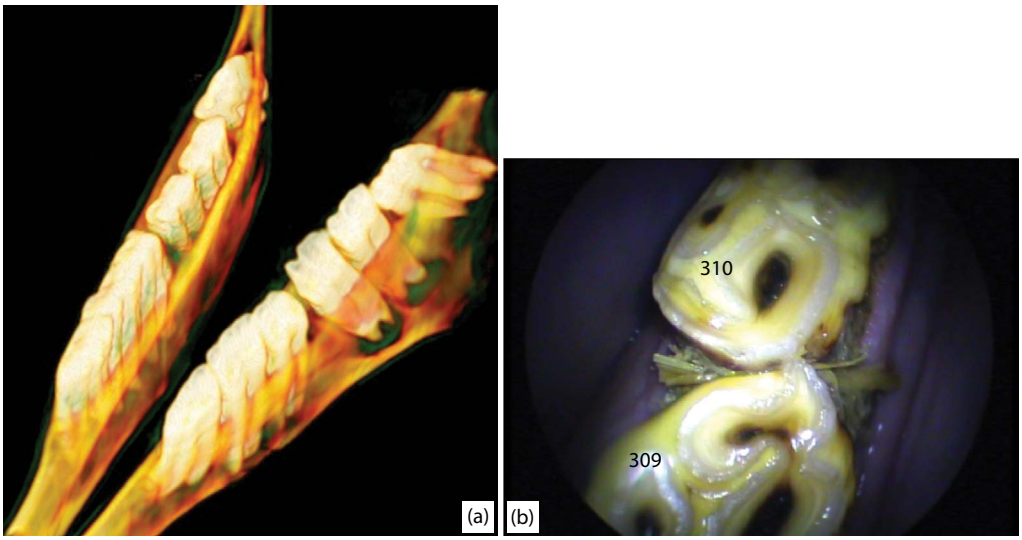


Fig. 11.5 **Cheek tooth malalignment: (a) CT image of mandible showing maleruption of 4th and 5th cheek teeth in each arcade, resulting in gaps in which food material can accumulate as seen in (b) oroscopic image of left mandibular arcade.**

- Removal: routine procedure undertaken +/– sedation +/– local anaesthetic block; time out of ridden work not required.
- Alternatively, clinical crown of prominent wolf teeth may be reduced to gingival level with motorized burr at time of routine dental examination/rasping, with no deleterious effect.

Dental malalignment

- Although each cheek tooth arcade is composed of six teeth, they are closely apposed and act as single grinding unit.
- Teeth may occasionally erupt out of line/position, resulting in loss of continuity of dental arcade (Figure 11.5a,b).
- Mid-arcade (3/4/5th cheek teeth) most commonly affected.
- Gaps between teeth can trap food and cause gum disease and oral pain/inappetence.
- Tend to be of clinical significance only when secondary gum disease worsens later in life; rare cause of oral pain in young horses in training.
- Removal of deciduous premolar 'caps' (prior to normal shedding) around the affected site may alleviate in medium term, but not curative.

'Parrot' mouth

- Congenital/developmental defect in which upper incisor arcade overshoots lower jaw (Figure 11.6).
- Severity ranges from mild-marked.
- Heritability plays a role but other factors probably involved.
- Surgical correction possible at early age (3–6 months) but rarely employed.
- By yearling stage unalterable and non-progressive.
- No special management required: eating/bitting unaffected and considered a cosmetic blemish only. Of little practical importance regardless of severity.

Tooth root abscess

Tooth root (dento-alveolar) infection is rare. In young racehorses usually affects a single cheek tooth in mid-lower arcade (mandible), but can also affect maxillary arcade. Infection involves one or more vital pulp horns with subsequent devitalization of tooth. External signs of disease variable; purulent discharge exits through path of least resistance (lower jaw, mouth or sinus), determined primarily by location of diseased tooth.

Causes

- Mandibular cheek tooth infection: may follow inhibited eruption of permanent tooth (and subsequent vascular disruption).
- Maxillary cheek tooth infection (rare in young horses): may be secondary to dental fracture or vital pulp compromise.



Fig. 11.6 **'Parrot' mouth.**

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Signs
- Mandibular: unilateral firm, painful focal swelling (of variable size) of lower aspect of mandible (distinguish from normal dental 'eruption cysts', which are typically symmetrically enlarged with minimal pain).
- More advanced disease may be associated with purulent draining tract.
- Usually no oral pain.
- Maxillary infections: unilateral facial swelling or unilateral purulent nasal discharge (arising from secondary sinusitis).

Diagnosis
- Clinical findings strongly indicative.
- Oral examination (oroscopy): useful to determine tooth involvement/vitality and plan treatment.
- Radiography/CT: useful for definitive diagnosis but can generally be reserved for cases non-responsive to initial antibiotic therapy.

Management
- 'Best practice' involves thorough diagnostic workup including radiography or CT +/– oroscopy; however, many cases managed successfully without diagnostic imaging.
- In horses in training initial treatment is usually systemic antibiotic medication (4–6 weeks); can be fed/trained as normal.
- Poor/short-lived response to antibiotic medication should prompt further investigation.
- Surgical extraction of tooth required only in small proportion of cases (fractured teeth more likely to require surgical intervention).

Prognosis
- Most cases respond favourably to antibiotic treatment providing dental fracture/fragmentation not present.
- Remission of swelling may take months.
- Rarely interferes with training.

WOUNDS

Wounds to tongue
Wounds to the rostral tongue usually the result of direct trauma from a bit or chifney. Typically a transverse cut on the dorsal surface of the tongue of variable length and depth; contamination with food is common. Lacerations to side of tongue may occasionally be caused by fractured mandibular 'cap' or cheek tooth.

Signs
- Acute oral discomfort/inappetence.
- Excessive salivation or bleeding from mouth.

Management
- Determined by severity and location.
- Most cases require antibiotic/anti-inflammatory medication and only minimal wound care (lavage) in acute phase.
- Deep/extensive dorsal injuries may benefit from sutured repair.
- Blood and nerve supply is located in underside of tongue; only the most severe injuries (with obvious signs of devitalization) should be considered for partial amputation.
- Amputation of tongue back to the level of frenulum possible without significant loss of function.
- Injuries to side of tongue require medical treatment only; inciting cause should be removed if still present.

Prognosis
- Most injuries heal well without primary closure.
- Permanent groove/defect/scar may result but tongue function unaffected.

Wounds to nostril
Sustained from interaction with stable fittings. Typically a full-thickness tear through the lateral or rostral margin of one nostril, of varying length.

Signs
- Severity and configuration of wound varies between individuals.
- Initial bleeding may be profuse.

Management
- Goal is to minimize scarring/stricture of nostril, as may have negative effect on future airway function at exercise.
- Primary repair (2- or 3-layer sutured closure) should be undertaken unless injury is old/grossly contaminated/obviously devitalized.
- Wounds often break down regardless of management and may have to be repaired by delayed closure.

Prognosis
- Excellent, with appropriate management.

Fractured mandible: Rostral
Fracture of incisive portion of lower jaw is most common in young colts and arises from oral play with stable

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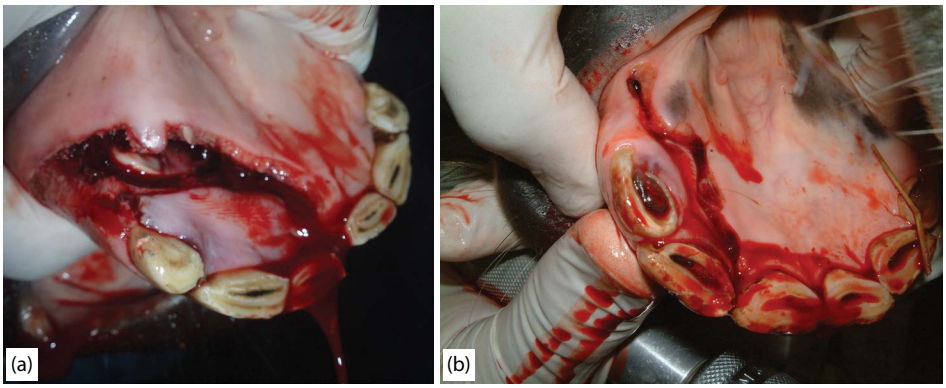


Fig. 11.7 **Typical appearance of rostral mandibular fracture at presentation (a) and when manually reduced prior to surgical repair (b).**

fittings. Usually consists of an avulsion of one or more lower deciduous incisor teeth with variable involvement of associated bone plate (Figure 11.7a). Invariably partially displaced and usually contaminated with food material at time of diagnosis.

Signs
- Oral discomfort is rare; eating and ridden behaviour usually unaffected and frequently only detected due to blood on stable fittings/feed manger.

Management
- Choice of treatment determined by severity, and quality of remaining attachment to jaw.
- Loose single teeth with minimal gingival/bone attachments may be simply removed; no wound aftercare is required.
- Most fractures require surgical repair for best outcome. Standing surgical procedure readily undertaken in first opinion setting: cerclage wire to realign fractured fragment (Figure 11.7b), removal of wire (under standing sedation) 4–6 weeks later.
- May be fed/exercised as normal following surgical repair.

Prognosis
- Excellent; no interruption to training.
- Some disruption to eruption of adult teeth can be expected; however, overall cosmetic outcome usually good.

Fractured mandible: Horizontal/ vertical ramus

Rare injury that arises from direct trauma such as kick or as a result of being cast in stable. Usually unilateral and most commonly involves the horizontal ramus. Fractures are usually incomplete or non-displaced. Fracture line may exit orally and some dental involvement is inevitable.

Signs
- Non-focal thickening of lower or lateral jaw on one side.
- Pain on palpation.
- +/– excessive salivation.
- Usually no evidence of external/skin wound.
- Oral examination may reveal generalized thickening of affected mandible +/– malodour +/– focal gingival bruising.
- May resemble cheek tooth infection in initial stages.

Diagnosis
- Clinical findings usually strongly indicative.
- Radiography/CT may assist diagnosis; however, fracture line often indistinct (especially in acute phase).

Management
- Unilateral fracture of mandible heals well without treatment, as the fragments are stabilized by muscles of mastication and remainder of jaw. Bilateral fracture requires surgical stabilization.
- Secondary infection (of mandible or adjacent cheek teeth) may arise if there is oral involvement: prophylactic antibiotic medication (4–6 weeks) may be warranted.
- Deterioration in appearance of jaw (or development of discharging tract) should prompt further investigation; occasionally surgical debridement or removal of affected cheek tooth may be required, although latter is typically delayed (months) until fracture line has stabilized.

Prognosis
- Good. Minimal interruption to training.

EYES

Conjunctivitis

The conjunctiva of the eye is very sensitive to inflammatory insult due to its vascular structure and large amount

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of lymphoid tissue. Conjunctivitis is usually a mild and transient condition of little concern for training. May be unilateral or bilateral, depending on underlying cause.

Causes
- Most commonly arises as a primary condition (bacterial/viral/trauma/foreign body).
- May be secondary to other eye disease such as corneal ulcer (p. 324), blocked nasolacrimal duct (p. 328) or uveitis.

Signs
- Reddening +/– swelling of conjunctival tissue.
- Usually with ocular discharge.
- Not usually associated with significant ocular pain; closed eye indicates presence of a more serious problem than conjunctivitis.

Diagnosis
- Examination of eye (+/– fluorescein staining) to rule out presence of corneal ulcer or foreign body.

Management
- Primary conjunctivitis: topical antibiotic medication 2–3x/day until resolution.
- Poor or short-lived response should prompt further investigation.

Prognosis
- Excellent. No interruption to training.

Corneal ulceration
Ulceration of the cornea is usually the result of trauma. A break in continuity of the corneal surface permits bacterial (less commonly fungal) colonization by either microbes normally resident on the cornea or by primary pathogens, and the combination of infection and inflammatory products at the site may cause progressive stromal degradation. Depth of ulcer ranges from superficial to full-thickness.

Cause
- Typically arises from direct trauma (e.g., from stable incident or accidental whip contact) or foreign body (seed/dirt).

Signs
- Painful eye (closed eyelids, excessive tear production, photophobia, conjunctivitis).
- Most ulcers are evident on close examination and appear as a focal defect in corneal surface +/– associated corneal oedema (opacity).

Diagnosis
- Fluorescein staining: retention of dye on exposed corneal stroma is definitive.
- Sedation +/– nerve blocking may be needed for full assessment.

Management
- Management guided by severity of stromal degradation/inflammation/ocular pain.
- Identification and removal of any foreign body.
- Simple ulcers: topical broad-spectrum antibiotic medication used prophylactically 2–3 x/day until resolution.
- +/– pain management for secondary ocular reflexes in acute phase: topical atropine sulphate (pupil dilation) and systemic (flunixin meglumine) +/– topical non-steroidal anti-inflammatory drug (NSAID; pain/inflammation relief).
- Removal from ridden exercise (to minimize exposure to dust/sunlight) until pain free.
- Eye should be monitored for healing.
- Complicated/infected ulcers (rare) are those that are deep, of large diameter or deteriorating rapidly ('melting'): require aggressive management to avoid long-term loss of vision. Treatment includes the above and topical antiproteases (EDTA); insertion of subpalpebral lavage catheter may facilitate frequent (up to every 2–4 hours) administration of topical drugs. Hospitalization may be warranted to permit intensive management.
- Poor response to initial antibiotic therapy should prompt diagnostic cytology and culture to isolate underlying pathogen and direct specific antimicrobial treatment.
- Fungal keratitis: topical (natamycin/miconazole/fluconazole/voriconazole) +/– systemic antifungal therapy.
- Surgery (conjunctival graft) may be considered if medical therapy fails.

Prognosis
- Overwhelming majority heal rapidly with simple treatment and generally display sore eye for <3 days. Interruption to training is minimal.
- Small proportion of cases are troublesome to treat or require hospitalization.

Corneal stromal abscess
Rare but serious condition that carries high risk of unilateral blindness or restricted vision. Abscessation within the cornea may arise from direct trauma or

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follow the healing of an ulcerated cornea when pathogens remain trapped below the superficial corneal layers. Superficial stromal abscesses may be primarily bacterial (*Pseudomonas* spp./*beta-haemolytic streptococci/staphylococci* spp.) or fungal; deeper abscesses are typically fungal (*Aspergillus* spp./*Fusarium* spp.). Fungal pathogens appear to have a propensity for deeper layers of the cornea and associated disruption to normal function of corneal endothelium can result in secondary corneal oedema or hydrops (bulging).

Cause
- Cause usually not determined in individual cases but may arise from direct trauma to cornea +/– foreign body penetration; haematogenous spread of pathogens also possible.

Signs
- Painful eye (closed eyelids, excessive tear production, photophobia, conjunctivitis) is typical in acute phase.
- Single or multiple focal areas of intra-corneal infiltrate, usually discoloured and often with change of external contour of cornea
- +/– associated corneal oedema (opacity); sometimes profound.
- +/– corneal vascularization.
- +/– anterior uveitis.

Diagnosis
- Clinical findings and slit-lamp observation of corneal stromal infiltrate usually definitive.
- Fluorescein staining: usually no (or weak) uptake of stain.
- Diagnostic cytology/culture warranted but does not always result in fungal isolation.

Management
- Requires aggressive management to salvage vision and prevent recurrence.
- Topical +/– systemic antifungal therapy is cornerstone of treatment: 4–6 week treatment course is typical. Concurrent topical antibiotic medication warranted if bacterial pathogens considered to be implicated.
- Topical medication administered through subpalpebral lavage catheter: voriconazole (or miconazole/natamycin) 4–6x/day or continuous pump lavage.
- +/– pain management for secondary ocular reflexes in acute phase: topical atropine sulphate (pupil dilation) and systemic (flunixin meglumine) +/– topical NSAID (pain/inflammation relief).
- Catheterisation necessitates removal from ridden exercise for duration of treatment.
- If good response to medical therapy does not result in initial weeks, surgery should be considered.
- Depth of abscess determines surgical option of choice: superficial keratectomy may assist drug penetration; deep abscesses may require deep lamellar endothelial keratoplasty.

Prognosis
- Guarded to good prognosis for retention of vision in affected eye with appropriate management.
- Superficial abscesses usually respond to medical management alone; deeper abscesses frequently require surgery.
- Some degree of permanent corneal scarring inevitable.
- Loss of vision or enucleation in small proportion of cases.

AIRWAYS

Sinusitis

Air-filled sinus cavities comprise a large proportion of the skull. These communicate indirectly with the nasal passages, through which drainage of sinus secretions occurs. Infection of a sinus cavity may be primary or secondary and is usually unilateral.

Causes
- Usually primary bacterial infection.
- Less commonly: secondary to tooth root infection (p. 321), sinus masses or head trauma.
- Rarely may have fungal component.

Signs
- Unilateral purulent nasal discharge.
- +/– unilateral malodour.
- +/– deformation of facial bones/ocular discharge on affected side in advanced cases.
- Horses are typically systemically well and demonstrate no exercise intolerance or respiratory noise.

Diagnosis
- Nasal endoscopy: usually possible to establish that discharge is entering nasal passage from nasomaxillary drainage angle.
- Radiography: increased opacity +/– fluid line(s) in affected sinus on laterolateral (LL) projection.
- Further diagnostic imaging (oroscopy/sinoscopy/CT) as required (Figure 11.8).

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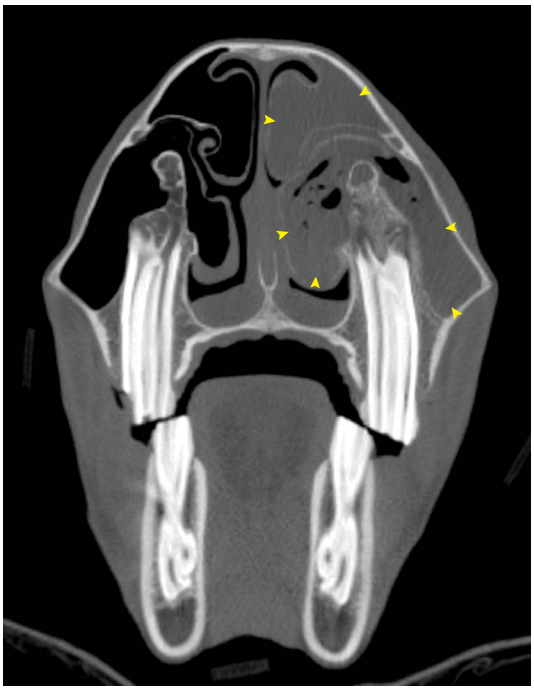


Fig. 11.8 Sinusitis: Transverse CT image showing soft tissue density filling maxillary sinuses on one side.

Management

- Presumptive medical treatment (without definitive diagnosis) often favoured in first instance.
- Antibiotic medication (2–4 weeks).
- Continued training plus feeding from floor (to assist drainage) recommended.
- If poor response to initial therapy: diagnostic investigation/treatment. Some cases require daily large-volume sinus lavage (small- or large-bore trephination +/– in-dwelling catheter: standing surgical procedure) until resolution (typically <1 week), or facial flap sinus surgery (rare).

Prognosis

- Most cases of primary sinusitis resolve with prompt antibiotic therapy; training rarely interrupted and little/no effect on performance.

Fungal (mycotic) rhinitis

Fungal growth may occur on any mucosal surface in the nasal passages or ethmoturbinate regions and is an uncommon cause of persistent nasal discharge. Typically unilateral.

Cause

- Inciting cause unknown but probably due to opportunistic colonization by fungal species (frequently *Aspergillus* spp.) from the stable environment.

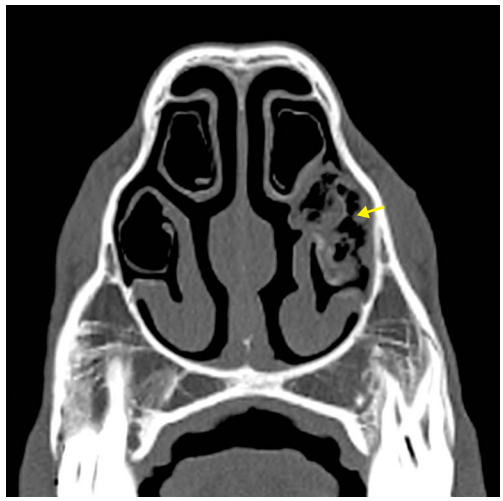


Fig. 11.9 Fungal rhinitis: Transverse CT image showing nasal conchal destruction (arrow).

Signs

- Persistent unilateral nasal discharge +/– malodour.
- Partially or non-responsive to antibiotic therapy.

Diagnosis

- Endoscopy: yellow/green plaque on affected mucosal surface +/– erosion/necrosis of underlying concha. Thorough examination of all nasal passages with narrow endoscope may be necessary.
- Transendoscopic capture of plaque material for laboratory analysis.
- Very rarely, advanced imaging (CT) may be required to localize lesion and differentiate from other causes of persistent nasal discharge (Figure 11.9).

Management

- Transendoscopic debulking of lesion and periodic lavage (usually more than three treatments required) with antifungal medication (enilconazole).
- Training may continue as normal.

Prognosis

- Excellent; most cases resolve with treatment and do not recur.
- Rarely interferes with training.

Ethmoidal haematoma

Rare non-neoplastic, slowly progressive mass originating in the ethmoidal turbinates (or less commonly in maxillary sinuses). Ulceration of surface of mass may result in nasal bleeding. Unilateral or bilateral; additionally, some

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unilateral masses may extend behind the nasal septum to encroach upon other side. Typically in older (≥3 YO) horses.

Causes
- Underlying cause unknown.
- Thought to arise from recurrent submucosal haemorrhage and subsequent distension of submucosal lining to become haematoma capsule.
- Gradual expansion of mass into air spaces of nasal passages or sinuses.

Signs
- Clinical signs dependent on location and size of lesion.
- Recurrent low-grade unilateral nosebleed (epistaxis), usually unrelated to exercise.
- +/– respiratory noise.

Diagnosis
- Endoscopy: smooth-walled mass may be visible extending from ethmoid turbinate labyrinth into nasal passage. Visualization of mass is only partial and does not permit full assessment of extent.
- Radiography: smooth-margined opacity in region of ethmoid turbinates +/– sinus fluid lines usually visible on LL projection.
- CT: permits full assessment of extent of lesion and allows accurate surgical planning (Figure 11.10a,b)
- Definitive diagnosis only through histology of mass lining (obtained at surgery).

Management
- Without treatment mass continues to slowly expand and clinical signs will worsen.
- Choice of treatment determined by size and location of lesion.
- Small/moderate-sized nasal lesions can be treated successfully under standing sedation with laser ablation or formalin injection. Repeated treatments usually necessary.
- Lesions with sinus involvement usually require surgical ablation (facial flap).

Prognosis
- Moderate recurrence rate following treatment.
- Good medium-term remission of nasal lesions usually possible with laser ablation.
- Surgery and formalin injection associated with mortality risk (small) due to potential exposure of central nervous system to infection or caustic agent.

OTHER

Submandibular lymphadenopathy

Enlargement (+/– abscessation) of one or more submandibular lymph nodes. The sublingual and submandibular lymph nodes are a normal part of the lymphatic system and lie in a chain under the jaw and throat. Enlarged lymph nodes are of concern primarily because they may signal the presence of infectious disease.

Generalized mild enlargement in response to viral/bacterial challenge is common in yearlings and rarely of concern. Enlargement and recurrent abscessation of a single lymph node is also common and usually not associated with systemic illness. Differentiation from the more profound/multiple abscesses seen with *Streptococcus equi* ('strangles') infection (p. 375) is important.

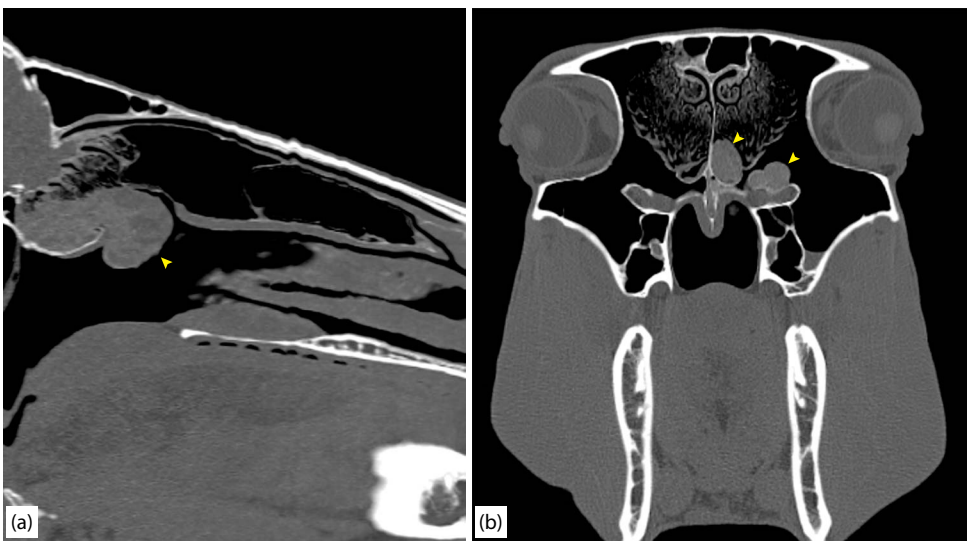


Fig. 11.10 Ethmoidal haematoma: Sagittal (a) and transverse (b) CT images showing smooth-margined mass arising from ethmoid turbinate labyrinth.

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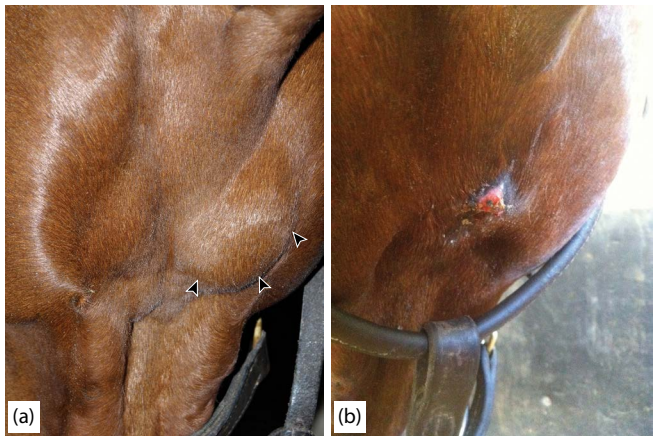


Fig. 11.11 Typical submandibular lymph node enlargement (a) and following spontaneous drainage (b).

Signs
- Solitary lymph node enlargement: may be large, sore to palpate and intermittently discharge purulent material between periods of relative quiescence (Figure 11.11a,b).
- Usually without fever/systemic illness/nasal discharge.

Diagnosis
- Clinical presentation and history are good indicators of risk of strangles.
- If multiple lymph node enlargement plus systemically ill +/– nasal discharge, +/– recent arrival on premises (days/weeks), precautions should be taken.
- Diagnostic tests: direct swabbing/culture of exudate +/– *Streptococcus equi* serology +/– guttural pouch/nasopharyngeal swabbing (culture and polymerase chain reaction [PCR]).

Management
- If strangles considered unlikely: daily warm fomentation; once draining, lavage +/– antibiotic medication into cavity reduces likelihood of recurrence.
- Systemic antibiotic medication should be avoided as generally delays maturation of abscess.

Prognosis
- Occasionally may recur or persist as low-grade chronic condition with intermittent drainage for many weeks/months.
- Does not interfere with training.

Blocked nasolacrimal (tear) duct
The nasolacrimal (tear) duct is formed from the convergence of two canaliculi in the medial canthus of eye and courses through first the lacrimal then the maxillary bones to emerge in the nasal cavity. The duct serves to carry tears from eye to nose and varies in diameter along its course. Direct or indirect obstruction may occur, resulting in overflow of tears from eye onto face. Unilateral or bilateral.

Causes
- Most commonly from direct blockage with inflammatory debris arising from eye (following conjunctivitis).
- Pinching of duct by the bony structures that surround it (tooth root abscess/sinusitis/suture exostosis) may also cause obstruction.
- Congenital atresia of duct is rare; typically with history of long-standing ocular discharge and absence of distal nasal punctum when examined.

Diagnosis
- Fluorescein dye placed in eye should emerge at nostril within 20 minutes if unobstructed.
- Imaging of duct necessitates contrast radiography/CT or specialized endoscopy and is rarely necessary.

Management
- Catheterization of nasal punctum and retrograde (nostril to eye) irrigation of duct with saline solution; readily performed in unsedated horse and can be used for combined diagnosis/treatment. Excessive pressure should be avoided.
- If unsuccessful: topical antibiotic/anti-inflammatory eye medication and further attempt in 1–2 weeks.
- Normograde (eye to nostril) irrigation may be attempted under standing sedation.
- Congenital atresia: surgical correction (rarely undertaken).

Prognosis
- Does not interfere with training.

Cheek/lip abscess
Acute, focal infection/abscess of cheek or lip at/near corner of mouth is encountered frequently. Unilateral.

Causes
- Usually follows small penetrating injury on inside of mouth (such as from a seed/grain).
- Less commonly may arise from bit trauma.

Signs
- Pain/guarding of cheek/lip; frequently with acute, severe head shyness (usually one-sided) and aversion to examination.

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Fig. 11.12 **Lip abscess.**

- May resent fitting of bridle/headcollar.
- +/– external swelling of cheek/lip (Figure 11.12).
- Palpable thickening, intensely painful to light pressure.
- Gingival wound usually very small or inapparent.
- +/– malodour.

Diagnosis
- Clinical findings definitive.
- Examination from opposite side of mouth generally easier to achieve.

Management
- Usually self-resolving in matter of few days.
- Daily warm fomentation warranted if abscess close to external surface of lip.
- Systemic antibiotic medication should be avoided as generally delays maturation of abscess.
- Systemic anti-inflammatory medication in acute phase may assist with continued training.

Prognosis
- Excellent; interruption to training generally brief (days).

Nasal atheroma (dermal inclusion cyst)
Uncommon developmental epidermal cyst at back of 'false nostril' and usually visible externally. Unilateral or bilateral. Generally remains static in size but may be very slow growing.

Signs
- Typically grape-sized fluid-filled swelling at top of flexible margin of nostril.
- Painless.

Diagnosis
- Clinical findings definitive.

Management
- Does not cause problems and treatment rarely indicated.

Prognosis
- Does not interfere with training and nostril/airway function considered to be unaffected unless very large.
- View as cosmetic blemish only.

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GASTROINTESTINAL CONDITIONS

Gastric ulcer syndrome (EGUS)

Gastric ulcers occur commonly in horses in training. Severity varies between horses and over time. Ulcers may be clinically silent or associated with a variety of signs including inappetence, light condition and poor performance.

The horse has evolved to graze through the majority of the day; in the natural state the relatively small (8–15 L capacity) stomach is rarely empty and a constant flow of saliva (produced during chewing) serves to buffer acidity. Only half of the stomach is lined by acid-producing glandular mucosa, and because of this (and the constant inflow of acid-buffering saliva) there are marked regional differences in acidity within the stomach. Ulcers frequently occur in non-glandular (squamous) mucosa and are usually more severe on the lesser curvature of stomach. Ulceration of the glandular mucosa may also occur, and the two conditions are now considered to be largely distinct: the presence/absence of one cannot be used as a predictor for the presence of the other. Squamous gastric ulceration is a multifactorial condition that essentially represents a mismatch between the acidic environment within the stomach and the protective features of the stomach lining. The causative factors involved in development of glandular ulceration are poorly understood but inflammation and gastric blood flow may have a role.

Causes

- Underlying cause of gastric ulcer syndrome in athletic horses is altered gastric environment arising from replacement of natural grazing state with stabling, concentrated nutrition and exercise.
- Nutritional factors: concentrated meals, high-starch diets and reduced roughage of horses in training creates more acidic stomach environment than in free-living horses.
- Exercise also plays important role: reduced mucosal blood flow; also increased intra-abdominal pressure during gaits faster than walking leads to greater bathing of the non-glandular lining with acidic stomach contents.
- Unlike in people, 'stress' and specific microorganisms (*Helicobacter* spp.) are not thought to be contributing factors.

Risk factors

- Occurs in majority of racehorses in training (up to 90% of horses in full work).
- Horses out of work (including broodmares) can also have ulcers.
- Prevalence of ulcers, and likelihood of more severe ulcers increases with exercise intensity.
- Up to 10–15% of horses in race training have moderate to severe gastric ulceration.
- Fasting, and intermittent access to water are both recognised to increase the risk of, and severity of ulcers.
- Increased risk of squamous ulcers with increased time between forage meals (>6 hours) compared with more frequent forage access.
- Frequent use of concentrated electrolyte pastes may contribute, although use of electrolytes with feed is considered to be safe.
- Drugs such as phenylbutazone or suxibuzone do not cause ulcers when used at recommended dose rates.
- Association with crib-biting (cribbing), however, mechanism is unclear.

Signs

- Most horses with ulcers exhibit no obvious sign of the condition.
- Clinical signs (when present) are non-specific and may include: poor appetite, failure to thrive, weight loss, colic around mealtimes, poor performance or behavioural changes such as irritability or sourness.
- Onset of signs usually insidious and may coincide with increase in workload.

Diagnosis

- Clinical findings non-specific and should not be relied on for diagnosis.
- Gastroscopy: is currently the only reliable method for diagnosis and grading of lesion severity. Restriction

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of food (8–16 hours) and water (2–4 hours) required to empty stomach prior to examination. Entire stomach (including greater and lesser curvatures, and pylorus) should be examined. Grading of severity of squamous ulceration is noted in Table 12.1 and Figure 12.1.
- Grading of glandular mucosa ulceration: descriptive recording of presence/location/distribution/appearance and severity (mild/moderate/severe) is recommended (Figure 12.2).
- Relevance of gastroscopy findings should not be determined in isolation; interpret with caution and in context of clinical presentation. While intuitive that more severe lesions are more likely to be significant, the relationship is not linear and there is a poor correlation between clinical signs and severity. However, squamous ulceration of grade 3 or 4 is generally considered significant +/– merits treatment.
- Potential future role of biomarkers (salivary/blood) for diagnosis, differentiation of glandular and squamous ulceration and monitoring of clinical progression.
- Blood haematology/biochemistry profile is unaffected by presence of equine gastric ulcer syndrome (EGUS) and is not a useful diagnostic aid.
- Faecal occult blood or albumin detection of no use as a diagnostic aid.

Management

- Accept that gastric ulcers are a near-inevitable consequence of stabling and exercising the athletic horse, although severity and significance varies between individuals.
- Suppressing acid is the cornerstone of management of ulcers (both squamous and glandular).

Table 12.1 **Gastroscopic grading of squamous gastric ulceration (Figure 12.1)**

GRADE	
0	No apparent mucosal abnormality
1	Mucosa intact but areas of reddening +/or hyperkeratosis
2	Small single or multifocal lesions
3	Large single or extensive superficial lesions
4	Extensive lesions with areas of apparent deep ulceration

- Some alteration of dietary management (provision of more/more frequent hay; turnout/grazing) may be beneficial but is not curative, and return to low-starch/high-fibre diet is incompatible with intensive training. While free access to forage is considered to reduce the risk of ulcers, strong evidence is lacking: pasture has much lower dry matter content than hay/haylage and the naturally grazing horse would consume many times the amount of pasture on a daily basis than could be offered as forage to a stabled horse. Significant healing/mitigation of gastric ulceration should not be expected through dietary management alone.
- Alfalfa hay may have protective role: high in calcium and protein and may buffer stomach acid.
- Medical treatment usually reserved for horses with both clinical signs and gastroscopic evidence of moderate-severe ulcers.
- Medical treatments/preventives generally need to be administered for duration of time that horse is in full training, with lapses only to permit racing free from drug metabolites (ulcers begin to recur within a few days off treatment). Medical management (including dose) tailored to individual is preferable to 'blanket' recommendations but not always practical in yard situation.
- Treatment categories are shown in Table 12.2. Omeprazole is currently the drug of choice for squamous ulcers; bioavailability of drug may vary with formulation. Efficacy may be enhanced if dosed early morning after overnight fast.
- Glandular ulcers: acid-suppressing therapies alone have limited efficacy, and should be combined with misoprostol (modulates inflammation & gastric blood flow) +/– mucosal protectants (such as sucralfate), with 8 weeks minimum duration of initial therapy before reappraisal. Some evidence of improved efficacy with injectable long-acting omeprazole.
- Antibiotics currently have no role in routine treatment of gastric ulcers.

Prognosis

- Clinically significant ulcers if untreated can affect training by limiting frequency/intensity of fast work or racing (failure to maintain body condition).
- With appropriate treatment body condition/other clinical features can be managed satisfactorily in majority (70–85%) of affected horses.
- Persistence of significant squamous ulceration beyond 3–4 weeks of omeprazole treatment warrants investigation of dosing/diet regime.

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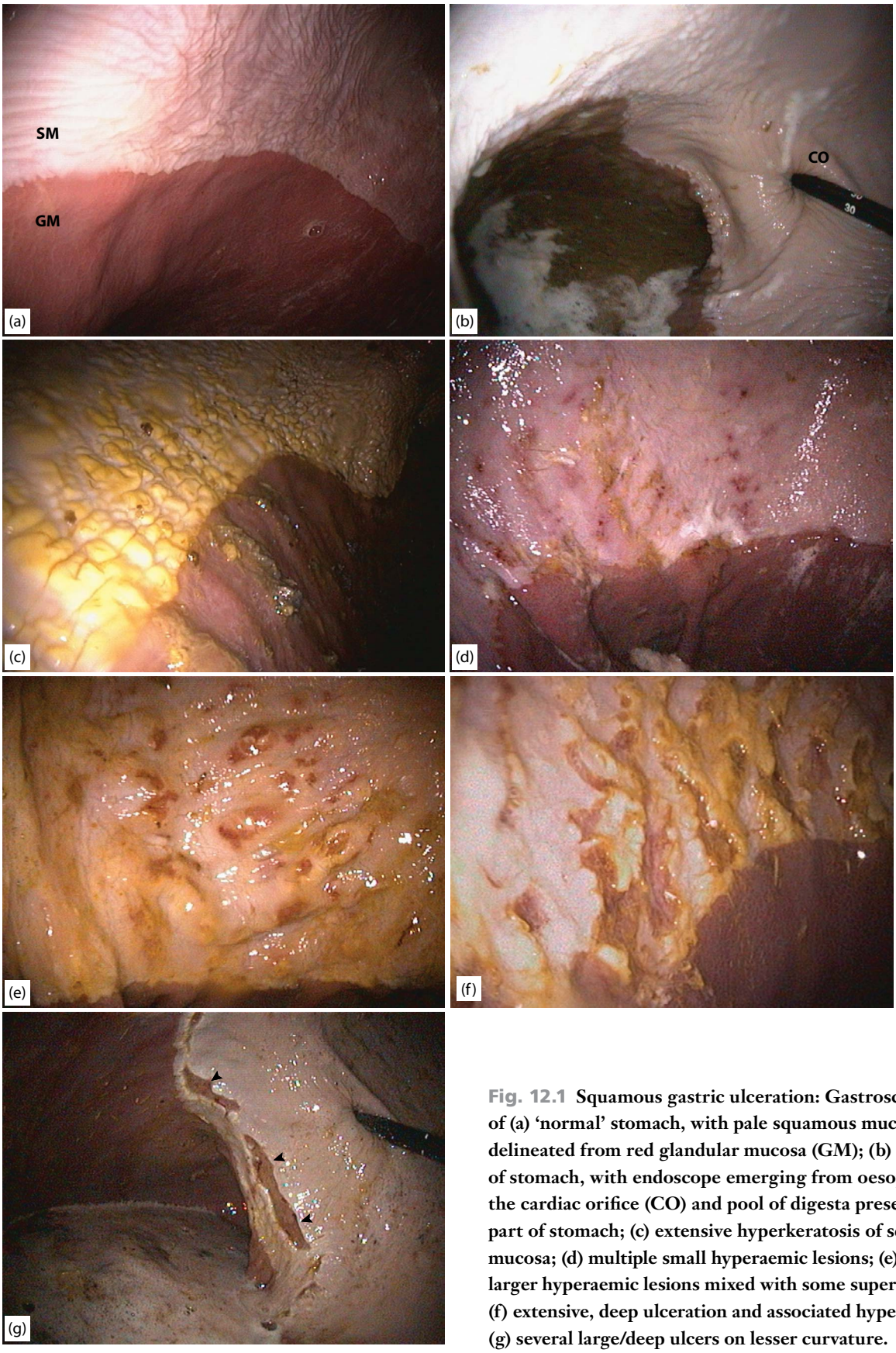


Fig. 12.1 Squamous gastric ulceration: Gastroscopic appearance of (a) 'normal' stomach, with pale squamous mucosa (SM) clearly delineated from red glandular mucosa (GM); (b) lesser curvature of stomach, with endoscope emerging from oesophagus through the cardiac orifice (CO) and pool of digesta present in glandular part of stomach; (c) extensive hyperkeratosis of squamous mucosa; (d) multiple small hyperaemic lesions; (e) multiple larger hyperaemic lesions mixed with some superficial ulcers; (f) extensive, deep ulceration and associated hyperkeratosis; (g) several large/deep ulcers on lesser curvature.

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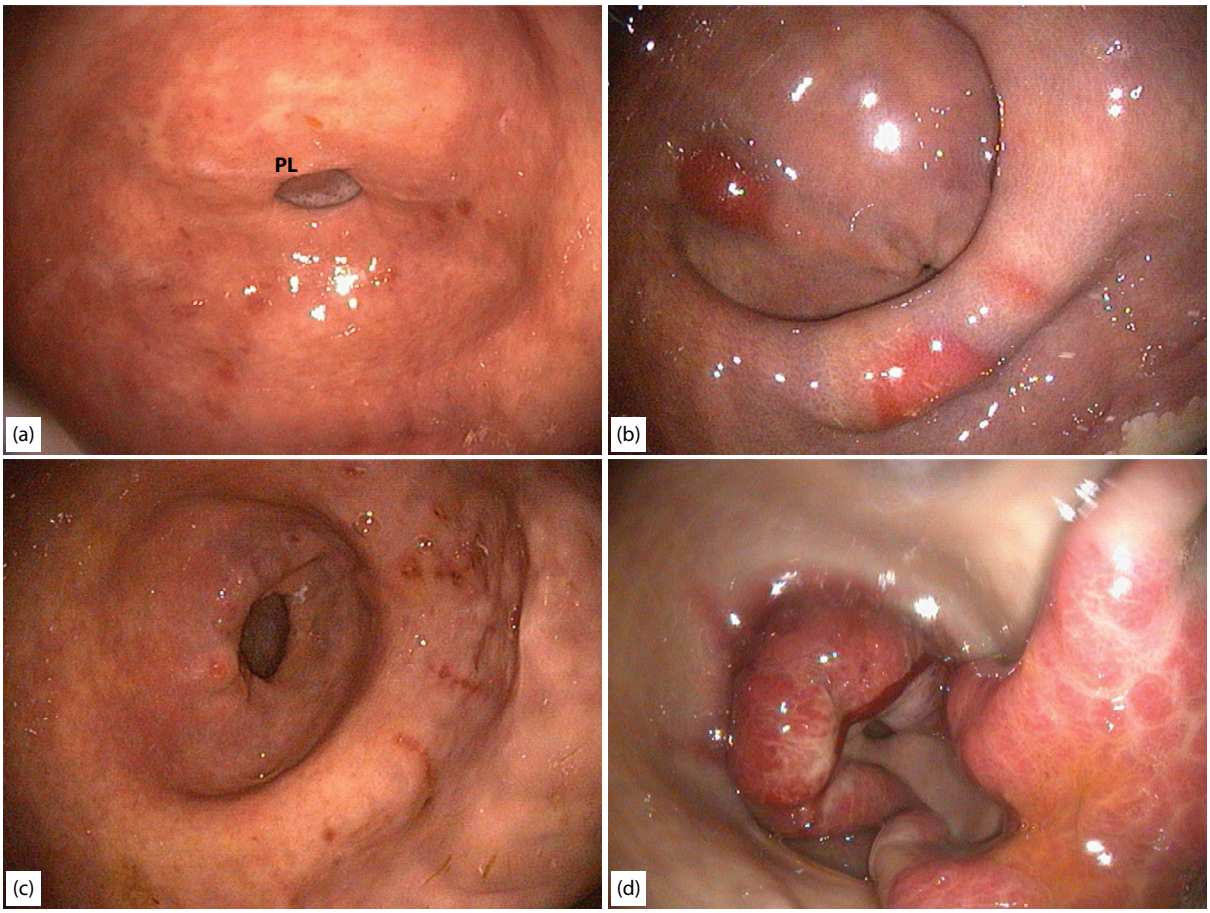


Fig. 12.2 **Glandular/pyloric gastric ulceration: (a) mild hyperaemia (PL: pyloric lumen); (b) discrete raised hyperaemic areas; (c) multiple small focal ulcers; (d) marked hyperaemic thickening of pyloric mucosa.**

Colic

Colic is a general term for abdominal discomfort or pain (Table 12.3). There are many different causes of colic ranging from transient motility disorders ('spasmodic' colic) to strangulating torsions that lead to rupture or fatal shock unless corrected surgically. Most colic episodes are self-resolving or require only simple medical management; however, when part of the bowel becomes twisted and blood supply is compromised, deterioration can be swift. Infection, parasites and gastric ulceration can also cause abdominal pain. General points concerning the main forms of colic encountered in racehorses are discussed in the next section.

Risk factors

- Risk of colic is relatively low in well-managed racing yards (typical incidence of 5% per year).
- Main risk factors are abrupt changes in diet or management.
- Microbial population of hindgut plays a major role in digestion; sudden alterations in diet can cause shifts in bacterial population with resulting changes in acidity, gas production and gut motility.
- Racehorses on high-concentrate/low-forage diet and little access to grazing are to some degree already on metabolic knife-edge.
- Windsuckers/crib-biters at greater risk (association with altered gut transit time rather than swallowing of air).
- Tapeworm burdens associated with increased risk of spasmodic colic.
- Sudden restriction of exercise (stable rest), such as following injury, is associated with increased risk of colonic impaction colic.

Signs

- See Table 12.3.
- Typical signs: pawing ground, flank-watching, inappetence, recumbency +/– rolling, kicking belly, stretching to urinate, sweating.
- Severity highly variable, ranging from mild/intermittent discomfort to severe uncontrollable pain.

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Table 12.2 **Treatment categories for gastric ulcers**

TYPE	ACTION	DRUGS	EFFICACY
Inhibitors of acid production	Lower acidity of stomach by blocking production	Omeprazole (proton pump inhibitor): the most potent anti-ulcer medication. Once-daily oral dosing; preferably given on 'empty stomach' (ideally after overnight fast). Tailoring management to individual is preferable but not always practicable. 'Maintenance' dose rates vary between formulations: buffered formulation 2 mg/kg; enteric-coated granule formulations 1 mg/kg; plain formulations 4 mg/kg (all once daily). Higher dose rates can be used either at outset of therapy or for 'poor responders' (i.e., start low dose and double if inadequate response). Long-acting injectable formulation available: weekly intramuscular injection. Ranitidine/cimetidine (histamine receptor antagonists). Require 2–3x/day oral dosing. Lengthy detection time restricts usefulness.	Good efficacy (70–80%) for healing of squamous ulcers across range of dose rates; healing occurs in 3 weeks (clinical effect usually more rapid) Glandular ulcers: poor efficacy with acid-inhibition drugs only Efficacy (for squamous ulcers) lower than omeprazole
Antacids	Neutralize acid within the stomach	Magnesium oxide/hydroxide/aluminium hydroxide are most common constituents.	Difficult to feed sufficient quantities to produce clinical effect; small amounts are non-therapeutic.
Mucosal protectants	Bond to exposed ulcer cavities and assist healing	Sucralfate: twice-daily oral dosing at 12 mg/kg. Should not be given before/at same time as omeprazole, as limits absorption of latter.	Adjunctive treatment for glandular ulcers. Assists healing but does not prevent ulcer formation.
Prostaglandin E analogues	Gastric acid inhibitor; anti-inflammatory; increases mucosal blood flow	Misoprostol: twice-daily oral dosing at 5 µg/kg, 1 hour pre-feeding.	Some evidence of good efficacy for glandular ulcers.
Nutraceuticals	Range of effects including increased mucus production, antioxidant activity	Various supplements: pectin-lecithin complex; sea buckthorn berry extract; fig extract; processed yeast (Yea-Sacc/*Saccharomyces cerevisiae*). Limited available information on appropriate dosage.	Some limited evidence of efficacy in prevention but should be considered as ancillary aids rather than sole therapy.

- Clinical parameters (pulse rate, demeanour, pain level, abdominal distension and gum colour/refill, gut sounds) are good indicators of severity.
- Should be differentiated from conditions that can present with similar signs: exertional rhabdomyolysis/pleuropneumonia/peritonitis.

Diagnosis

- Relevant history may assist diagnosis: recent management changes/current level of exercise/presence of normal faeces/previous episodes of colic.
- Typical pulse rate for mild/spasmodic colic 40–60 bpm; >60 for colic possibly requiring hospitalization and >80 for horses with severe cardiovascular compromise requiring immediate hospitalization and supportive care or surgery.
- Rectal palpation (displacement/distension of abdominal organs) and nasogastric intubation (for gastric reflux) assist diagnosis. Transcutaneous abdominal ultrasound, peritoneal tap (abdominocentesis) and blood analysis also useful.

Management

- See Table 12.3.
- Pain is single most important factor in determining whether a colicking horse requires referral to a surgical facility.
- Short-acting pain control may be needed to conduct initial examination: alpha-2 agonists (xylazine/detomidine) potent and fast acting.
- Initial response to analgesics (typically phenylbutazone) +/– motility modifying medication (hyoscine butylbromide) determines management. Flunixin is used in some circumstances to differentiate need for surgical intervention but is a powerful and potentially masking analgesic and should be used with caution.
- Majority of cases (>90%) resolve with initial analgesic therapy +/– walking/lunging. Failure to manage pain

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Table 12.3 **Classification of common types of racehorse colic**

TYPE	DESCRIPTION	TREATMENT	PROGNOSIS
'Spasmodic' colic	Most common form of colic; often at feeding times. Usually transient. Mild-moderate pain and gut sounds usually increased	Spasmolytic and anti-inflammatory medication followed by short period of walking/lunging. May be fed/managed as normal following episode. Failure of initial medication/exercise to resolve pain warrants further investigation or referral	Majority of episodes resolve with medical treatment/exercise
Colonic (pelvic flexure) impaction	Natural 'hairpin' bend in large colon is most common site of blockage; predisposed by narrowing of intestine at bend and local 'motility pacemaker' at pelvic flexure. Most common in horses recently confined to stable (e.g., following injury); may reflect change in intestinal motility or feed intake. Low-grade intermittent discomfort (scraping, lying quietly) over several hours +/– few/hard faeces. Diagnosis simple with rectal palpation: impaction readily palpable (but not accessible with enemas)	Nasogastric tubing with water +/– mineral oil (2–4 L) +/– magnesium sulphate (0.5–1 g/kg) to hydrate and lubricate gut contents. Restrict feed and hay until impaction cleared. Resume light exercise if feasible. Frequency of nasogastric tubing determined by size/severity of impaction: most simple cases resolve with one or two treatments	Average clearance time 2 days; small proportion require hospitalization for intensive management or surgical intervention
Surgical colic	Continuing signs of pain +/– deteriorating clinical parameters (high pulse rate) in the face of initial medication warrant referral to surgical facility. Types of colic requiring hospitalization include large colon displacements (some may be managed medically), large colon volvulus/torsion and small intestinal strangulations or impactions	Treatment determined by lesion type	Prognosis best with early intervention. Short-term survival (to hospital discharge) 70–85%. Survival rates lower for strangulating lesions than for simple obstructions and lower for small intestinal or caecal involvement than for large or small colon involvement. Once through immediate post-surgery period, prognosis for long-term survival (>1 year) is good: around 85%. Approximately 10% of horses require second colic surgery during hospitalization, and long-term survival for these horses is low (around 20%). Most (>80%) horses that survive surgery return to full use; typically require 2–3 months out of ridden exercise following surgery
Recurrent colic	Repeated episodes of colic (over weeks/months) warrants further investigation. Determine whether pattern exists (i.e., at times of increased stress or anticipation of feeding). Medical investigations include determining parasite burden (worm egg count/tapeworm serology), presence of gastric ulcers and haematology/biochemistry. High incidence of colic within a yard may indicate nutrition/feeding problem	Identify cause/risk factor: modification of management may resolve problem. Specific medical treatment as required	Prognosis dependent on diagnosis

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with initial treatment indicates need for further investigation/treatment.
- High heart rate (>60 bpm) or severe/unremitting pain and deteriorating clinical signs warrants immediate transport to surgical facility; prognosis for survival declines considerably with elapsed time.

Prognosis
- See Table 12.3.

Parasites
Horses harbour nematode parasites in their gastrointestinal tracts throughout life. Parasite populations are replaced constantly or seasonally depending on parasite species and environmental factors. Although horses in training generally have limited access to pasture, intestinal parasite burdens occur and in some circumstances can cause weight loss, colic or intestinal upsets. Immunity to parasites increases with age but most horses in training are susceptible. The major types of parasites are detailed in Table 12.4.

Diagnosis
- Adult parasites (roundworms and strongyles) are occasionally noted in faeces, but this is a poor method of detection of worm burden.
- Diagnosis of large strongyle or roundworm burden requires faecal analysis for presence of eggs.
- Sampling technique: collect at least 3 faecal balls (or parts thereof) at random from fresh (<12 hours old) dung pile; expel air from plastic bag before sealing, and keep cool (<6°C) until processing.
- Positive faecal egg count (FEC) confirms presence of laying adult females, but of no value in detecting presence of larval stages (encysted or otherwise).
- Small strongyles (cyathostomes): blood sampling (enzyme-linked immunosorbent assay [ELISA] test) for detection of specific antibodies has high sensitivity and specificity, but routine use in 'herd' management of young, stabled horses in training not considered essential/advisable at present time. As with other antibody-based testing, false positives may occur for up to 5 months post-deworming.

Table 12.4 **Major types of gastrointestinal parasites in horses**

TYPE	SOURCE OF INFECTION	LIFE CYCLE	EFFECTS
Small strongyles (cyathostomes)	Grazing	Adults live in large intestine and eggs passed in faeces. Larvae develop on pasture; when ingested burrow into large intestine wall ('encysted' larvae) later to emerge into large intestine as adults. Common	Seasonal emergence of large numbers of dormant larvae from intestinal wall can cause severe diarrhoea and weight loss, particularly in young horses
Large strongyles	Grazing	2–5 cm long red worm. Adults live in large intestine and eggs passed in faeces. Larvae develop on pasture; when ingested penetrate intestine wall and migrate through blood vessels or to liver, returning to large intestine to become adults. Rare	Feeding and migration of larvae can cause damage to small intestinal mucosa (anaemia, weight loss), blood vessels (thrombosis/colic) or other organs (liver damage/peritonitis)
Roundworms (ascarids)	Grazing	Large white worm. Adults live in small intestine and eggs passed in faeces. When ingested, larvae penetrate intestinal wall then migrate to liver and lungs. Larvae coughed up, swallowed and develop into adults in small intestine. Common	Large numbers of worms may cause obstructions and poor growth in foals, which have low resistance. Mature horses show few signs, and routine control in horses >1 YO not required
Tapeworms	Ingestion of forage mites in hay/bedding/grazing	Adults live near caecum (ileocaecal valve) or small intestine/stomach. Segments full of eggs shed and ingested by intermediate host (forage mite), in which development to larvae occurs. Ingestion of forage mites (and larvae) by horse. Common	May cause colic (spasmodic and ileal impaction)
Pinworms	Ingestion of faecal-contaminated feed/water	Adults live in colon/rectum and emerge to lay eggs around anus. Eggs stimulate irritation/tail-rubbing and larvae are ingested directly or through contaminated feed/water. Common	Tail-rubbing

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- Tapeworms: ELISA testing (saliva/blood) is method of choice (faecal analysis of little use due to intermittent shedding of eggs). Antibody levels remain detectable for up to 5 months post-deworming and false positives occur.
- Routine blood profile (haematology/biochemistry): poor method of detection of worm burden; larval cyathostomes sometimes associated with low plasma protein; relevance of eosinophil count unknown.
- Tail rubbing/perineal pruritis may be a sign of pinworm *(Oxyuris equi)* infestation; however, this is an unrelated species to the main parasites of concern and should not be used to guide deworming strategy.

Management

- Repeated, frequent use of dewormers is unnecessary and contributes to anthelmintic resistance in wider population.
- Widespread use of dewormers has resulted in genetic selection for parasites resistant to certain drug classes. No evidence that rotation of dewormer drug class prevents development of resistance.
- Most dewormers are broad spectrum but none have 100% efficacy.
- Majority of adult horses in training have only small parasite burdens; a small proportion of horses are responsible for the majority of worm egg shedding (broadly, <20% horses are responsible for >80% shedding).
- Larger worm burdens more likely to be associated with clinical disease.
- Parasite control programme should ideally be 'surveillance-based': identify and selectively treat horses with significant burdens to minimize shedding, while reducing reliance on dewormers.
- FECs to identify 'shedders': categorize into low (<200 eggs per gram [epg]), moderate and high FECs. Note that FECs are not a particularly accurate indication of worm burden in individuals (only take account of egg-producing mature female worms) and are best utilised as a population management aid.
- General rule: strategic treatment of all horses in late autumn and/or spring with broad-spectrum larvicidal dewormer effective against strongyles and tapeworms. Additional treatments through spring/summer as required based on faecal sampling (and only for horses with FEC >200 epg); frequency/timing determined by risk categorization of population (horses with pasture access higher risk).
- Avoiding unnecessary treatment of horses with low (<200 epg) FEC helps maintain a 'drug susceptible' population of worms and limits development of dewormer resistance.
- With appropriate application, generally more cost-effective than 'blanket' interval dewormer dosing.
- Treatment intervals: usually based on 'egg re-appearance period' of each drug class: benzimidazoles (fenbendazole) 4 weeks; macrocyclic lactones (ivermectin/moxidectin) 6–12 weeks; tetrahydropyrimidines (pyrantel) 4 weeks.
- Regular (twice weekly) removal of faeces from communal grazing areas to prevent contamination of pasture.

Diarrhoea

Diarrhoea is usually the result of colonic dysfunction and may arise from several different causative agents. Mild, self-limiting diarrhoea without systemic illness is common in racing yards and generally requires no intervention. Much rarer is severe diarrhoea associated with systemic illness or chronic diarrhoea associated with weight loss; infectious or parasitic disease may be responsible and hospitalization is sometimes necessary.

Diarrhoea is usually associated with abnormally rapid transit of food through the large intestine; this limits re-absorption of water/electrolytes and digestion of fibre. Large fluid losses can occur, leading rapidly to dehydration unless replenished. Disruption of the intestinal mucosal barrier can lead to systemic inflammatory response and secondary effects such as organ dysfunction and laminitis.

Causes

- Transient loose faeces/mild diarrhoea: may arise from altered fermentation in large intestine due to change in diet (or poor adaptation to high-carbohydrate/low-fibre ration).
- Antibiotic-associated diarrhoea: normal balance of microflora in hindgut may be disrupted by antibiotic therapy, leading to proliferation of pathogenic microbes. Antibiotics associated with greatest risk vary with geographical region: penicillin/gentamicin, enrofloxacin, doxycycline and ceftiofur implicated on occasion. Risk greatest in horses under stress or with change of antibiotic class during treatment. Occurs in approximately 0.6% of horses in general population treated with antibiotics.
- Acute severe diarrhoea +/– systemic illness: may arise from infectious microbes *(Salmonella*, *Clostridium difficile/perfringens*, *Neorickettsia*, *Coronavirus)*.
- Chronic persistent diarrhoea (with weight loss): may arise from parasitic (small strongyles) or infiltrative (inflammatory or neoplastic) disease.

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Signs

- Diarrhoea: severity varies from loose faeces to projectile diarrhoea.
- +/– depression/inappetence.
- +/– fever.
- Rarely, may exhibit mild colic at onset or just prior to breaking with diarrhoea.
- Clinical assessment can be reasonable guide to dehydration and the need for fluid replacement: raised pulse rate/delayed capillary refill time/dullness/reduced urine output indicate >5% dehydration.

Diagnosis

- Clinical severity and history guide diagnostic efforts.
- Mild diarrhoea/no systemic illness: further investigation generally not required.
- Moderate diarrhoea +/– mild illness (inappetence/colic): blood analysis to determine well-being and hydration status.
- Severe diarrhoea/suspected infectious cause: diagnostic sampling for attempted identification of pathogens. Multiple (3–5) faecal samples for culture or polymerase chain reaction (PCR) +/– immunoassay for clostridial toxins; interpretation of results should account for false positives and negatives and specific characteristics of individual pathogenic species.
- Chronic/wasting diarrhoea: hospital-based investigation for parasitic/infiltrative disease.

Management

- Usually necessary to make treatment decisions without benefit of knowing underlying cause.
- Fluid/electrolyte imbalances (dehydration) and absorption of bacterial toxins can occur rapidly.
- Goal of treatment is primarily supportive: replace fluid and electrolyte losses, reduce systemic and intestinal inflammation, re-establish normal intestinal function.
- Blood analysis abnormalities reflecting endotoxaemia (leucopenia) or significant dehydration merit intervention.
- Probiotics: efficacy doubtful; current evidence does not strongly support use.
- Intestinal 'binding' agents may reduce systemic absorption of toxins from the gut: smectite administered by frequent (every 6–12 hours) nasogastric intubation or oral administration. Activated charcoal is a non-specific binding agent used in other species but is unlikely to have significant impact on the equine hindgut.
- Mild diarrhoea in otherwise healthy horse: usually self-limiting and requires no treatment; address nutrition (e.g., starch overload, inadequate fibre) if thought to be contributing (see also Chapter 22).
- Moderate diarrhoea without dehydration: anti-diarrhoeal treatment warranted: clay/smectite (mucosal protectant) +/– bismuth subsalicylate by nasogastric tube (as required but up to 4x/day).
- Mild-moderate dehydration: fluid replacement therapy with isotonic fluids (by nasogastric intubation providing small intestine is motile).
- Severe diarrhoea +/– systemic illness: life-threatening condition requiring hospitalization and immediate supportive care (large-volume intravenous fluids/anti-endotoxic therapy/gastrointestinal protectants). Supportive measures (distal limb cryotherapy) to prevent laminitis may be warranted.
- Antibiotic therapy generally contraindicated other than in severe cases with suspected/confirmed clostridial/*Salmonella* infections, or those with secondary septic disease at other sites (lungs, jugular veins, liver). Choice of drug dependent on diagnostic findings and laboratory susceptibility patterns. Has potential to further disrupt or delay recolonization of gut by beneficial microbiota. Faecal microbiota transplantation has potential role in management of some cases.
- Biosecurity measures if infectious disease suspected.

Prognosis

- Mild diarrhoea is usually transient and does not interfere with training.
- Severe acute diarrhoea requiring hospitalization associated with high cost of treatment and guarded prognosis for life.
- Antimicrobial-associated diarrhoea: high (approximately 20%) mortality rate due to systemic endotoxaemia or laminitis.
- Survivors have good chance of returning to training but often require lengthy convalescence due to debilitation.
- Chronic/wasting diarrhoea: prognosis dependent on underlying disease. Parasitic disease usually responds to therapy; neoplastic infiltrative disease has poor prognosis.

Oesophageal obstruction ('choke')

Acute obstruction of the oesophagus with food. Breathing is not affected. Potential for backing up of food/saliva with spill over into windpipe. Potential complications include secondary pneumonia and (rarely) oesophageal perforation or stricture.

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Cause
- Usually caused by a food bolus that has been incompletely chewed or bolted quickly.

Risk factors
- Any age/stage of training.
- Most commonly occurs after post-race feed.
- Dental abnormalities (oral pain due to loose premolar cap/fractured tooth/sharp overgrowth) may inhibit normal mastication.

Signs
- Obstruction noted during/immediately after feeding.
- Typically present with acute discomfort, coughing, stretching of neck and presence of food-flecked saliva at one or both nostrils.
- Intermittent reflexive spasm/stiffening of neck following swallowing (Figure 12.3).

Diagnosis
- Clinical findings definitive.

Management
- May resolve without treatment.
- Potential for development of pneumonia if food material enters lungs; warrants immediate veterinary attention.
- Withhold food and water until obstruction cleared.
- Nasogastric tubing and gentle/repeated lavage of obstruction by stomach pump while under heavy sedation (to lower head).
- Antispasmodic medication (hyoscine butylbromide) frequently used to assist clearance but efficacy doubtful. Oxytocin may reduce oesophageal muscle tone.
- If choke has been present for some hours, prophylactic antibiotic medication should be considered and horse monitored in following days for signs of pneumonia (fever/coughing).



Fig. 12.3 'Choke': Typical appearance of neck during transient reflexive spasms.

Prognosis
- Good; once obstruction cleared can be managed as normal.
- If horse suffers from repeated episodes, investigation of underlying cause (feeding/management/dental pathology) warranted.
- Rarely, damage to mucosa may result in oesophageal stricture.

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UROGENITAL CONDITIONS

FEMALE REPRODUCTIVE SYSTEM

The oestrous cycle

Overview

- Fillies and mares are seasonally polyoestrous, with reproductive inactivity through winter and regular oestrous cycles through summer (largely brought about by increasing day length – 'long day breeders').
- Cycle length (ovulation to ovulation) is typically 21–22 days.
- 'Oestrus' ('heat'/'season'; period of sexually receptive behaviour): typically lasts 5–7 days and coincides with development of an ovarian follicle, which produces oestrogen.
- Ovulation (release of a mature egg from the follicle) occurs in the last 24–48 hours of heat period.
- By day 6 after ovulation the follicular remnant becomes the corpus luteum.
- 'Dioestrus': progesterone produced by the corpus luteum dominates for the next 14–15 days: 'heat' behaviour is absent.
- If not pregnant, corpus luteum lysed by prostaglandin and cycle repeats.
- Progesterone level in luteal phase >1.0 ng/mL and during oestrus <1.0 ng/mL.

Transitional cycles

- 'Transitional' phases occur between dormant winter phase (anoestrus) and active summer cycling.
- Irregular pattern of heat periods during transitional phase is common.
- Spring transitional phase: several non-ovulatory waves of follicular development.
- Onset of transitional phase is stimulated by increasing day length.
- Failure to commence regular heat period may warrant further investigation.

Oestrus and performance

- The oestrus cycle is associated with large fluctuations in endogenous sex hormones (oestrogen and progesterone).
- These hormones have a wide range of complex effects on multiple body systems, with the potential for some of these to impact athletic performance or response to training loads.
- While oestrogen has some positive effects on skeletal muscle (both anabolic and enhanced energy metabolism), the impact of the oestrus cycle on training or race performance has been poorly researched in racehorses. In human athletes there is a potentially small negative effect on performance in the early follicular phase of the cycle, although objective evidence is inconsistent and shows considerable variation between individuals.

Managing oestrus

Signs of being in season (oestrus) vary widely between individuals. In most fillies some degree of receptive behaviour is noted but daily management is not affected. A small proportion may be so dominated by the hormonal effects of the heat period that receptive behaviour overrides other activity, thereby interfering with training or racing. It may be desirable to manage oestrus in these circumstances.

Transitional oestrus

- Self-limiting condition, usually requires no intervention.
- Acceleration of transitional phase (by stimulation of the first ovulation of the year) may be possible through use of medication (medication regulations influence method of choice).
- Daily oral administration of synthetic progestin (altrenogest) for 8–15 days +/– single administration of prostaglandin at end of treatment. Ovulation usually within 15 days of end of treatment.
- Use of GnRH analogue (deslorelin) may hasten ovulation.

Control/suppression of oestrus

Oestrus control is achieved either through timing of ovulation to ensure oestrus does not coincide with a particular

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race target ('strategic avoidance'), or suppression of oestrus behaviour altogether.

- Manipulation of timing of oestrus cycle ('short cycling'): achieved by administration of prostaglandin (optimally 5–6 days following ovulation); ovulation determined by serial monitoring of blood progesterone levels. Horse typically returns to oestrus 3–4 days after prostaglandin administration. Possible to bring cycle forward by up to a week (Figure 13.1).

Oestrus suppression is generally achieved by prolonging the luteal phase by either interfering with natural luteolysis or through administration of exogenous progesterone. There is no single technique with complete efficacy and method of choice will depend on individual circumstances and regulatory constraints:

- Oxytocin: administration of exogenous oxytocin during dioestrus (Figure 13.1) disrupts luteolysis, prolonging corpus luteum activity. Methods include daily intramuscular (IM) injections (60 IU) either from days 7–14 after ovulation (requires reproductive assessment to determine exact day of ovulation) or for 29 consecutive days regardless of stage of oestrus cycle. Slow-release formulation oxytocin injections can also be given on days 7 and 10 after ovulation with similar efficacy. Good efficacy (60% to >70%) and may suppress oestrus for 2–3 months. No deleterious effect on future fertility.
- Intrauterine methods: implantation of inert intrauterine devices that mimic pregnancy may result in prolonged dioestrus (average 2 months). A modular magnetic pessary device has demonstrated good efficacy, safety and no apparent deleterious effect on future fertility; other methods used previously (e.g., glass marbles) are no longer recommended due to reports of negative effects on uterine health.
- Altrenogest: daily oral administration keeps filly in dioestrus. Oestrus occurs 5–10 days after end of

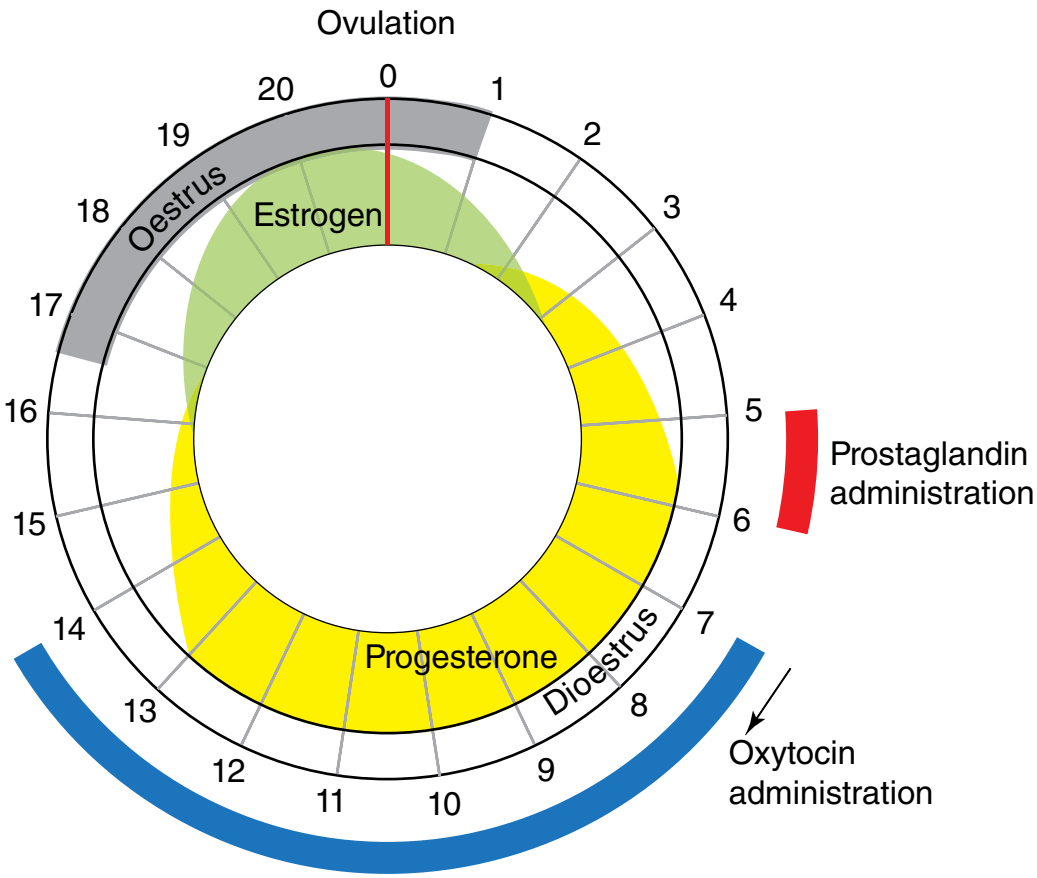


Fig. 13.1 The oestrus cycle: An illustration of the typical relationship between periods of varying sexual receptivity (oestrus/dioestrus), key hormones (oestrogen/progesterone) and optimum times of hormonal interventions to manipulate/suppress oestrus.

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treatment. Trace levels of anabolic steroids trenbolone/trendione are present in some altrenogest formulations.
- Immunocontraceptive vaccine: long-term (3 to >6 months) suppression of oestrus possible following two-dose protocol of gonadotrophin-releasing factor (GnRF) analogue vaccine (antibody response to GnRF). Safe and efficacious at suppressing ovarian activity although oestrus behaviour can persist. Normal follicular activity may not resume for extended period (and may be permanent in some animals); hence use is contraindicated in fillies with a potential breeding future.

OESTRUS MANAGEMENT

- With intervention it may be possible to bring cycle forward ('short cycling') by approximately 1 week.
- Administration of prostaglandin to cause early end to luteal phase of cycle and thereby bring filly back into season. Prostaglandin must be given during period when corpus luteum is sensitive to it; this small window of opportunity is calculated from day of ovulation and treatment may commence with daily injections from days 2–3, or alternatively single administration at 5–6 days post-ovulation.
- Determining timing of ovulation generally easier with serial blood sampling than repeated per-rectum examinations in horse in training.
- Blood sample every 48 hours while filly is in season; if progesterone is 0, hasn't ovulated yet.
- Efficacy of this intervention may vary due to several factors and recommended to follow up with blood sample to check if filly returns into season.

Manipulation of breeding season

- Shortening the winter 'inactive' phase of the reproductive cycle and advancing the spring/summer phase of ovarian activity is advantageous both to prepare fillies for covering and to move the normal spring transitional period forward to avoid interference with racing in those fillies that are problematic during transition.
- Important for fillies/mares to experience shortening daylength in autumn; however, exposure to 'long days' (>15 hours) using artificial light from around winter solstice (peak light-receptive period) onwards can advance the breeding cycle (success of approximately 90%).
- Long-day stimulation needs to occur for minimum of 8 weeks to be effective; typical to continue light stimulation from commencement through to when daylight hours naturally extend in spring.
- Can either commence immediately with 15–16 hours of light or build up incrementally.
- Light supplementation should be used at end of day/evening (ineffective in morning).
- Artificial light can either be environmental (barn lighting) at minimum 100 lux (fluorescent or incandescent) or short wavelength (465–485 nm) blue light applied to single eye with headpiece.

Pregnancy and racing

Racing regulators generally permit racing up to 120 days of pregnancy. The primary consideration regarding pregnancy is the effect of intense exercise on fetal health; however, the effect of pregnancy on athletic performance is also of interest:

- Effects of exercise on fetal health have been little researched in horses; however, in people high levels of fitness to late term are possible with no adverse effect on maternal/fetal well-being.
- Training/racing the pregnant filly is safe; however, attention should be paid to administration of any drugs that may have effects on pregnancy/fetus. Risk associated with intra-articular corticosteroids unquantified but likely to be negligible.
- Effects of pregnancy on performance have not been investigated in horses.
- Considerable physiological changes occur during pregnancy, but these are poorly understood in racehorses: include increased blood volume and cardiac output.
- Evidence from human athletes is ambiguous; however, aerobic power appears to be maintained well in pregnancy and may even be enhanced; some evidence that anaerobic work capacity diminishes in late term.

Mastitis

Infection of the mammary gland. Usually affects only one side of the udder; each teat is fed by two (rarely three) mammary glands.

Causes

- Increased mammary serum production caused by high oestrogen levels may predispose to infection.
- Cyclical or seasonal factors usually responsible; however, high levels of exogenous (plant or fungal) oestrogens sometimes found in hay/haylage.
- Serum leaking may permit secondary bacterial colonization: aerobic bacteria (usually *Streptococcus zooepidemicus*) that exist as normal flora on skin of udder generally responsible. Fungi rarely implicated.

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Risk factors
- Uncommon condition.
- Occurs in fillies in and out of training.
- Risk factors not known.

Signs
- Onset usually acute.
- Painful, firm enlargement of affected gland(s) +/– oedematous filling running forward from udder along belly (Figure 13.2).
- +/– abnormal (purulent/serosanguineous) mammary secretion(s).
- +/– hindlimb stiffness/lameness.
- +/– fever.

Management
- Differentiate mastitis (painful) from benign udder enlargement/lactation (latter needs no treatment).
- Systemic antibiotic +/– anti-inflammatory medication.
- Milking/'stripping out' affected teat once/twice daily may assist resolution through removal of bacteria and debris. Sedation/physical restraint may be necessary if resented.
- +/– oxytocin (10–20 IU).
- Use of intra-mammary antimicrobial products possible but caution required as smaller teat aperture/shorter cistern than cows.
- Continued ridden exercise usually possible.

Prognosis
- Excellent: rapid response to antibiotic therapy is typical and interruption to training minimal.



Fig. 13.2 Mastitis: Swollen left teat (arrowheads) with associated oedema running forward to caudal abdomen.

Pneumovagina/urovagina

'Windsucking' of air (pneumovagina) and pooling of urine in the vagina (urovagina) are separate conditions that may be encountered in fillies in training and occasionally require intervention. Pneumovagina causes a vaginal sucking/gurgling noise during exercise. Both pneumovagina and urovagina may be associated with a secondary vaginitis/vaginal discharge and occasionally irritability and discomfort is observed.

Causes
- Pneumovagina: the vaginal lips and internal vestibular sphincter act as seals against aspiration of air into the reproductive tract. The vaginal seal may be compromised or more readily breached in fillies with light body condition (lack of perineal fat) or poor vulvar conformation; movement at exercise disrupts the weakened seal, allowing vagina to fill with air.
- Urovagina: downward cranial slope of vagina or poor vestibular/vulvar conformation can lead to incomplete voiding of urine. Pooling of urine within vagina may cause inflammation and vaginal discharge.

Management
- Pneumovagina: surgical closure of the upper vaginal lips (Caslick's vulvoplasty), leaving sufficient opening for urination.
- Urovagina: if urine pooling is suspected to be secondary to previous Caslick's, vulva should be re-opened. If due to conformation/condition, specialist surgical intervention is the only treatment: rarely warranted in fillies in training.

Prognosis
- Rarely interfere with training.
- Secondary infection associated with both conditions may have implications for fertility.

MALE REPRODUCTIVE SYSTEM

Castration

Techniques

Castration may be performed as an open (non-sterile) surgical procedure under standing sedation, or under general anaesthesia (GA) using either the same surgical approach or an aseptic sutured technique. The benefits of standing castration are lower cost and lower risk of serious complications, whereas aseptic sutured castration under GA offers lower risk of interruption to training due to post-surgical infection.

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Management

- Standing castration: large surgical incisions, prophylactic antibiotic/anti-inflammatory medication and early return to ridden exercise (trotting/light cantering resuming within 2–5 days) reduces risk of postoperative swelling/infection. Daily cleansing of scrotum +/– greasing of hindlimbs to prevent scalding.
- Sutured (GA, aseptic) castration: 1 week of walking prior to resumption of ridden exercise. No/little wound care required.
- Fertility may persist for up to 6 weeks post castration.
- Behavioural changes may not be observed for weeks/months depending on prior temperament and age at castration.

Complications (Table 13.1)

- Standing castration: approximately 20% of horses require some treatment (re-opening of incisions +/– further antibiotic therapy) for postoperative swelling or infection. Risk of serious complication (death/eventration of intestine) very low regardless of age. Excessive haemorrhage in immediate post-surgery period almost invariably resolves with short period (1–2 days) of restricted exercise +/– administration of acepromazine.
- GA castration: risk of minor complications (infection, swelling) much lower than standing castration; however, risk of death arising from GA is 0.05–1%.

Table 13.1 **Complications associated with castration**

COMPLICATION	DESCRIPTION	TREATMENT	OUTCOME
Post-castration haemorrhage	Minor bleeding (Figure 13.3a) is common in hours following open castration. Persistent and profuse bleeding is rare and is usually result of incomplete crushing of testicular artery	Bleeding usually minor and of no concern. If drips/small stream, no treatment required but restrict exercise until stabilized. Profuse bleeding: administration of acepromazine may decrease severity. Persistent profuse bleeding: clamp cord, leaving forceps in place for 24–36 hours; if unsuccessful, surgical exploration may be required (exceptionally rare).	Excellent. Intervention rarely required
Post-castration swelling	Minor swelling around scrotum/sheath (Figure 13.3b) common in immediate days following standing castration	No treatment required. Swelling of sheath (prepuce) indicative of insufficient daily exercise	Resolves with exercise
Eventration	Abdominal cavity and scrotum communicate; creates potential for intra-abdominal contents to herniate through surgical wound. Very rare. May involve small intestine or omentum. Usually occurs within 4 hours of surgery	Prolapsed omentum (Figure 13.3c): transect under standing sedation. Eventration of small intestine: surgical emergency, requires cleaning/protection of exposed intestine and immediate surgical referral	Excellent survival rates (>85%) for small intestinal eventration with appropriate management
Infection	Swelling of one/both sides of scrotum at 1–2 weeks following open castration is common; usually coincides with incision healing and subsequent lack of drainage. Painful scrotal/inguinal thickening +/– fever +/– hindlimb lameness or stiffness	Systemic antibiotic +/– anti-inflammatory therapy (to effect). Re-establish drainage +/– flushing scrotal wound (antiseptic solution) as required	Most cases resolve with 1–2 weeks of antibiotic therapy. Very rarely, infection of spermatic cord non-responsive to medical therapy (funiculitis) can develop over months and necessitate surgical removal of infected stump.
Infection (peritonitis)	Self-limiting (5–7 days) non-septic peritonitis is common after open castration and requires no treatment. Septic peritonitis is rare: recurrent pyrexia/colic/weight loss/diarrhoea in weeks following castration	Broad-spectrum antibiotic +/– anti-inflammatory therapy +/– hospitalization	Good with appropriate management

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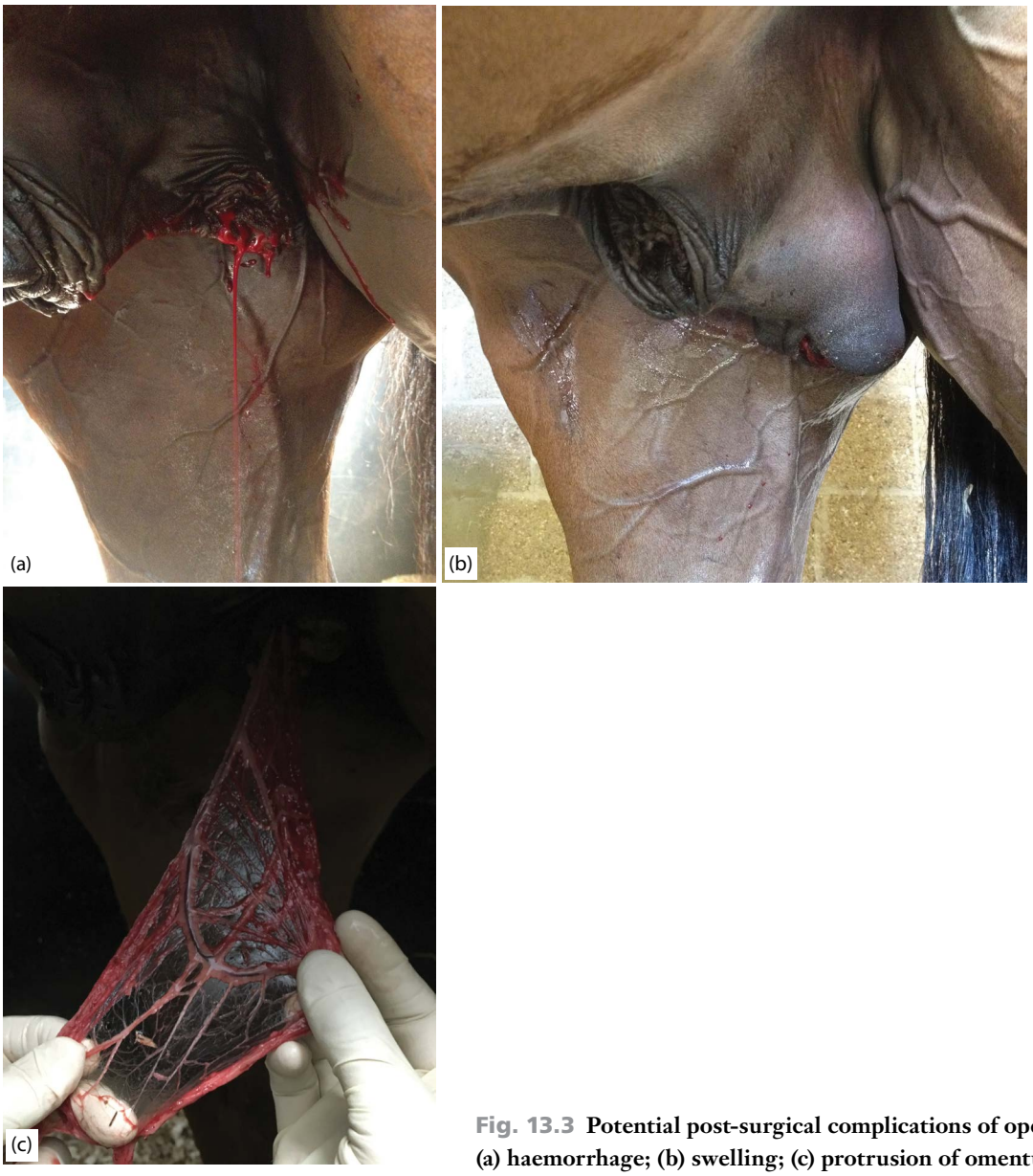


Fig. 13.3 **Potential post-surgical complications of open castration: (a) haemorrhage; (b) swelling; (c) protrusion of omentum through wound.**

Cryptorchids ('rigs')

Cryptorchidism is a failure (partial or total) of one or both testicles to descend into the scrotum. The retained testicle is typically much smaller than the external testicle. Usually only one testicle is affected. Affected testicle is more likely to be intra-abdominal if left-sided than if right-sided. Bilateral cryptorchids and monorchids (one testicle completely absent) are very rare.

Causes

- Testes develop within abdomen of fetus and are programmed to migrate from their origin near each kidney through the internal and external vaginal rings of the abdominal wall to the scrotal sac.
- Migration guided by structure known as the gubernaculum and subject to hormonal control.
- Final descent of testes into scrotum occurs either just prior to, or in days after, birth.
- Failure of descent can result in testicle being retained anywhere along migratory path, from fully intra-abdominal (usually near body wall) to within inguinal canal.
- Cryptorchids ('rigs') classified as abdominal, incomplete abdominal or inguinal.

Risk factors

- Genetic factors thought to play a role, but direct heritability unlikely in most cases.

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Effects
- Abdominal testicles are generally sterile but produce testosterone.
- Cryptorchids display normal colt behaviour.
- Retained testicle almost never responsible for lameness/gait abnormality (although frequently blamed).

Diagnosis
- Diagnosis based on clinical evaluation and history is usually sufficient.
- Thorough examination may require sedation; if inguinal, the high testicle may be partially palpable.
- Further confirmation may be required for surgical planning (or when possibility of previous hemicastration exists); passport should be checked for declaration of previous surgery.
- Transabdominal ultrasonography: high sensitivity and specificity for detection of both abdominal and inguinal testes.
- Blood hormonal assays: range of tests available and choice dictated by individual circumstances; multiple tests may be warranted in legal cases. Serum oestrone sulphate (only for horses >3 YO) or anti-Müllerian hormone (AMH) (any age) both have high sensitivity for detection of retained testicular tissue. Stimulation of testosterone production by administration of human chorionic gonadotrophin (hCG); blood samples before, and 1 and 24 hours after intravenous (IV) injection of 6000 IU hCG highly sensitive for the detection of testicular tissue but not in young horses (<18 months) or during winter.

Management
- Of little concern for training aside from planning for appropriate castration technique.
- Full descent of testicles occurs within weeks of birth in most normal horses; however, descent of an inguinally located cryptorchid testicle may occasionally occur in young adult life (yearling to 2 YO).
- Determining likelihood of full descent of retained testicle is not possible.
- Hormonal treatment (hCG) to assist descent of inguinal testes has low efficacy.
- Castration technique determined by location of retained testicle: inguinal or scrotal surgical approaches under GA, or standing laparoscopic removal (if abdominal).

URINARY TRACT PROBLEMS

Urinary tract problems are rare in horses in training. Although horses are sometimes presented for examination by training staff because of concerns over irregularities of urination, drinking or associated behaviour, these almost invariably are found to be non-pathologic and transient.

Polyuria/polydipsia
- Normal maintenance water requirements for a horse in training vary according to workload, diet and climate but typically 60–70 mL/kg bodyweight (BWT)/day (around 30 L/day).
- Detailed history and measurement of daily water intake may assist investigation of excessive drinking/urination.
- Blood analysis, including biochemistry (blood urea nitrogen, creatinine and glucose concentrations) and urinalysis (specific gravity and presence of glucose/protein), generally permits differentiation of urinary tract disease from non-renal causes (psychogenic polydipsia).
- Excessive drinking +/– urination in healthy racehorses may result from 'psychogenic polydipsia', a form of stable vice. Can also arise from high dietary protein (such as if fed on legume rather than grass-based forage) or salt, or administration of corticosteroids or diuretics.
- Psychogenic polydipsia: only abnormality is low urine specific gravity (<1.005).
- Water deprivation testing can also be undertaken to determine whether horse is capable of concentrating urine; should not be used if evidence of renal insufficiency on blood analysis.
- Generally: if horse appears healthy (no weight loss, pyrexia etc.) and no clinical concern of disease/pathology, treatment/management is usually unnecessary.
- Treatment of psychogenic polydipsia (if desired): restriction of water to maintenance requirements and improving level of stimulation in the stable environment.

Urinary tract infections
- Urinary tract infections very rare in young racehorses.
- May occur secondary to urinary obstructions (bladder stones/sediment).
- Can affect lower (urethra/bladder) or upper (kidney) urinary tract.

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- Lower urinary tract signs: include straining to urinate, frequent small-volume urination, urine scalding.
- Upper urinary tract signs: include weight loss, fever, mild colic.
- Diagnosis: mid-stream urine sample (cytology/culture), ultrasonography, endoscopy.
- Treatment: antibiotic medication, guided by bacteriological results.

INTERSEX DISORDERS

Disorders of sexual development are very rare but may be encountered during investigations into infertility or aberrant testosterone levels. Sex differentiation is a complex process that begins with chromosomal makeup (determined at fertilisation). Gonadal development and subsequent hormonal production then contribute to the external characteristics of sex such as body type, bone mass and behaviour. Genital development has a natural tendency towards female differentiation in the absence of testosterone, with masculinization being an active process requiring both testosterone production and tissue sensitivity to it.

Developmental abnormalities can occur at any stage of this process; therefore, a great variety of intersex conditions exist. X-chromosome monosomy, and male-to-female sex reversal are the most common genetic abnormalities. Affected horses typically have a disparity between their genetic sex (e.g., XY sex chromosome constitution) and their sex phenotype: they may be outwardly normal but sterile/subfertile, have ambiguous or underdeveloped internal sexual organs or external genitalia, display inappropriate behaviour or have abnormal testosterone levels. Diagnosis may sometimes be presumptive based on clinical findings and blood levels of reproductive hormones, but definitive diagnosis often requires exhaustive genetic analysis. The heritability of various forms of the intersex condition has been poorly investigated; however, both recessive and sex-limited dominant transmission are recognized.

The key considerations regarding intersex disorders are gender assignment to satisfy concerns of racing regulatory bodies, and determination of fertility status in the case of horses intended for breeding. Once racing authorities have been made aware of the ambiguous sex status of a racehorse, restrictions may be imposed on the type of races the horse in question is permitted to enter. This is usually done on an individual basis and approach may vary between jurisdictions. Classification as male or female in many affected animals is far from simple; human sporting bodies have moved away from arbitrary categorization based on chromosome analysis to regulations that take into account testosterone levels, external phenotype and sporting distance/discipline, and these regulations continue to evolve due to scientific and societal influences. Equivalent refinement of regulations in respect of intersex disorders does not currently exist in horseracing.

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NEUROLOGICAL CONDITIONS

VERTEBRAL COLUMN CONDITIONS

'Wobbler' syndrome (cervical vertebral compressive myelopathy/ cervical stenotic myelopathy/ cervical vertebral malformation)

'Wobbler' syndrome is a syndrome in which malformation or instability of the cervical vertebral column leads to compression of the spinal cord, causing neurological gait deficits of varying severity.

Impingement on the cord may be constant (static) or may worsen with changes in head/neck position (dynamic). Sites most commonly affected are C3–C5 for dynamic compression and C5–C7 for static compression, although generalized narrowing of the canal is usually present regardless of the actual lesion location.

The peripheral location of nerve tracts involved in voluntary control of skeletal muscle means that upper motor neuron signs (incoordination, weakness +/– spasticity) define the syndrome. Because of the more superficial location of nerve tracts servicing the hindlimbs they are usually more severely affected than the forelimbs.

Causes

- Developmental condition.
- Likely that there are multiple causative pathways that result in similar clinical presentations.
- Probably result of both genetic and nutritional factors, with rapid growth, high energy intake and micronutrient imbalances (both during pregnancy and in early life) leading to abnormal bone/cartilage maturation in some genetically susceptible horses.
- Patterns of heritability thought to be complex.
- Maturation of cervical vertebrae continues until around 5 years of age (at which point vertebral body growth plates close).
- Compression of spinal cord can occur through generalized narrowing of spinal canal or malformation/malalignment of certain vertebrae.
- In older horses, static compression may arise from arthritis of articulating process joints (APJs) in neck base, with bony impingement through the intervertebral space.

Risk factors

- Prevalence in young Thoroughbred population estimated at 0.5–2%.
- More common in males than females (3:1).
- Large, rapidly growing individuals appear at greatest risk; growth plate enlargement and developmental orthopaedic disease implicated.

History

- Onset of signs usually occurs between 6 months and 3 YO and is typically insidious.
- Acute-onset disease, and signs that fluctuate in severity are also encountered.
- Riders may raise concern about stumbling, poor action or lack of hindlimb propulsion.
- Atypical cases occasionally occur in which weakness (+/– signs of discomfort) or collapse can occur acutely during/after exercise in a horse with no prior signs of neurological dysfunction.

Signs

- Incoordination (ataxia, Table 14.1) and postural muscle weakness (paresis) are main presenting signs: may walk as though sedated ('truncal sway').
- Cranial nerve function and mental status are normal.

Table 14.1 **Grading of ataxia**

GRADE	SIGNS
0	Normal
1	Minimal neurological deficits noted with normal gaits; requires manipulative tests (tight circles/backing up/crossing legs/tail sway) for detection
2	Mild deficits noted at walk; more obvious response to manipulative tests; may stumble/drag toes at trot.
3	Readily apparent at walk
4	Very ataxic, may fall if turned tightly or backed
5	Recumbent

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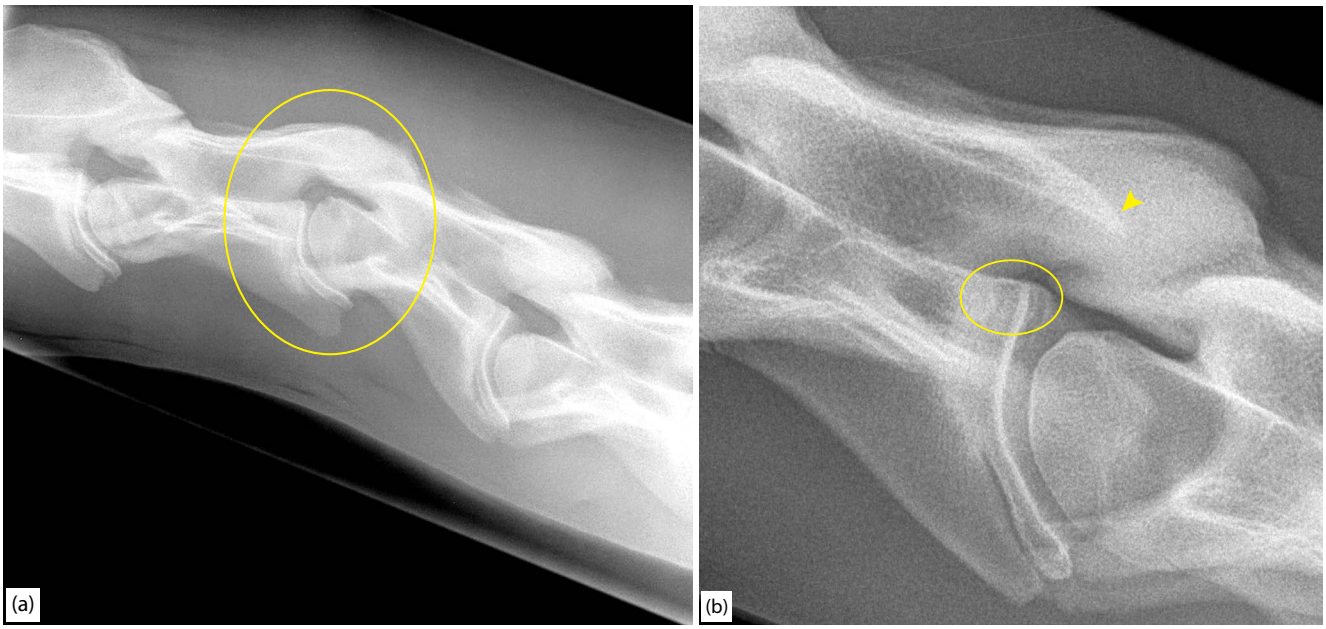


Fig. 14.1 **Radiological features associated with 'wobbler' syndrome: (a) vertebral malalignment (circled) (at C3/C4 site); (b) caudal extension of dorsal lamina (arrowhead) and flare of caudal physis (circled).**

- Ataxia is usually of greater severity in hindlimbs than forelimbs.
- When turned in tight circles: exaggerated circumduction of outer hindlimb +/– interference or crossing over of hindlimbs.
- When backed up: dragging of hindlimbs and weakness.
- When walked forward with head elevated (particularly downhill): inconsistent hoof placement +/– spasticity all limbs +/– dragging of forelimbs.
- When walked forward and tail pulled each side: lateral weakness and overcorrection on release.
- +/– loss of tail tone.

Diagnosis

- Presumptive diagnosis has traditionally been based on clinical findings and radiography.
- Standing radiography may support clinical suspicion; however, sensitivity is too low (<50%) for definitive diagnosis and limitations should be recognized. Advanced imaging (contrast myelography/ MRI/CT) enhances diagnosis.
- Radiological features: flare of caudal physis ('ski-jump')/caudal extension of dorsal lamina/ vertebral malalignment most useful for diagnosis (Figure 14.1a,b). Caution when interpreting neck base arthropathy, as common in normal horses.



Fig. 14.2 **Measurements used for intravertebral sagittal diameter ratio: Minimum intravertebral diameter (a) and vertebral body width (b).**

Measurement of intravertebral sagittal ratio (ratio of minimum sagittal diameter of canal to width of corresponding vertebral body) allows assessment of vertebral canal stenosis (Figure 14.2). Ratio ≤50% of any vertebra from C4–C6 (≤56% at C7) is strongly

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linked to disease, although operator variation is in the order of 5–10%.
- Multiplanar imaging (CT/MRI) offers significant diagnostic advantages over radiography (including avoidance of superimposition, and identification of spinal nerve compression); however, technical limitations (size of equipment, inability to evaluate dynamic compression) and challenges in determining clinical relevance of suspected compressive sites may balance usefulness. Imaging under general anaesthesia (GA) associated with increased complication risk relative to standing radiography.
- Transcranial stimulation (performed on sedated horse) is diagnostic test with highest specificity for spinal cord disease. Measures latency time between magnetic/electrical stimulation through spinal cord and onset of muscle contraction; this time period is prolonged and variable in horses with spinal cord disease.

Management
- Horses with ataxia graded ≥2 are generally considered unsafe to ride and may require care in handling, especially when sedated.
- Euthanasia may be considered in moderately/severely affected horses due to uncertainties around safety/return to athletic use, and suspected genetic influence making retirement for breeding an unfavourable choice. Insurance status may necessitate interim period of conservative management prior to final assessment.
- Diagnostic imaging findings may assist determination of best treatment path (and prognosis): degenerative changes affecting axial surfaces of articular process joints much less likely to respond to non-surgical management.
- Conservative management may stabilize condition in proportion of cases: stable rest (4–8 weeks), dietary changes and anti-inflammatory (corticosteroid +/– non-steroidal anti-inflammatory [NSAIDs]) medication. Improvement in neurological signs may result, but complete recovery sufficient to resume training is uncommon.
- Dietary management aimed at slowing growth while maintaining balance of key nutrients: restriction of energy and protein intakes to 75% of National Research Council (NRC) recommendations (+/– supplementation with vitamins A, E); low carbohydrate (CHO) diet.
- Surgery (fusion of adjacent vertebral bodies at affected sites) may be considered for valuable individuals and can result in return to athletic use. Uncertain outcome, lengthy recuperation (6–12 months), frequently incomplete recovery (by 1–2 neurological grades) and concerns over ethical aspects of breeding from affected horses mean that surgical intervention is rare in racehorses, although high success rates have been reported.
- In skeletally mature horses with ataxia arising from degenerative pathology of the APJs (not the typical situation encountered in young racehorses), intra-articular medication of affected sites (guided by CT myelography) may result in clinical improvement.

Prognosis
- Prognosis for racing (and other athletic disciplines) generally considered to be poor.
- Up to 30% of horses with milder grades (1–2) of ataxia may race following conservative/medical management.
- Surgery: better success rate than conservative/medical management in all but the lowest grades of ataxia, but associated with significant serious/fatal complication rate (implant infection/failure, neurological deterioration, upper airway obstruction).
- Surgery: majority (>75%) of horses improve by 1–2 grades; return to athletic use is more likely in horses with dynamic compression, younger horses and those in which clinical signs were only present for <1 month.

VIRAL INFECTIONS

Equine herpes virus 1 myeloencephalopathy

Equine herpesvirus (EHV)-1 is endemic throughout horse populations worldwide and most horses (>80%) are exposed at some point early in life and become latent carriers. Multiple viral strains exist and strains associated with neurological disease and hypervirulence are recognized; however, current understanding of relationships between strain types, horse immunity factors and type/severity of disease is limited. The virus most frequently causes respiratory disease but occasionally is associated with neurological signs. Outbreaks of ataxia and weakness caused by the neurological form of EHV-1 are rare but occur sporadically in racing yards. Severely affected horses may become recumbent and require euthanasia, and disruption to yard operations is inevitable due to movement restrictions until spread of disease has been controlled.

Causes
- Neurotropic strains of EHV-1 cause vasculitis in the central nervous system (CNS) with resulting widespread ischaemic necrosis of neural tissue.

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- Non-neurotropic strains can also cause myeloencephalopathy (less common).
- Patient factors, including immune status, stress and genetic susceptibility, may play a role in development of disease but poorly understood.
- Dynamics of virus latency and re-activation also poorly understood; horses may be infected with multiple EHV-1 strains at same time, making attribution of clinical disease to specific strain(s) challenging.
- Disease spreads readily between horses through direct (respiratory/nasal route) and indirect (people, surfaces: environmental persistence for up to days/weeks) contact.

Risk factors
- Usually adult horses; any stage of training.
- Previous vaccination for EHV-1/4 does not currently preclude development of neurological disease.

History
- Acute-onset ataxia.
- Recent respiratory disease (within past 1–2 weeks) in affected individual or yard is frequently reported.

Signs
- +/– initial fever, followed within 7 days by neurological signs in some horses.
- ataxia +/– paresis; usually symmetric and affects hindlimbs to greater extent (may 'dog-sit').
- +/– urinary incontinence (bladder paralysis).
- Ataxia may be transient or progress to recumbency.
- Respiratory disease and limb oedema may be noted in larger population at time of outbreak.

Diagnosis
- Virus detection: polymerase chain reaction (PCR; nasopharyngeal swab/blood sample); or detection of antibody response (seroconversion ≥4-fold increase in titre in samples 10–20 days apart).

Management
- In case of neurological EHV: strict adherence to statutory requirements and current recommendations from regulatory bodies, including isolation of premises and biosecurity measures such as minimising shared airspace between known cases and potential contacts.
- Monitoring rectal temperatures twice daily in all in-contact horses, with further PCR testing for horses that develop fever and to monitor shedding in known clinical cases.
- Avoid stress/strenuous exercise in at-risk horses until disease status known.
- Movement restrictions for 1 month; lifting of restrictions only when either serological monitoring determines no further activity within at-risk group or >14 days from onset of signs in last clinical case.
- Cases demonstrating neurological disease: anti-inflammatory (NSAID) +/– antiviral medication. Typically NSAIDs at full dose while pyrexic, and tapered dose for further 3–5 days.
- +/– dexamethasone in acute phase (generally not advised with concurrent use of NSAIDs).
- Pyrexic/close contact horses (+/– EHV-positive test) without neurological signs: prophylactic antiviral (valacyclovir) medication is recommended to reduce chance of developing neurological disease; best effects if given pre-inoculation.
- Booster vaccination in the face of an outbreak is a risk-based decision. Generally inadvisable to vaccinate any horses that may have been recently exposed as may potentiate neurological form of disease. Vaccination of wider non-contact group may potentially reduce shedding/spread.
- No current vaccines prevent the neurological form of the disease; however, evidence that virus shedding (and therefore severity of outbreak) may be lower in vaccinated yards.

Prognosis
- Ataxic horses that remain ambulatory have good prognosis for return to full function but may take months.
- Recumbency >24 hours is poor prognostic indicator but intensive supportive care of recumbent horses generally rewarded with partial recovery (guarded prognosis for return to athletic use).
- Mortality 5–30%.
- Horses recovered from neurological EHV do not appear to pose any greater risk of future infection to susceptible populations.

Equine protozoal myeloencephalitis

Equine protozoal myeloencephalitis (EPM) is a neurological disease caused by interaction with protozoal parasites (primarily *Sarcocystis neurona*; less commonly *Neospora hughesi;* possible link with *Toxoplasma gondii*). Disease is currently restricted to horses living in or originating from the Americas (geographical limit of main parasite *S. neurona*).

Causes
- Parasitic life cycle is complex, involving definitive host (opossum in the case of *S. neurona*) and any of multiple intermediate mammal hosts.

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- Sporocysts are passed in opossum faeces and the infective stage may contaminate feed or water.
- Birds, insects and other vectors may act to disseminate the parasite.
- Horse (along with other mammals) is a 'dead end' host in which the parasite does not replicate but rather may cause disease during its colonization.
- Following ingestion, parasite passes from gastrointestinal tract to bloodstream then eventually localizes in CNS.
- Many factors involved in development of clinical disease including stress at/after infection, number of parasites ingested and location within CNS (spinal cord, brain stem or brain) of protozoal activity. Not all horses infected with parasite will develop disease, and factors influencing progression to severe neurological disease poorly understood.
- Most horses mount an effective and long-lasting immune response against parasite and never develop clinical disease. 'Horizontal' transmission between horses does not occur.
- Life cycle and modes of transmission of *N. hughesi* poorly understood.

Risk factors

- Majority of horses (approximately 30–80% depending on location) within the USA have serological evidence of exposure to *S. neurona*; however, clinical disease only develops in small proportion (approximately 0.14%): typically sporadic and rarely involves more than 1 horse on a farm/yard, although clusters can occur.
- Likely that some horses are more susceptible than others to disease; risk factors unknown.
- Risk greatest in young (<5 YO) horses.
- Transmission between horses does not occur (horses do not produce any infective stages).
- Thought that transplacental transmission also does not occur.

History

- Onset of clinical disease usually insidious, but can occasionally be acute.
- Disease is predominantly limited to the Americas.
- Occasional clinical case encountered in Europe/Australasia in horses originating from the Americas; clinical signs may arise many months or even years after original exposure to parasite.

Signs

- Neurological signs vary widely and determined by location and extent of lesions within CNS.
- Most commonly involves spinal cord (rather than brain).
- May mimic other diseases such as wobbler syndrome (p. 349).
- Primary characteristic that differentiates it in many cases is asymmetry: of neurological dysfunction or muscle wastage.
- Ataxia, weakness, stumbling, abnormal gait; milder presentations may be confused with lameness.
- Selective loss of muscle of face, limbs or topline.
- Less commonly, brain signs: head tilt, seizures; also difficulty swallowing.
- Gradual progression in severity and range of signs is typical, although sudden deterioration may also occur.

Diagnosis

- Definitive diagnosis currently difficult (in live horse) due to lack of specificity of current tests.
- Differentiation from some neurological diseases (wobbler syndrome; EHV-1 neuropathy) may be relatively straightforward on clinical/history/diagnostic testing grounds, whereas others are more complex (viral encephalomyelitides, neoplasia, toxicities, CNS infections, head trauma, migrating parasites).
- Presumptive diagnosis based on thorough neurological examination combined with diagnostic serological testing (paired serum cerebrospinal fluid [CSF]).
- Blood serology alone does not differentiate exposure to *S. neurona* (common) from clinical disease (rare); serological testing as a screen in healthy horses/pre-purchase context inadvisable due to very low positive predictive value in absence of neurological signs.
- Negative serum test generally useful to exclude disease (high negative predictive value for clinical disease).
- Combination of blood and CSF testing currently used to determine likelihood of disease: quantitative assays that determine ratio of antibody in blood/serum to CSF to confirm intrathecal antibody production. Molecular (PCR) testing of CSF may assist diagnosis.

Management

- Affected horses pose no risk of infection to other horses.
- Due to difficulties associated with definitive diagnosis, presumptive treatment may be appropriate.
- Early treatment is important for successful outcome.

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- Anti-protozoal medication (ponazuril; diclazuril; sulfadiazine/pyrimethamine): duration of course dependent on manufacturers' advice and clinical progress but typically 6–8 weeks.
- Anti-inflammatory medication (NSAIDs/corticosteroids/antioxidants) may be used to manage some clinical signs in short term.

Prevention

- Minimizing exposure to infective protozoal stages: securing feed from possible contamination by opossums and rodents.
- Intermittent prophylactic use of anti-protozoal medication in youngstock on farms with high exposure rates to *S. neurona* may be justified in some circumstances, although standard protocols have not been established.
- Affected horses are not a biosecurity risk to others (no horizontal transmission).

Prognosis

- Clinical cases if not treated will deteriorate, although may take months/years.
- Better prognosis for cases treated early in course of disease.
- Majority (60–70%) of cases treated promptly show significant clinical improvement, with up to a third of these recovering completely.
- Best chance of complete recovery for less severely affected horses.
- Relapse in small proportion of cases.

Arboviral encephalitis

Arboviral encephalitis is a broad term for a group of viral diseases (West Nile virus [WNV], Eastern [EEE], Western [WEE] and Venezuelan [VEE] equine encephalomyelitis, Murray Valley encephalitis [MVE], Japanese encephalitis [JE], Ross River Virus [RRV]) spread by blood-feeding insects (most commonly mosquitoes) and causing predominantly neurological signs. Arboviruses replicate in the body of the transmitting insect; the natural reservoir for disease is primarily the wild bird population (also rodents, other mammals, macropods for some diseases) with horses being a 'dead-end' host for the virus. Horse-to-horse and horse-to-human transmission is highly unlikely because of the low levels of virus circulating in infected animal (although can occur with VEE). Seasonal appearance of clinical cases is typical.

The geographical distribution of the main equine arboviruses within and between countries varies seasonally with climatic conditions and spread of natural hosts and insect vectors. Currently:

- WNV: Americas, parts of Europe, Africa, Middle East, Australasia.
- EEE and WEE: Americas.
- VEE: Central and South America.
- MVE: Australia.
- JE and RRV: Australasia.

Causes

- Reservoir of infection in natural host (predominantly wild birds, but also rodents and other mammals, macropods), with transmission of virus to horse by mosquito bite. Insect vector(s) and major host(s) differ between viruses.
- Inflammation of CNS may ensue after initial viraemia, resulting in neurological signs.
- Proportion of infected horses that develop clinical disease and severity of disease varies between viruses and virus strains.
- Incubation period 3–15 days.
- Horses that recover from infection generally develop long-term immunity.

Signs

- Vary in type, severity and progression between both causative viruses and individual horses.
- May include combinations of: fever, depression, weakness, ataxia, cranial nerve deficits, altered mental state (depression, aggression or hyperexcitability), head pressing, blindness, convulsions.
- Severe cases may progress to recumbency +/– death within 2–3 days.
- Neurological signs not always symmetric.
- WNV: neurological morbidity approximately 10%.

Diagnosis

- Presumptive diagnosis often based on clinical presentation and knowledge of disease status of locality.
- Should be differentiated from other diseases with similar signs: EPM/EHV/wobbler syndrome/rabies/botulism/bacterial meningitis.
- Similarities in clinical presentation and serological cross-reactivity can complicate diagnosis.
- Serology (fourfold change in antibody titre in paired samples 10–14 days apart) often strongly indicative.
- PCR testing sensitive but transient/short-lived viraemia may result in false negatives.

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- IgM enzyme-linked immunosorbent assay (ELISA; testing for recent antibodies).
- Definitive diagnosis may require postmortem sampling; care in handling tissues of horses suspected of having the disease, as human infection possible.

Management

- No specific therapies exist.
- Supportive care: hospitalization/fluids/anti-inflammatory medication.
- WNV: hyperimmune plasma early in disease may improve outcome.
- Severe clinical signs or rapid deterioration may warrant euthanasia.

Prevention

- Vaccination (WNV/EEE/WEE/VEE/JE) for animals living in, or travelling to, endemic areas.
- Primary vaccinations/annual boosters should be completed before onset of risk period for mosquitos.
- Minimize exposure to mosquitoes: stabling and lighting management/use of insect deterrents and insecticides/elimination of standing water.

Prognosis

- WNV: around 30% of horses with clinical disease die or require euthanasia.
- Recumbent horses have poor prognosis for survival.
- Mortality rate of EEE, VEE and WEE can be high (50–90% for EEE, the most pathogenic).
- Many horses that recover from clinical disease (up to 30% for WNV) have residual neurological deficits (e.g., gait abnormalities) that can persist for months/years.
- MVE: low morbidity and mortality (approximately 10%) rates.

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SKIN CONDITIONS

INJURIES

Interference injuries

See Chapter 4 p. 45.

Girth 'gall' ('girth sore')

Acute bruising/myositis/abrasion of girth region. Usually most severe within 12 hours of incident and diminishes over subsequent days.

Cause

- Overtight/pinching girth.

Signs

- Swelling/oedema at/just behind girth (on lower midline or one/both sides) (Figure 15.1).
- +/– mild palpable tenderness.
- Hair loss unusual, and usually no break in skin.
- Occasionally focal dermatitis.
- Occasionally a firm, painful enlargement of underlying pectoral muscle(s).

Diagnosis

- Clinical findings definitive.



Fig. 15.1 **Girth gall.**

Management

- Topical (if unbroken skin) +/– systemic non-steroidal anti-inflammatory drug (NSAID) or corticosteroid medication in acute phase to reduce swelling.
- Severe swelling (rare): may benefit from 1–2 days out of ridden exercise.

Prognosis

- Resolves rapidly and rarely interferes with training.

INFECTIONS

Cellulitis/lymphangitis

[See also Peritarsal/inguinal infection, p. 269].

Ascending generalized infection of (usually) single limb with resulting inflammation/swelling. Most frequently affects cannon/fetlock region. Severity and progression varies.

Cause

- Typically but not invariably associated with small entry point for infection (puncture, cut or focal dermatitis).

Signs

- Filling/oedema of limb.
- Severity varies and in worst cases may extend to upper limb.
- Generalized pain response to palpation.
- Mild (most commonly) to severe lameness; typically warms up with forced exercise.

Diagnosis

- Clinical findings usually definitive.
- Diagnostic procedures rarely required.
- If lameness severe and/or unremitting: consider investigation to rule out synovial sepsis

Management

- Antibiotic and anti-inflammatory medication.

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- Exercise assists resolution and disperses filling: appropriate level of exercise determined by severity, but in general forced walking during acute phase and return to ridden exercise as soon as possible.

Prognosis
- Most cases respond favourably to treatment, with little interruption to training.

Pastern dermatitis ('cracked heels'/'mud fever')
Inflammation/infection of heel/low pastern region of one or more limbs; characterized by dry or wet dermatitis, crusting and variable localized filling. Severity and response to treatment varies. Often involves white limbs. Some horses appear more prone to condition than others.

Causes
- Multifactorial.
- Generally an opportunistic infection by bacterial pathogen(s) due to breakdown in local skin defences.
- Factors that contribute to development: excessive wetting or wet-dry cycles; poor cleaning following exercise; inappropriate skin dressing; skin trauma from interference or boot rubs; some track surfaces appear to predispose.
- Frequently a seasonal problem (winter/wet conditions).
- Bacterial species generally free-living in environment: most commonly involves mixed bacterial population; may include *Staphylococcus* spp.

Signs
- Patch of dry or wet crusting/matted hair (Figure 15.2a,b).
- +/– cellulitis/inflammation of pastern.
- +/– localized pain on palpation.
- +/– lameness (only with secondary cellulitis).
- May crack/bleed during exercise.

Diagnosis
- Clinical findings definitive.
- Bacteriological investigation (culture) rarely useful.

Management
- Management guided by recent history of previous treatment.
- Variety of approaches to treatment: most incorporate cleaning/debridement and topical medication.
- Initial topical application of debriding/keratolytic cream useful to lift crusts, followed by daily/twice-daily application of antibiotic cream until resolution.
- Daily management: light barrier cream (paraffin) for exercise; gentle cleansing (chlorhexidine solution) and drying post-exercise, followed by application of topical antibiotic cream.
- Choice of topical antibiotic cream determined by local experiences and efficacy.
- Clipping of any long hair around site may assist treatment.
- Avoid aggressive cleaning/irritation of site.
- +/– systemic antibiotic medication if cellulitis present.
- Some limited evidence that pine wood shavings may be advantageous over other bedding types as inhibits growth of bacterial pathogens involved in disease (possibly due to presence of polyphenols).

Prognosis
- Rarely interferes with training but significant daily care often needed.
- May persist for weeks/months in troublesome cases.



Fig. 15.2 Pastern dermatitis ('cracked heels'): (a) typical wet dermatitis in 'crease' of pastern; (b) extensive crusting and matting of hair.

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- Susceptible horses require careful management/vigilance to prevent recurrence; generally a favourable response to prompt treatment.

Bacterial boils and 'runners'

Acute, localized skin infection characterized by raised papule (usually solitary) and intense pain. Most commonly found in areas of tack contact/pressure, particularly withers or flank.

Causes

- Bacterial (usually staphylococcal) infection.
- Usually an opportunistic infection caused by breakdown of local skin barrier.

Signs

- Raised oedematous papule often with small central 'head'.
- +/– single or multiple raised lymphatic 'runners' (Figure 15.3).
- Usually intensely painful to palpate and may interfere with tack.

Diagnosis

- Clinical findings definitive.

Management

- Systemic antibiotic (3–5 days) medication +/– topical antibiotic ointment.
- Fomentation may encourage rupture and early resolution.

Prognosis

- Resolves rapidly with treatment.



Fig. 15.3 **Bacterial 'boil' (arrow) with engorged lymphatic vessels ('runners') on flank.**

- Interruption to training depends on lesion location/severity and potential interference with tack (but usually short term, <2 days).

Bacterial folliculitis

Bacterial infection of hair follicle(s), most commonly at sites prone to sweating +/– pressure from tack (back/flanks/neck).

Signs

- Multiple small firm dermal papules that usually develop crusting 'heads' and focal secondary hair loss (Figure 15.4a,b).

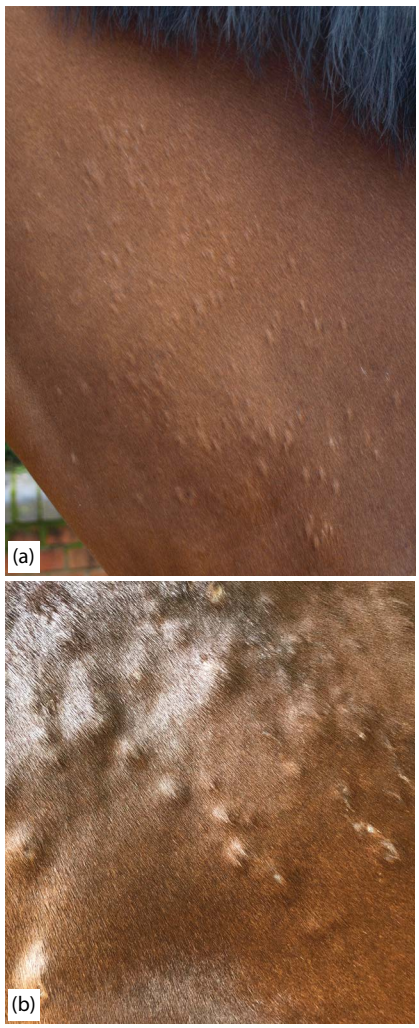


Fig. 15.4 **Bacterial folliculitis: (a) multiple small papules, some with crusting 'heads', as commonly encountered on one/both sides of neck; (b) less commonly, larger firm crusting nodules/papules are encountered that may slowly spread over extensive area.**

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- May resemble fungal infection (dermatophytosis/'ring worm'/'girth itch').

Diagnosis
- Clinical findings usually definitive.
- Monitoring progression of disease over initial days usually permits differentiation from fungal infection (easy loss of hair with latter).

Management
- Topical medicated/antibacterial shampoos or chlorhexidine solution, taking care to gently raise scabs/heads.

Prognosis
- Good. Rarely interferes with training.

Dermatophytosis ('ringworm'/'girth itch')
Contagious fungal skin infection causing crusting and hair loss (due to localisation in hair follicles). Most commonly occurs at girth, flank, neck or face (areas in contact with tack). Infections most common in young horses, as previous exposure leads to reasonable (but not complete) resistance to re-infection in older horses.

Causes
- *Trichophyton* spp. most commonly implicated but *Microsporum* spp. also encountered.
- Fungal spores are resistant to environmental conditions and may persist for years.
- Contagious through direct or indirect contact (environment, tack, grooming equipment).
- Usually also requires lowered local skin defences through abrasion, sweating or pressure.

Signs
- Time period from exposure to development of skin lesions approximately 10–14 days.
- Initial appearance is of clusters of raised hairs, progressing rapidly to crusting and hair loss (Figure 15.5a–d).
- Lesions may be solitary/focal or extensive.
- Crusting/scaling is usually 'dry'; less commonly manifested as wet dermatitis.
- May resemble other types of dermatitis (Bacterial folliculitis, p. 359), particularly on neck; can usually be differentiated by ready plucking of hair from lesion edges and rapid progression over few days.

Diagnosis
- Clinical findings (and progression) sufficient for diagnosis in most cases.
- If definitive diagnosis required: hair/crust plucks from lesion edges reveal fungal hyphae and spores on microscopic examination, although false negatives can occur.
- Polymerase chain reaction (PCR) testing (hair/skin) highly sensitive, specific and rapid.
- Fungal culture rarely warranted.

Management
- Lesions eventually resolve spontaneously, but treatment is undertaken to minimize spread.
- Presumptive treatment warranted if justified by clinical signs: better to treat as precaution than risk spread.

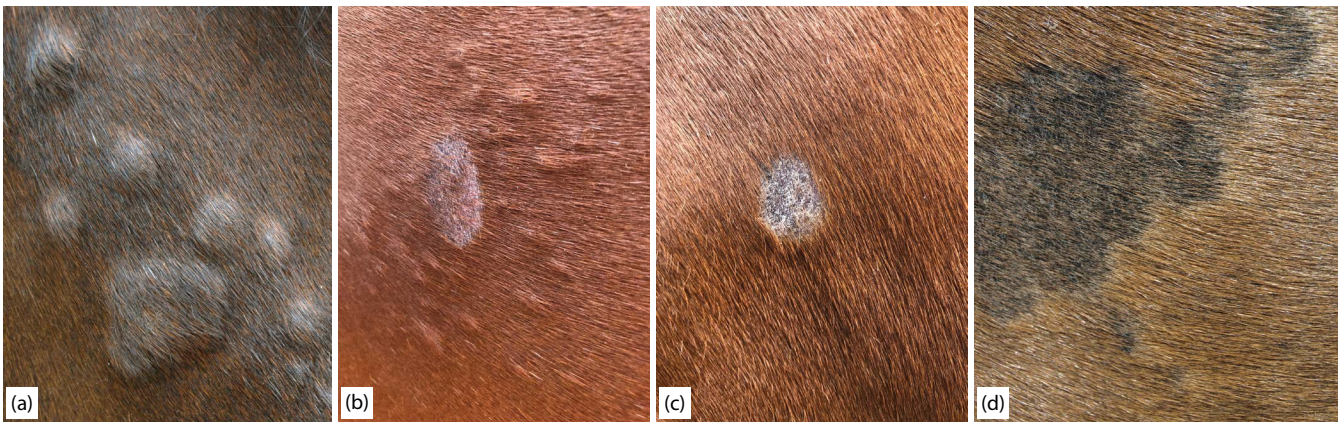


Fig. 15.5 Dermatophytosis ('ringworm'). Typical progression of lesions: (a) raised hairs of very early lesions, followed by (b) dry crusting and easy hair loss; (c) early healing phase in which hair on periphery of lesion is no longer shed, but lesion remains crusted; (d) post-infection phase with growth of new hair.

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- Topical fungicidal medication (enilconazole/miconazole/lime sulphur): focal or entire coat depending on signs.
- 2–3 treatments at 3- to 4-day intervals recommended for common topical medications.
- Application of keratolytic/debriding cream useful to lift crusts prior to antifungal treatment.
- Lesions in close proximity to sensitive sites (eyes, muzzle, ears, girth) best treated with topical fungicidal cream.
- During infectious period care should be taken to disinfect tack/rugs/grooming kit (sporicidal disinfectants/fumigation) to prevent spread between horses.
- May infect stable staff/riders: caution with handling of active lesions and vigilance for skin lesions.
- Oral antifungal medication (griseofulvin): empirical support for use; however, efficacy has not been demonstrated scientifically and routine use in individual cases not recommended.
- Vaccination available in some jurisdictions, but practicalities limit widespread use.

Prognosis
- Most cases resolve over 1–2 weeks with treatment; rarely interferes with training.
- New lesions may continue to appear for some time after treatment: due to time delay between infection and resulting hair loss rather than ineffectual medication.
- May interfere with racing and international travel while lesions are visibly active.

Dermatophilosis ('rain scald'/'rain rot'/'Streptothricosis')
Skin infection characterized by crusting/wet dermatitis that occurs most commonly over areas of the body subject to wetting/sweating such as quarters or back; less frequently on front of hind cannons. Usually in horses at pasture or with winter coats; uncommon in stabled horses.

Cause
- Opportunistic colonization of wet/damaged skin by microbe present in environment *(Dermatophilus congolensis)*.

Signs
- Wet dermatitis in which exudate causes hard matting of hair.
- Crusts may come away leaving painful moist/raw/pustular lesions.

Diagnosis
- Clinical findings usually definitive.
- Laboratory work (cytology, PCR) can be undertaken if necessary but rarely required.

Management
- Medicated (keratolytic) washes/creams or chlorhexidine cleansing to remove matted hair/crusts, followed by topical antiseptic/antibiotic creams until resolution of dermatitis.
- Topical tea tree oil has documented efficacy against *D. congolensis*.
- Systemic antibiotics may assist healing if lesions widespread.

Prognosis
- Good. Rarely interferes with training.
- If treatment delayed or inappropriate, permanent alopecia/white hairs may ensue.

OTHER CONDITIONS

Urticaria ('hives')
Common clinical manifestation of allergy (rather than single disease entity, or a skin disease per se). Allergic response (generally Type 1 hypersensitivity) to inhaled/ingested/injected/contact allergen(s). Typically acute onset. Severity varies and duration ranges from transient (few days) to many weeks/months (rare).

Causes
- Inflammatory response causes increased vascular permeability and leakage of protein and fluid into skin, resulting in skin oedema/'wheals'/papules.
- Inflammatory cascade complex and may be influenced by both immunological and non-immunological (physical, genetic, chemical) factors.
- Wide range of potential causes including (most commonly) immunological/allergic (e.g., reactions to foods; insect bites; drugs; vaccines; parasites); less commonly physical (e.g., temperature variations; sun exposure), infectious (systemic or skin infections) or psychogenic.
- May be associated with change in feed/bedding/management; however, inciting cause rarely determined with confidence.

Signs
- Diagnosis is straightforward on clinical grounds; determining inciting 'trigger' far more challenging

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Fig. 15.6 Urticaria: (a) typical appearance with generalised raised wheals; (b) less common ring-/'donut'-shaped lesions.

and not necessarily assisted by immunological (intradermal or serological) testing.
- Multiple raised patches of hair/flat oedematous wheals/papules throughout coat (Figure 15.6a,b). Generally circular but sometimes ring-shaped.
- Most prominent over face/neck/flank, usually affects both sides of body; pressure from blanket may keep lesions away from trunk.
- Transient improvement (hours) may be noted following exercise.
- Small proportion of cases develop severe exudative wheals, with resulting secondary crusting/hair loss.

Management
- Remove inciting cause, if identified.
- Majority of mild cases require no treatment (if not worsening) and self-resolve over days/weeks. Training may continue unabated.
- Corticosteroid medication: dexamethasone (single IV administration) will usually resolve clinical signs for 3–4 days and transient cases may require no further treatment.
- Recurrent/chronic cases: may require low-dose oral prednisolone (1 mg/kg BWT/day until lesions resolve, then tapered reduction over days/weeks); alternatively, repeated dexamethasone dosing as required.
- Use of a stretch-material head and neck cover (in addition to rug) useful to keep pressure on high-visibility skin areas and may permit satisfactory (cosmetic) management long-term thereby eliminating need for medication.
- Other treatments include antihistamines; tricyclic antidepressants; nutritional (fatty acid supplementation: linseed/flaxseed oil).
- Rarely, lesions of variable severity may persist for many months: may be manageable by minimizing time spent in stable environment and determining whether dietary factors are involved (trial elimination of dietary components can take many weeks).
- Allergen-specific immunotherapy (based on intradermal testing) may reduce need for corticosteroid use in problematic cases, but is rarely employed due to duration of treatment (many months), cost and slow (+/– uncertain) onset of effect.

Prognosis
- Good: most cases resolve rapidly or can be managed satisfactorily.
- Occasional chronic cases require long-term medication +/– altered stabling/dietary management.
- Rarely interferes with training, although drug administration needs to be planned around racing engagements.

Papillomatosis ('warts')
Common viral (papillomavirus) skin disease. Moderately contagious; incubation period approximately 2 months. Seen predominantly in young (yearling/2 YO) horses, as older horses develop good immunity. May bleed if traumatized but generally benign and of no importance.

Signs
- Single or (more commonly) multiple grey/pink vegetative lesions (Figure 15.7a,b).
- Most common sites are lips and muzzle, lower limb and groin/sheath.

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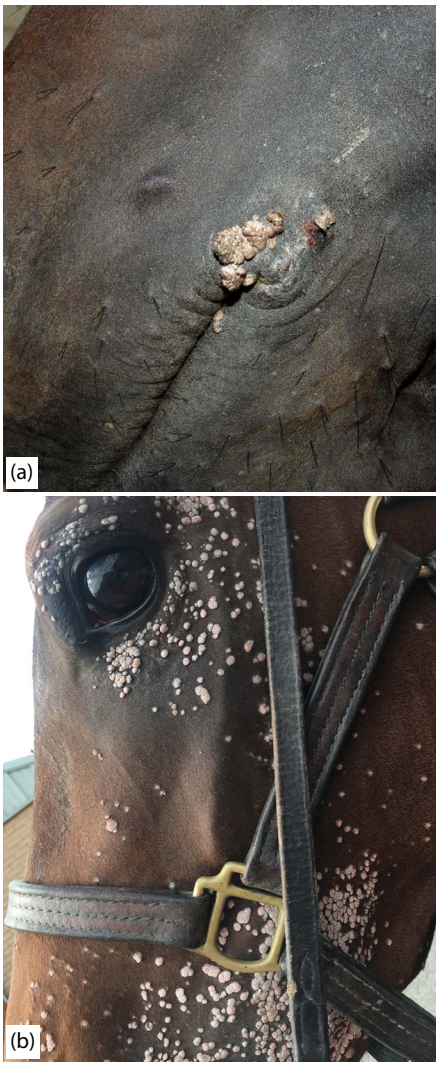


Fig. 15.7 **Papillomatosis ('warts') (a) at corner of mouth (where prone to trauma), and (b) more extensively along face.**

Diagnosis
- Clinical findings definitive.

Management
- Usually no treatment required.
- Spontaneous regression in 2–4 months.
- Troublesome warts suffering from repeated trauma (corners of mouth, pastern) may warrant removal (sharp dissection/cautery/topical caustic or 5% imiquimod ointment).
- Good efficacy reported with autogenous vaccines but rarely used.

Prognosis
- Good. Rarely interfere with training and resolve (with no recurrence) within months

Aural plaque (aural acanthosis)

Type of papillomatosis ('warts') affecting inner aspect of the ears. Uncommon. Generally benign and of no importance. Caused by exposure to papillomaviruses. Poorly researched condition and understanding of transmission and natural course of lesions is limited.

Signs
- Multiple grey/pink raised vegetative lesions inner aspect of ear; may coalesce to form large plaque (Figure 15.8).
- Unilateral or bilateral.
- Not usually associated with other signs, but affected horses occasionally display sensitivity around ears/head shyness; in such cases not usually possible to determine whether sensitivity is arising from plaques or from previous attempts at treatment.

Diagnosis
- Clinical findings definitive.

Management
- Treatment should be avoided/discouraged in all but very rare cases of severe head/ear hypersensitivity, as may precipitate behavioural changes/headshaking.
- Good efficacy and low recurrence rate reported with topical 5% imiquimod ointment (treatment duration approximately 2 months), but associated with immediate adverse effects of inflammation, soreness.



Fig. 15.8 **Aural plaque.**

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Prognosis
- Good: almost invariably a cosmetic blemish only, with no impact on training.

Sarcoids
Fibroblastic skin tumours that can take several forms and often behave unpredictably. Do not metastasize but may spread locally. Spontaneous resolution can occur but highly unusual; most lesions are slowly progressive.

Causes
- Multifactorial disease process.
- Arise from exposure to bovine papillomaviruses; flies appear to have role in transmission: predilection sites are those most prone to fly-bite (belly/sheath/face).
- Risk factors for individual horses (such as genetic susceptibility, impaired immune response to virus) poorly defined.

Signs
- Lesions may be solitary or multiple. Classified into following recognizable forms depending on appearance (Figure 15.9a–d):
  - Occult: affected sites (most usually around mouth/eye/groin) appear as poorly defined areas of thick/hard skin, sometimes with change in overlying hair.
  - Verrucous: classic 'warty' appearance; anywhere on body.
  - Nodular: firm, non-painful nodules with normal overlying skin. Most commonly around eye, sheath and groin.
  - Fibroblastic: similar appearance to proud tissue with areas of ulceration/haemorrhage and often extensive local spread.
  - Mixed: lesions with features of any of above.

Diagnosis
- Can generally be distinguished from other common skin conditions (warts, proud flesh) on clinical appearance, although solitary nodules (+/– ulcerated margin) may resemble other nodular disease (mast cell/basal cell tumours).
- Undertaking biopsy to confirm diagnosis is inadvisable: aggressive re-growth/spread is common following partial excision.

Management
- If appearance is suggestive of sarcoid, should be managed as such.
- Most sarcoids do not interfere with training during racehorse career; treatment usually reserved for lesions that interfere with tack or are growing rapidly.
- Unless a sarcoid is to be removed in its entirety, it should be left alone (biopsy generally contraindicated).
- Because of the considerable risk of aggressive re-growth, important that initial therapy (if undertaken) is appropriate.
- Choice of treatment is determined by location of lesions and economic factors.
- Solitary, well-defined sarcoids with relatively small skin 'pedicle' are readily ligated (rubber ring/thread ligature) to cut off blood supply, resulting in loss within 1–2 weeks; generally do not recur.
- Several treatment options available for more serious lesions: good efficacy (>75% complete remission) with laser excision, topical ointments (5% imiquimod/5-fluorouracil/bloodroot) and intra-lesional chemotherapy and radiotherapy.
- Periocular lesions: iridium-192 interstitial brachytherapy (90% success rate; time to maximum

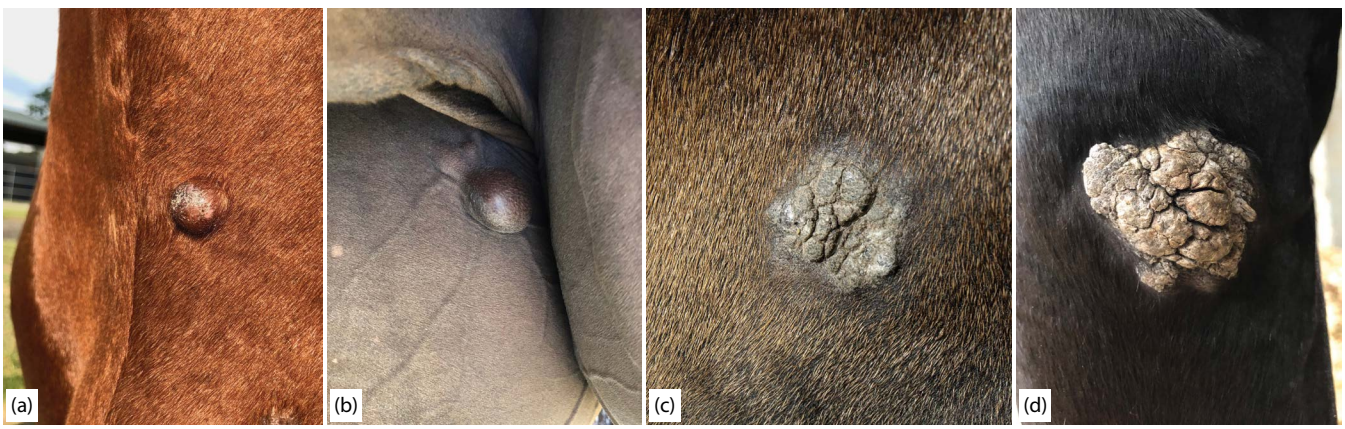


Fig. 15.9 **Sarcoids: (a, b) nodular; (c, d) verrucous.**

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resolution up to 2 years) or topical 5% imiquimod cream.
- Electrochemotherapy and photodynamic therapy are possible alternative treatment options.

Prognosis
- Sarcoids rarely interfere with training but can have implications for re-sale.
- Recurrence after treatment is unusual, but more likely for head/neck lesions, verrucose sarcoids and in horses with multiple lesions.
- Remission may occur in the absence of treatment, but very rare.

Eosinophilic collagen necrosis/granuloma
Common, non-painful focal nodular skin lesion(s). Typically over back and flank. Usually do not progress or enlarge and may remain unchanged permanently or resolve spontaneously over months.

Cause
- Cause unknown but risk factors include pressure of tack and insect bites.

Signs
- Small, firm and non-painful dermal nodules.
- Solitary or multiple.
- Hair loss not a feature unless rubbed by tack.

Diagnosis
- Clinical findings definitive.

Management
- Usually not treated, as cosmetic blemish only.
- Perilesional or systemic administration of corticosteroids may result in improvement/resolution but rarely warranted.

Prognosis
- Usually remain a long-term minor skin blemish but do not interfere with training.

Tail rubbing/pruritus
Pruritus over quarters or tail base may result in persistent irritation and traumatic dermatitis of tail. Severity varies between individuals; can affect horses of all ages. Usually of cosmetic importance only.

Causes
- Frequently undetermined as typically a cycle of trauma/infection/irritation.
- Often part of a more generalized pruritus.
- Uncommonly initiated by tail rub/dermatitis sustained during transport.
- Other causes of pruritus include worms (*Oxyuris equi* resides in lower bowel and deposits eggs on perianal skin) and *Culicoides* allergy; however, these are rare in racehorses.
- Presence of skin lesions elsewhere on body may indicate parasitic or allergic origin.

Signs
- Broken and lost hairs at tail base with some crusting dermatitis.
- +/– pruritus when quarters/tail base stimulated.

Diagnosis
- Diagnostic work usually not undertaken as more practical to treat than diagnose.
- Care should be taken to determine whether horse is truly pruritic, as this will direct management.

Management
- Deworming (unless recently treated).
- Initial treatment should be directed at breaking the dermatitis/irritation cycle.
- Repeated washes with keratinolytic/antibacterial medicated shampoo (selenium sulphide products also have antipruritic/antifungal action; tea tree oil anecdotally useful) +/– daily topical emollient cream +/– tail bandaging resolves most cases.
- Systemic corticosteroid (prednisolone) medication may be warranted in acute phase.

Prognosis
- May persist for weeks/months but does not interfere with training.

Linear keratosis
Rare dermatitis characterized by linear bands of crusting (hyperkeratosis), generally on neck, shoulder or flank.

Cause
- Unknown; likely to be an autoimmune disorder.

Signs
- Vertical (continuous or broken) line of hair loss, crusting or white hairs that resembles a 'run-off scald' (Figure 15.10).
- Usually unilateral.
- Pruritus and pain are not features.

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Fig. 15.10 Linear keratosis.

Diagnosis
- Clinical findings definitive.

Management
- Treatment not usually needed (or effective).
- Steroid or keratolytic creams may reduce severity of some lesions temporarily (if desired).

Prognosis
- Life-long condition, although spontaneous resolution may occur.
- Does not interfere with training.

Harvest mite dermatitis
Seasonal pruritic dermatitis caused by the biting larvae of the harvest mite (*Trombicula autumnalis*). Primarily in horses with access to pasture (chalky soil) in late summer/autumn; however, mites can also be found in hay and stabled horses may therefore be affected at any season in mild climates. Not contagious but more than one animal may be affected.

Signs
- Areas of crusting/hair loss on face or lower legs, with characteristic orange 'droplets' of dried serum.
- Pruritus (rubbing face, stamping legs) is a key feature.
- Larvae rarely seen as only on horse for brief periods to feed.

Diagnosis
- Clinical findings strongly indicative.
- Confirmation by microscopic examination of crust/debris for mites.

Management
- Topical insecticidal washes.

Prognosis
- Responds well to treatment.

Lice
Infestation with biting (*Damalinia equi*) or sucking (*Haematopinus asini*) lice causes irritation/itching and is most common in winter and spring. Contagious through direct or indirect (rugs/grooming kit/environment) contact. Horses free from clinical signs may be carriers of lice.

Signs
- +/– pruritus and resulting dermatitis.
- Rubs, hair loss and scurf typically along base of mane/tail or over quarters.
- Close examination may reveal eggs (small/pale/oval 'nits') attached to hairs of mane (Figure 15.11) or lice in coat.
- Lice may be present in the absence of clinical signs.

Diagnosis
- Clinical findings usually definitive.
- Coat groomings can be placed on a dark surface and examined for movement of lice.

Management
- Insecticidal powders/washes/topline treatments; treatment on 2 occasions 7–10 days apart usually recommended. Good efficacy reported with single topical treatment of deltamethrin.
- In-contact horses should be treated if feasible.

Prognosis
- Good. Resolves rapidly with appropriate treatment.



Fig. 15.11 Lice eggs: Small white eggs adhering to hairs of mane.

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Culicoides hypersensitivity ('sweet itch')

Allergic dermatitis caused by biting midges (*Culicoides* spp.). Seasonal (summer). Rarely seen in racehorses.

Signs

- Thickened scaly skin at base of mane/tail with broken hairs.
- Pruritus.

Diagnosis

- Clinical findings usually definitive.

Management

- Topical insecticidal washes/topline treatments.
- Insect repellents applied to belly (most common site for the midge bites that initiate inflammatory cascade).
- Emollient/keratolytic creams.
- Systemic corticosteroid therapy may be useful to manage initial pruritus.

Prognosis

- Most cases can be managed satisfactorily.
- Rarely interferes with training.

Dermoid cysts

Congenital cysts within the skin, thought to arise from aberrant placement of tissue during embryonic development. Presence noted early in life. Generally remain unchanged in appearance throughout training.

Signs

- Small, non-painful skin nodules of similar size and appearance to eosinophilic collagen necrosis (p. 365) but differentiated by being found invariably on dorsal midline of back (Figure 15.12).
- Not associated with hair loss but few white hairs may be observed over each nodule.
- Single or multiple.

Diagnosis

- Clinical findings definitive.

Management

- Generally no treatment required.
- Exceptionally, may interfere with tack and in these cases surgical excision is curative.

Prognosis

- Remain present throughout life but do not interfere with training.

Intertrigo

Uncommon exudative dermatitis of contact areas of skin in high groin (or, rarely, axilla). Most commonly seen following open castration or in colts with large testicles. May arise secondary to 'chafing' or contact with post-castration granulation tissue/exudate.

Signs

- Areas of shallow ulceration or granulation, focal in early stages but often coalescing to involve extensive regions of in-contact skin (Figure 15.13)
- May be associated with considerable local inflammation/soreness and wide-based hindlimb gait.
- Progression of dermatitis can be rapid.



Fig. 15.12 **Dermoid cysts: Typical appearance of small nodules and white hairs on midline of back.**



Fig. 15.13 **Intertrigo.**

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Diagnosis
- Clinical findings definitive.

Management
- Most cases respond favourably to simple non-medical cleaning +/– restricted exercise (to minimise chafing). Concurrent management of castration wound infection/exudation/granulation (presumed contributory to onset) important; trim any tissue protruding from castration wound.
- Daily cleansing with dilute antiseptic solution and careful drying of affected site followed by application of barrier (zinc oxide) cream; often painful to undertake in initial stages and may require sedation.
- Minority of cases can be troublesome and require meticulous management to optimize healing: topical antiseptic/antibiotic/antifungal ointment +/– systemic NSAID indicated in initial stages and may be guided by laboratory findings.

Prognosis
- Full resolution may take weeks and some interference with training can be expected in initial stages.

Calcinosis circumscripta

Rare, benign subcutaneous calcified growth typically found overlying a joint; most commonly the stifle. Cause is unknown. Unilateral or bilateral. Detection of mass typically occurs during yearling/early training phase or at routine pre-purchase radiography.

Signs
- Firm, non-painful subcutaneous mass over lateral aspect of stifle.
- Size of mass varies but can be extensive.
- Overlying skin not involved, of normal appearance and can be moved freely over lesion.
- Rarely associated with lameness.



Fig. 15.14 Calcinosis circumscripta lesion overlying lateral aspect of stifle (CdCr).

Diagnosis
- Clinical presentation definitive.
- Radiography: heterogenous/granular radiopaque body lateral aspect of stifle (Figure 15.14).

Management
- No treatment required in most cases.
- If considered to be causing lameness, or large and growing (rare): surgery. Close association with stifle joint capsule means surgical excision is not without risk of complication including wound breakdown

Prognosis
- Rarely interferes with training.
- Life-long condition; however, growth of mass, if it occurs, is very slow and of little concern.

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MISCELLANEOUS CONDITIONS

Stable vices (stereotypic behaviours)

Stable vices or stereotypic behaviours are habitual and repetitive behavioural patterns that serve no apparent function. Thought to be a 'stress-relieving' mechanism. They include the following:

- Crib-biting (cribbing): grasping of fixed object with incisor teeth, contraction of ventral strap muscles of neck and production of a 'grunt'.
- Windsucking: as for crib-biting (+/– grasping of fixed object) with swallowing/gulping of air.
- Weaving: habitual swinging of head/neck from side to side with associated shifting of weight.
- Box walking: habitual pacing/walking in or around stable.
- Other variations of tongue/lip play, wood chewing, stall kicking and hole digging.
- Coprophagy (eating of faeces): may be a habitual behaviour but also may be secondary to nutritional deficiencies (such as insufficient fibre in diet).

Causes

- Actual neurobiological mechanism(s) responsible for these behaviours (and reasons why only some individuals affected) are poorly understood, but in brief if horses are prevented from executing some natural behaviours then development of alternative behaviours can occur due to the sensitization of the underlying neural systems involved.
- Horses in free-range conditions have strong motivation to move and forage for a large proportion of the day as well as continual social interactions; stable vices are thought to result largely from absence of this natural behaviour.
- Some evidence that gastrointestinal (gastric ulcers, intestinal transit times) and dietary (low forage, high concentrate) factors are important in some horses.
- Management at weaning can have a strong influence: feeding of concentrates and housed (as opposed to pasture) weaning both associated with greater risk of development of stable vices. Stress, gastrointestinal factors and frustrated sucking behaviour may play a role.
- May be copied from other horses, although learned behaviour likely to be less of an influence than other risk factors.
- Certain behaviours may be precipitated by stress (e.g., change of yard).

Effects

- Main stable vices are generally considered to be an 'unsoundness', with negative impact on saleability/value.
- Racetrack performance is unaffected.
- Horses that crib-bite or windsuck are at increased risk of developing colic; however, little known about the link and not necessarily a cause-effect relationship.
- Air swallowed while windsucking does not transit the gastrointestinal tract and in itself does not cause colic; more likely to relate to undefined factors such as intestinal transit times/gastric ulceration/increased abdominal pressure.
- Stall kicking and door banging may cause injuries or cosmetic blemishes such as acquired bursae over front of carpus.

Management

- Once a horse has displayed a stable vice for some time, management changes frequently have little or no effect.
- If recognised early, may be beneficial to investigate/alter nutritional +/– environmental factors.
- Stabling that permits some social contact or visual interaction with other horses may be useful in minimising/preventing behaviour.
- Large mirror placed on stable wall or obstacles (tyres) on floor may limit weaving/box walking behaviour.
- Provision of salt/mineral lick or interactive feeding devices/toys may reduce crib-biting.
- Investigation/treatment of gastric ulceration if thought to be implicated.

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- Anti-crib devices (e.g., crib strap, muzzle) have good efficacy but do not address underlying causes and in some circumstances can increase stress. Use should preferably be limited to severely affected horses or those prone to recurrent colic/poor feed intake. Part-day use, or combination with other environment-enriching methods (toys, increased forage) may be considered.
- Companion animals (sheep/goat/pig/other equine): empirical evidence of good efficacy for otherwise unmanageable cases.
- Surgery (neurectomy of ventral branch of spinal accessory nerve plus myectomy of omohyoideus/sternothyrohyoideus muscles): good (not total) efficacy for preventing crib-biting but rarely undertaken in racing yards. Preoperative cribbing duration associated with surgical success (lower success rates for horses that have cribbed >3 years before surgery).
- Separation from other horses (to limit potential for others to 'learn' behaviour) not warranted.
- While attempts to modify stereotypic behaviours may be worthwhile, ultimately if they fail it is unlikely that the affected horse's training career or welfare will be adversely affected.

Headshaking

Poorly understood condition characterised by frequent, unpredictable and sometimes violent head movements (typically vertical), often with other signs of apparent nasal/facial irritation such as snorting, sneezing, rubbing nose. Rare in racehorses (most common in middle-aged horses, of all breeds and disciplines). Condition is often worse at exercise than rest, and may be triggered by seasonal or environmental factors.

Causes

- Diverse range of unrelated conditions may manifest as nasal/facial irritation: these include behavioural problems, dental and sinus disease, ear mites, neck injury, guttural pouch mycosis, equine protozoal myeloencephalitis.
- Most common cause, however (generally diagnosed by multi-disciplinary investigation to rule out the above), is trigeminal-mediated headshaking: acquired disorder with similarities to human 'trigeminal neuralgia'. Functional abnormality of the sensory nerve that emerges from infraorbital foramen on each side of face (infraorbital branch of the trigeminal nerve) resulting in lower threshold for nerve activation than normal horses. Not currently known what precipitates this nerve disorder.

Effect

- Effect on ability to train/race depends on severity. When intermittent and with mild/moderate signs, unlikely to interfere with training. In severe cases, horse may become unrideable.
- Little is known about disease progression over medium to long term.

Management

- Diagnosis of (trigeminal-mediated) headshaking is one of exclusion and requires extensive diagnostic investigation that may incorporate CT, endoscopy, bone scan and nerve blocking.
- Prognosis for complete resolution has generally been considered poor, with range of scientifically unvalidated treatments being used for treatment.
- Wide individual response to all management strategies: 'trial-and-error' approach to treatment in individual cases.
- First approach to treatment should be use of a nose net: non-invasive, inexpensive, risk-free. Mechanism of action thought to be through non-painful chronic sensory stimulation leading to closing of nerve 'gates' to painful stimuli. Significant improvement can be expected in about 25% cases.
- Horses that do not respond favourably to nose net should be considered for neuromodulation therapy. Moderate success rates for short- to medium-term remission reported with percutaneous electrical nerve stimulation performed under standing sedation.
- Oral supplementation with magnesium (or magnesium + boron) has some reported efficacy and is non-invasive and safe. Mechanism of action currently unknown.
- Medical therapies include use of gabapentin and carbamazepine, although inconsistent results and regulatory constraints limit usefulness.

Elevated GGT (liver enzyme) syndrome

Poorly understood condition characterised by raised gamma-glutamyl transferase (GGT) activity often in association with non-specific evidence of poor performance or ill thrift. GGT is an enzyme found in many tissues, but raised serum levels are considered highly specific to the liver. Link between elevated GGT and poor athletic performance in some individuals has been recognised for a long time but still much uncertainty about relationship, particularly as does not appear to arise from liver 'disease' as such. Elevated GGT is not always clinically relevant and may be encountered as an incidental finding in a proportion of horses without any obvious performance (or clinical) problems.

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Causes
- Cause currently unknown but likely to be complex/multi-factorial.
- Primary cause not thought to be related to any known viruses.
- Often presumed to be a sign of 'overtraining'; however, overly simplistic as no clear understanding why particular animals are affected while others subjected to the same (or greater) training loads are not.
- Oxidative stress (resulting from exercise stimuli) also proposed as possible cause; however, affected horses appear to be able to maintain antioxidant status satisfactorily in the face of challenges and do not demonstrate increases in markers of oxidative damage.
- Physiological changes that occur with intense exercise may be implicated: repeated depletion (and repletion) of glycogen stores in the liver has plausible role.
- Increased hepatic glutathione synthesis/recycling +/– mild exercise-related cholestasis may be important part of the syndrome.

Risk factors
- Prevalence in horses in training estimated at approximately 2%.
- No apparent association with age.
- More commonly encountered in fillies.
- Typically encountered in horses in full training/high-speed exercise.

Signs
- Elevated serum GGT not always associated with clinical signs; however, 'syndrome' characterised by non-specific signs of underperformance, difficulty keeping body condition, reduced appetite.
- Usually no other haematological/biochemical abnormalities noted.

Diagnosis
- Blood sample/biochemistry: reference range for serum GGT is <40 IU/L for 'normal' horses. GGT activity ≥80 IU/L considered to be abnormally elevated and potentially related to poor performance.
- Mild elevations of GGT (40–60 IU/L) above reference range, without other clinical or blood chemistry abnormalities unlikely to be of significance.

Management
- At present no definitive treatment(s) exist.
- Management determined by individual circumstances and can range from unaltered training to extensive rest +/– nutritional supplements.
- When high GGT detected in horses already identified as performing poorly or losing body condition, strong indication to allow period of time out of full training: ideally pasture turnout or light exercise. Optimal length of time not known; however, unlikely to see return of GGT to normal levels with <6–8 weeks of rest.
- When high GGT detected in horses that are performing satisfactorily and clinically normal, vigilance is warranted, but continuation in full training is not necessarily inadvisable. Nutritional supplementation (see later) recommended.
- Supplementation with antioxidants (such as coenzyme Q10, vitamins C, E) +/– omega-3 amino acid-rich nutrients remains chief recommendation, but lack of clear association with antioxidant deficiency makes value of such supplementation uncertain.

Prognosis
- Guarded: not currently possible to predict which horses will respond favourably to rest/supplementation. Recurrence of high GGT/poor condition occurs upon a return to high-speed work in significant proportion of cases.

Synchronous diaphragmatic flutter ('thumps')
Unusual but transient condition seen in the immediate period after high-intensity exercise/racing, characterised by spasmodic, regular 'twitch' of flanks that resemble human 'hiccups'.

Causes
- Caused by abnormal firing of the phrenic nerve, which provides motor innervation to the diaphragm. Nerve runs in close proximity the heart, and when in hyperexcitable state electrical depolarizations of heart may trigger synchronous contractions of diaphragm.
- Multiple possible causes of phrenic nerve hypersensitivity: most are metabolic and likely to be related to transient alterations of electrolyte levels (low calcium, potassium, chloride) +/– acid-base balance.

Risk factors
- Rare.
- More likely to occur after prolonged exercise in hot/humid conditions (greater electrolyte disturbances resulting from sweat losses).

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Signs

- Sudden onset of involuntary, rhythmic and sometimes audible ('thump'/'hiccup') twitch of one/both flanks.
- Severity variable.
- Typically in the immediate post-exercise period, and most frequently in horses visibly fatigued or heavily sweating.

Diagnosis

- Clinical presentation is definitive.
- No necessity for haematological/biochemical assessment in majority of cases but warranted if recurrent.

Management

- Most cases resolve spontaneously; offer free choice food and water.
- Intra-gastric or intravenous administration of polyionic electrolyte solution if faster resolution desired.

Prognosis

- Excellent. Most cases do not recur.
- Attention to hydration and electrolyte supplementation in pre-exercise/pre-race period for horses that have previously experienced an episode is sensible.

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Equine influenza

Equine influenza (EI) is the most important viral respiratory disease in the racehorse population worldwide. Similar in many respects to human influenza A; antigenic drift and strain mutation ensure outbreaks occur periodically even in vaccinated populations due to vaccine failure. Virus damages cells lining the upper and lower respiratory tract leading to impairment to normal respiratory clearance mechanism and secondary bronchiolitis.

Signs

- Fever (often high, and lasting several days), nasal discharge and coughing. Bronchiolitis/increased tracheal mucus detected endoscopically.
- Severity of signs depends on immune status of individual: in a naïve population signs may be dramatic, while in a partially immune population may be mild/subclinical. Vaccinated horses usually experience milder and shorter duration disease.
- Serious disease in vaccinated populations is highly unusual. Mortality may occur (rarely) in immunocompromised foals.

Transmission

- Highly contagious.
- Incubation period 1–5 days.
- May shed virus for 7–10 days after infection. Carrier status does not exist. Decreased shedding in vaccinated horses.
- Direct and indirect (hands, tack, surfaces, shared airspace) spread through respiratory route.
- Young or inadequately vaccinated horses at greatest risk.
- Survival of virus in environment for hours/days.
- Dry, cool conditions appear to favour spread.
- Windborne spread (kilometres) possible and has been recorded when windspeed >30 km/hour.

Diagnosis

- Usually clinically indistinguishable from other common viral/bacterial respiratory infections.
- Diagnosis requires laboratory testing.
- Nasopharyngeal swabs: Polymerase chain reaction (PCR) detection of virus from day 1–10 of infection. Reduced shedding in vaccinated horses can limit detection.
- Blood sampling: seroconversion (≥4-fold rise in antibodies) occurs by day 8; typically obtain 2 samples 10–14 days apart.

Management

- Biosecurity measures to minimise spread.
- Supportive care includes non-steroidal anti-inflammatory medication for fever; rest (general guideline: complete rest for number of days equivalent to days displaying fever) +/– antibiotic medication for horses with persistent or secondary respiratory disease.
- Usefulness of anti-viral therapies poorly investigated; rarely used in practice.
- Most horses fully recover within 1–2 weeks. Care to monitor yard respiratory health in weeks following infection due to negative impact on lower airway defences.
- Booster vaccinations for at-risk horses may be useful in outbreak situation.

Prevention

- Vaccination is a requirement of most racing jurisdictions and is generally highly effective at limiting the impact and spread of EI in racehorse populations. Severity and spread of disease is much reduced in vaccinated horses.
- International surveillance panel monitors epidemiological shifts in active strains and makes recommendations to vaccine manufacturers for vaccine updates, to minimise risk of vaccine breakdowns.
- Current vaccines provide reasonable but not total protection against emerging strains and boosting 2–3 times/year is recommended.
- Good biosecurity measures, with heightened vigilance when local infectious respiratory disease suspected/reported.

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Equine herpesvirus

There are several types of herpes viruses that affect horses. Types 1 and 4 are of greatest importance and best understood; knowledge of types 2 and 5 is evolving, but these latter are commonly found in upper airways of healthy racehorses and unlikely to play a major pathogenic role in this population. EHV is endemic and is notable for its ability to remain dormant in previously infected individuals (latency in lymphocytes and the trigeminal ganglia), with stressors prompting periodic reactivation and shedding of virus. Variable strains of EHV-1 and 4 are involved in outbreaks of disease; virulence (and clinical signs that develop) differs depending on strain characteristics and individual horse immune factors.

Signs

- EHV 4: primarily associated with signs of respiratory disease (coughing, nasal discharge, fever). Can occasionally cause individual abortions.
- EHV 1: can cause respiratory disease (as noted earlier) as well as neurological disease and contagious abortions.
- Neurological form (usually caused by neurotropic strains): may be preceded by respiratory disease +/– fever. Progressive ataxia (usually hindlimb but may progress to forelimb or complete recumbency) +/– urinary overflow incontinence.

Transmission

- Most horses are exposed early in life; estimated that 80–90% horses have been infected by either EHV 1 or 4 by the age of 2 years.
- Main reservoir of infection is reactivation of latent virus in outwardly healthy 'carrier' horses.
- Reactivation episodes may be prompted by stressors such as travel, sales or racing.
- Incubation period 2–10 days.
- Virus spread by direct or indirect contact with respiratory secretions; shedding of virus occurs from initial fever for up to 2 weeks depending on immunity.
- Virus may survive in environment for weeks.

Diagnosis

- Laboratory diagnosis: virus isolation is gold standard (nasopharyngeal swabs/blood sample); also PCR and seroconversion (≥4-fold increase in titre in samples 10–20 days apart).
- Currently no reliable test for carrier status.

Management

- Neurological/paralytic EHV: strict adherence to statutory requirements including isolation of premises. Movement restrictions for 1 month, with lifting of restrictions only when serological monitoring determines no further activity within at-risk group or >14 days from onset of signs in last clinical case. (See also Chapter 14, p. 351.)
- Respiratory EHV: generally no treatment required (antibiotic medication may be needed for persistent respiratory disease/secondary bacterial infections). Biosecurity measures as for neurological/paralytic EHV recommended, but rarely implemented as less serious outcomes of respiratory EHV rarely prompt diagnosis/management.

Prevention

- EHV is endemic and all horses are potentially carriers.
- Routine vaccination with current vaccines may limit susceptibility of a yard to outbreak of infection (reduced virus shedding) but does not currently prevent either respiratory or neurological disease from occurring in individuals.
- Routine disinfection of stables and transport advisable.

Equine rhinitis virus

Equine rhinitis viruses A (ERAV) and B (ERBV) are endemic in the young racehorse population and can be associated with upper and lower respiratory tract disease. Current understanding about overall role in respiratory health of racehorses, interaction with other pathogens, persistence and transmission of infection is limited.

Signs

- Range of non-specific signs of infectious respiratory disease: coughing/nasal discharge/ocular discharge/enlarged submandibular lymph nodes/+/– fever.

Transmission

- Most horses are exposed to ERAV or ERBV either early in life or within the first few months in a training yard; yearlings commencing training appear to be the most at-risk group.
- Seroprevalence in horses ≥2 years of age is high.
- Primary route of transmission likely to be respiratory (direct and indirect contact), although virus is shed in both faeces and urine, so potential for environmental contamination.
- Viral shedding commences 3–4 days following infection and may persist for weeks.

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Diagnosis
- Usually clinically indistinguishable from other common viral/bacterial respiratory infections.
- Diagnosis requires laboratory testing (but rarely undertaken).
- Detection of virus (nasopharyngeal swabs/PCR) or evidence of seroconversion (acute and convalescent blood sampling)

Management
- Clinical disease is usually mild and short-lived and requires no treatment.
- As for other infectious respiratory disease a short period of reduced exercise, +/– antibiotic therapy for secondary/persistent bacterial lower respiratory tract infection may be warranted.
- Acquired immunity appears to be long-lasting; clinical disease in older (≥3 YO) horses is uncommon.

Prevention
- Given their presumed low pathogenicity, there is little current clinical incentive to prevent circulation of ERVs in the racehorse population.
- Ubiquity of the viruses in young horses also means prevention would necessitate any effective vaccination programme (if available) initiated early in life.

Strangles
Strangles is a highly infectious bacterial respiratory disease caused by *Streptococcus equi*. Endemic in wider equine population. Up to 10% of horses that have recovered from infection may become clinically silent carriers, with infection reservoir in guttural pouches and intermittent shedding of bacteria for months/years.

Signs
- +/– initial fever.
- +/– inappetence.
- Nasal discharge.
- Swollen submandibular and pharyngeal lymph nodes, with eventual abscessation and discharge of purulent material.
- Severity of disease varies depending on immune status/age of horse.

Transmission
- Direct transmission between horses through respiratory secretions and open abscesses.
- Shedding does not begin until 1–3 days after initial fever (and then persists 2–3 weeks in most horses).
- Incubation period 1–3 weeks.
- Indirect transmission via equipment, tack, staff and shared water sources.
- In ideal conditions may survive in water for weeks; likely to be much shorter in sunlight or on soil.

Diagnosis
- Clinical presentation of enlarged/abscessed pharyngeal lymph nodes +/– nasal discharge is strong indicator of possible infection.
- Laboratory detection of bacteria (PCR-preferred; or culture) needed for confirmation: from abscess aspirate or discharge/nasopharyngeal swabs/guttural pouch sampling.
- Bacteria rapidly invades lymph nodes and is then frequently not easy to isolate from throat/nose during early stages of disease.
- Repeated nasopharyngeal swabbing (three swabs at weekly intervals) or single endoscope-guided guttural pouch washes submitted for PCR or live culture is recommended. False-negative PCR tests possible. Sensitivity of three nasopharyngeal swabs (PCR) is around 90%.
- Blood serology (enzyme-linked immunosorbent assay [ELISA] test): good sensitivity and specificity for previous exposure (positive from 2 weeks to around 6 months following exposure), although false positives and false negatives occur. Weak positives may represent recent infection or residual antibody response and should be re-sampled in 7–14 days.
- Detection of (clinically silent) carriers is challenging: not currently possible to distinguish carriers from non-carriers using either serology (antigens A and C) or blood inflammatory markers. Current best protocol to identify carriers: PCR testing of bilateral guttural pouch washes and single nasopharyngeal swab, taken on single/same occasion.

Management
- Suspicion of clinical strangles should prompt strict isolation of premises and adherence to statutory requirements and current recommendations from regulatory bodies.
- See Chapter 21 (p. 435) for general quarantine principles.
- Horses may be infectious for 6 weeks after discharges clear (with persistent shedding for months/years from guttural pouch carriers) so rigorous testing necessary to release horses/premises from quarantine.
- Division of all horses on premises into risk groups: 'Red' for clinical disease; 'Amber' for direct or indirect contacts with those in 'Red' group; 'Green' for horses

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with no known direct/indirect contact. Physical separation, no crossover equipment/water source, and use of dedicated staff for each group. Blood ELISA testing of 'Amber' and 'Green' groups. Testing of clinical cases (three nasopharyngeal swabs or single guttural pouch PCR) following resolution of disease before release into wider population. Latter testing best performed >30 days after last clinical signs; testing earlier likely to result in detection of convalescing horses still positive for presence of pathogen.
- The short delay (1–3 days) between onset of fever and when a horse becomes infectious permits further identification/isolation of animals before they can spread disease, with appropriate vigilance (twice daily monitoring of temperature).
- Treatment of clinical cases not necessary (other than supportive care) and is dependent on stage/severity of disease and determined on individual basis. Most infected horses clear the disease naturally, with shedding of bacteria ceasing rapidly after full recovery.
- Possible to halt disease in early cases (pyrexia) with 3–5 days of antibiotic medication (antibiotic of choice is penicillin); however, benefits of antibiotic use are poorly investigated and some concern that it may delay maturation of abscesses or diminish the development of protective immunity. If used, should be restricted to acutely infected horses with high fever and before abscesses are evident. Antibiotics should not be used prophylactically.
- Cases with lymph node abscessation best managed with non-steroidal anti-inflammatory drugs (NSAIDs) rather than antibiotics.
- Treatment of guttural pouch carriers: large volume/repeated lavage of guttural pouches with benzylpenicillin/gelatin solution.
- Following outbreak: rest contact pastures for 4 weeks, disinfection of wooden surfaces and water troughs.

Prevention
- Vaccines for *S. equi* available; however, limitations exist regarding efficacy, availability and consequent impairment of serological surveillance.
- Good biosecurity practices such as isolation and/or swabbing/blood sampling of all arrivals considered to be high risk should minimize likelihood of disease entering yard.

Equine viral arteritis
Equine viral arteritis (EVA) is a viral disease causing respiratory signs, abortion and vasculitis. Primarily a disease of concern to breeding establishments, with shedding by carrier stallions and transmission at mating leading to abortion.

Signs
- Infection not always accompanied by clinical signs; majority are inapparent/subclinical.
- Severity of signs influenced by challenge dose, viral strain, environmental conditions and individual horse factors.
- Fever/depression.
- Filling of lower legs.
- Conjunctivitis and puffiness around eyes.
- Nasal discharge.
- Swelling of scrotum or mammary glands.

Transmission
- Main route of transmission is venereal at mating.
- Respiratory spread through direct or indirect contact also occurs and has been largely responsible for some outbreaks.
- Incubation period 2–14 days (6–8 days venereal).
- Infected stallions remain long-term carriers, while mares show transient disease (respiratory signs +/– abortion).

Diagnosis
- Laboratory diagnosis: virus isolation/PCR/seroconversion.

Management
- Strict adherence to statutory requirements and current recommendations from regulatory bodies.
- Isolation of clinical cases and contacts.
- No treatment available; most cases recover without complication.
- Castration of infected stallions may be considered.

Prevention
- Blood sampling prior to breeding.
- Vaccination of breeding stallions.
- Certification of seronegativity prior to vaccination is important to facilitate international travel.

Equine infectious anaemia ('swamp fever')
Equine infectious anaemia (EIA) is a viral infection with worldwide distribution that can cause severe anaemia and death. Recovered horses remain infected for life and act as a reservoir of infection.

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Signs
- Highly variable (may be subclinical).
- Acute disease: fever/weight loss/diarrhoea/depression/ataxia/jaundice.
- Chronic disease: recurrent fever/anaemia/weight loss.
- Severe, unexplained anaemia should prompt investigation.

Transmission
- Virus persists in monocytes (blood cells) for life in recovered animals.
- Recurrence of disease may occur following immunosuppression/stress.
- Transmission is through infected blood/plasma, although other routes of infection (respiratory/mucosal) are possible.
- Insect vectors may play a large role: predominantly biting flies (mechanical transmission only: no viral replication in insect vector)
- Insects involved in transmission do not normally travel large distances (separation of horses >200 m breaks transmission).
- Other possible routes of transmission include contaminated veterinary or dental equipment, contaminated blood products (plasma) and transplacental (mare to foal) transmission; these routes are primarily responsible for outbreaks in previously disease-free areas.
- Incubation period variable but typically 1–3 weeks; can be ≥90 days.
- Risk and speed of transmission within populations appears to be linked to route of transmission: insect vector/natural spread considered slow, while spread involving human intervention (biological products, or airborne transmission) may be rapid.

Diagnosis
- Laboratory diagnosis.
- Coggins blood test (agar gel immunodiffusion test): detection of antibodies possible from 7–14 days post-infection. False negatives are possible. Is the official test for EIA for international movements, but testing guidelines may be revised as detection techniques evolve.
- ELISA blood test: used for rapid screening but associated with false positives/negatives; Coggins test should always be used for suspected clinical cases/contacts.
- PCR: has a role in detection of recently exposed/'serologically silent' horses but low viral loads in carriers currently limits use.

Management
- Strict adherence to statutory requirements and current recommendations from regulatory bodies including isolation of premises.
- No treatment available.
- Compulsory slaughter of affected horses and quarantine of direct contacts is requirement in most jurisdictions.

Prevention
- No vaccine available.
- Routine serological monitoring of breeding stock and blood donors.
- Caution when travelling to countries with endemic disease.

Contagious equine metritis

Contagious equine metritis (CEM) is a bacterial infection of the reproductive tract caused by *Taylorella equigenitalis*. Main Thoroughbred populations largely free of the disease apart from sporadic outbreaks; endemic in many non-Thoroughbred breeding populations. Economically important disease, although causes only mild clinical signs (endometritis/vaginitis/cervicitis).

Signs
- Fillies in training: very low risk group but infections can occur through indirect transmission; usually no external sign of infection.
- Mares: mucopurulent vaginal discharge +/– temporary infertility (early return to oestrus) following covering.
- Stallions: no external signs of infection.

Transmission
- Direct venereal contact at mating or teasing.
- Indirect spread through handlers/equipment.
- Some mares become long-term carriers without showing further signs: pathogen resides in clitoral tissues (rarely in uterus).
- Stallions can become long-term (months/years) carriers without showing signs of disease: pathogen resides in the urethral sinus/fossa and sheath.
- Foals of carrier mares may be infected at birth and remain carriers.

Diagnosis
- Genital swabbing to isolate pathogen.
- Swabbing protocol determined by breeding/import requirements and risk status of horse/country
- Fillies/mares: clitoral sinuses and fossa +/– endometrial/cervical swab during oestrus.

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- Colts/stallions: distal urethra, urethral fossa and sinus, penile sheath.
- *T. equigenitalis* is difficult to culture: use of antibiotics in week prior to swabbing or delay in processing of swabs >48 hours can reduce reliability of testing. Culture time: 3 days for growth, 7 days for negative.
- PCR highly specific and sensitive and more rapid than culture but currently not widely accepted as pre-export test.
- Testing usually combined with screening for other bacterial causes of endometritis *(Klebsiella pneumoniae/ Pseudomonas aeruginosa)*.
- Serological testing currently not reliable for detection of infection in all circumstances.

Management

- Notifiable in many jurisdictions: strict adherence to statutory requirements and current recommendations from regulatory bodies.
- Positive culture from filly/mare may be treated with repeated antiseptic washing (clitorectomy for intractable cases), followed by re-testing protocol to ensure clear of infection.

Prevention

- Prevention dependent on avoiding contact with infected individuals.
- Genital swabbing prior to breeding (or international travel).

Piroplasmosis

Piroplasmosis is a tick-borne disease caused by the protozoal parasites *Theileria equi* and *Babesia caballi*. *T. equi* is most common, but dual infections also occur. Parasite colonizes red blood cells causing cell destruction and anaemia. Disease occurs in horses living in (or transported to) regions where parasite is endemic; however, also problematic when healthy but seropositive horses fail testing requirements for international shipping. Horses born to mares from endemic regions may be seropositive.

The disease is widespread throughout southern, central and eastern Europe, Asia, Africa and Central and South America. High seroprevalence within horse populations in endemic zones.

Signs

- Acute disease: fever/severe anaemia/jaundice/oedematous filling of legs, abdomen/depression/raised respiration and pulse rates/red urine (haemoglobinuria)/abortion.
- Chronic disease: milder signs including recurrent fever/anaemia/weight loss.
- Most infections of horses born in endemic regions are non-fatal.
- Fatality rate in other horses: 10–50%.

Transmission

- Ticks are natural means of spread between horses (several species are recognized vectors for disease).
- Incubation period: *T. equi* 12–19 days, *B. caballi* 10–30 days.
- Other possible routes of transmission: mechanical spread through contaminated veterinary equipment (e.g., needles) or blood products.
- Once recovered from infection, many horses become long-term carriers with no outward sign of infection.
- Occasionally stress/other disease may precipitate a relapse in a carrier.
- Infected horses pose no risk of infection to other animals in the absence of appropriate tick vector.

Diagnosis

- Laboratory diagnosis to detect antibodies to parasite(s).
- Indirect fluorescent antibody test (IFAT) preferred.
- ELISA also used as screening test.
- PCR testing may have increased role in future.

Management

- Strict adherence to statutory requirements: some countries free of disease require quarantine, export or compulsory slaughter to prevent parasite from becoming established in local tick population.
- Acute disease: good efficacy of antiprotozoal drugs for control of acute infection.
- 'Seropositive but healthy': some antiprotozoal drugs have shown reasonable efficacy for elimination of infection of *B. caballi*; *T. equi* is relatively resistant, with lower success rates of treatment.
- Dosage of antiprotozoal drugs (imidocarb dipropionate, diminazene aceturate) required for attempted complete elimination of infection is close to toxic threshold: severe side-effects (colic/diarrhoea) common and supportive care necessary.
- Success of elimination regime often cannot be determined for some months as antibodies remain detectable for lengthy periods.

Prevention

- No vaccine available.
- Management to minimize risk of contact with ticks in endemic regions.
- Avoid practices that might spread infected blood between horses.

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African horse sickness

African horse sickness (AHS) is a viral infection spread by bite of infected midges. African horse sickness virus (AHSV) causes damage to cellular lining of blood vessels, resulting in widespread effusion and haemorrhages in body cavities/lungs.

The disease is endemic in sub-Saharan Africa. Previous outbreaks in the Middle East, southern Europe and Southeast Asia. *Culicoides* midges involved in transmission are widely distributed throughout northern Europe and the UK and spread of the related midge-borne Bluetongue virus (sheep/cattle) has demonstrated risk of spread of AHSV, facilitated by changing climatic conditions.

Signs

- Several forms of disease.
- High mortality rates in susceptible horses.
- Acute respiratory form: short incubation period/high fever/respiratory distress/frothy nasal discharge/inflamed conjunctivae/highest (>90%) mortality.
- Subacute cardiac form: swollen eyelids/oedematous shoulders/neck/chest/heart failure/moderate (>50%) mortality.
- Mixed form: mixed respiratory and cardiac signs; high (>70%) mortality.
- 'Fever' form: fever/swollen eyelids/no mortality.

Transmission

- Infectious but non-contagious: spread by the bite of infected *Culicoides* midges.
- Weather conditions and climate change thought to contribute to spread: warm, wet weather, wind dispersal.
- No direct horse-to-horse transmission.
- Variable incubation period.

Diagnosis

- Laboratory diagnosis: blood testing for antibodies (ELISA/PCR). Also virus isolation.

Management

- Strict adherence to statutory requirements and current recommendations from regulatory bodies including isolation of premises, compulsory slaughter and implementation of large exclusion zone.
- No treatment available.

Prevention

- At present vaccination restricted to Africa or reserved for outbreak events in disease-free countries.
- Current (attenuated live) vaccines do not entirely prevent subclinical infection.
- Potential for new-generation vaccines with greater safety/efficacy to have future role in limiting disease and facilitating international travel.

Dourine

Dourine is a serious venereal disease caused by the protozoan parasite *Trypanosoma equiperdum*. It is found in Asia, Africa, Southern and Eastern Europe and South America.

Signs

- Signs develop over weeks/months.
- Variable severity.
- Swelling/oedema of genitalia; may extend to perineum and lower abdomen.
- Purulent vaginal or urethral discharge.
- +/– conjunctivitis.
- Urticaria-type plaques over body.
- May progress to neurological signs (progressive weakness, paralysis).
- Loss of condition (despite good appetite).
- High (>50%) mortality outside of endemic areas.

Transmission

- Sexually transmitted (natural mating or artificial insemination [AI]).
- Most commonly from stallions to mares.
- Mare to foal transmission possible.
- Parasite cannot survive outside living host.

Diagnosis

- Laboratory diagnosis required.
- Blood testing for antibodies (complement fixation test [CFT]/IFAT).

Management

- No effective treatment.
- No available vaccine.
- Usually notifiable in non-endemic areas; strict adherence to statutory requirements and current recommendations from regulatory bodies including isolation of premises.
- Prevention of transmission through identification of infected animals (blood testing for antibodies).
- Horses should not leave endemic areas without certified freedom from disease.
- As no long-term cure, surviving infected horses are subject to breeding and movement restrictions indefinitely (or euthanasia).

Glanders

Glanders is a serious, potentially fatal bacterial infection (caused by *Burkholderia mallei*) that causes nodular

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abscessation/ulceration in the respiratory tract and lymphatic system. May infect people. It occurs in the Middle East, parts of Asia, Africa and South America.

Signs
- Development of signs may be acute or chronic.
- Three main forms of disease: nasal (nodules/ulcers in nasal passages/purulent nasal discharge/coughing), pulmonary (coughing/difficulty breathing/weight loss) and cutaneous (also known as 'farcy': abscessation/rupture of subcutaneous lymph tracts).
- All forms of disease may be associated with fever and debilitation.

Transmission
- Subclinical carrier/latent state exists.
- Ingestion of water or feed contaminated with respiratory/cutaneous discharge from infected/carrier horses.
- Incubation period highly variable and dependent on route/dose/host factors: days to months.

Diagnosis
- Laboratory diagnosis required.
- Blood testing for antibodies (CFT): accurate, sensitive to infections of >1 week duration (including carrier state).
- Culture of organism can be difficult and may require repeat sampling of purulent discharge.

Management
- No available vaccine.
- Notifiable in non-endemic areas; strict adherence to statutory requirements. Compulsory slaughter policy is typical as antimicrobial treatments are ineffectual at eliminating carrier state.

Hendra virus infection
Viral disease apparently originating from fruit bats (genus *Pteropus*) that can cause acute severe/fatal respiratory and/or neurological signs in horses. Other animals, including humans, may be seriously/fatally affected, and it is the threat to human health that is the primary concern in management of diseased horses. Currently only reported in Australia, with disease events being sporadic and infrequent.

Causes
- Hendra virus is a paramyxovirus that has an affinity for vascular tissues, initially causing damage to blood vessel walls with further effects on lungs, neural tissue and other organs.
- Primary reservoir of virus appears to be fruit bat populations.

Signs
- Can cause a range of signs depending on which organ system sustains the greatest vascular damage.
- Acute-onset fever +/– severe respiratory signs +/– severe neurological signs.
- Respiratory signs: respiratory distress (increased respiratory rate, pulmonary oedema); when close to death white or blood-stained frothy nasal discharge.
- Neurological signs: varied including ataxia, central signs (altered consciousness/'dazed' wandering/head tilt), weakness, collapse.
- Other signs may include depression, elevated heart rate, muscle trembling, colic.
- Rapid progression to death is a feature.
- Some characteristics resembling AHS (p. 379).

Transmission
- Modes of transmission (between bats, and from bats to horses) uncertain.
- Likely that exposure of horses is at least partly a chance event, and likely to come from contact with water or food contaminated with infected body fluids/excretions from bats.
- Horse-to-horse transmission may occur and thought to result from close direct contact, or indirect contact with contaminated items.
- Incubation period 5–21 days (large majority <15 days).
- Key consideration for human health: infected horses may transmit disease to in-contact people up to 3–5 days before onset of clinical signs. The examination of apparently healthy horses therefore poses some risks to vets in endemic regions.
- Bat-human, and human-human transmission has not been recorded.

Diagnosis
- Laboratory diagnosis: detection of virus/viral antigen or specific antibodies. Samples include blood, nasal, oral and/or rectal swabs in live horse; +/– fresh and fixed tissue specimens from postmortem.
- High potential for serious/fatal infection in people exposed to infected material: approach to laboratory specimen collection should reflect this (risk analysis to prevent exposure, use of full personal protective equipment). Consult local/current professional guidelines.

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Management

- Management (of clinical cases, and quarantine/movement control of premises) determined by risk assessment and statutory requirements. Infected dogs and cats pose a transmission risk and are incorporated into management plan.
- High risk to people (and high human fatality rate) associated with handling of infected horses/material, and this risk is primary concern in management.
- No specific antiviral therapies exist.
- Severe disease: euthanasia.
- Mild disease: horses may recover with supportive care.

Prevention

- Vaccination for animals living in, or travelling to, endemic areas is strongly advisable on equine and human health grounds.
- Measures to limit exposure to fruit bat body fluids/contaminants: feed/water containers under cover; excluding horses from the vicinity of bat food fruit/flowering trees.

Prognosis

- Case fatality rate in horses is approximately 75%.

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Part 4

MANAGEMENT OF THE RACEHORSE

Population and Individual Health

CHAPTER **18** Selection of the racehorse

CHAPTER **19** Prevention of injury

CHAPTER **20** Poor performance

CHAPTER **21** Herd health

CHAPTER **22** Nutrition

CHAPTER **23** Fluid and electrolyte therapy

CHAPTER **24** Blood analysis

CHAPTER **25** Transport

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SELECTION OF THE RACEHORSE

OVERVIEW

Pre-purchase examinations vary in content with type of animal, intended purpose and client demands, and may comprise any combination of physical inspection, assessment of airway function, blood analysis, radiographic, ultrasonographic or even advanced (magnetic resonance imaging [MRI], computed tomography [CT], positron emission tomography [PET]) imaging. Examinations of horses for private sale typically follow the recognized standard of 'five-stage' inspection encompassing pre- and post-exercise phases (+/– supplementary diagnostic imaging), while examinations in the context of public auctions typically have a truncated examination depending on type of horse and circumstances of sale.

Pass or fail?

The pre-purchase examination represents a 'risk assessment' of the potential for a horse not to fulfil the particular purpose for which it is being considered for purchase. The clinician's role is to conduct a thorough examination, assimilate any adverse findings in the context of the horse's individual characteristics (e.g., stage of career, conformation, body type) and interpret the relevance of these findings to and for the purchasing client. Every purchasing client has different perceptions of risk and what is acceptable to one buyer may 'fail' for another. A buyer's risk profile may be influenced by their experiences and biases, those of their veterinary advisor(s), as well as horse factors such as pedigree and residual value should a racing career not be fulfilled. Criteria for horses purchased ('pinhooked') specifically for public or private resale at a future date must necessarily be more stringent than for horses bought primarily for racing, as the presence (or development, with time elapsed/further training) of potentially significant faults may prove an obstacle to resale. Acceptability of orthopaedic conditions that require some patience in handling or that may necessitate veterinary intervention to maintain soundness is determined largely by the client's requirements. Significance of clinical and imaging findings can vary markedly depending upon age of animal, stage of training, intended purpose and future racing jurisdiction: some training conditions (such as dirt tracks, fast or poorly maintained ground and certain racetrack training grounds) are considered to be less forgiving of pre-existing carpal or fetlock pathologies, and some racing jurisdictions have regulatory stipulations that might prevent horses with particular lesions from racing. It is therefore important for the clinician to establish clearly the purpose for which the horse is being bought and the level of acceptable risk, and familiarise themselves with any relevant regulations pertaining to orthopaedic and non-orthopaedic conditions (or prior drug use) in the destination jurisdiction.

Expectations

Purchasing an unbroken yearling is a high-risk investment when considering the likelihood of racetrack or resale earnings providing a return over and above the combined cost of purchase and training. This risk is considerably lower for the horse in training that has typically been selected for examination/purchase on the basis of demonstrable athletic ability. Expectations of racing potential for a yearling should be considered in the context that only 40–50% of horses will race at 2 YO, and around 20% horses will not have started a race by the end of their 3 YO season. Regardless of how comprehensive any pre-purchase veterinary examination is there will be latent conditions or anatomical, physiological and mental/behavioural aspects of the horse that cannot be adequately identified or tested, that in some cases can preclude the fulfilment of a racing career.

Conflicts of interest

Prior to undertaking a pre-purchase examination the clinician should attempt to establish the identities of both vendor and potential purchaser to avoid any possible conflict of interest. Clinicians should avoid knowingly undertaking examinations on horses that have been under their direct care and if such a situation is unavoidable, the examination should only proceed on the understanding that the potential purchaser is informed of this conflict and that the vendor gives permission for the clinician to

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declare all relevant history relating to that animal. To not do so may be viewed as a conflict of interest and breach of professional ethics should regulatory or legal proceedings arise from a post-purchase dispute.

Clarification of the vendor-purchaser relationship as stated earlier is generally not possible in the context of public auctions for a range of reasons including opacity around ownership shares, horses being presented by sales consignors rather than owners, and time constraints limiting the level of veterinary input available for assessment of each animal. In these challenging circumstances best practice is for clinicians to refrain from undertaking pre-bid examinations on horses that are, or have been, under their direct care or for which they have prior knowledge that might compromise their impartiality or objectivity.

All information gathered during the course of the examination is solely reportable to the potential purchaser. While the vendor has no right to this information, judicious sharing of pertinent findings (with permission of the purchasing agent) is courteous and usually beneficial to all parties.

Conditions of sale at public auction

Conditions of sale at public auction differ between sales companies and type of sale. Most have clauses regarding return of lots due to undeclared upper airway conditions (or prior surgery), stable vices, use of selected classes of drugs and prior exposure to diseases that may prevent international movement and it is important to be familiar with the relevant conditions when undertaking pre-bid inspections. Conditions of sale are amended periodically in response to scientific and legal developments, and even when familiar with a particular auction it is advisable for the clinician to check the text of these conditions (published in the respective sale catalogue or online) for updates. Legislation in some countries permits the return of animals with latent defects beyond the scope of published conditions of sale; however, preventing such situations arising in the first instance through competent examination is always preferable.

THE VETTING PROCEDURE

Key elements of a full (horse in training) pre-purchase examination are outlined in the following sections. For practical reasons it is not possible to incorporate all of these elements in pre-bid inspections conducted at public auction.

Preliminaries

- Establish the intended purpose for which the horse is being examined; imaging requirements and relevance of findings may differ between jurisdictions.
- Communication with vendor prior to examination regarding level of exercise required/recent medication.
- Verbal or written (preferred) statement from vendor regarding stable and ridden vices, history of previous surgery, exercise-induced pulmonary haemorrhage (EIPH), heart irregularities or any other significant veterinary intervention, previous administration of important proscribed drug classes and any current racing sanctions.
- Establish recent racing form.
- Verify identification from passport and record vaccination history.

Physical examination: Summary

- Condition and conformation: apparent fitness, overall health and obvious conformational faults.
- Eyes (Table 18.1): horses with severely impaired vision can function seemingly normally in training with few or no behavioural irregularities. Gross assessment, direct ophthalmoscopy and evaluation of ocular reflexes. Menace reflex is reasonable test of vision (requires intact sensory and motor pathways), unlike pupillary light reflex.
- Heart: rate, rhythm and murmurs. Auscultation from both sides of chest at rest and immediately following exercise. Heart rate is commonly recorded but rarely of importance.
- Mouth: teeth (evidence of stable vices); corners of mouth (bit damage); tongue (bit/chifney damage).
- Neck: throat and submandibular space (scarring/lymph nodes); jugular grooves (patency of jugular veins/recent injection); poll and wither (head shyness/injury).
- Girth, topline and belly: sarcoids (interference with tack); muscular pain/asymmetry of topline and pelvis; abdominal scars (previous surgery); tail (tone and injury).
- Genitalia: both testicles fully descended/palpably normal (colts); mammary glands and vulval conformation (fillies).
- Limbs: detailed palpation weight-bearing and with limb in flexion.
- Feet: foot symmetry, balance, hoof quality and type of shoeing.
- Observation of temperament, or management features pertaining to temperament (stabling, exercise).
- Observation of features of stabling/bedding that may indicate presence of stable vices or management for lower respiratory tract disease.

Action

- Examination at walk and trot in hand before exercise.

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Table 18.1 **Significance of ocular findings**

	FINDING	CAUSE	SIGNIFICANCE
Globe	Small/shrunken globe	May be congenital (+/– concurrent lens luxation and blindness) or acquired (secondary to inflammatory disease)	Vision usually severely affected
Cornea	Oedema	May be primary/secondary, temporary or permanent.	Effect on vision dependent on extent. Not usually progressive.
	Keratitis	Multiple causes (viral/bacterial/fungal/immune) and variable severity.	Most respond favourably to treatment; some conditions are recurrent.
	Ulceration	Multiple causes (trauma/infection)	Most respond favourably to treatment with little/no permanent effect on vision.
Anterior chamber	Uveitis	Inflammation of anterior chamber/iris.	Frequently recurrent. Considered high risk for development of future episodes of vision-affecting disease.
	Synechiae (adhesions)	Indicates previous inflammation (uveitis) in eye	Considered high risk for development of future episodes of vision-affecting disease.
Iris	Coloboma	Congenital focal weakening/prolapse of iris.	No effect on vision.
	Corpora nigra and iris cysts	Can reach large size and occasionally detach to cause inflammation of eye	Generally considered benign and no risk for vision.
Lens	Cataracts	Anterior (Y-shaped) and posterior suture opacities are common (up to 20%).	Not progressive and do not significantly affect vision. Central capsular cataracts may interfere with ophthalmoscopic examination of retina, but do not normally affect vision (some interference with vision possible when pupil constricts in bright daylight).
	Luxation	May be anterior or posterior; always associated with cataract and inflammation	Blindness or severely affected vision.
Fundus (retina)	Peripapillary chorioretinopathy ('butterfly' lesions)	Localized loss of retinal pigment. Very common; may arise from previous inflammation.	Not progressive and do not affect vision.
	Focal chorioretinopathy	'Bullet hole' loss of retinal pigment	Variable outcome but when extensive may be a risk for future vision impairment
	Optic disc atrophy or loss of vascularization	Loss of blood supply to optic nerve	Usually indicates blindness
	Retinal detachment	Multiple causes (trauma/congenital/viral)	Severe impairment to vision

- Exercise component should ideally incorporate fast work so that dynamic respiratory obstructions and propensity for EIPH can be assessed.
- Second examination at walk and trot following cooling-off period.

Flexion tests

- Flexion tests are non-specific and response varies between horses.
- May exacerbate subclinical lameness.
- Force and duration of flexion can strongly influence results; important that standardized approach is used and 'positive' responses (obvious lameness which persists for ≥3 strides) are compared against opposite limb and interpreted with caution.

Ancillary tests

- Blood/serum samples obtained for analysis for presence of anti-inflammatory +/– other classes of drugs (e.g., sedatives, diuretics).
- For horses that are intended to travel internationally, appropriate blood +/– reproductive tract samples according to the specific entry requirements of receiving country.
- Assessment of upper airway function, limb ultrasonography and radiography as required.

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VETTINGS: ASSESSING THE SIGNIFICANCE OF CHRONIC MUSCULOSKELETAL PATHOLOGIES

- Many racehorses carry (and perform successfully with) chronic musculoskeletal pathologies.
- Risk determinations for future impact on career should synthesize diagnosis, lesion severity (clinical and imaging), horse and purchasing client factors; the importance of similar imaging features can vary widely between horses.
- In general: if a horse has been racing successfully in spite of a chronic and stable musculoskeletal pathology, then risk of that pathology worsening in short- to medium-term future (to extent that training/racing is impeded) is low, providing that other major variables (such as training conditions) remain constant.
- Risk of a chronic musculoskeletal pathology becoming problematic in future may increase with change in training conditions or racing jurisdiction (e.g., if latter has greater regulatory scrutiny/sanction for horses with musculoskeletal abnormalities).

History

- While not always a compulsory component of the pre-purchase examination, a full and frank disclosure of veterinary history (particularly with regard to timing/frequency of orthopaedic medications) by the vendor can greatly assist the interpretation of any radiological abnormality or lameness that is encountered during the examination.

Reporting

- A written report detailing the identification of the animal, scope of the examination, procedures undertaken, available history/declarations, findings and interpretation of findings regarding the suitability of the horse for the intended purpose should be made available to the purchasing client.
- It is frequently the case that multiple findings (e.g., radiological lesions) of low/negligible significance may be encountered. The extent to which these need to be/should be communicated to purchasing clients in the context of public auction pre-bid inspections is an area of legal uncertainty, given that many clients consider the filtering/distilling of such information to only the clinically important points to be the primary role of their trusted veterinary advisor. Regardless of how such findings are reported it is very important that their presence is at least recorded in contemporaneous notes or worksheets in the event that future claims arise that lesion(s) were missed.

PRE-PURCHASE RADIOGRAPHY

Pre-purchase radiography is a standard procedure for yearlings at premier public auctions and many horses in training of medium/high value. Pre-purchase radiography should be considered a survey of the common radiographic lesion predilection sites, rather than a comprehensive warranty of future soundness. Several important orthopaedic conditions encountered in racehorses are either radiologically silent or occur at sites not routinely surveyed during a pre-purchase examination: the absence of radiological faults does not necessarily equate to the absence of significant pathology.

The radiographic protocol (minimum specified views) varies with the age of the horse and the requirements of the sales company/purchaser, and reflects the relative importance of certain pathological conditions at various stages of training. The proximity of image acquisition to sale date is usually stipulated by the conditions of sale (typically within 3–6 weeks), and examining clinicians should still be alert to pathology that may arise in the intervening period. Examples of internationally-recognised protocols are listed in Table 18.2.

Supplementary projections

The need for additional radiographic projections is dictated by clinical findings. Bony enlargements/splints not adequately covered on standard projections may warrant lesion-oriented obliques to assess activity. The importance of fetlock condylar pathology in horses in training makes a strong case for inclusion of multiple flexed dorsopalmar/plantarodorsal projections (Appendix 4) into any comprehensive protocol for horses that have trained. Multiple DLPaMO + skyline distal row projections of the carpus (Appendix 4) are beneficial for assessment of the middle carpal joint when activity at this site is suspected.

Radiographic survey of the back (or neck) is not recommended as part of a standard pre-purchase protocol; there is no evidence of a correlation between radiographic appearance of the dorsal spinous processes of the back and future risk of clinical disease in asymptomatic horses.

Digital images

Due to the large file size of acquired images, image compression is used both to facilitate data archiving and to permit transmission of images to remote servers. Image compression can either be reversible ('lossless') or irreversible ('lossy'). The type and level of compression that is diagnostically (or legally) acceptable varies with application and current guidelines are neither definitive nor universal to all situations. The often low range of contrast in equine skeletal imaging calls for a conservative approach

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Table 18.2 **Standard protocols for pre-purchase radiography**

SITE	PROJECTION	YEARLING	HORSE IN TRAINING
Front fetlock	Dorsopalmar (DPa) elevated 20^0	●	●
	Dorsolateral oblique (DLPaMO)	●	●
	Dorsomedial oblique (DMPaLO)	●	●
	Standing lateromedial (LM)	●	●
	Flexed lateromedial (flexed LM)	●	○
	Flexed dorsopalmar (flexed DPa)	○	⊕
Hind fetlock	Dorsoplantar (DPl) elevated 30^0	●	●
	Dorsolateral oblique (DLPlMO)	●	●
	Dorsomedial oblique (DMPlLO)	●	●
	Standing lateromedial (LM)	●	●
	Flexed plantarodorsal (flexed PlD)	○	⊕
Knee (carpus)	Dorsopalmar (DPa)	●	●
	Dorsolateral oblique (DLPaMO)	●	⊕
	Dorsomedial oblique (DMPaLO)	●	●
	Standing lateromedial (LM)	●	●
	Flexed lateromedial (flexed LM)	○	●
	Skyline distal row (flexed $D35^0PrDDiO$)	○	⊕
Hock (tarsus)	Dorsoplantar (DPl) slightly (10^0) lateral	●	◔
	Dorsoplantar (DPl)	○	●
	Dorsolateral oblique (DLPlMO)	●	●
	Dorsomedial oblique (DMPlLO)	●	●
	Standing lateromedial (LM)	●	●
Stifle	Lateromedial (LM)	●	●
	Caudolateral oblique (CdLCrMO)	●	●
	Caudocranial (CdCr)	●	●
Front foot	Lateromedial (LM)	◔	●
	Upright pedal ($D60^0Pr$-PaDiO)	○	●

Green, standard protocol; half-green/half-white, inclusion dependent on specific requirements/clinical judgement; +, more than single projection recommended.

to image compression; the quality of the viewed image is also frequently limited by the resolution of the viewing screen as much as by file size.

It is recommended that the Digital Imaging and Communications in Medicine (DICOM) format is used where possible for diagnostic, pre-purchase and teleradiology applications of radiographic and ultrasonographic images, as this involves no image degradation and permits full adjustment of window levels and other post-processing. By contrast, window level of JPEG and TIFF formats is set at the time of creation of the image and cannot be altered by the viewer; additionally, it is possible for JPEG images to be manipulated such that lesions are obscured. JPEG images should only be used as part of a pre-purchase examination if there is no visual loss of image quality due to compression.

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Image quality and interpretation

Radiographic series are assessed methodically not just for pathology but with attention paid to diagnostic quality, completeness, date of acquisition and accurate labelling, and any deficiencies noted. Should positioning, exposure or superimposition of anatomical structures limit full assessment of important predilection sites for pathology, further radiographic images should be obtained (this is usually possible even in the context of public auctions when time is limited). The relevance of certain radiological lesions to future soundness may differ between individuals with similar defects, and clinical evaluation of the affected site is essential for accurate interpretation in many cases. The relative importance of radiological findings can also differ considerably depending on stage of career. As a general rule, developmental orthopaedic disease (osteochondrosis: osteochondritis dissecans [OCD]/bone cyst) and abnormalities associated with the proximal sesamoid bones (PSBs; sesamoiditis, foal sesamoid fractures) are viewed more critically in the yearling because any influence on trainability remains untested. When these same faults are detected in a horse that has been trained to the 'breeze-up'/'ready-to-run' stage and the horse remains clinically unaffected they can be interpreted more leniently, and when detected in a horse-in-training with recent racing form they can often be disregarded (or at least considered low risk for future interruption to training). Radiological signs of active arthritic change in important high-motion joints (fetlock/middle carpal joint) should be interpreted in the context of physical findings but may be significant at any age.

Risk associated with radiological findings at pre-purchase survey is estimated through a combination of clinical judgement and understanding of relevant scientific and empirical evidence. Radiological irregularities may be 'normal' variants of anatomic structures, incidental findings with negligible risk of interfering with training, clinically silent pathologies that may have future clinical relevance or pathologies that are already clinically apparent. The potential for lesions to progress or regress with future training can vary widely between individuals and is affected by a host of unquantifiable factors including those relating to conformation, maturity, genetics, training approach and location/track characteristics, and risk assessment at the time of radiographic survey is necessarily an approximation only. The prevalence of radiological abnormalities in yearlings presented for sale at public auction (Table 18.3), and their impact on future performance, has been investigated in a small

Table 18.3 **Prevalence and significance of radiological abnormalities in public-auction yearlings**

SITE	FINDING	PREVALENCE (PUBLISHED FIGURES)	RISK FOR TRAINABILITY	RISK FOR RESALE (AS HORSE IN TRAINING)
Carpus	Dorsal medial carpal disease	1.9[g]–2.7[b]%	Low-medium; dependent on severity and training location. Conflicting published evidence regarding likelihood to start a race. May develop carpal lameness in training	High; dependent on severity
	Chip/fragment/spur (middle carpal joint)	2.5[b]–4[a]%	Medium. May develop carpal lameness in training	High
	Distal radial cyst	0.09[b]%	Medium	Medium
	Radial carpal bone cyst	?	Low-medium. Most remain clinically silent but those that develop lameness can be problematic to manage	Medium
	Ulnar carpal lucency/cyst	8.33[d]–22.2[a]%	Negligible.	Negligible
	Accessory carpal bone fragmentation/fracture	0.08[a]–0.4[b]%	Low/negligible (if clinically silent)	Low-medium depending on severity and intended market
	Distal intermediate carpal bone modelling/ fragment	?	Low	Low

(Continued)

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Table 18.3 **Prevalence and significance of radiological abnormalities in public-auction yearlings** ***(Continued)***

SITE	FINDING	PREVALENCE (PUBLISHED FIGURES)	RISK FOR TRAINABILITY	RISK FOR RESALE (AS HORSE IN TRAINING)
Hock	DIT/TMT spur	9.92[d]–35.4[a]%	Negligible. No association with performance	Low/negligible
	OCD (tibiotarsal joint, all sites including distal talus fragmentation)	4[c]–8.7[a]% (distal intermediate ridge most common: 3%)	Low. No association with performance	Low
	Wedged-shaped or collapsed T3	1.1[g]–1.2[b]%	Mild severity: low. Marked/collapsed: medium-high: may be less likely to start a race and may predispose to slab fracture	Mild: negligible Marked: medium/high
	T3 or CTB fracture (including incomplete/ distal/slab)	1.4[g]%	Low. No association with performance	Chip/incomplete: negligible Complete slab: low-medium
Front fetlock	Sagittal ridge OCD (lucency)	17.4[b]–37.1[a]%	Low/negligible No association with performance	Negligible
	Chip/fragment: dorsoproximal P1	0.7[a]–3.2[g]%	Low/negligible	Low
	Chip/fragment: palmaroproximal P1	0.4[a]–2[d]%	Low (dependent on type/size). No association with performance	Low/negligible
	Bone cyst (distal MC3 or proximalP1)	0.5[g]–0.7[b]%	Statistical risk undetermined; may remain clinically silent but those that develop lameness can be problematic to manage.	Medium-high
	Supracondylar lysis (moderate-severe)	0.1[a]–2.1[b]%	Medium. Conflicting published evidence but possible reduced likelihood to start a race (at 2 or 3 YO).	Low-medium
Hind fetlock	Sagittal ridge OCD (>10 mm length)	0.7[a]–1.6[b]%	Low-medium. Limited evidence of statistical association with fewer starts/ less likely to start/greater time to first start. In practice, not recognised as a significant clinical problem.	Low
	Chip/fragment: dorsoproximal P1	1.6[d]–3.3[b]%	Low. Statistically may be less likely to start a race[j]. In practice not recognised as a significant clinical problem.	Low
	Chip/fragment: plantaroproximal P1	5.9[b]–7.1[d]%	Low/negligible (dependent on type/size). No association with performance	Low/negligible
	Bone cyst (distal MT3 or proximal P1)	0.2[b]–0.4[i]%	Statistical risk undetermined; may remain clinically silent but those that develop lameness can be problematic to manage.	Medium-high

(Continued)

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Table 18.3 **Prevalence and significance of radiological abnormalities in public-auction yearlings** ***(Continued)***

SITE	FINDING	PREVALENCE (PUBLISHED FIGURES)	RISK FOR TRAINABILITY	RISK FOR RESALE (AS HORSE IN TRAINING)
Pastern joint	Lucencies/bone cysts (distal P1/proximal P2; midline/condylar)	0.9[k]–2.4[f]% (forelimbs:hindlimbs 40:60%)	Midline small-medium lucencies: negligible. Bone cysts: no overall statistical association with performance; however, risk may vary with lesion size/location/degree of articular communication.	Low-high (dependent on size/appearance)
	Palmar/plantar chip/ fragment	0.06[k]%	Low-medium (greatest risk if concurrent arthritic change). No association with performance at 2 or 3 YO. May (rarely) cause lameness in early training.	Low-medium
Proximal sesamoid bone: forelimb	Marked sesamoiditis (≥3 irregular vascular channels >2 mm)	2–3.6[a]%	Medium (determined by SLB involvement). May be associated with reduced performance at 2 and 3 YO	Low-medium
	Fracture	0.4[g]–1.5[a]% (medial most common)	Low-high (dependent on location/ severity). May be associated with reduced performance at 2 and 3 YO	Medium-high
	Elongated/abnormal shape	5.6[b]–10.3[g]%	Low/negligible. No effect on performance	Low
	Modelling (osteophytes/ enthesophyte[s])	1.6[b]–6.6[g]%	Low-medium (determined by SLB involvement). May be associated with reduced performance at 2 and 3 YO	Low-medium
Proximal sesamoid bone: hindlimb	Marked sesamoiditis (≥3 irregular vascular channels >2 mm)	2[e]–3[a]%	Medium (determined by SLB involvement). May be associated with reduced performance at 2 and 3 YO	Low-medium
	Fracture	1.4[g]–2.9[b]%	Low-medium (dependent on location/severity). Statistically no detrimental effect on performance	Low-medium
	Elongated/abnormal shape	2.8[b]–7[g]%	Low	Low
	Modelling (osteophytes/ enthesophyte[s])	4[a]–9[g]%	Low. No effect on performance	Low
Stifle	OCD (defect/fragment): lateral trochlear ridge	3[c]–5.1[b]% (lesion >40 mm in 0.5%)[a]	Horses with smaller lesions (total length <6 cm) undergoing surgery prior to 2 YO season have similar trainability to normal horses; longer time to first start. Horses with more extensive lesions (total length >6 cm) less likely to race	Low-medium (dependent on radiological appearance/clinical history)

(Continued)

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Table 18.3 **Prevalence and significance of radiological abnormalities in public-auction yearlings** ***(Continued)***

SITE	FINDING	PREVALENCE (PUBLISHED FIGURES)	RISK FOR TRAINABILITY	RISK FOR RESALE (AS HORSE IN TRAINING)
Stifle	OCD (defect/fragment): medial trochlear ridge	0.3[b]–0.7[a]%	Medium	Low-medium (dependent on radiological appearance/clinical history)
	Medial femoral condyle: bone cyst	2[c]–5.6[a]%	Medium >6 mm deep: less likely to race at both 2 and 3 YO (implies risk of interrupted training)	Low-high (dependent on radiological appearance/ clinical history)
	Medial femoral condyle: shallow (<3mm deep) lucency	6.4[h]–16[c]%	Low-medium. Small proportion (3.6[h]%) may develop into bone cyst. Statistical association with fewer starts at 2 YO.	Low
	Patellar fracture	0.1[a]%	High	High
Front foot	P3 cyst	2.3[b]%*	Medium-high. Less likely to start a race.	High
	P3: proliferative modelling (toe)	6.1[a]%	Low. No effect on performance	Low
	P3: proliferative modelling (dorsal)	14.5[a]%	Low/negligible	Negligible
	P3 fracture	0.3[a]%	Low-medium (dependent on type)	Low-medium (dependent on type)
	Bipartite navicular bone	?	High. Usually cause lameness (untreatable) in training; small number of horses remain clinically unaffected and race.	High

* Published prevalence appears to differ significantly from that observed in clinical practice.

[a] Jackson M, Vizard A, Anderson G *et al.* (2009). A prospective study of presale radiographs of Thoroughbred yearlings. RIRDC No 09/082.

[b] Kane AJ, Park RD, McIlwraith CW *et al.* (2003). Radiographic changes in Thoroughbred yearlings. Part 1: Prevalence at the time of the yearling sales. *Equine Vet J* 35(4):354–365.

[c] Oliver LJ, Baird DK, Baird AN *et al.* (2008). Prevalence and distribution of radiographically evident lesions on repository films in the hock and stifle joints of yearling Thoroughbred horses in New Zealand. *NZ Vet J* 56(5):202–209.

[d] Furniss C, Carstens A, van den Berg (2011). Radiographic changes in Thoroughbred yearlings in South Africa. *J S Afr Vet Assoc* 82(4):194–204.

[e] Spike-Pierce D, Bramlage LR (2003). Correlation of racing performance with radiographic changes in the proximal sesamoid bones of 487 Thoroughbred yearlings. *Equine Vet J* 35(4):350–353.

[f] Vargas J (2011). Racing prognosis of Thoroughbred yearlings with subchondral bone lucencies in the proximal interphalangeal joint. *Proc Am Assoc Equine Pract* p. 243.

[g] Miyakoshi D. *et al.* (2017). A retrospective study of radiographic abnormalities in the repositories for Thoroughbreds at yearling sales in Japan. *J Vet Med Sci.* 79(11):1807–1814.

[h] Pérez-Nogués M. *et al.* (2021). Progression of shallow medial femoral condyle radiographic lucencies in Thoroughbred repository radiographs and their influence on future racing careers. *Equine Vet J* 53(2):287–293.

[i] Russell J. *et al.* (2017). Heritability and prevalence of selected osteochondrosis lesions in yearling Thoroughbred horses. *Equine Vet J* 49, 282–287.

[j] Robert C. *et al.* (2013). Influence of juvenile osteochondral conditions on racing performance in Thoroughbreds born in Normandy. *Vet J* 197, 83–89.

[k] Santschi M. *et al.* (2015). Prevalence of radiographic abnormalities of the proximal interphalangeal joint of young Thoroughbreds and associations with early racing performance. *J Equine Vet Sci* 35, 225–231.

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Table 18.4 **Risk matrix for radiological findings at pre-purchase survey**

PROBABILITY		SEVERITY	
Rare	May occur in exceptional circumstances	Low	Likely to remain clinically inapparent but may interfere with resale (no impact on training)
Unlikely	Could occur occasionally	Low	May become clinically apparent but no impact on training
Possible	Expected to occur occasionally	Medium	May go lame +/– interrupt training
Likely	Expected to occur regularly	Medium-High	May go lame and require significant input (+/– surgical) +/– interfere with training/racing
Almost certain	Expected to occur frequently	High	If goes lame, treatment options limited/ prognosis for athletic soundness guarded/poor

number of studies (USA, Australia, New Zealand, South Africa, Japan). There are only a few consistent or strong relationships between radiological findings and key measures of performance that include likelihood to start, time to first start and career placings/prize-money. It should be noted that none of these studies investigate the aspect that is most important to potential purchasers: that of the actual risk of a radiological abnormality causing a future related orthopaedic problem. Additionally, most of these studies sample a selected (premier sales) population and do not take into account previous or subsequent surgical intervention, and numbers of many less common conditions are insufficient to draw statistically robust conclusions about relevance to future soundness. Regardless of the general indirectness of these 'risk associations', risk may still be attached to some abnormalities on an individual basis because of clinical experience of potential problems arising from similar lesions, and the consequences for that individual should lameness arise. Risk is best expressed in a risk 'matrix' (probability of a negative outcome arising, and likely severity of that negative outcome; Table 18.4); that associated with osteochondrosis lesions ('bone cysts'/OCD) found on radiographic survey is found in Table 18.5; however, in the absence of good data these should be considered only approximate guidelines at best.

Table 18.5 **Risk* matrix for previously untreated osteochondrosis lesions† detected at yearling pre-purchase survey (based on population prevalence)§**

LESION	ESTIMATED PROBABILITY OF DEVELOPING SIGNIFICANT ASSOCIATED LAMENESS	POTENTIAL SERIOUSNESS FOR RACING CAREER IF SIGNIFICANT LAMENESS DEVELOPS
DIPJ cyst	<15%	High
Pastern joint cyst (non-midline)	<10%	High
Fetlock joint cyst	<20%	High
Stifle cyst	<20%	Medium-High
Stifle OCD	<20%	Medium-High
Tibiotarsal joint OCD	<5%	Low

* Further refinement of risk based on radiological features generally possible.

† If clinically silent at time of detection.

§ Unpublished data, P. Ramzan.

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Radiological lesions encountered in yearlings

A wide range of lesions, both developmental and acquired, are encountered in radiographic surveys of yearlings. Many of the developmental lesions (such as bone cysts) are typically clinically silent before the commencement of training. A brief description of each, to supplement information already provided in Table 18.3 (and Chapter 6), with comments pertinent to pre-purchase assessment, follows. While some associations between lesion size and racing outcomes have been reported in the literature it is important to recognise that in practice measurement of lesions is rarely undertaken and that radiological interpretation is best informed by many factors beyond simply size, including imaging features relating to how 'settled' a lesion site appears, the presence of associated joint modelling/fragmentation and clinical evidence of joint inflammation.

Distal interphalangeal ('coffin') joint cysts

Bone cysts with distal interphalangeal joint (DIPJ) communication may be found in P3 (most commonly) or P2. P3 cysts are not always radiologically evident, and when they are it is generally the 'upright' or standing DPa pedal projection that is most useful. Evidence of a cystic lucency may first be noted at the edge of field of view on the lateromedial (LM) projection of the fetlock, and if so further projections should be obtained to further assess (Figure 18.1a,b). Statistical risk of developing lameness from a DIPJ cyst has not been quantified; if a cyst becomes clinically active it can pose a serious threat to a racing career. Risk cannot currently be categorised by radiological appearance or location of the cyst.

Pastern joint (PIPJ) cysts

Small midline lucencies in distal P1 (Figure 18.2a) are generally considered a normal variant and can be disregarded. Other lucencies more typical of bone cysts can be found centrally or in the condyles of distal P1 (Figure 18.2b–d), or in proximal P2. Risk of developing lameness associated with proximal interphalangeal joint (PIPJ) cysts has not been quantified but is considered low; however, should a cyst become a clinical problem the risk to that individual's racing career can be serious. Lesion size/appearance/degree of articular communication may permit some refinement of risk assessment; additionally the presence of osteoarthritic modelling of the margins of the associated joint is a strong indicator that the cyst could be problematic. Cysts in the condylar region of distal P1 tend to become narrower/smaller as they resolve (Figure 18.2e) and when encountered at the time of public auction with this narrow appearance are highly unlikely to cause clinical problems in training.

Pastern joint (PIPJ) fragments

Fragmentation +/–/or enthesophyte(s) at back of PIPJ (Figure 18.3; see also p. 143) is a rare finding; risk of developing associated unsoundness in future training is not considered to be high although if lameness does arise it can be problematic to resolve; risk cannot be fully assessed prior to commencement of full training. As with PIPJ bone cysts, concurrent radiological signs of osteoarthritis (OA) are associated with increased risk of future lameness. Origin of these fragments is uncertain, but typical location at the site of insertion (on palmaroproximal P2) of the middle scutum (the thick fibrocartilaginous pad over which the deep flexor tendon slides) indicates insertional pathology/avulsion in many cases. Generally of long-standing appearance at time

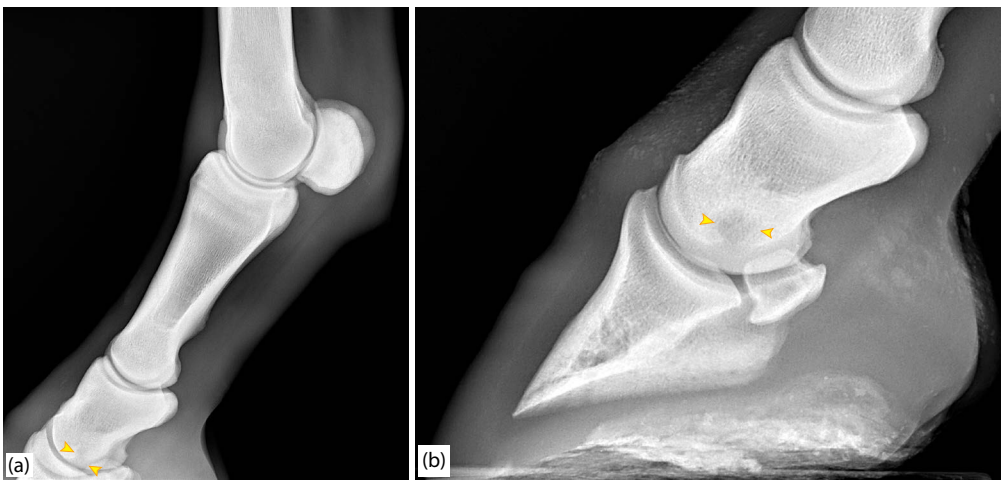


Fig. 18.1 **LM fetlock projection may permit detection of DIPJ lucency at edge of field of view (a); suspect lesions are better defined with further radiographs centred on foot (b).**

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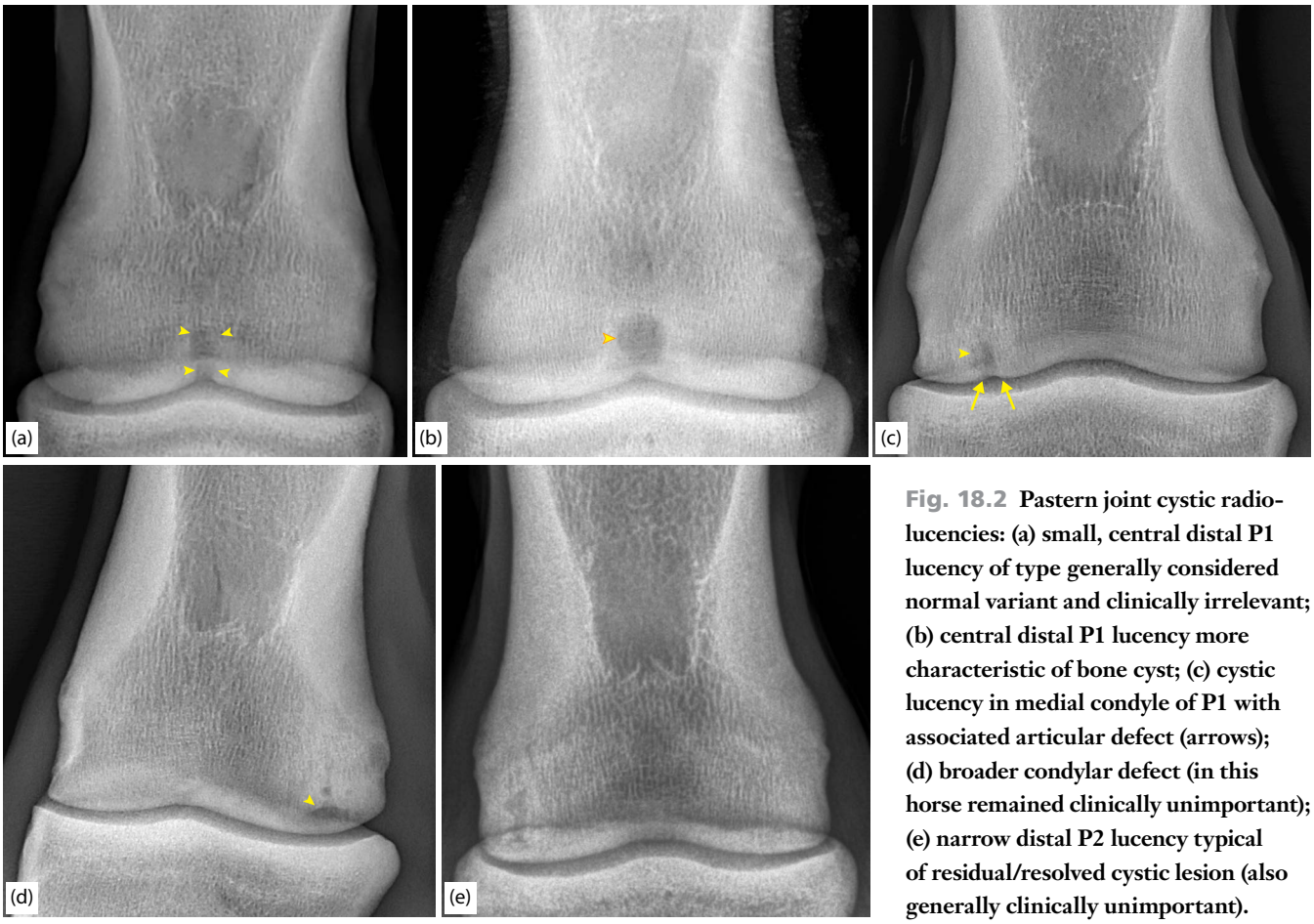


Fig. 18.2 **Pastern joint cystic radiolucencies: (a) small, central distal P1 lucency of type generally considered normal variant and clinically irrelevant; (b) central distal P1 lucency more characteristic of bone cyst; (c) cystic lucency in medial condyle of P1 with associated articular defect (arrows); (d) broader condylar defect (in this horse remained clinically unimportant); (e) narrow distal P2 lucency typical of residual/resolved cystic lesion (also generally clinically unimportant).**



Fig. 18.3 **Plantar PIPJ fragmentation (DLPlMO).**

of detection (at yearling stage) so presumed to be result of undetected injury early in life.

Proliferative entheseal (non-articular) modelling of the palmar aspect of P1 is an unrelated condition, but is encountered occasionally. Even when pronounced, it is generally clinically silent and remains irrelevant to athletic soundness throughout training; should only be considered potentially important if radiological or clinical (e.g., concurrent digital tendon sheath effusion) features suggest ongoing activity.

Pastern joint (PIPJ) osteoarthritis

Arthritic change (Figure 18.4) involving the PIPJ margins (generally the dorsal margin) is a rare finding that when identified in a yearling is considered to represent at least medium risk to future trainability, as should lameness subsequently develop when the animal enters training the prognosis for return to sustained athletic soundness is guarded.

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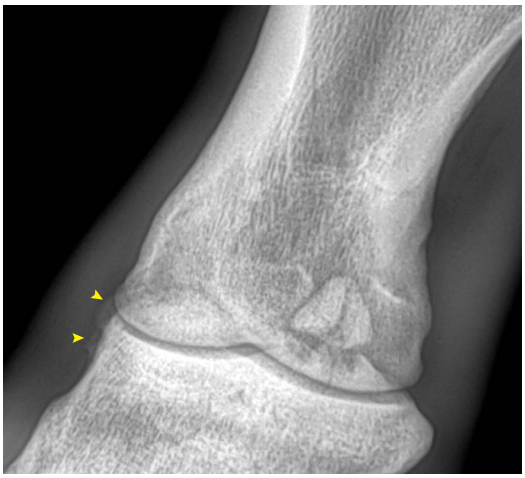


Fig. 18.4 PIPJ osteoarthritis: Immature/active modelling of dorsomedial joint margin (arrowheads); note also concurrent palmar fragmentation similar to that in Figure 18.3 (DLPaMO).

Fetlock joint cysts

Bone cysts may occur in the distal cannon or proximal P1 and vary widely in size and appearance. Risk of developing lameness associated with fetlock cysts has not been quantified but is generally considered low; however, should a cyst become a clinical problem the risk to that individual's racing career can be serious. Judging risk of developing associated lameness is subjective and influenced by degree of apparent articular involvement, surrounding bone activity (or apparent radiological 'stability') and location of primary articular defect (Figure 18.5a–d); some lesions represent a high risk due to lesion location (such as in/near parasagittal groove). Additional radiographic projections are often needed for better assessment. Many examples exist of horses with radiologically obvious lesions having successful/trouble-free racing careers. However, an additional consideration in this joint is the extreme stresses that are placed upon the fetlock in race training; bone cysts in distal cannon may sometimes predispose to

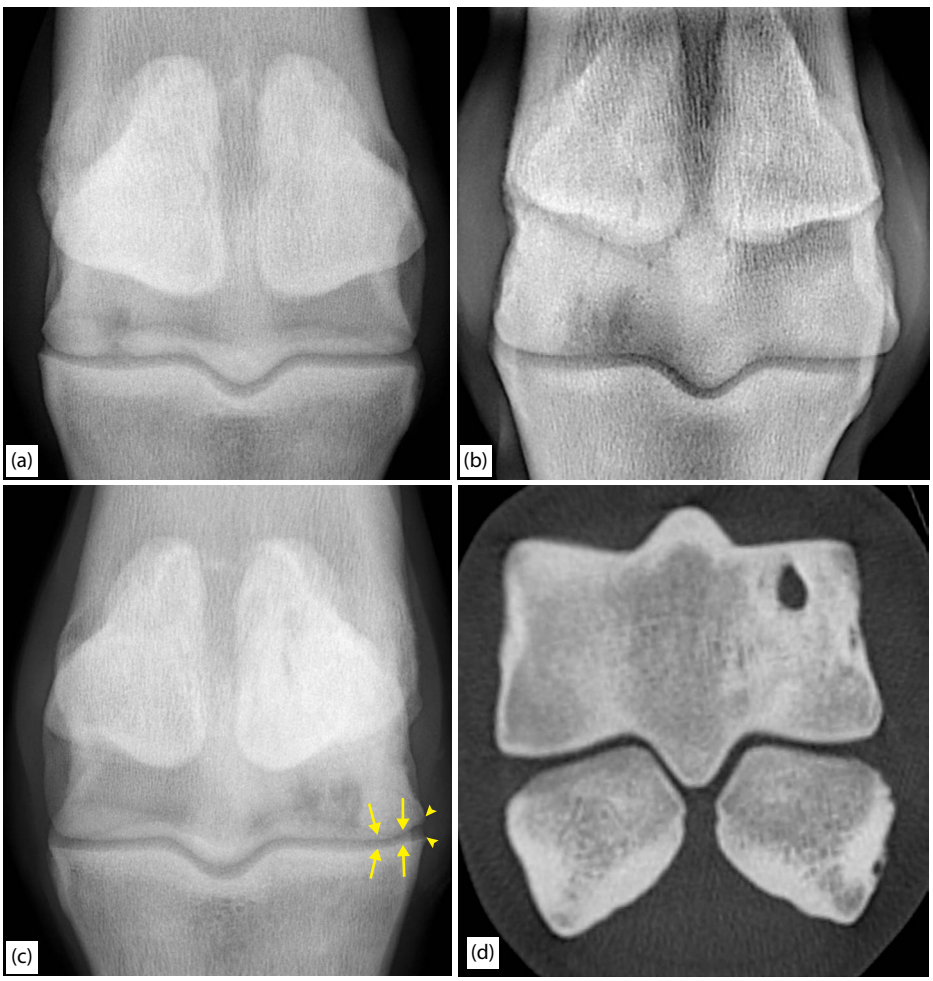


Fig. 18.5 Examples of fetlock cystic radiolucencies in distal MC3: (a) lesion of typical appearance (DPa); additional radiological features likely to represent high risk of poor athletic outcomes are seen in (b) extensive, poorly defined defect affecting much of condyle with associated marked increased density of distal cannon and conspicuity of metacarpal vessels (flexed DPa), and (c) large lytic defect with secondary articular modelling (arrowheads) and narrowing of joint space (arrows) on affected side; (d) transverse CT image showing cyst positioned dorsally (and therefore presumed to represent lower risk of future associated lameness/fracture than palmar lesion).

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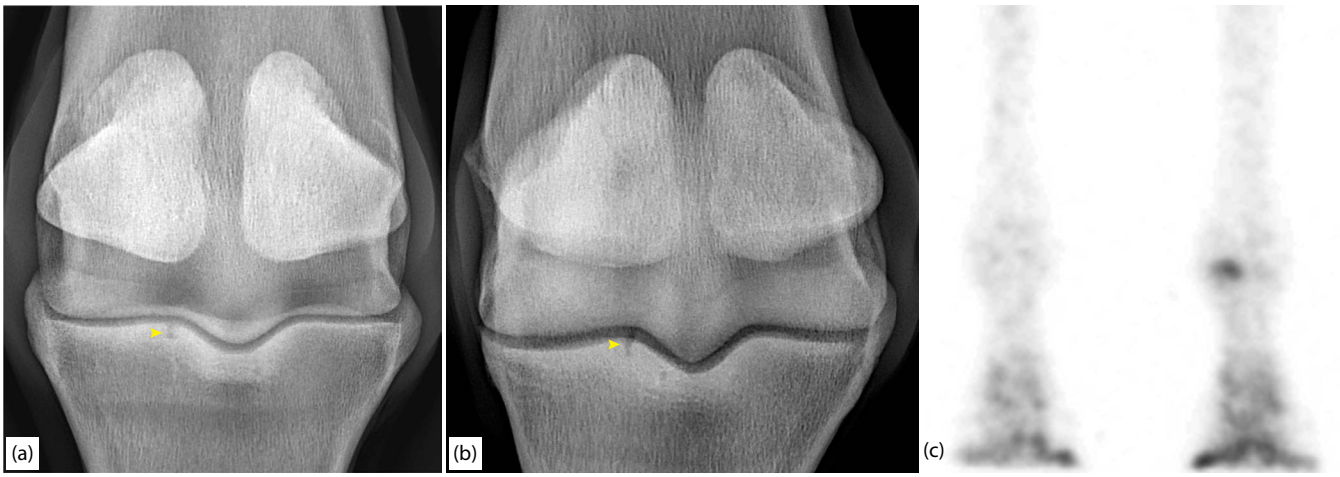


Fig. 18.6 Proximal P1 short linear cystic radiolucency: Typical radiological appearance in non-midline position and visible on both standing DPa (a) and flexed DPa (b) projections. Usually an incidental finding of no clinical importance but occasionally associated with joint inflammation: IRU in (c) from horse with persistent synovitis/mild lameness assumed to be associated with lesion.

development of condylar fracture pathology when located in the palmar/plantar aspect of the joint (rather than the typical location in mid-distal cannon).

Very small, linear cystic lucencies (Figure 18.6a,b) are an occasional finding in the proximal articular margin of P1. Their origin is uncertain. These are considered very low risk for future impact on soundness, although a small proportion can develop associated mild lameness +/– joint inflammation (Figure 18.6c). Their (typically) non-midline location distinguishes them from short incomplete fractures, and they are not considered a risk factor for future P1 fracture (or for progression to larger cystic cavity).

Fetlock sagittal ridge OCD

Sagittal ridge OCD lesions (visible on flexed LM and standing DPa projections) can generally be disregarded as unimportant to future athletic soundness. If there is concurrent presence of joint effusion +/– radiological evidence of activity involving the articular margins of the fetlock more caution may be merited. Rarely, radiological lesions are encountered in the region of the dorsal sagittal ridge that appear to straddle OCD and bone cyst pathologies, with a marked lucency +/– surrounding sclerosis noted on DPa projections (Figure 18.7a,b). These latter lesions are considered medium/high risk in yearlings (and 2 YOs) as they may cause significant career interruption if they become clinically active in early training.

Fetlock dorsal osteochondral fragmentation ('chip' fracture)

Osteochondral fragmentation/chip fracture involving the dorsal articular margin of the fetlock (see also p. 129) is

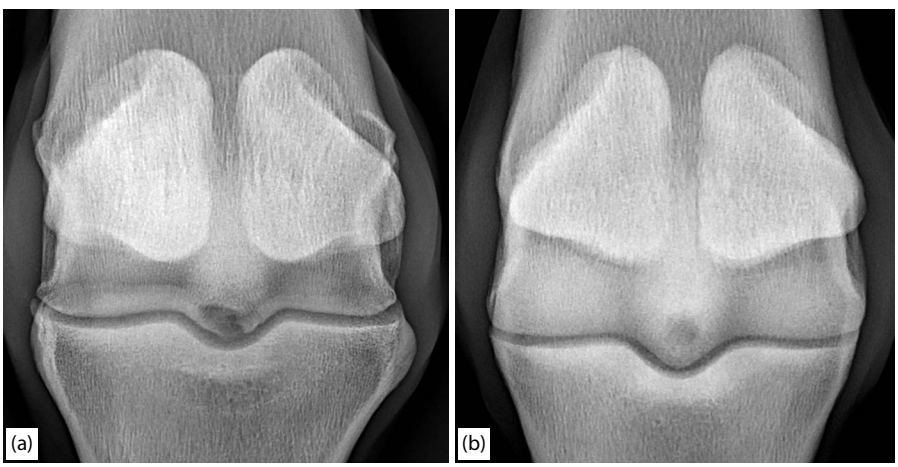


Fig. 18.7 Sagittal ridge osteochondrosis: Extensive radiolucent defects such as marked/irregular OCD-type lesions (a) and those with an appearance more consistent with bone cysts (b) are associated with increased risk of lameness. Fetlock in image (a) also has some active modelling of medial joint margin.

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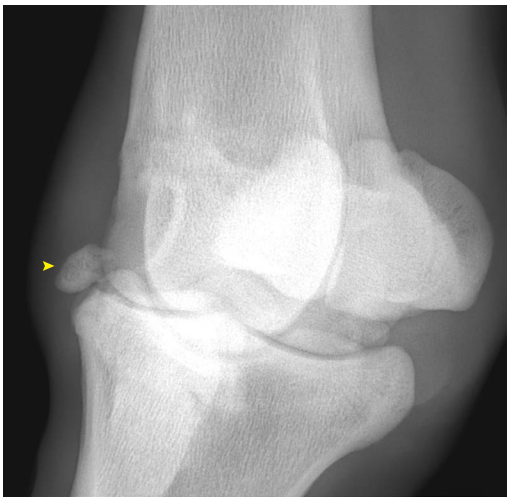


Fig. 18.8 **Large, displaced fragment from dorsoproximal hindlimb P1 of long-standing appearance: radiological blemish but rarely clinically significant.**



Fig. 18.9 **Large plantar process fragment with associated articular defect detected as clinically silent incidental finding in a mature racehorse: similar fragmentation was present in contralateral hindlimb.**

considered very low risk for interfering with future athletic soundness providing there is no concurrent joint effusion or radiological evidence of other degenerative change involving the fetlock. Larger, atypical fragments arising from juvenile paddock accidents (Figure 18.8) are uncommon but are generally clinically silent and unimportant for future soundness. A statistical association between presence of dorsal chip fracture in hind fetlocks and reduced likelihood to race has been reported; however, specific risk of developing lameness has not been quantified and is likely to be very low. The main concern arising from the detection of dorsal chip fracture in yearling radiographs has generally been the potential for the lesion to interfere with resale (rather than the potential to cause a clinical problem); however, attitudes to this are likely to be more lenient in future.

Fetlock plantar osteochondral fragmentation

Osteochondral fragments at the back of the hind fetlock arising from proximal P1 (see also p. 141) or of indeterminate origin are common, and regardless of radiological appearance are typically clinically inapparent and not associated with future unsoundness or effects on racing performance. Even larger lesions arising from juvenile injury to the plantar process with apparent residual articular irregularity (Figure 18.9) are generally benign and in almost all cases do not interfere with training.

Fetlock supracondylar lysis

The underlying cause of the radiological feature described as 'supracondylar lysis' (Figure 18.10) is unknown, and in the absence of good understanding of any underlying

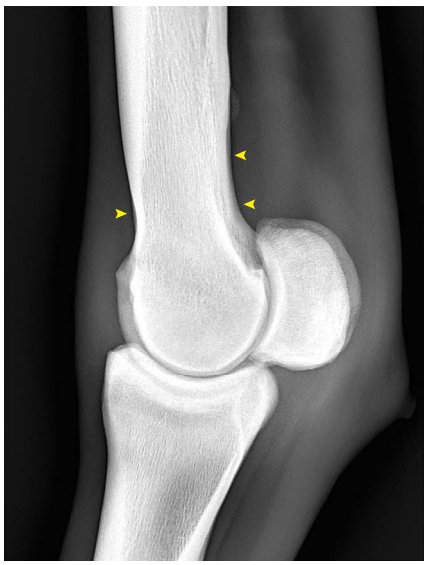


Fig. 18.10 **'Supracondylar lysis': Characterised by relative concavity of caudodistal +/– craniodistal margins of cannon when assessed on LM radiograph.**

pathological process or validated grading of severity, its presence should not generally be given undue emphasis when assessing risk to future trainability. Most affected fetlocks are clinically unremarkable with no prior history of problems, and do not develop associated lameness in training. Unless severe or associated with other radiological lesions (such as arthritic change), it can generally be considered a low-risk finding (but in case of latter, medium-high risk).

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Sesamoiditis

Radiological evidence of sesamoiditis (see also p. 144) is best considered an indirect marker of previous (or potential) pathology at the suspensory ligament branch (SLB)/bone interface rather than as a risk to future soundness per se. Basing pre-purchase assessment on radiological appearance alone without contribution of clinical/ultrasonographic information (including assessment of physique/conformation) is not advised. Radiological features (in combination with clinical appraisal) are a reasonably good indicator of the likelihood of significant SLB pathology and can be used to judge when ultrasonographic imaging should be incorporated into the examination (Figure 18.11). Certain features such as large lucencies and enthesophytes are strongly indicative of prior/existing SLB insertional pathology; however, if ultrasonographic appearance of the associated SLB is satisfactory the risk posed to future trainability is considered to be low. SLB insertional lesions

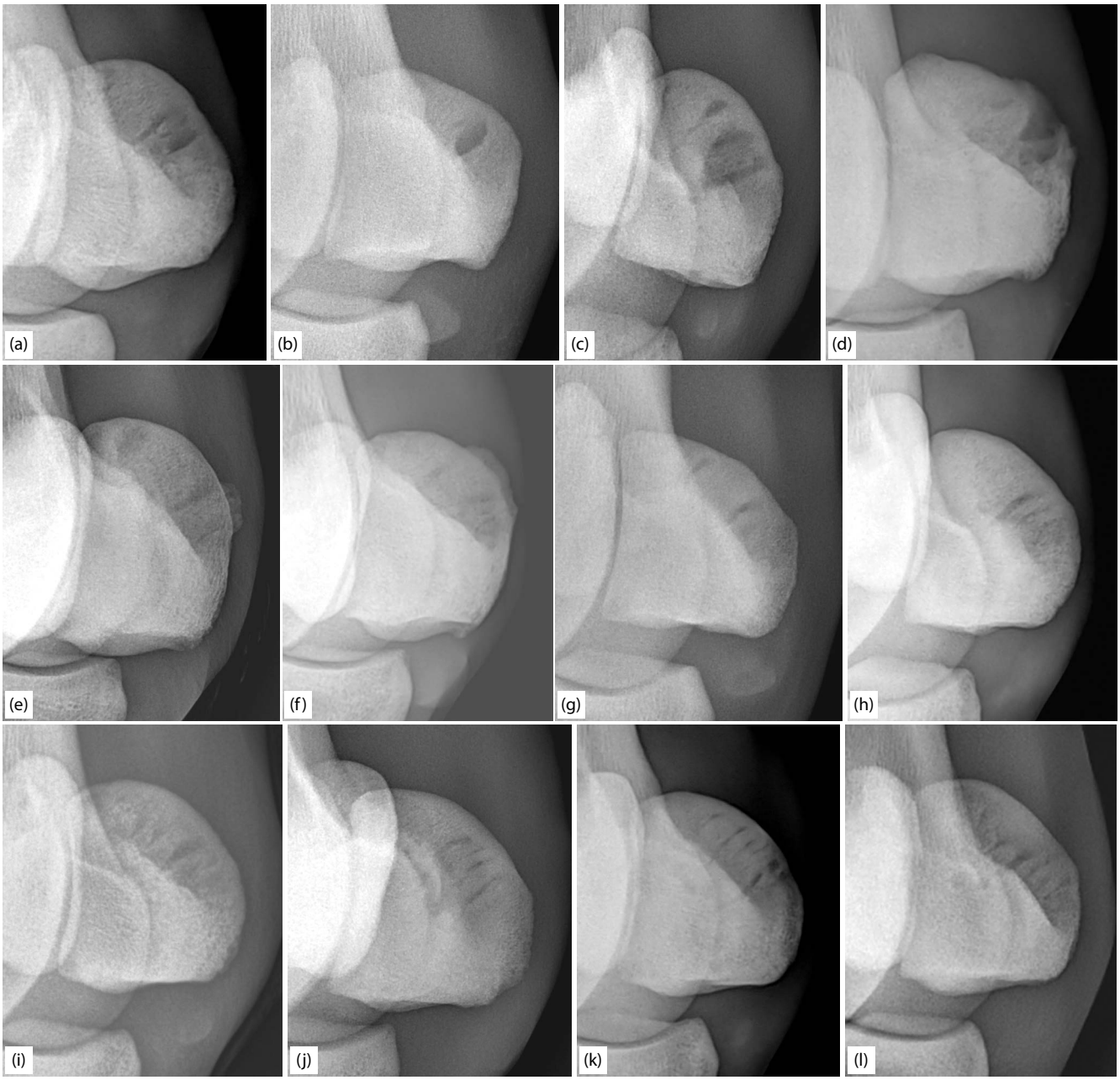


Fig. 18.11 **Examples of radiological appearance of PSB that are (a–f) highly likely, (g–l) possibly, and** *(Continued)*

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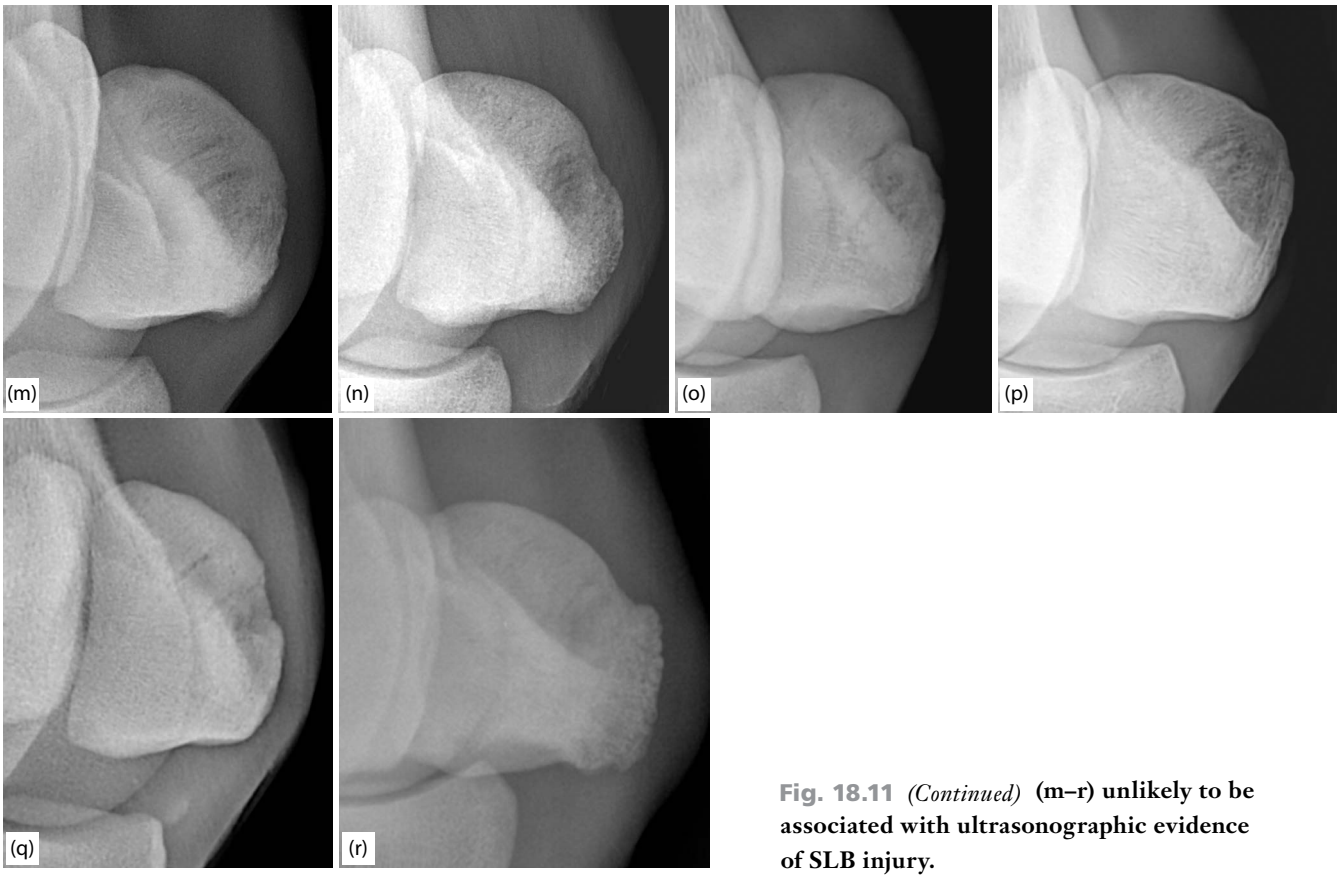


Fig. 18.11 *(Continued)* **(m–r) unlikely to be associated with ultrasonographic evidence of SLB injury.**

may occur in the complete absence of clinical/radiological abnormality but are then generally considered low risk for serious future interruption to training.

Sesamoid fracture

Sesamoid fractures encountered at the time of yearling public auction are typically long-standing, being the result of injury sustained early in life. When fractures have healed fully with bony union the radiological appearance is of an enlarged, elongated or irregularly shaped sesamoid bone (Figure 18.12a–c), and providing the bone margins are smooth and there is no associated SLB pathology the risk of future unsoundness arising from the site is low.

When sesamoid fractures have healed by non-bony union and fragments remain visible, or when bone quality appears suboptimal (Figure 18.12d) there may be a risk of future unsoundness depending on clinical and radiological appearance, location and SLB involvement. Apical sesamoid fractures in hindlimbs (Figure 18.12e,f), if clinically silent, are considered low risk, whereas forelimb fractures can range from low to high risk depending on individual factors. Fetlocks that have undergone surgical removal of apical sesamoid fragments (Figure 18.12g) are judged on an individual basis but are generally considered lower risk than if the fragment remains in situ; however, for all sesamoid fractures (but particularly forelimb) it is not possible to comprehensively assess the risk of future unsoundness until the young racehorse has commenced full training.

Sesamoid: Bone cyst

Bone cysts involving the PSB (p. 139) are very rare. They may involve the axial, dorsal or distal border of the bone and vary in appearance; they may be encountered in both hindlimbs (more commonly) and forelimbs. The origin of these lesions is unknown; some may arise from previous septic pathology, whereas others may be truly developmental. The current evidence base is insufficient to categorize risk to future trainability; it is known that some individuals develop significant lameness while others remain clinically unaffected throughout a racing career.

Middle carpal joint 'spur'/chip/OA

Presence of degenerative changes (including 'spurring' or fragmentation) involving the dorsomedial articular margins of the radial carpal or third carpal bones (p. 177) is

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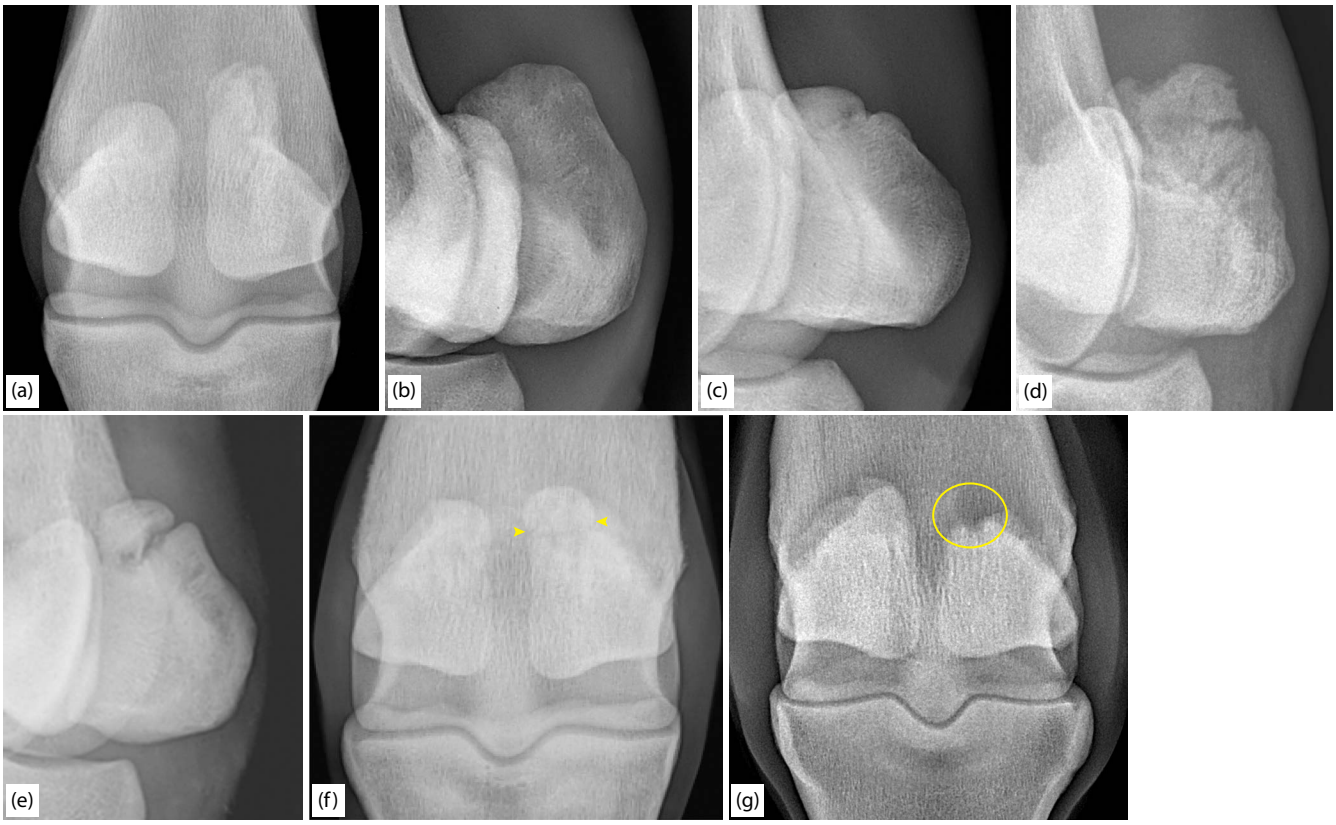


Fig. 18.12 Examples of previous PSB fracture: Elongated lateral PSB (a), enlarged PSB (b) and irregular apical contour (c) of long-standing/settled appearance, all the result of healed foal injuries. PSB in (d) is also the result of a foal injury; however, in contrast to (b) has a highly irregular appearance (and subsequently fractured in pre-training). Long-standing but malunited apical PSB fracture (e, f) in hind fetlocks, and absence or altered profile of apex in horses that have previously undergone surgical removal of fragments (circled) (g) are usually clinically inapparent (prior to commencement of training).

a potentially serious blemish in a yearling. Although it does not necessarily follow that such findings will lead to problematic carpal lameness in training, if the horse has already demonstrated a propensity to develop pathologic change at this important site even before training has commenced then it must be considered a significant risk. Additionally, once carpal degenerative changes are present they will remain evident (and frequently worsen) radiologically over time and may therefore limit opportunities for resale. Degenerative changes occur on a spectrum of severity and some refinement of risk assessment may be possible depending on radiological features that indicate activity/quiescence at the site (Figure 18.13a–d). More than any other anatomical site, relative importance of radiological findings in the middle carpal joint is interpreted in the context of conformation, body type, precocity/maturity and training intentions as these factors strongly influence whether further deterioration is likely (or likely to be significant) under training loads. 'Skyline' projections of the distal carpal row have not traditionally been incorporated in yearling radiographic surveys; however, use of this projection may permit more complete assessment of risk of future carpal pathology.

Modelling/fragmentation involving the more lateral articular margin of the same joint (the intermediate carpal bone) (Figure 18.14a–c) is commonly encountered in yearlings and, providing it does not occur concurrently with radial carpal bone pathology, can be viewed with much greater leniency than the latter.

Other carpal findings

Modelling of the antebrachiocarpal joint is generally considered far less important than of the middle carpal joint, and mild articular lipping/spurring (of either proximal row of carpal bones, or distal radius) (Figure 18.15a,b) is unlikely to represent increased risk of carpal lameness in

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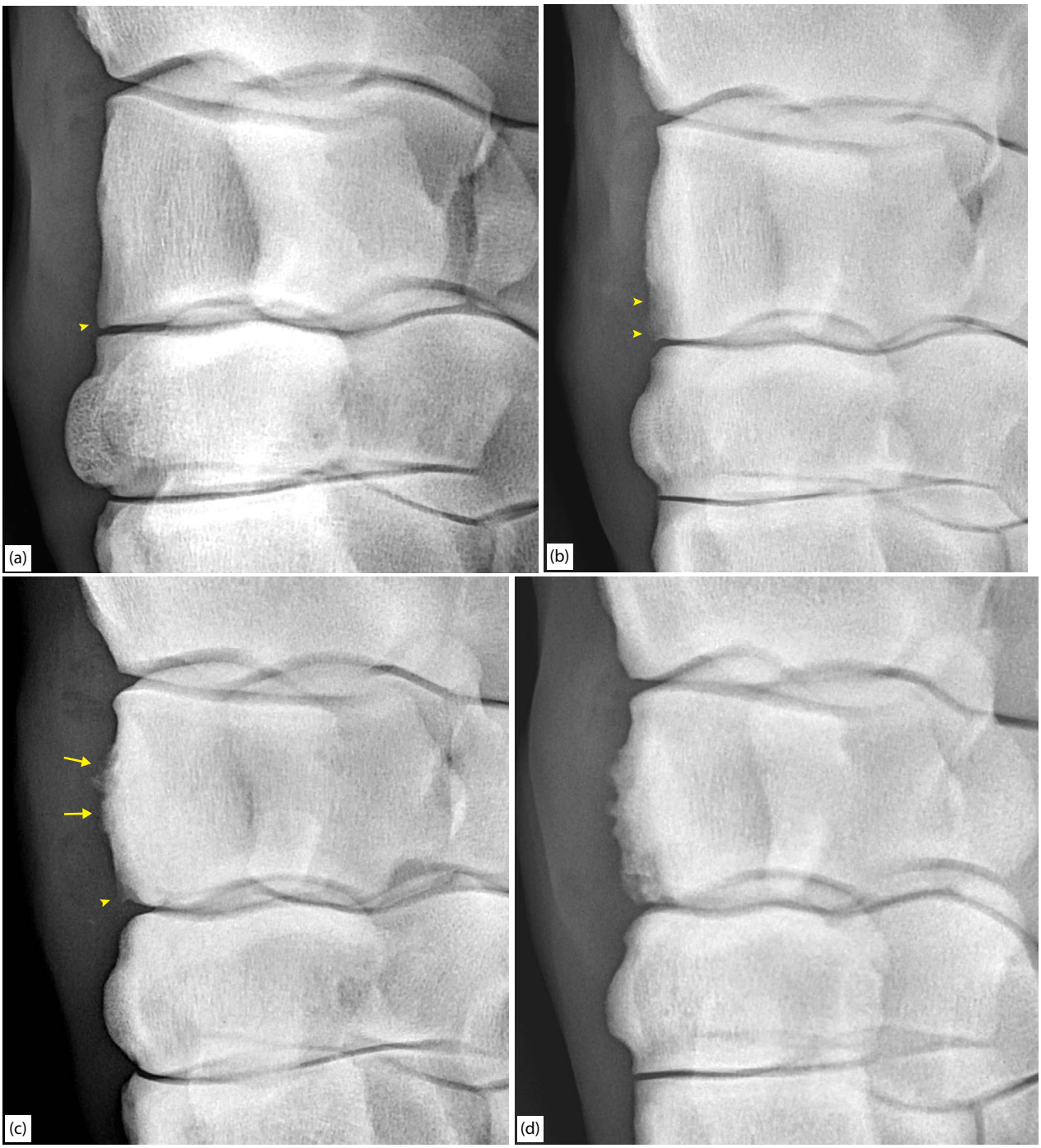


Fig. 18.13 Radiological features (on DLPaMO) of distal radial carpal bone modelling used to assess risk: (a) 'angular' profile but regular bone density through distal quadrant (settled/inactive appearance); (b) lytic appearance with poorly defined outline of bone (consistent with current activity/likelihood of further radiological +/– clinical deterioration); (c) 'cut back' profile with some lysis of margin (arrowhead) but overall increased radiodensity of distal quadrant, also entheseal modelling of dorsal joint capsule attachment (arrows) (consistent with chronic middle carpal joint activity); (d) more advanced changes than in (c), with probable fragmentation of distal articular margin.

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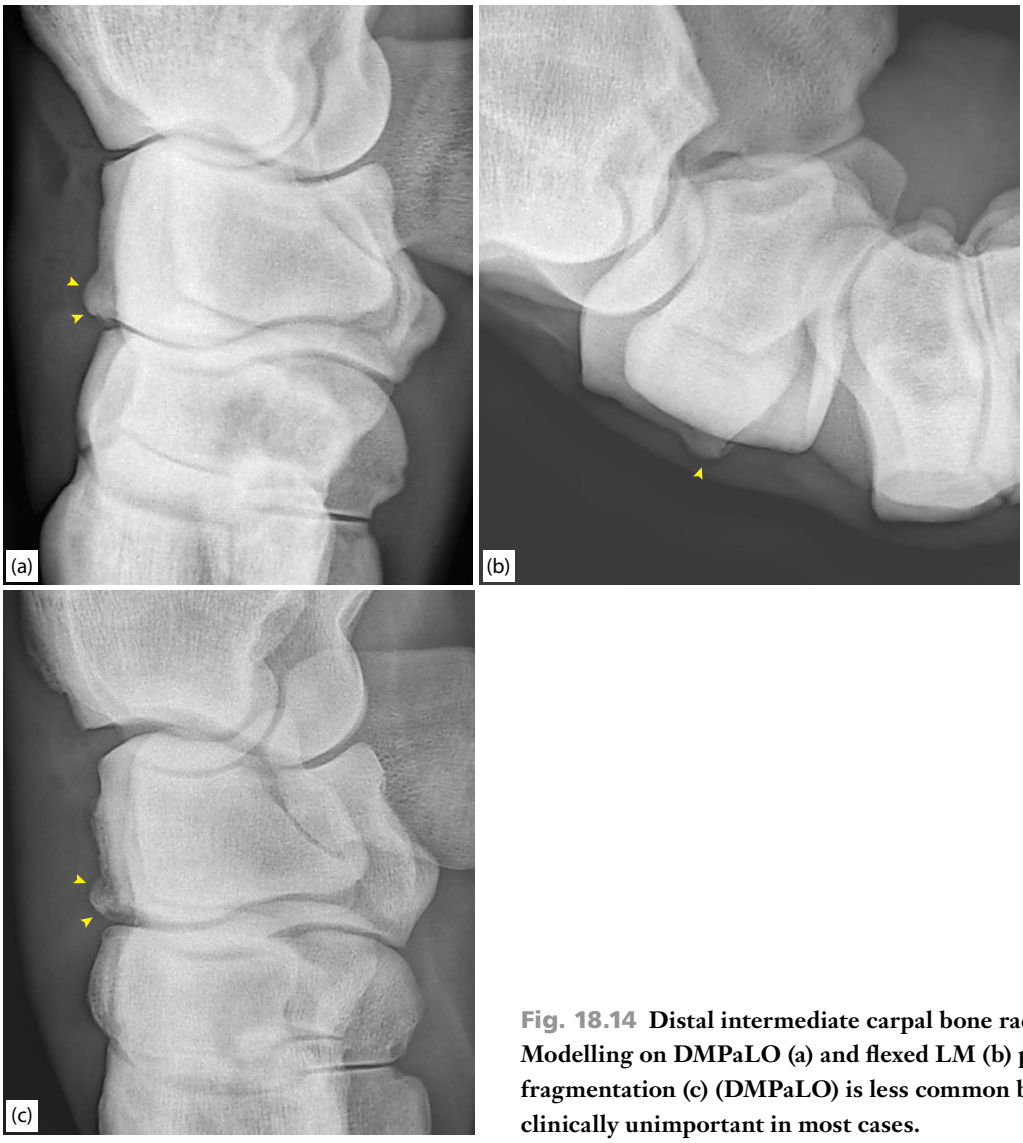


Fig. 18.14 **Distal intermediate carpal bone radiological findings: Modelling on DMPaLO (a) and flexed LM (b) projections; obvious fragmentation (c) (DMPaLO) is less common but also considered clinically unimportant in most cases.**

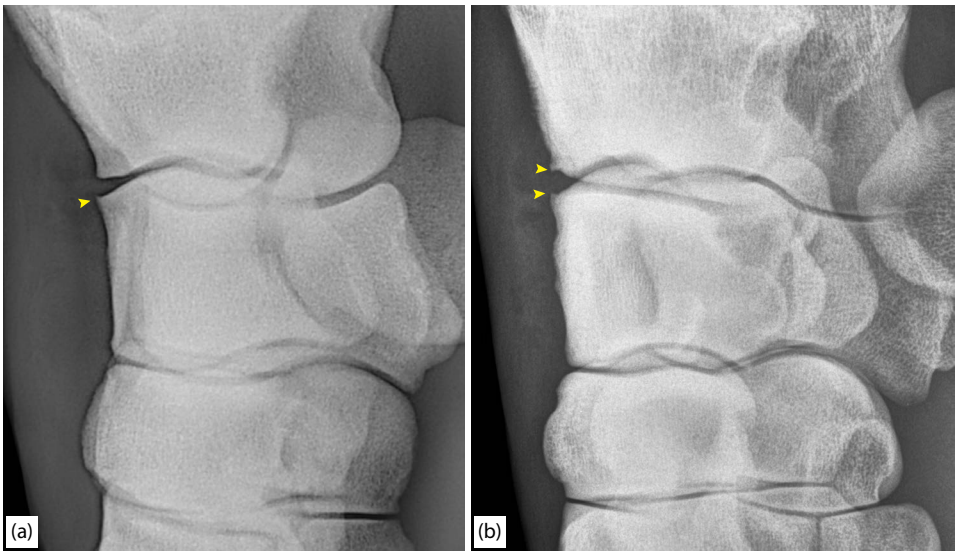


Fig. 18.15 **(a,b) Mild articular modelling ('spurring') of ABC joint.**

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Fig. 18.16 **(a) Ulnar carpal bone cyst; (b) proximal margin fragmentation of accessory carpal bone.**

training. More advanced modelling/fragmentation may sometimes be the result of juvenile paddock injury and is not necessarily of significance, but should be judged on an individual basis.

Bone cysts +/– small osteochondral fragments associated with the ulnar carpal bone (Figure 18.16a) are very common and of no importance for future athletic soundness. Bone cysts in the important medial load-bearing bones (radial and third carpal) rarely cause lameness but may be problematic to manage should they do so. Small fragments from the proximal margin of the accessory carpal bone (Figure 18.16b) are generally insignificant (even when there is concurrent mild articular modelling of the ABC joint), although if associated with carpal sheath effusion should be viewed with caution. Other irregularities at the back of the knee are encountered only rarely and importance should be judged on an individual basis.

Radial physitis

The primary risk posed by radial physitis (Figure 6.148, p. 191) is that until it settles fully (generally judged by the absence of a pain response to palpation +/– radiological resolution) any existing conformational defect will continue to worsen (Figure 18.17), with subsequent potential effects on future soundness (particularly carpal lameness). Judging the likely severity of any final 'incorrectness' is dependent on several factors and relevance will vary between individuals.

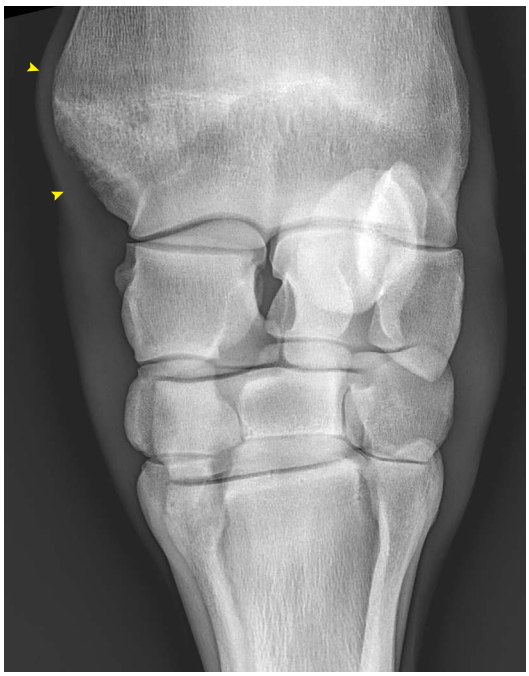


Fig. 18.17 **Radial physitis: Residual (and permanent) prominence of distomedial radius following severe physitis.**

Radial osteochondroma/physeal exostosis ('spur')

True radial osteochondroma (p. 189) is rare in yearlings; more common is a variety of bony protuberances and physeal reactions at the back of the lower radius

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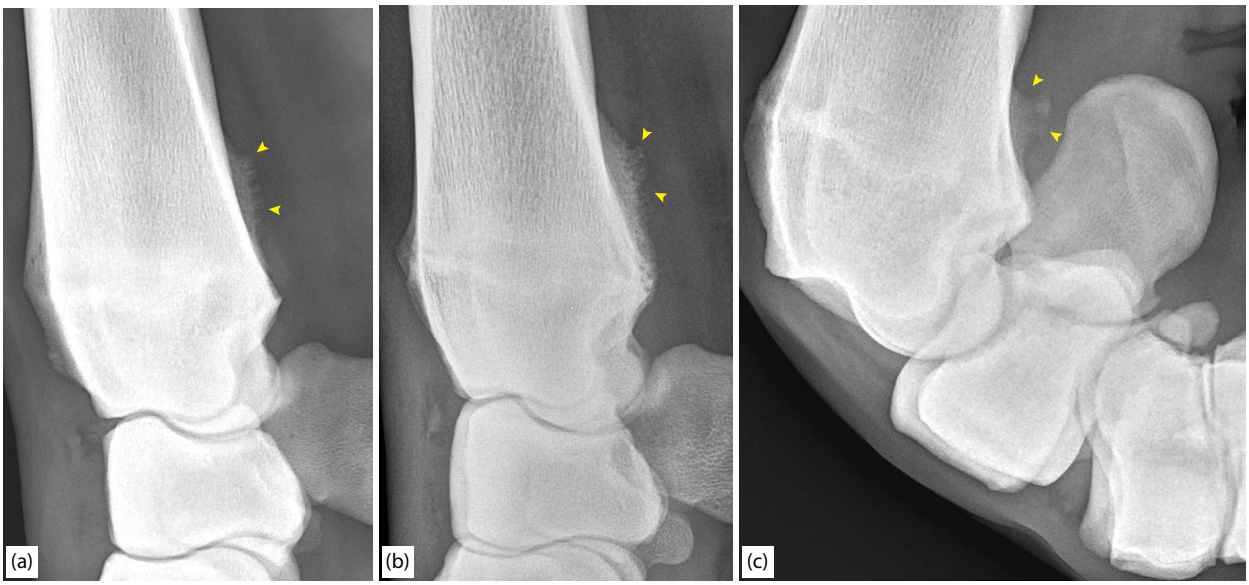


Fig. 18.18 **(a-c) Examples of bone protuberances commonly seen in region of distal radial physis above back of carpus; these are rarely clinically important.**

(Figure 18.18a–c) that should be judged on an individual basis. Generally considered low risk, as most are clinically irrelevant and in the unlikely event that secondary injury involving the soft tissue structures of the carpal sheath should ensue, there is a good surgical solution.

Distal hock joint 'spur'/fragmentation

'Spurs'/osteophytes at the front of the tarsometatarsal (TMT) joint arising from the upper cannon range are very common and range in size from small to very large (Figure 18.19a,b). They are considered clinically insignificant regardless of appearance. Similarly, dorsal fragmentation involving T3 (generally the distal articular margin) (Figure 18.19c) is relatively common and not associated with increased risk of future unsoundness.

Distal hock joint osteoarthritis

Minor osteoarthritic change involving the TMT +/– distal intertarsal (DIT) joint (Figure 18.20a,b) is considered low risk. When the degree of arthritic activity (mixed

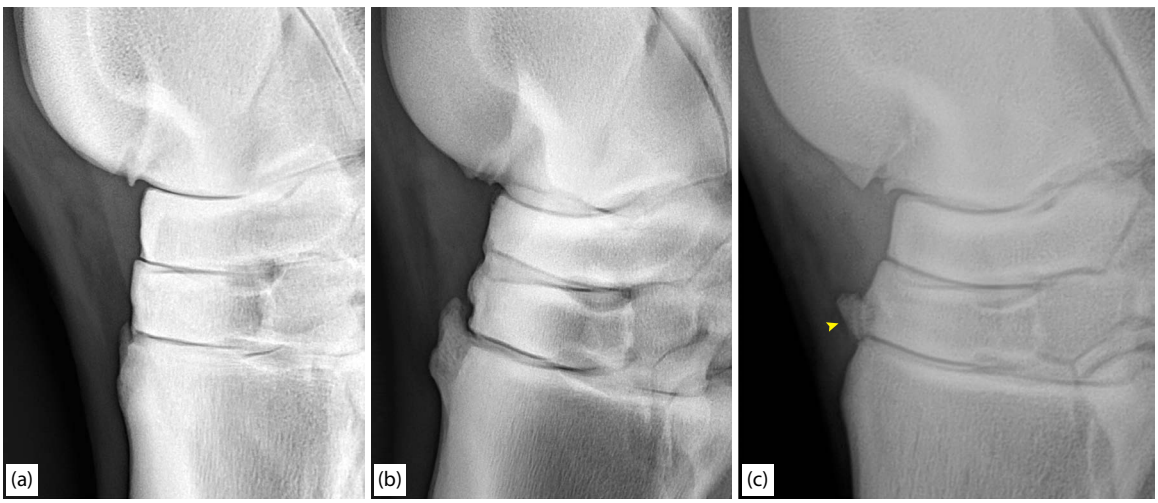


Fig. 18.19 **Proximal metatarsal spurs in the lower hock range from small (a) to large (b) but are almost invariably clinically unimportant regardless of size; (c) dorsodistal fragmentation of T3 is a juvenile injury and similarly of little importance to future athleticism.**

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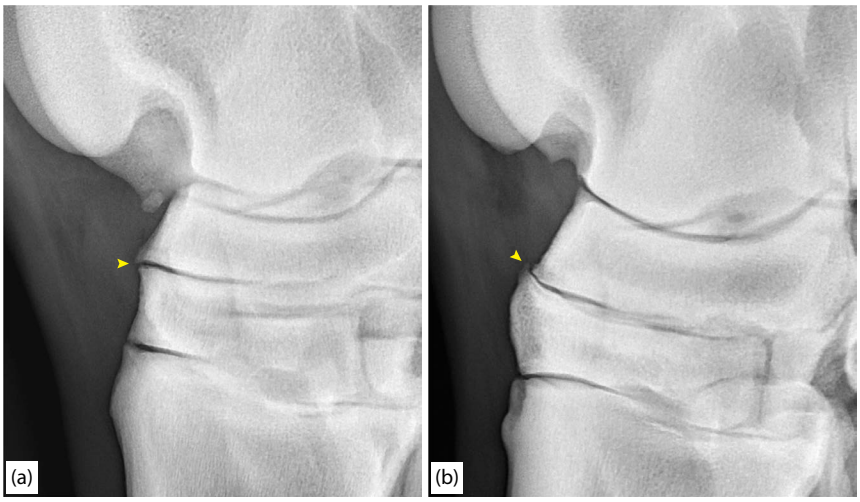


Fig. 18.20 **(a,b) Modelling of DITJ of mild/moderate severity; there is no evidence of more generalised degenerative/ankylosing change along the joint (DMPlLO).**

proliferative and destructive changes) in the DIT joint is marked (Figure 6.170a,b, p. 207), caution should be advised about the potential to develop problematic lameness in training (although this is a rare event).

Third tarsal bone collapse/'wedge'-shape

Mild forms of 'wedge'-shape conformation of the third tarsal bone (T3) are common and can be disregarded (Figure 18.21a). When T3 is more noticeably malformed (Figure 18.21b) it may predispose to slab fracture (p. 208) in training, although the risk of this outcome has not been quantified. Interpretation should account for the possibility that projection angle may influence perception of T3 shape. Horses with markedly collapsed T3 (arising from neonatal period) (Figure 6.175a,b, p. 209) are at medium to high risk for developing career-threatening tarsal lameness or fracture.

Tibiotarsal joint OCD

OCD lesions/fragments are encountered at a variety of sites in the tibiotarsal joint. In yearlings at premier sales it is usually the case that radiological evidence of prior surgery (rather than existing fragments) is noted (Figure 18.22a) and in these cases, providing there is no clinical abnormality, risk to future trainability is low/negligible. When distal intermediate ridge fragments remain in place (Figure 18.22b), risk of future unsoundness remains low although individual assessment should be based on radiological and clinical appearance. Medial malleolar fragments may be difficult to detect (best visualised on D10^{0}LPlM projection) and can occasionally cause joint effusion but rarely career interference. Small fragments at other sites (distal talus; trochlear ridges) (Figure 18.22c) are generally considered unimportant.

Stifle: Bone cyst

Individual refinement of risk to future soundness posed by medial femoral condyle bone cysts may be possible based on radiological appearance, clinical exam (presence/absence of joint effusion) and sometimes history (serial radiographs, or of surgical intervention) provided by vendor; however, in general the future behaviour of cysts should be considered

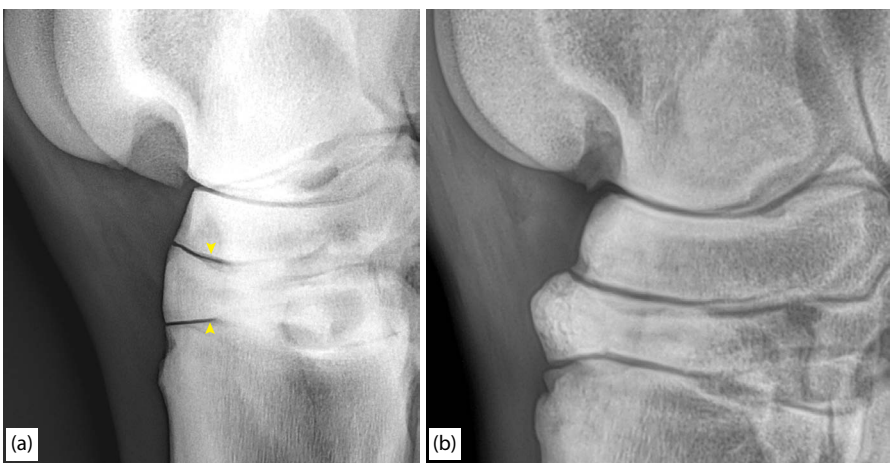


Fig. 18.21 **'Wedge'-shaped appearance of T3 of mild (a) and moderate (b) severity.**

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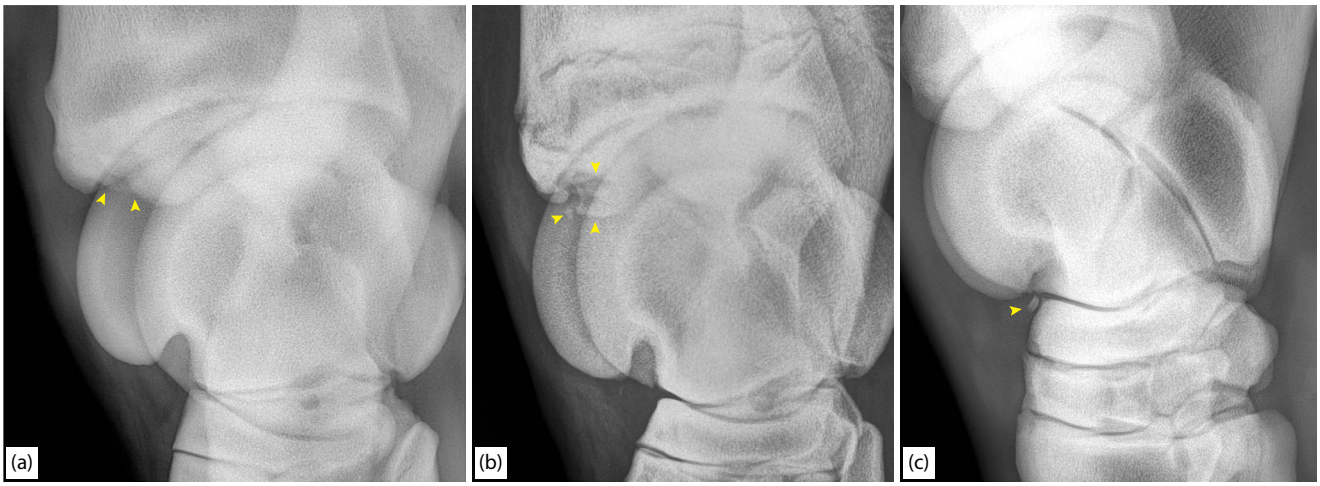


Fig. 18.22 **(a) Irregular articular margin of distal intermediate ridge of tibia of settled appearance (consistent with previous surgery) (DMPlLO); (b) same site with OCD fragments in place; (c) small osteochondral fragments in vicinity of distal talus are clinically unimportant.**

unpredictable (at least until a horse has completed its first cycle of full training). Radiological features subjectively associated with higher risk of future lameness include broad articular involvement, marked sclerotic change in the surrounding bone of the medial femoral condyle and narrowing or modelling of the joint space (Figure 18.23a; also Figure 6.196, p. 230). Radiological features consistent with resolving pathology (Figure 18.23b) are viewed more leniently. Statistical risk of developing lameness has not been quantified but is likely to be low; should a cyst become a clinical problem the risk is primarily of interruption/loss of 2 YO season (rather than career-ending). Detection of stifle cysts can be impaired if radiographs are underexposed (Figure 18.23c,d).

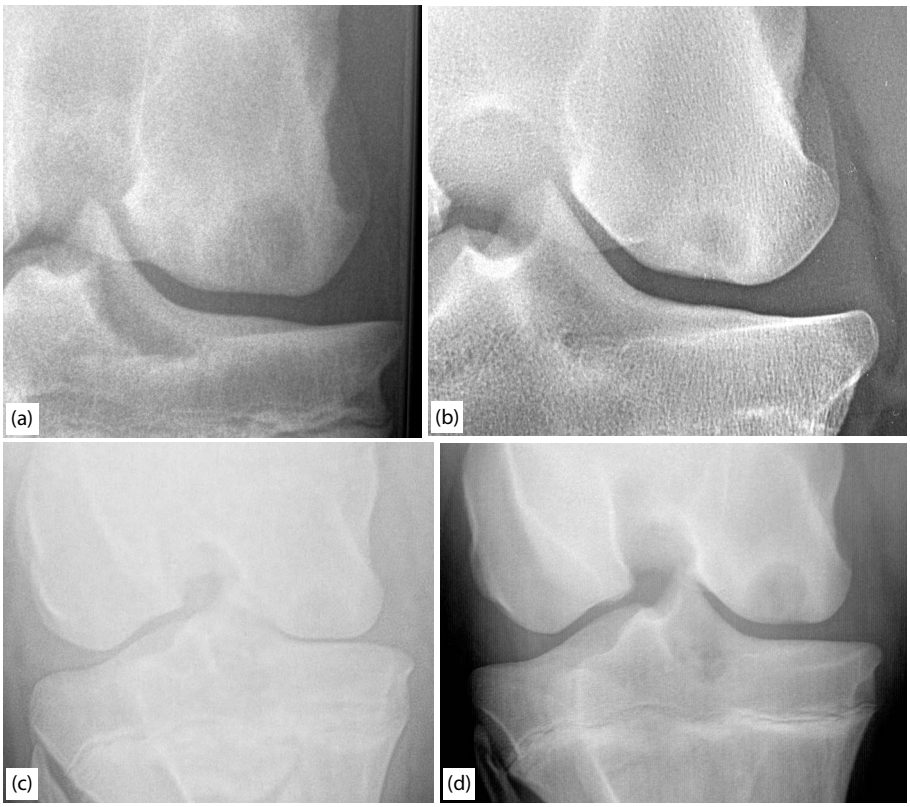


Fig. 18.23 **Radiological features of stifle cysts used to assess risk: (a) large/indistinct with apparent broad articular involvement; (b) more regular trabeculation throughout medial femoral condyle, and smaller articular involvement. Radiographic quality important for detection/assessment: Cystic defect is poorly differentiated in underexposed radiograph (c) but clearly defined with appropriate exposure (d).**

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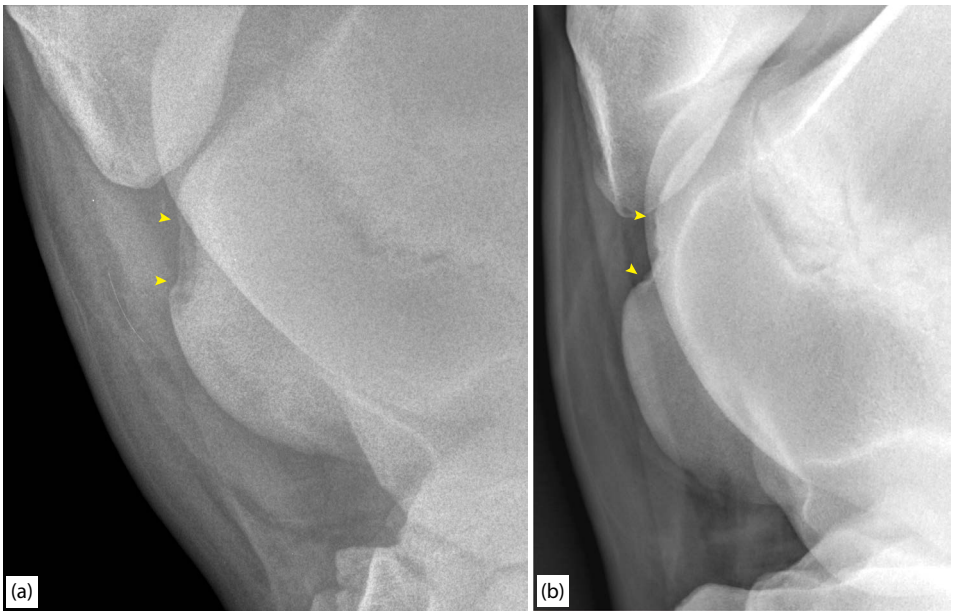


Fig. 18.24 **(a,b) Defects in proximal portion of lateral trochlear ridge of stifle, consistent with previous surgery to remove OCD fragment(s).**

Stifle: Shallow medial femoral condylar lucency

Shallow (<3 mm) lucencies in the medial femoral condyle (Figure 6.197, p. 231) are a relatively common finding. As for stifle cysts some radiological features (depth, apparent articular defect, surrounding bone appearance) may permit subjective refinement of risk assessment. While behaviour of shallow lucencies is unpredictable, only a small proportion (<5%) of affected horses subsequently develop a bone cyst at the same site.

Stifle OCD

When a yearling has previously undergone surgery to remove stifle OCD lesions it remains obvious radiologically throughout life (Figure 18.24a,b); providing the surgical site appears settled and is not extensive, the risk of developing associated problems in training is low/negligible. Horses with small fragments that have not been treated surgically are generally also low risk; however, more extensive lesions +/– clinical evidence of joint effusion are associated with increased risk of developing lameness and interruption to training.

PRE-PURCHASE ULTRASONOGRAPHY

Ultrasonographic assessment of the forelimb cannon/fetlock region is commonly included in pre-purchase examinations of horses-in-training and high value yearlings. Sensitivity for detection of previous/current tendon and SLB injury is high; interpretation of proximal suspensory ligament appearance is more subjective, but major lesions are usually readily apparent. Examination of other areas (e.g., pelvis) may be indicated if clinical abnormality or reported history demands it. Ultrasonography is commonly incorporated into yearling inspections to assess integrity of SLB–sesamoid bone interface if radiological evidence of sesamoiditis is present, and is integral to full risk assessment of horses with both clinically silent and clinically active SLB injuries (p. 146). Subtle ultrasonographic irregularities (Figure 6.96a–d, p. 149) and normal variants (Figure 18.25) are common, and in the absence of clinical/radiological abnormality are generally not considered to influence future injury risk. Such SLB ultrasonography is well suited to yearling public auctions as it can generally be conducted rapidly in the unsedated animal.

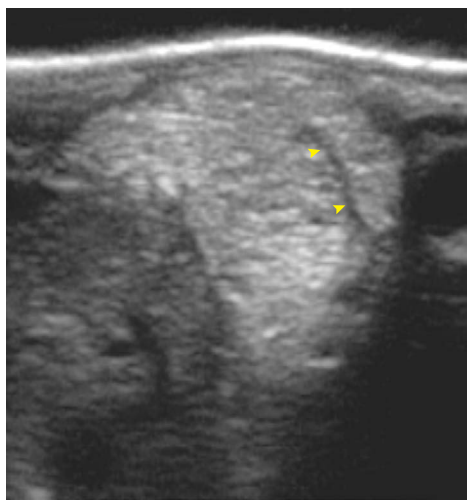


Fig. 18.25 **Normal anatomical variant in palmar insertional portion of SLB resulting from divergent bands of fibres (transverse ultrasonogram); does not represent injury.**

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Satisfactory image acquisition is usually possible without clipping the limb. Sequential cross-sectional and longitudinal images of the major structures of the palmar cannon (flexor tendons, inferior check and suspensory ligaments) and of the SLBs are obtained of both forelimbs and assessed for symmetry, size, echogenicity and fibrillar pattern. Particular care should be taken to assess the most important sites of injury: superficial digital flexor tendon (SDFT), suspensory ligament origin (proximal metacarpus) and SLB insertion.

ASSESSMENT OF AIRWAY FUNCTION

Physical examination/history

- Vendor declaration on unsoundness of wind or previous upper airway surgery should be sought.
- Careful palpation of throatlatch for detection of surgical scars, congenital laryngeal abnormalities and laryngeal muscle atrophy. External scars are generally very difficult to detect.

Resting endoscopy

- The upper airway conditions of greatest importance to the racehorse (Chapter 8, p. 277) are dynamic in nature and cannot be fully assessed at rest, as function at racing speeds is the determinant of any potential impairment to athletic performance.
- Despite this resting endoscopy is still the mainstay of respiratory tract assessment at public auctions and most private horse-in-training examinations. Useful for detection of recurrent laryngeal neuropathy, congenital abnormalities and arytenoid chondritis, previous laryngeal and aryepiglottic fold (but not palatal) surgery. Not useful for palatal, pharyngeal or aryepiglottic fold function. Significance of resting endoscopic findings in the yearling are summarised in Table 18.6.
- Laryngeal function is graded according to symmetry and synchrony of movement of the arytenoid cartilages (p. 280); observation of full range of cartilage movements assisted by inducing swallowing or nasal occlusion.
- While there is significant intra- and inter-observer and intra-horse variability for exact laryngeal function grade, agreement is good when simply determining whether 'meets conditions of sale' or 'doesn't meet conditions of sale'. Overall, the sensitivity and specificity of resting endoscopy for predicting laryngeal function at exercise is excellent.

Table 18.6 **Diagnosis and pre-purchase interpretation of upper airway conditions in yearlings**

CONDITION	DETECTABLE WITH RESTING ENDOSCOPY?	SIGNIFICANCE OF RESTING ENDOSCOPIC FINDINGS FOR FUTURE PERFORMANCE
RLN	Yes	Good statistical correlation between resting grade and likelihood of future impact on career; however, refinement of risk is enhanced with dynamic endoscopy. Small proportion of 'outliers' from both ends of spectrum with contradictory resting/dynamic function (Figure 8.7, p. 285).
DDSP	No	Displacement/replacement of palate occurs regularly as normal feature during resting endoscopy but cannot be used as predictor of performance-limiting DDSP at exercise.
VLAC	Frequently yes (with sufficient stimulation/nasal occlusion); may evade detection without sustained stimulated abduction.	Dependent on severity; mild cases with good corresponding arytenoid function unlikely to affect performance. Many cases detectable with resting endoscopy if stimulation is sufficient; however, dynamic endoscopy may be required for full assessment.
MDAF	No	Not detectable with resting endoscopy.
Chondritis	Yes	Dependent on severity.
Epiglottic structure	Yes	Not significant (unless markedly abnormal).
4-BAD	Yes	Highly significant. May have racing career but highly unlikely to be good performers.
Pharyngeal collapse	Yes/maybe	Dependent on severity. Dynamic endoscopy required for full assessment.
PLH	Yes	Not significant: normal feature of maturation.

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- Horses that have previously undergone laryngeal re-innervation surgery continue to have observable laryngeal dysfunction at rest, and the success of treatment can only be assessed with overground endoscopy.
- Sedative drugs are in common use at public sales as behaviour-modifying agents and have the potential to temporarily weaken observed laryngeal function.
- Epiglottic appearance (size/flaccidity/convexity) may vary considerably with maturity and between rest and exercise and is generally a poor predictive aid for future dynamic upper airway obstruction. Conflicting evidence of significance in literature; however, some associations between severe grades and dorsal displacement of the soft palate (DDSP)/medial deviation of the aryepiglottic fold (MDAF) reported and as a result markedly flaccid or short epiglottis usually viewed with caution in the untrained yearling.

Videoendoscopy

- Availability of recorded resting endoscopy of upper airway (typically provided by clinician acting for vendor) is commonplace at public auction.
- To be considered of diagnostic quality, recordings should include identification of horse, permit visualisation of the entire larynx, be of sufficient duration and display full range of arytenoid movements (both resting, and abduction induced by swallowing/nasal occlusion). It is recommended that length of time of direct observation of larynx should be not less than 45 seconds. Recommended guidelines can be found in Table 18.7.
- Throats that are 'fixed' open may indicate use of exercise (high respiration rate evident) or other means (e.g., nasal twitch, auditory stimulation) to stimulate laryngeal dilation and are problematic to interpret, as are videos in which arytenoid cartilages are not permitted to relax fully (e.g., introduction of flush fluid/stimulation of swallowing whenever cartilage begins to adduct). Accepted laryngeal grading systems all necessitate observation of arytenoid movement and resting laryngeal symmetry, and absence of 'relaxed' phase of laryngeal movement from video is a significant contributor to discrepancies between pre- and post-sale interpretations of function.

Table 18.7 **Recommended pre-sale videoendoscopy guidelines**

Timing of examination	Recommended time interval between video acquisition and first day of sales session in which horse sells: ≤10 days
Horse identification	Horse identification, date of examination, name of veterinary surgeon/practice embedded/on-screen digital annotation
Image quality	Video recording should comprise a single, unbroken and unedited stream of footage with good (bright, clear) image quality.
Endoscopic technique	Either nostril may be used for introduction of endoscope; however, endoscope should be maintained centrally in nasopharynx to minimise parallax error when assessing the laryngeal cartilages. Orientation of laryngeal image should be vertical at all times. Clear, unobstructed visualisation of entire epiglottis, arytenoid cartilages and vocal cords/ventricles at some point during recording. Endoscope should be advanced close enough to larynx to allow thorough assessment of the mucosal surfaces (for detection of ulceration/chondritis). Visualisation of larynx not only at maximal abduction but also during periods of full relaxation is strongly recommended so that the full range and synchrony of arytenoid movements can be assessed. Footage that only records fully abducted 'fixed open' arytenoid position, or endoscopic techniques that prevent relaxation of the larynx may be unacceptable to examining clinicians. Minimum 45 seconds (preferably 60 seconds) of footage of direct visualisation of larynx (i.e., exclusive of passage of endoscope through nasal passages): this time should be used constructively to record the full range of arytenoid movements. Full arytenoid abduction may be demonstrated by variety of means including stimulation of swallowing via introduction of flush solution or nasal occlusion. It is recommended that footage contains good visualisation of at least 3 swallows. For horses with resting arytenoid asymmetry, nasal occlusion is preferable to allow better assessment of ability to sustain abduction and possible presence of arytenoid apex collapse/ventroaxial luxation of corniculate process of arytenoid cartilage (VLAC).

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- Non-diagnostic videoendoscopic recordings justify endoscopic reappraisal.
- Endoscopic reappraisal also warranted for 'intermediate' grades of laryngeal function, as ability to directly observe and stimulate arytenoid movements can allow for risk assessment more specific to purchasing client's needs than might be possible from video recording.
- It follows that vendors should expect (and should be encouraged to permit) requests for permission from examining clinicians to undertake endoscopic re-examinations in a proportion of animals, for a range of reasons including laryngeal grade, client risk parameters and video quality.

Wind testing

- Presence and character of abnormal respiratory noises not necessarily specific for type of respiratory obstruction (p. 279).
- Any use of tack that might affect upper airway function (noseband/tongue-tie) should be noted.
- Correlation between respiratory noise made on the lunge and that made when exercised in straight lines has been incompletely investigated.
- A horse that makes an inspiratory noise in both directions on the lunge is no more likely to have poor laryngeal function than one that makes a noise in only one direction.

Laryngeal ultrasonography

- Objective assessment of intrinsic laryngeal muscle atrophy/degeneration (p. 283).
- May be used as adjunct to resting endoscopy to determine significance of 'intermediate' grades of laryngeal function, but generally has poor predictive value for future laryngeal function in most young horses and used infrequently.

Overground endoscopy

- Used routinely in situations in which concern exists over resting laryngeal function/respiratory noise/previous respiratory surgery/racing form.
- Success in determining the true nature/severity of any dynamic upper airway obstruction depends on the endoscopic examination being conducted at near-racing speeds (and arguably distances) to replicate upper airway pressures and fatigue that can lead to loss of pharyngeal/laryngeal stability.

LARYNGEAL FUNCTION AND PRE-PURCHASE ASSESSMENT

- **Can a yearling with good laryngeal grade at resting endoscopy fail a wind test?** Yes. Small proportion of yearlings with 'good' resting grades exhibit some dynamic collapse with overground endoscopy.
- **Can a yearling with poor laryngeal grade at resting endoscopy pass a wind test?** Yes. Approximately 15% of horses with Havemeyer grade 3, and 2% of horses with Havemeyer grade 4 throats exhibit good dynamic laryngeal function.
- **Does overground endoscopy always give definitive answers about current or predicted laryngeal function?** No. While overground endoscopy permits far better assessment of overall throat function than is possible with resting endoscopy, there may still be significant variability over time and between assessments (particularly in young horses).
- **Can a horse with abnormal (resting +/– dynamic) laryngeal function have a successful career?** Yes, however, they are likely to earn far less over a career than horses with 'normal' and 'intermediate' grades. Examples exist of horses with poor laryngeal function becoming elite performers but they are statistically far less likely to do so. They are also more likely to undergo future throat surgery.

PUBLIC AUCTION VETTINGS: THE YEARLING

Pre-bid inspections of yearlings at public auction are often undertaken under challenging circumstances due to constraints of time and convention. Examinations typically consist of brief physical inspection in the stable followed by observation at walk (examination at trot is not generally accepted practice and reserved for horses with radiological lesions or suspected lameness).

Physical examination

Particular attention to enlargement of PSBs (plus deep palpation of SLB insertions), distal radial growth plates and effusion of important high motion joints (including DIPJ). Major conformational faults or physical traits that might affect trainability are noted. Palpation of the throat may reveal the likely presence of congenital laryngeal abnormality (4-BAD, p. 296). Opportunities to observe action are limited and importance is placed on stride length and indicators of neurological dysfunction/incoordination when turned, as well as foot placement and balance.

Airway function

Upper airway is frequently inflamed +/– pharyngeal lymphoid hyperplasia (p. 294): may obscure full visualization of

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abducted arytenoid cartilages; intermittent displacement of the soft palate is common. Attention should be paid to mucosal lesions indicative of arytenoid chondritis (p. 294).

Radiography

[See p. 388]. Particular attention should be paid to the following:

- Developmental orthopaedic disease (osteochondrosis: OCD/bone cysts): significance varies with location and appearance.
- PSBs: fractures and sesamoiditis.
- Articular modelling of important high motion joints (middle carpal joints/fetlocks).

Ultrasonography

Useful to assess SLB–sesamoid interface for subclinical ligament injury; clinical and radiological assessment (Figure 18.11) can be used to select individuals that should be scanned. 'Juvenile' SDFT tendinitis (p. 158) is a common finding and importance is dependent on tendon dimensions and destination jurisdiction. Tendon dimensions should be considered permanent, i.e., tendons enlarged due to 'juvenile tendinitis' do not return to 'normal' size regardless of management/maturity.

PUBLIC AUCTION VETTINGS: THE 'BREEZE-UP' 2 YO

'Breeze-up' ('ready-to-run') auctions differ from other formats in that the horses being offered for purchase are unraced 2 YOs that must complete a short fast-exercise 'breeze' prior to sale. Although untested on the racetrack, these horses have undergone a full training preparation and consequently many of the unquantifiable risks attached to imaging/physical defects seen in yearlings (particularly developmental findings such as osteochondrosis lesions) can therefore be interpreted with greater objectivity. There is a greater likelihood of encountering both adaptive changes (such as carpal modelling) and training-related injuries: the presence of some of these is not necessarily viewed negatively, and the likely/desired racing jurisdiction and purchasing client's goals and risk profile play a large role. Horses presenting with stress injuries or dorsal metacarpal disease ('sore'/'bucked' shins, p. 165), or even lameness of unknown origin (where it is presumed a stress injury is responsible) may be acceptable if client's expectations for the horse can allow a rest period, as the majority of these conditions heal fully and carry an excellent prognosis for return to full soundness. Training loads during preparation for these sales is sufficient for horses to develop fetlock fatigue pathology, and condylar prodromal/unicortical fissure fractures (p. 110) are regularly encountered at radiographic screening.

Physical examination

Unlike at yearling auctions, examination of action at trot is an accepted practice. Track conditions for the breeze may influence subsequent physical examination findings in some cases. Sore shins are common in breeze-up 2 YOs: may be associated with lameness and thereby complicate interpretation of physical findings. Of little long-term significance but may interfere with training in immediate post-sale period. Hindlimb lameness due to tibial stress injury may also manifest following fast exercise.

Airway function

Format of breeze-up auctions permits observation at exercise and assessment of respiratory noise. Endoscopy/video endoscopy is used for resting laryngeal assessment as for yearlings.

Radiography

Training adaptations should be expected and give a better indication of whether a horse may be troubled by future orthopaedic problems at certain sites (carpus, fetlock) than is possible at yearling stage. This also allows better risk assessment for suitability to train/race in certain jurisdictions. Particular attention should be paid to the following:

- Carpus: middle carpal joint changes (if present) can be expected to progress with further training. May have implications for resale later in career and may be predictive for future carpal lameness in training.
- Fetlock: active joint margin modelling is not uncommon; determine significance (for training and resale) on individual basis. Sesamoid bone changes (sesamoiditis, old fragments/fractures) can generally be viewed much more leniently than at yearling stage if clinically silent.
- Tibia: distal tibia may be visible on LM projection of hock and endosteal/periosteal callus of caudodistal cortex (p. 224) may indicate presence of stress injury.

Ultrasonography

Clinical findings, value of horse and risk profile of purchasing client determine whether ultrasonographic assessment is incorporated into the vetting. Unlike 'horse-in-training' auctions in which some time may have elapsed between last fast work/race and sale, all breeze-up 2 YOs will have galloped during the sale and successful

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masking of signs of soft tissue injury is therefore less likely. Clinically apparent SLB injuries can be scanned to give more precise risk assessment, although clinical findings are the best guide to short-/medium-term risk posed by such lesions. Sub- or mildly clinical SLB lesions are generally of less importance (as the horse has demonstrated capacity to train to race speeds without developing clinical injury). Ultrasonographic assessment of the proximal suspensory ligament (and main body of ligament) is useful as chronic lesions at these sites may occur with minimal clinical abnormality noted at the time of sale.

PUBLIC AUCTION VETTINGS: THE HORSE-IN-TRAINING

Level of fitness and recent racing form are of considerable importance for interpretation of findings. It is possible to be less critical of certain imaging/clinical faults in a fit horse that has run recently than in a horse unraced for some months, and in fact judging the full significance of the latter for future soundness is rarely possible. Many significant injuries (including SDFT tendinitis and stress fractures) can be difficult to detect clinically after even short periods of rest and/or anti-inflammatory measures and imaging is important to mitigate risk in horses that have not had a racetrack appearance for more than 2–3 weeks prior to the date of examination.

Physical examination

Examination at trot is an accepted practice; action should be considered in the context of level of fitness/excitability. Recent lameness may be diminished/abolished with short periods of rest or intra-articular medication prior to sale. Particular attention should be paid to joints, tendons and ligaments, especially unilateral effusion/thickening of any structure. Also assessment of feet for evidence that they may have been problematic in past or may require particular attention in future (use of composite materials/atypical or remedial shoeing/foot imbalances/hoof wall defects or cracks).

Airway function

Particular attention should be paid to endoscopic evidence of previous surgical intervention (larynx/aryepiglottic folds); palpation of throatlatch region for surgical scars (often subtle)/laryngeal muscle atrophy.

Radiography

Requirement for pre-purchase radiography is dictated by needs of buyer and/or intended racing jurisdiction. Particular attention should be paid to the following:

- Carpus: middle carpal joint changes are common and are considered in the context of clinical findings. Assess for possible future interruptions to training arising from chip/slab fracture rather than previous activity. Long-standing osteochondral fragmentation (Figure 18.26) or arthritic change of settled appearance may be acceptable in absence of clinical abnormality.
- Fetlocks: long-standing osteochondral fragmentation or arthritic change of settled appearance (Figure 18.27) may be acceptable. Assessment for fetlock subchondral pathology is arguably the most important aspect of imaging the horse in training at public auction. Problematic subchondral lesions or prodromal/unicortical condylar fissure fractures (p. 110) are reliably detectable only with 'flexed dorsopalmar/plantarodorsal' projection (Appendix 4) and multiple projections should be taken if concern exists over possible presence of lesion. Increased lateral condylar density of hind fetlocks (Figure 18.28) is a relatively common feature and although likely representative of subchondral adaptation/pathology, is not necessarily viewed negatively (particularly if symmetric, and clinical appraisal satisfactory). Evidence of prior injury (metacarpal/tarsal condylar or P1 fracture) may include surgical implants or residual bone 'scar' from conservatively managed injuries (Figure 18.29a,b), and importance of these to future soundness is assessed on an individual basis; residual



Fig. 18.26 Long-standing distal radial carpal bone chip fracture (in this case was clinically silent/unimportant).

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articular defects (particularly following condylar fracture) can be associated with ongoing lameness. Other features occasionally noted on fetlock screening (supracondylar lysis, Figure 18.10; 'flat' palmar metacarpal condyle(s), Figure 18.30) are generally unimportant unless concurrent lameness/joint inflammation is present. Modelling of dorsoproximal P1 (Figure 18.31) is generally considered an incidental finding of no concern but if associated with focal lucency or thickening of subchondral bone plate (or lameness) may indicate presence of previous/current



Fig. 18.27 **Dorsal articular modelling ('lipping') of fetlock (DLPaMO).**



Fig. 18.28 **Increased radiodensity of lateral metatarsal condyle (circled) in hindlimb fetlock, typical of many horses in training and frequently not of clinical importance (flexed DPl).**

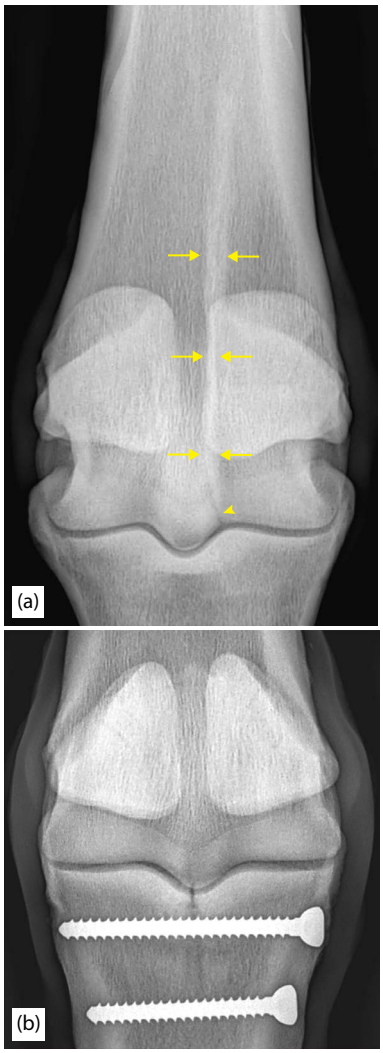


Fig. 18.29 **(a) Linear radiopacity (arrows) and residual articular defect (arrowhead) in hindlimb cannon associated with previous metatarsal condylar fracture; (b) residual defect at proximal articular margin of surgically repaired P1 fracture.**

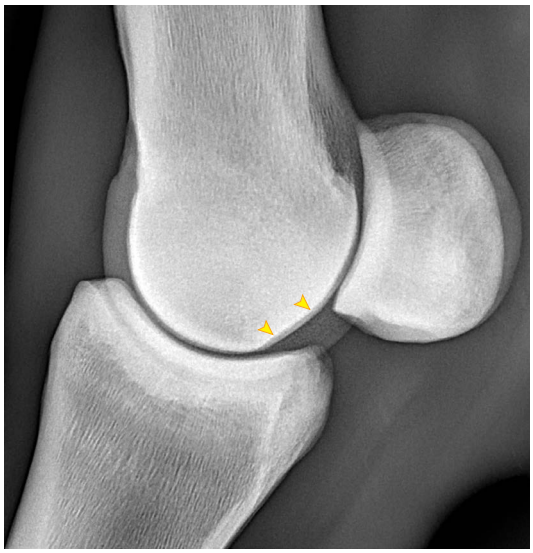


Fig. 18.30 **'Flat' profile to palmar metacarpal condyle(s) on LM projection.**

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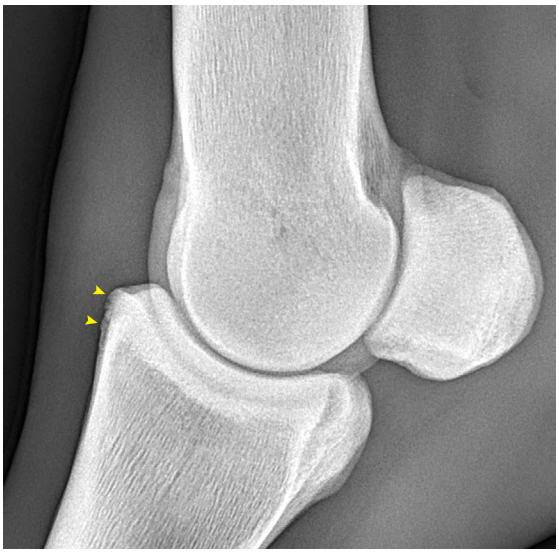


Fig. 18.31 Modelling of dorsoproximal P1 in hindlimb fetlock.

short incomplete P1 fracture. Latter is difficult to confirm in pre-purchase context.

Ultrasonography

Clinical findings, value of horse and risk profile of purchasing client determine whether ultrasonographic assessment is incorporated into the vetting. The heat and swelling typically associated with many tendon and suspensory ligament (proximal/body) injuries can successfully be minimised or abolished with rest and anti-inflammatory measures and reliance on clinical judgement alone (without ultrasonography) carries with it a risk of undetected injuries. Sub- or mildly clinical SLB lesions are commonly encountered; these do not necessarily preclude purchase; however, client risk profile and destination jurisdiction are important factors.

CONFORMATION AND GAIT

Overview

Much importance is placed on limb conformation ('correctness') and body type when assessing juvenile horses. The form of a horse's musculoskeletal system defines its function: the alignment of joints, angulation of limbs and orientation of musculature will determine its distribution of weight, movement of limbs and biomechanical efficiency and thereby its potential for both athleticism and orthopaedic soundness/unsoundness. However, as with most other aspects of selection (pedigree and genetic analysis, imaging, upper airway function, stride and performance analytics), conformation should be considered a contributory rather than the predominant factor in determining future athletic success. There is a great range of expression of conformation types in the juvenile Thoroughbred population and the interplay of conformation with the many other factors that contribute to athletic success is a largely unresearched field. Most current evidence is anecdotal, and assessment of both severity of faults and potential importance for future athleticism is open to multiple interpretations.

CONFORMATION

- Very little known about associations between static conformation and biomechanical loading/injury risk; virtually nothing known about 'dynamic conformation'.
- While highly likely that conformation affects injury risk and power generation/athletic capacity, there is no certainty about what degree of defect is important relative to other intrinsic and external factors.
- Predisposition to injury should not be considered the same as predetermination.
- Judging the importance of conformation to any particular individual remains more of an art than science at present.

Conformation assessment

Limitations

Assessment of conformation is highly subjective. Accurate measurement of conformation is difficult and current scientific knowledge is both limited and based largely on information derived from yearlings vetted at public auction, a population of animals already selected for saleability. While terminology relating to limb conformation (Table 18.8) is widely recognised, grading of severity has been neither standardised nor validated. Conformation assessment relies on visual appraisal of alignment/misalignment of external anatomical landmarks, yet these external landmarks frequently do not correlate with the presumed position of underlying bony features and certainly misrepresent the complexity of rotation and alignment that can exist. Only multiplanar radiographic/tomographic imaging has the capacity to truly map out the interrelationships of bones and joints and such imaging has no practical place in selection of the racehorse. Use of videography and motion analysis has the potential to improve the objectivity of conformation assessments but is also limited by reliance on external landmarks.

Practical assessment

Assessment should encompass not only alignment of the lower limbs but their relationship with the upper limbs (humeral, scapular and pelvic/femoral angulation), width of chest/trunk, height of withers relative to hindquarters

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Table 18.8 **Conformational faults: Outline description and significance**

CONFORMATION	DESCRIPTION	SIGNIFICANCE
Offset ('bench') knee	Carpus and/or cannon laterally offset (with varying severity) relative to radius.	May worsen from yearling to adulthood. Greater loading through medial bone column: higher likelihood of developing splint enlargement/C3 sclerosis. Association with risk of developing fetlock problems (in same limb). Moderate-marked severity: associated with significant risk of future carpal lameness (specifically pathological loading through dorsomedial carpus, and third carpal bone sclerosis) +/– radiological deterioration that may affect resale
Carpal valgus	Lateral deviation of lower limb originating at knee. Angle of radius-midcarpus-mid-fetlock is >180°.	Mild-moderate carpal valgus of little concern & some evidence that may reduce risk of carpal injury.
Back at the knee	Proximal row of carpal bones positioned caudal to front edge of radius, resulting in 'concave' appearance to knee when viewed from side.	Improves from yearling to adulthood. Anecdotally associated with greater risk of overextension injuries involving antebrachiocarpal joint.
Over at the knee	'Convex'/'buckling' appearance to knee when viewed from side due to incomplete extension of affected limb(s). Typically a result of flexural limb deformity/tendon contracture rather than structural incongruity of bony column of forearm/carpus.	May worsen from yearling to adulthood. When severe, considered a serious fault that may lead to unsoundness. Associated with greater risk of pastern fracture.
Fetlock toe-out (forelimb)	Fetlock valgus (lateral deviation originating at fetlock). Very common (small amount of outward rotation through the fetlock is normal).	May predispose to interference injuries if severity > mild.
Fetlock toe-in (forelimb)	Fetlock varus (medial deviation originating at fetlock).	Greater risk of developing lateral suspensory ligament branch injury. When severe, considered a serious fault that may lead to unsoundness.
Upright forelimb pastern	Straight/upright appearance of fetlock when viewed from side.	If marked severity: greater risk of injury in same limb (but unquantified). Biomechanical effect: likely reduced energy absorption during loading.
Long/sloping pastern	Fetlock appears hyperextended/slack when viewed from side.	If marked severity: greater risk of injury in same limb (but unquantified).
Straight through the hock	Large tarsal angle (when viewed from side)	Greater risk of developing hindlimb lameness (especially hock/proximal suspensory ligament). Biomechanical effect: likely reduced energy absorption during loading +/– reduced propulsion.
Sickle hocks	Small tarsal angle (when viewed from side)	Greater risk of developing lower hock joint osteoarthritis or curb but impact on trainability likely to be low.
Uneven feet	(One upright/boxy, other flat-heeled); may develop either from 'sidedness' when grazing, chronic lameness or DIPJ contracture.	Merits caution if marked: determine significance on individual basis. Correction usually not possible/advisable

(indicating likelihood of further growth), length of back, body type and condition. Limb conformation is assessed statically (from the front, rear and both sides with the horse stationary and weight-bearing on all limbs) and dynamically (observation from front and rear while the horse is walking). The latter is crucial to fully assess limb flight and foot placement and dynamic irregularities in limb motion. Conformational faults should be classified as deviations from 'normal' and graded by severity as mild, moderate or marked. Identifying the part of the limb/

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articulation from which any deviation (such as an outward rotation) arises is important. Assessment of foot balance (both from footfall when walking and during limb palpation with foot raised) can add to the understanding of any conformational or gait abnormalities noted and whether corrections have been attempted in the past.

Significance

The importance of conformation lies primarily in its influence on limb loading (and thereby predisposition to injury) and, less quantifiably, on force generation during locomotion (particularly the hindlimb). Conformation assessment is of greater importance at yearling examinations (prior to training) than in horses that have trained at least one season (or to breeze-up sale consignment) and in which any adaptive effects have become tangible either clinically or on imaging. Consideration of body type, severity of fault and activity of growth plates and prior experience of closely related animals may help determine the importance of limb conformation in a particular yearling.

Conformational faults may be complex with horses displaying combinations of misalignment and rotation even in the same limb, and very little is known about the summation or negation of risk that may result from these combinations. Advice on likely significance should be given on the basis that there is little statistical foundation even for conclusions drawn about simple/common faults. It should also be noted that long-established veterinary practices relating to surgical and non-surgical correction of angular limb deviations are based on presumed effects on future saleability (+/– musculoskeletal durability) rather than any solid scientific foundation. Opinions on conformation/body types most suitable for different racing distances (sprint/middle-distance/staying) are widely held but currently supported a limited evidence base. Smaller/shorter horses are more likely to race as 2 YOs (and have more career starts) and base-narrow horses are less likely to run as 2 YOs. Yearlings that are taller or heavier than average are more likely to become elite performers.

There are some general trends of association between certain conformation types and development of specific injuries; however, exceptions to all rules exist. Most conformational faults do not carry such a greatly increased risk of related problems that they should preclude purchase; however, in general the significance for future unsoundness is greater the more severe the fault. The faults that in practice appear to carry the greatest risk to future trainability are knees that are offset or over-at-the knee, and toe-in front fetlocks. Offset knees of medium or greater severity consistently lead to adaptive changes (particularly increased density/sclerosis) developing in the important load-bearing portions of the middle carpal joint (third carpal bone/radial carpal bone) that can predispose to lameness or carpal fracture. It is recommended that most other conformational faults (unless severe) be viewed leniently and in the context of the overall body type/maturity/intended use.

The relationship between conformation and future racing performance/athleticism is complex and to date researched correlations between most metrics have been weak. Areas of interest include measurements such as shoulder and coxal (hip) angle and their potential relationships with stride length and power generation. There is evidence that the gait characteristics of a juvenile horse are correlated to that of the mature animal, and features such as length of back are known to affect lateral bending of the spinal column at faster paces. Use of commercial analytical aids to assist selection of juvenile horses may yield associations in future, although proprietary interest could limit any such associations being subjected to scientific review. At present, excluding severe conformational faults, pedigree is a far greater influence on racetrack performance than conformation.

Influences on conformation

In general horses tend to be proportional, such that length of limb bones is related to height. Bone length changes little beyond 12 months of age, and by 18 months most yearlings are nearly (95%) full adult height. Several major factors contribute to the mature conformation of any horse. Genetics plays a large role: the main conformational faults are heritable to a variable extent depending on anatomical location. Traits associated with the carpus appear to be strongly heritable: these include offset and back-at-the-knee conformation. Conversely, foot conformation is considered to have low heritability.

Biomechanical loading through early life can influence adaptation and growth of bones, ligaments and tendons, and explains why limb conformation is frequently dynamic and has the potential to change (for better or worse) until full skeletal maturity occurs. Considerable self-correction of many carpal and fetlock conformational faults encountered in foals occurs by yearling stage; carpal valgus is a noteworthy example of a deviation that improves in many horses over this time. Corrective farriery (trimming/balancing +/– hoof extensions) and surgery is routinely used to assist realignment of the growing limb. From yearling stage into adulthood, limb conformation generally changes little: straightening of toe-out hind fetlocks and back-at-the-knee conformation occurs, while offset knee/toe-in conformation may worsen. Foot

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balance has a tendency to change with age and development. Weanlings and yearlings typically have an upright hoof/pastern angle (sometimes verging on 'boxy'), with this angle becoming lower ('broken-back') by 2 or 3 YO.

Corrective surgery

If corrective surgery for a fault has resulted in a normalization of conformation without untoward effects (such as on dynamic foot balance), then as a general rule any importance to potential trainability can simply be judged on the yearling's limb conformation as presented at time of sale.

Gait analytics

Commercial gait analysis services are increasingly used by prospective purchasers at 2 YO breeze-up/ready to run public auctions. These services use video analysis of the breeze to model gait, biomechanical and speed features of individual horses and allow comparisons against large datasets. The goal of such analytic tools is to determine the relative probability of an individual horse becoming an above-average (or elite) or below-average performer, and thereby inform purchasing decisions with data that extend beyond simple speed figures. The true predictive value of these tools is commercially sensitive information and unless subjected to scientific peer review the validity of such analysis is likely to remain an area of uncertainty.

HEART SIZE

Accurate measurement of cardiac dimensions in the live horse may be undertaken with echocardiography. As a general rule heart size is a poor predictor of athletic capacity in the flat racehorse. The following points are relevant:

- Heart size is strongly related to body size.
- Chamber dimensions and heart muscle mass increase considerably with training.
- Heart dimensions therefore often change markedly between yearling and mature (trained) racehorse.
- Top-performing sprinters often have hearts ≤ average size.
- Heart size is a more important predictor of endurance capacity; not relevant in yearlings/2 YOs but may be useful in National Hunt (hurdle/steeplechase) racehorses.
- 'Heart scores' derived from electrocardiogram (ECG) assessment of ventricular depolarization (QRS duration) may bear some relationship to heart size, but correlation with performance is weak.

GENETICS OF SELECTION

A horse's athletic potential remains largely unknown until completion of a training programme and tested against peers during fast exercise in training/racing. Given that athletic ability is largely determined by cardiovascular and muscular capacity and that around 35% of variation in performance is heritable, there has been considerable research interest in identifying gene variants coding for desirable traits that may contribute to performance. Genes associated with energy production and utilization at a cellular level may have a direct bearing on a horse's anaerobic and aerobic capacity and total oxygen uptake. The myostatin gene is of particular interest: it is directly involved in regulating skeletal muscle growth and has been linked to racing performance, optimal racing distance and growth potential. Variants of the myostatin gene have been shown to be highly predictive of distance aptitude: horses with CC (sprinting) and CT (middle-distance) genotypes are in general more precocious and more successful as 2 YO than TT (staying) genotypes.

It is important to note that while genetics play a role in the potential athleticism of any horse, epigenetic factors (the many and varied non-genetic influences that can affect gene expression) are at least as important. Such factors include early-life health, nutrition and exercise, and later on pre-training and race conditioning. A horse with superior genetics that has had a suboptimal juvenile development period (either through influences of management, environment or disease) may easily prove to be a less effective athlete than a horse with inferior genetic makeup that has been reared and trained optimally.

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CHAPTER 19

PREVENTION OF INJURY

OVERVIEW

Greater public awareness of and concern for the welfare of animals participating in sport has familiarised horseracing stakeholders with the phrase 'social licence to operate'. Society at large now demands that those governing the racing industry are held accountable for the welfare of racehorses and that if horseracing cannot be conducted in a way that is ethically acceptable then societal support will be withdrawn. One of the aspects of horseracing that is subject to the greatest public scrutiny is racetrack fatalities and specifically musculoskeletal breakdown; as guardian of racehorse health the equine clinician therefore has an important role in ensuring legitimacy and sustainability of the sport. Racehorse musculoskeletal breakdown is also a major contributor to the occupational risk of injury or death faced by jockeys (among the highest of all sports) and mitigating this risk is therefore a priority for all stakeholders.

The great majority of serious or catastrophic musculoskeletal injuries incurred by the flat racehorse during high speed exercise (whether that is during racing or training) involves the forelimb fetlock joint. The most common pathologies implicated across all major racing jurisdictions are fracture of the proximal sesamoid bones or lower cannon. It is recognised that in most cases fractures at these sites are not the result of a 'bad step' or 'unfortunate accident' but of focal pathology that develops over considerable periods and leaves the affected bone at heightened risk of failure when put under high loads. It follows that there are opportunities for early detection of prodromal injury that can be utilised by both primary care clinicians and regulatory bodies to reduce serious injury rates. How and when these opportunities are best utilised is dependent on the point during injury development at which reliable detection of abnormalities becomes possible, which can vary with several intrinsic and extrinsic factors. Successful risk reduction programmes have been introduced in several jurisdictions worldwide; however, even with enhanced regulatory scrutiny 'preventable' serious injuries continue to occur, highlighting that there is room for improvement through the application of new technologies and evidence-based processes.

While reduction of injury risk and early detection have consequent welfare benefits for the racehorse in terms of reduced mortality and morbidity, it should be recognised that total elimination of injury is not possible in any athletic pursuit, equine or otherwise. Training involves repetitive physical 'overloading' to stimulate positive musculoskeletal and physiological adaptations, and the complex interplay between multiple intrinsic and extrinsic factors cannot be controlled sufficiently to eliminate the risk of injury altogether. However, greater understanding of the pathogenesis of key injuries is leading to consideration of how modification of factors ranging from training programmes to racetrack design might reduce the prevalence of certain fatigue pathologies in future. In terms of preventing fatigue injuries from reaching their potentially serious/catastrophic end-stage there are greater opportunities, with methods to best achieve this determined by whether interventions are at the primary care or regulatory level. Key elements are the assessment of risk, application of screening tests/procedures and implementation of prophylactic measures and risk management strategies.

MANAGING RISK

Defining risk

Regulatory risk assessment or mitigation protocols can have ethicolegal and welfare consequences, and should therefore have terms of reference that clearly describe objectives and limit any ambiguities of language. The questions that follow may not have defined answers but should be considered carefully:

What is risk?

Risk is the chance or likelihood that an undesirable effect or event will occur. In the context of pre-race musculoskeletal screening of racehorses the primary concern of regulators relates to the likelihood of significant exacerbation of any injury/condition to the extent that the horse's

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safety or welfare might be compromised. Other concerns are whether the condition in question could significantly affect a horse's racing performance, or the well-being/welfare of the jockey, other horses and other jockeys. With these parameters it should be possible to delineate which musculoskeletal pathologies (or categories thereof) encountered during the assessment process preclude a horse from competing and which are acceptable.

What is 'unacceptable' risk?

Perception of risk is highly subjective and frequently differs markedly between owner, trainer, regulator and the public, as well as between racing jurisdictions and even races of different quality/profile. Most stakeholders would agree that a moderate to high risk of catastrophic injury occurring is unacceptable, but given that such categorizations have no quantitative value it may be preferable to frame risk in terms of whether a horse is likely or unlikely (or more/less likely than the average horse in the field) to sustain or exacerbate injury. Expressing risk in terms of both probability and severity (such as used in risk matrices) reduces the subjectivity inherent to this area: for instance, it might be unacceptable to race a horse with a particular injury judged to have a low probability of deteriorating during high-speed exercise but for which the consequences of that deterioration (if it were to happen) are severe/catastrophic, whereas a horse with pathology at higher risk of deterioration but for which the consequences of deterioration are minor might be permitted to race. Current understanding of the risks posed by key racehorse pathologies is outlined in Table 19.1.

What is the risk period being considered?

When making a judgement on the risk of serious injury posed by a particular clinical or imaging abnormality it is necessary to qualify that judgement in terms of time frame. This will necessarily depend on the nature of the abnormality/lesion and the degree of confidence about the diagnosis (which may be a function of the imaging modality(s) used). Musculoskeletal pathologies are dynamic and it follows that risk of incurring serious injury is also dynamic in nature. In general it can be considered that the certainty that an abnormality or lesion will *not*

Table 19.1 **Common orthopaedic conditions and associated immediate risks* with racing/high-speed exercise**

REGION	CONDITION	INCREASED RISK OF CATASTROPHIC INJURY?	INCREASED RISK OF UNEXPECTED POOR PERFORMANCE?	RISK OF SERIOUS EXACERBATION OF INJURY?
Fetlock	Palmar/plantar osteochondral disease (POD)	No	No†	No†
	Condylar fissure fracture	Yes§	Yes	Yes§
	Dorsoproximal P1 chip	No	No	No†
	SLB insertional desmopathy	No†	No†	No†
Cannon	SDFT tendinitis	No†	Yes	Yes
	SL desmopathy	No†	Yes	Yes
	Dorsal metacarpal disease ('bucked shin')	No	No	No
	Dorsal fatigue fracture	Yes	Yes	Yes
Carpus	MCJ osteoarthritis/chip fracture	No	No†	No†
	C3 sclerosis	No†	No	No†
	C3 sagittal plane lucency	No†	No†	Yes
	C3 frontal plane lucency	Yes	Yes	Yes
	ABC joint osteoarthritis/chip fracture	No	No	No†

* Based on empirical evidence from UK/European flat racing population.

† Dependent on imaging features/lesion grade and assuming static clinical presentation in pre-race period.

§ Unless determined otherwise with multiplanar/functional imaging.

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lead to serious injury diminishes with time elapsed since last assessment. Level of confidence about the decision may be high for the immediate period (typically an imminent race) but quite reasonably may no longer be relevant or accurate beyond a few weeks depending on the amount of high-intensity exercise (and response to that exercise) undertaken during that period.

What injuries can be prevented?

It is recognised that the great majority of serious/fatal injuries sustained at high speed are the result of fatigue pathology and therefore have a prodromal phase during which early detection may be theoretically possible. Factors that limit early detection include location and type of pathology, presence of detectable clinical signs, level of vigilance and available diagnostic modalities. Of the fetlock pathologies, a large proportion of condylar fractures may be detectable prior to complete fracture with good vigilance and use of ambulatory (radiographic) imaging; prevention of serious proximal sesamoid bone fracture has previously been problematic but is now possible with advanced imaging (positron emission tomography [PET]). By contrast, prevention of fractures that occur acutely following a monotonic loading event (such as most proximal phalangeal fractures) or due to risk factors inherent to track surface conditions should not be expected.

Where and when?

Prevention of serious injury demands assessment of musculoskeletal 'health'. When and how this assessment occurs strongly influences its efficiency and accuracy: generally it is regarded that longitudinal assessments (such as those that occur in primary care) with the benefit of access to veterinary history have greater value and are more likely to be accurate than those conducted in a single point in time (such as pre-purchase examinations or regulatory checks in the pre-race period).

Ethics of risk management

Ethics relate to the moral standards and principles that govern professional conduct and are considered to be distinct from legal obligations. Managing the risk of serious injury in a domestic animal bred and trained for athletic competition poses many ethical challenges for both the primary care and regulatory clinician, not least because concepts of what constitutes animal welfare are complex. Veterinary codes of professional conduct give primacy to protecting animal health and welfare but also support decisions based on the needs and circumstances of the client, including that of confidentiality. Musculoskeletal pathologies are an inevitable consequence of athletic training, and while the primary care clinician may be well placed to manage risk it cannot be eliminated altogether. For the equine clinician there is both the responsibility to safeguard welfare for an animal that cannot 'consent' to risk, and the recognition that the racehorse is bred solely for the purpose of racing.

Ethical conflicts are not the preserve of veterinary medicine and similar tests of moral judgement can be found in human sports medicine. In sports medicine as in racehorse medicine there is often a high degree of uncertainty over what might be significant in terms of risk for a particular individual: decisions may have to be made in the absence of a sufficient evidence base to ensure certainty about the outcome. Choosing to avoid immediate risk may not ensure long-term well-being or career success, and vice versa. Solutions are rarely absolute, and shared (rather than paternalistic) decision-making is favoured. However, it is not always the case that collaboration in decision-making or even 'informed consent' results in the best outcome for the patient: the highly competitive athlete may choose a path that is detrimental to their health. Indeed, while it is often stated that racehorses cannot 'choose' to race, the question of free choice is by no means settled in human sports: it is considered that in very few situations (a notable exception being concussion) is the athlete free from external influences and therefore truly autonomous. Conflicts of interest are also commonplace in elite sport and particularly team sports, where the physician may have a contractual relationship with the player's employer (club) in addition to the duty of care to the player. Team success is not always compatible with the player's best health or career interests, and both team management and player may justifiably claim some rights to both accept or limit risk.

The equine clinician must navigate these challenges with the awareness that injury risk assessment has potential ramifications not just for the horse but for trainer and connections, and the wider racing community. These stakeholders share risk and the responsibility for taking it, while having diverse perspectives. A trainer may approach a decision in a way that seems risk-taking, but is influenced by knowledge of that horse's training or injury history, or a need to act in the best interests of the owner. The regulatory body may approach a decision in a way that seemingly disregards the individual horse's welfare to protect the wider interests of racing. Ultimately, effective veterinary care (whether primary or regulatory) relies upon respect between all parties and trust that decisions will be based as far as reasonably possible on scientific merit.

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ELEMENTS OF RISK ASSESSMENT

Assessment and stratification of risk in the racehorse is a complex process and reflects the multifactorial pathway to musculoskeletal failure; there is no single good test to mitigate risk and assessment therefore relies on a range of elements.

Profiling

'Profiling' is the process of assessing whether an individual has characteristics statistically associated with susceptibility to the target condition or injury. Some of these 'risk' characteristics may be specific to a particular track or jurisdiction (such as horses that have travelled in that are not habituated to local conditions), whereas others may be recognised more widely across the racehorse population (e.g., entire males, horses that commenced racing at ≥3 YO, or that are having their first start after a long layoff). Determining whether any given horse has a profile 'of interest' for the purposes of injury screening is therefore dependent on many factors.

Large datasets have been interrogated with a view to creating statistical models that might be used to assist screening racehorses for risk of serious injury; however, because of the low incidence rate of fatal race day injuries all such models have to date demonstrated poor positive predictive ability. While these models are currently of little practical use in isolation, they can have a valuable role in screening programmes by facilitating identification of subpopulations to be subjected to additional scrutiny. In order not to incorrectly classify horses (as low or high risk) the validity and predictive ability of any profiling 'thresholds' should be assessed fully prior to application.

Genetic/molecular testing

Testing for genetic and molecular biomarkers associated with risk of serious injury has future potential as an adjunctive procedure to assist injury prevention protocols.

Clinical examination

Veterinary pre-race physical inspection is a cornerstone of regulatory oversight of racehorses and is enshrined in rules of racing across most jurisdictions. Veterinary pre-race inspection usually takes the form of palpation of the distal limbs and a trot-up in hand to assess action/gait; however, the ambiguous terminology and lack of defined parameters typical of these rules means that their implementation is highly subjective and open to wide interpretation. While clinical examination can have value in the assessment of the individual, findings should be viewed in the context that horses with obvious clinical abnormalities may not carry an inherently greater risk of injury than horses without clinical abnormality (and vice versa).

Palpation

Palpation of the limbs is undertaken to detect clinical abnormality relating to joints, tendons and ligaments. Signs of inflammation may be modified by the use of bandages or cryotherapy and evidence of their application should be taken into account during assessment. While it is recognised that horses with palpable abnormality involving the suspensory ligament or flexor tendon at pre-race regulatory inspection may be at increased risk of sustaining injury during the race, use of palpation as a screening test has poor specificity and should therefore not be used in isolation to pass a horse as fit or otherwise to race.

Lameness

Lameness and injury risk: While it is recognised that lameness is an important clinical sign of musculoskeletal injury, in the context of regulatory screening it has little intrinsic value as a test of whether a horse is fit to race or not: lameness in the racing Thoroughbred is common while the outcome of serious/fatal injury is rare. Equally, the appearance of soundness should not lead to the presumption that a horse is in good musculoskeletal health: many factors can contribute to a horse presenting with an acceptable gait at pre-race inspection, including warm-up, medication or light recent training. Greater use of objective gait analysis may not resolve this fundamental problem: the premise that below a certain quantifiable threshold or within a set of descriptive constraints a lameness or gait abnormality might be deemed low/lower risk for development of serious injury is not supported by what is known about the presenting signs of prodromal injury for some of the most important conditions involving the fetlock (which frequently present with only a subtle or mild lameness). In assessing 'soundness' prior to a race the use of unambiguous language should be encouraged: documenting lameness as a gait abnormality or using imprecise terms such as 'scratchy' or 'poor mover' does not assist subsequent analysis of serious incidents or future risk assessment.

Although regulatory pre-race assessment of gait/lameness is a poor screening test, longitudinal monitoring by the primary care clinician can by contrast be of great value in identifying horses for further diagnostic investigation. Empirical evidence supports recent, uncharacteristic change in gait/lameness as meriting enhanced vigilance for the development of injury. Horses with chronic musculoskeletal pathologies commonly establish a 'baseline' gait during training and while some

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fluctuation may occur depending on stage of season or level of workload, a significant deviation from 'normal' for a particular animal can signal worsening (or development of new) pathology. In this context a subtle lameness that is new might easily be associated with a much greater risk of impending serious injury than a lameness of much greater severity that is long-standing. Such assessment requires familiarity with an individual horse's gait and its response to training stimuli. Longitudinal analysis of race day (or high intensity workout) stride parameters may also permit identification of at-risk animals if sufficient data is acquired over time.

Lameness and performance: Whether the presence of lameness (at any gait) has an effect on racing performance, or on risk of underperformance is largely unknown. Limited available evidence favours lameness as being statistically unimportant for both areas of interest, although clearly it can be of significance at an individual level.

CAN RACEHORSES PERCEIVE DEVELOPING INJURIES?

- Some horses appear to display behavioural changes (subdued demeanour at exercise/reluctance to train/unexpected poor performance) that suggest awareness of presence of injury or impending injury.
- Such features may be the first sign of developing injury and can precede the development of clinical abnormality by days/weeks.
- Subjective and subtle nature of these signs means that detection (if it occurs) is through attentive rider/staff familiar with the horse.
- Most typically associated with tendon (superficial digital flexor tendon [SDFT]) injuries but also occasionally with condylar fissure fractures, prodromal third carpal bone injuries, prodromal pelvic stress injuries.

Imaging

Diagnostic imaging has an important role in risk assessment. A range of imaging technologies is available and choice of modality will depend on many factors including site of concern, suspected pathology, available facilities, time and economic constraints. Much attention to 'imaging-based' risk assessment has focused on the fetlock region due to the contribution of injuries at this site to overall serious/fatal injury statistics. There is currently no single imaging technique that can or should be applied to all screening situations. All the diagnostic modalities useful for fetlock imaging (radiography, magnetic resonance imaging [MRI], computed tomography [CT], positron emission tomography [PET], scintigraphy) can be applied in the standing horse; however, they each have strengths and weaknesses in terms of both practical application and interpretation of resulting images. It is also the case that even within some modalities (notably CT) there may be a wide range of capabilities due to technology used and sometimes expertise of those applying it; cone beam CT, for example, has inferior diagnostic capacity for many key racehorse pathologies than fan beam CT (or indeed radiography). The great majority of cases of potential incipient injury detected during screening of racehorses in full training require imaging that combines both structural/architectural and functional/physiological assessment to stratify risk of future injury, and this currently necessitates the use of multiple modalities (not all of which may be available at the same geographical location). Bone fatigue injury is a dynamic process and, aside from the presence of unequivocal fracture (cortical breach), there is no current consensus on imaging criteria that should be used to define risk. Ongoing developments in this field mean that recommendations are likely to be revised periodically.

SCREENING

What is screening?

Screening is defined as selecting individuals at risk of a disease, injury or condition from a larger population not unified by the presence of the condition of concern. The concept is generally viewed as the application of a 'filter' to a population to select sub-groups or individuals of interest for further investigation and specifically to assist early detection (and consequently better management) of the disease or injury. In the assessment of health it is considered that screening tests are not necessarily definitive about the diagnosis (presence or absence) of the target condition, but rather they are advantageous in terms of cost, ease or speed of applicability or lower invasiveness for application to larger populations. While the perception in the context of racehorse injury is of screening 'out' horses at risk, in fact it is a process of subjecting a population to tests and scrutiny in order to select 'for' features that positively identify those animals.

Features of a good screening programme

The ideal screening test is considered to be one that correctly identifies individuals with the condition of concern, does not falsely identify individuals without the condition, is safe to use and satisfies cost-benefit criteria. The unambiguous and accurate prediction of the future health status of the tested individual is the goal, but in practice the design of any screening programme entails trade-offs that balance accuracy and practicality. The validity and usefulness of any test is usually judged in terms of its sensitivity and specificity. *Sensitivity* is defined as the

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ability of a test to correctly identify individuals with the target condition (and therefore determines the likelihood of 'false negatives'), whereas *specificity* is defined as the ability of a test to correctly identify individuals *without* the target condition (and therefore determines 'false positives'). The *positive predictive value (PPV)* of a test is the probability that individuals with a positive result actually do have the target condition (i.e., the proportion of individuals with a positive test that do have the condition), whereas the *negative predictive value (NPV)* of a test is the probability that individuals with a negative result do not have the target condition. These criteria are interrelated and it is generally the case that improved accuracy of a test according to one measure comes at the cost of reduced accuracy in other areas.

Determining which screening test(s) is appropriate for a particular target condition takes into account factors such as the prevalence of the condition, the severity of the outcome if the condition is not detected, the speed of progression and treatability of the target condition and the risk of negative consequences ensuing if an individual is incorrectly identified as being at risk (false positive). For conditions that are potentially life-threatening or progress rapidly if undetected, a screening test with a high NPV is desirable, although this would typically generate more false positives. Aside from choice of screening test, the way that it is administered can also alter its PPV and NPV: the setting of thresholds that are more or less stringent, or the way a population is sampled are examples.

Formulation of a screening programme requires consideration of practical, ethical and legal aspects in addition to scientific validity of the tests. Screening in any health sphere is considered to represent a tension between societal interests and those of the individual. Early detection (if economically and technically viable) of an important condition may be beneficial if that information can be used to assist treatment and improve outcomes, but the information gained through a screening process can also prove to be a burden if it results in unnecessary investigations or treatment. Testing should be applied consistently, and planning around information security and confidentiality, communications and potential conflicts of interest should be instigated. Subjecting a diverse population to screening inevitably yields information that may be unexpected or secondary to that being sought. It is important therefore that screening programmes have inbuilt steps for managing unexpected outcomes, as well as processes to minimise the undesirable consequences of false-positive and false-negative test results. There should be coherent protocols for the management of both positive and inconclusive test results, such that the proportion of the sampled population that inevitably ends up in this situation can be dealt with fairly and have a pathway to return to competition once treated or cleared. To ensure that fitness-to-compete determinations are made on clinical grounds and to safeguard the external perception of impartiality, decisions should be taken by regulatory clinicians independent of the sporting executive.

Screening for injury risk in the racehorse

In seeking to identify racehorses at risk of serious/catastrophic injury it is important to recognise that at race-start level the incidence of serious injuries is very low in most jurisdictions, and that good data on the prevalence of horses in the wider training population with 'prodromal' injuries are largely absent at present. Any screening programme aimed at identifying a condition that occurs very infrequently in a population is by definition likely to incur a higher rate of 'false positives', and therefore it can be expected that for every horse spared from catastrophic injury through screening many horses at 'normal risk' would need to be scratched as well. Despite advances in diagnostic imaging pertaining to the lower limb of the equine athlete, there is currently no single modality that affords the high accuracy and ease of applicability necessary for use as an effective screening tool on the wider population. Applying a range of criteria and tests (profiling, clinical and diagnostic) sequentially can, however, allow subpopulations with higher proportions of 'at-risk' animals to be identified, thereby increasing the predictive ability of tests targeted at those groups, and this is likely to remain the optimum strategy for injury mitigation for the immediate future (Figure 19.1). A rational screening protocol would involve an entry review process in which a horse's recent veterinary and training/racing history could be scrutinised, allowing selection of horses with a higher risk profile for enhanced clinical +/– imaging assessment.

The focus of the regulatory body will generally be on the reduction of race day injury rates, and the imperative of mitigating risk of catastrophic injury might be viewed as outweighing any consequences for those horses scratched unnecessarily from competition. However, there may be significant welfare implications for such horses: regulatory sanction may curtail career or racing opportunities and force retirement, sale or even euthanasia; additionally there is an economic cost to be borne by trainers and owners. If rates of false positives are high, stakeholders may lose trust in the legitimacy of musculoskeletal screening. Any cost-benefit analysis of a screening process therefore needs to take full account

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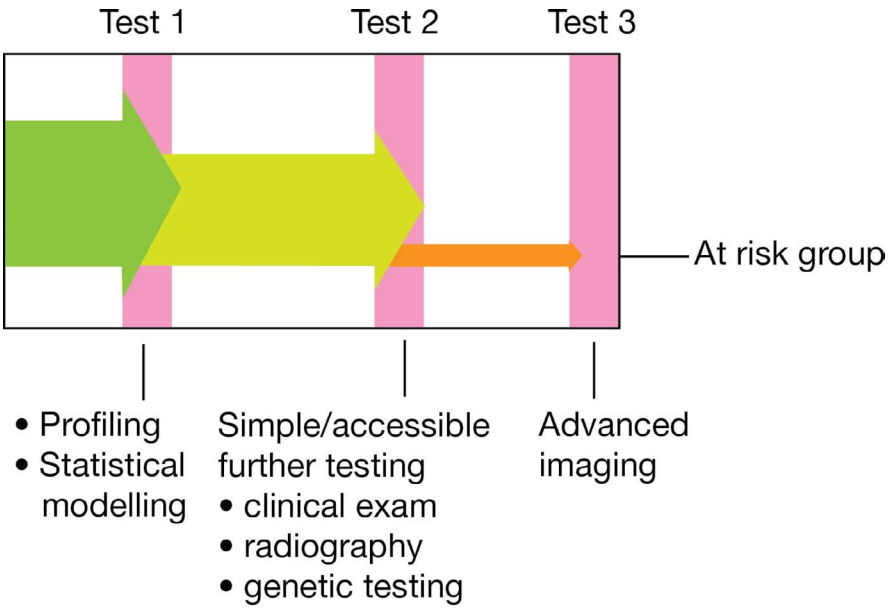


Fig. 19.1 **Sequential application of tests/profiling criteria to improve overall effectiveness (and minimise negative consequences) of injury prevention screening programme.**

not solely of 'fatalities saved' but of the negative consequences for 'low-risk' horses that fail a screening test. Expectations of the public regarding the likely success of screening protocols should also be managed carefully: race day risk of serious injury can be minimised but never eliminated completely.

Given the inadequacies of 'snapshot' clinical assessment in determining injury risk, expanded protocols that include assessments over a longer time period (including out-of-competition and post-race examinations) and profiling to identify 'horses of interest' are recommended. Chronic musculoskeletal conditions are very common in horses in training and many horses may present with clinical or imaging abnormalities yet are not at increased risk of serious injury. Enhanced communication and development of trust between primary care and regulatory clinicians permits pertinent information to be shared about such horses to improve the overall accuracy of the risk assessment process. It is incumbent on trainers/owners to comply with (and respect the outcome of) regulatory protocols, and there is an equal obligation for regulators to base decisions on objective, evidence-based criteria and apply them with consistency and transparency.

INJURY PREVENTION IN PRIMARY CARE

Assessment of injury risk is almost invariably better undertaken whilst the horse is 'at home' than at the racetrack when decision-making is likely to be compromised by time and resource constraints. The primary care clinician is best placed to identify animals of concern either directly or through the vigilance of yard staff and riders, to monitor gait (both in-hand and ridden) over time and in response to training stimuli, and to intervene in timely fashion with diagnostic blocking or imaging when deemed appropriate. For the great majority of horses adequate risk assessment can be accomplished in a first opinion setting (i.e., in the training yard), with only a small proportion of horses requiring referral for advanced imaging. Trainer and clinician familiarity with individual horses allows early recognition of deviation from 'baseline' clinical features and is superior to single point-in-time appraisals of risk that limit regulatory and pre-purchase examinations. To maximise the impact of injury prevention during training, however, it is necessary for there to be a regular veterinary presence in the yard, and a high level of mutual trust between trainer, staff and clinician so that potential injuries can be presented for veterinary attention promptly.

'CLINICAL BASELINE'

- Familiarity with a horse's normal gait (at various stages of training) can improve efficiency and accuracy of diagnostic investigations.
- Recent lameness or deterioration/change in action (particularly in response to high-speed exercise) is an important indicator of increased serious injury risk. This can be identified clinically or through use of gait/stride analysis.
- Interpretation of initial screening imaging findings may be influenced by recent clinical history: subtle findings may be viewed with more caution if associated with strong clinical concern.

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Education and awareness

Raising awareness of trainers and primary care clinicians to the predisposing factors that can lead to serious injury and the measures that can be taken to mitigate it has been shown to be effective in reducing injury rates. Some useful aspects are outlined in the following sections.

Training strategies

- Applying exercise regimes that optimize bone conditioning and readiness for racing, such as judicious exposure to high-speed exercise during 2-YO season.
- Allowance in training programmes for individual variation in maturity and response to training.
- Recognising that conditioning response differs between track types: training exposure to track surface that horse is intended to race on is encouraged; also reduction in workload when changing to new surface is advisable.
- Recognising that cumulative high-speed exercise without sufficient allowance for rest and repair (particularly if over short period of time) increases risk of serious injury.
- Training to fatigue is a risk factor for injury: muscle activity normally serves to dampen loading forces on limb and when muscles fatigue, greater strains experienced by bones/joints/tendons/ligaments.
- Awareness of variation in condition or injury rates related to local training tracks.

Vigilance

- Awareness of stages of training considered 'high risk' for specific injuries: particularly early in first season of training, and when stepping up to high-speed exercise following a layoff.
- Familiarisation with features of individual horses' action throughout the training season permits recognition of new/uncharacteristic lameness.
- Regular pre- (+/– post-) race inspections (in-hand trot) for routine monitoring: quality of recovery from high-speed exercise/racing can be very useful guide to clinical progress of chronic musculoskeletal pathologies.
- Anti-inflammatory/analgesic interventions that minimise/eliminate lameness limit ability to detect early signs of impending injury: avoid blanket/routine use of systemic anti-inflammatory medications and limit intra-articular medications to sites targeted following diagnostic workup.

Data collection

- Access to good data is a prerequisite to undertaking any meaningful audit or comparison of injury rates (whether that is between trainers, between jurisdictions or between time periods).
- Quality/availability of data is determined by level of veterinary access to training population and degree to which diagnosis is attained when horses present with lameness/injury; however, always useful to record even brief clinical notes for animals examined during routine inspections.
- Data acquisition assists early recognition of problems arising from racing/training surface conditions.

Accountability and feedback

- Confirmed diagnosis in the case of any serious/catastrophic fracture and feeding postmortem information back to yard provides opportunity for trainer and primary care vet to review internal processes/modifiable factors and establishes some accountability for 'preventable' injuries.

CASE STUDY: PRE-RACE/POST-RACE LAMENESS IN UK FLAT RACING YARDS

- Single-observer pre- and post-race examination data from two large UK flat racing yards (3000 pre-race checks; 80% races on turf, 20% synthetics).
- Common to observe lameness at pre-race examination: approximately 8% starters with ≥grade 2/10 lameness
- Moderate lameness (≥3/10) uncommon (1.5% starters); most of these previously subjected to diagnostic work (imaging +/– blocking).
- Majority of lameness unilateral forelimb.
- Similar proportion of lame/sound horses racing on turf and synthetics.
- 55–60% horses have same lameness score pre- and post-race; 10–15% horses better and 20% worse at post-race (day following race) check.
- Horses racing on synthetics more likely to have a better post-race lameness score than horses racing on turf.
- Correlation between finishing position and change in lameness score: lower finishing position less likely to be lamer post-race.
- No significant correlation between pre-race lameness (common) and racetrack serious injury (rare).
- Pre-race lameness an unreliable predictor of post-race lameness, and poor predictor of serious injury risk.

Managing the individual horse

While much of the discourse around risk assessment in the racehorse pertains to statistical risk and is derived from large population datasets, it is questions immediately related to the management of the individual equine athlete that ultimately preoccupy the primary care clinician. Generic injury prevention protocols are by their nature

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non-specific and do not account for the great variety of pathology encountered in racing practice. It is important to recognise that scientific understanding of the many biological and external predisposing factors for injury (and the interactions between them) is incomplete and that in all but the most overt cases of injury a range of management options is available. Diagnostic, treatment and training decisions can all therefore be justifiably influenced by individual circumstances, including severity of pathology, stage of career, response to medication, imaging progression and trainer/owner wishes. Consideration of the following factors is relevant:

Individual susceptibility

- Early-life factors (nutritional imbalances, restricted exercise due to illness), conformational faults and body type (and speed of maturity) may predispose some animals to musculoskeletal problems. Recognising predisposing factors and monitoring musculoskeletal responses to training loads can help to minimise establishment of chronic fatigue and articular pathologies.
- Awareness of injury trends with seasonal/training cycles: enhanced vigilance in early training and at time of first exposure to high-speed exercise recommended.
- Monitoring response to interventions: whether increased/decreased training stimuli, or medication.

Medication

- Most serious racetrack fractures arise from pre-existing pathology.
- Analgesic/anti-inflammatory measures (systemic and intra-articular medications, cold therapy) may impair recognition of early warning signs of impending injury and permit pathology to progress closer to catastrophic failure during racing or fast work.

When to image

- Determining when appropriate to intervene with diagnostic blocking +/– imaging influenced by many factors.
- Enhanced clinical vigilance (+/– periodic diagnostic imaging) for horses displaying changing/deteriorating action or chronic lameness.
- Screening ambulatory imaging can mitigate risk substantially: guided by clinical presentation but radiography of fetlock(s), ultrasonography of pelvis commonly utilised. Radiographic screening used to good effect in clinical practice to detect early injuries and can dramatically reduce serious fracture rates.
- Radiographic screening prior to intra-articular medication of fetlocks/knees recommended: historically most horses that sustain serious musculoskeletal breakdown in the immediate period following medication have not been imaged.
- Screening radiography of fetlocks (flexed dorsopalmar/plantar radiographic projection, Appendix 4) recommended for horses scheduled to depart primary care for period of competition away from home training track: helps to manage risks associated with altered training conditions and difficulties associated with remote decision-making.

WARNING SIGNS CHARACTERISTIC OF IMPENDING SERIOUS FETLOCK INJURY

- New (days/weeks) unilateral lameness.
- Lameness subtle/mild when trotted in hand but more noticeable when ridden.
- Onset (or worsening) of lameness may coincide with exposure to high-speed training loads.
- Fetlock free from obvious palpable abnormality.
- Lameness may improve/disappear with few days of light exercise, then recur with return to full training.

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POOR PERFORMANCE

Racetrack performance on any given occasion is influenced by a large number of intrinsic and extrinsic factors (ranging from athletic potential and fitness, to race pace, track condition, jockey tactics and quality/fitness of competitors). When a horse performs poorly, or counter to expectations, it is frequently difficult to determine with certainty whether the underperformance is genuinely arising from a horse-related problem or whether other factors are to blame. Knowledge of the horse's fitness, well-being and ability, training history and pre- and post-race communications with jockey therefore make the trainer best placed to judge whether a possible veterinary issue should be investigated. Poor performance has the potential to encompass a wide range of potential pathologies, and the clinician should seek to establish at the outset the trainer's level of concern and broad parameters for the scope of the investigation.

CAUSES OF POOR PERFORMANCE

There are many potential causes of poor performance. The most common group by far is dynamic upper airway obstruction, followed by lower airway disease (exercise-induced pulmonary haemorrhage [EIPH] and lower airway inflammation) and musculoskeletal injury. Once these conditions have been ruled out through diagnostic investigation, very few cases remain unresolved; however, these latter can often prove to be challenging, expensive and frustrating to identify. When investigating individual poor performance, caution should be exercised in attributing possible significance to 'yard-wide' risk factors: if, for example, a nutritional fault or air hygiene concern is detected in the yard, it can be expected that effects would be seen more broadly in the yard population (rather than just the individual).

Common causes

Key features of the main causes of poor performance are detailed in the following sections.

Upper respiratory tract obstruction

- Dynamic upper airway obstruction (Chapter 8) is the most common cause of poor racetrack performance.
- Frequently involves collapse of more than one structure.
- Not always associated with respiratory noise.
- Diagnosis typically requires overground endoscopy; some obstructions may be intermittent or only evident under race conditions and definitive diagnosis not always possible.

Exercise-induced pulmonary haemorrhage

- Readily assessed at resting post-exercise (preferably post-race or high-speed exercise) endoscopic examination of trachea.
- Clinical significance in relation to reported poor performance should not be assumed: racehorses may perform satisfactorily at elite level despite presence of moderate to marked EIPH.
- Interpretation aided by determining whether EIPH episode was isolated/uncharacteristic (or not); horse's scoping history may assist, otherwise reserve judgement until follow-up assessment after further high-speed exercise undertaken.
- In general; if degree of EIPH noted on visual inspection of trachea is mild, or only detectable through laboratory analysis of lower airway samples (tracheal or bronchoalveolar lavage), highly unlikely to be the primary cause of poor performance.

Lower respiratory tract disease

- Generally readily assessed through post-exercise endoscopic examination of trachea.
- As for EIPH, clinical significance in relation to reported poor performance should not be assumed.
- Response to treatment typically determines accuracy of diagnosis (i.e., infectious or non-infectious lower airway inflammation).

Musculoskeletal injury

- Undetected or subclinical bone stress injuries may be linked to both unexpected 'acute' and slowly progressive poor performance.

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- Orthopaedic injuries that may cause poor performance yet exhibit few/no obvious abnormalities (or lameness) at clinical appraisal include pelvic and spinal stress injuries and fractured ribs. Screening bone scan may be justifiable to assist diagnosis.
- Chronic articular/subchondral bone pathologies may also be associated with insidious-onset (but not acute) poor performance; however, their high prevalence in the 'normal' racehorse population should prompt caution in attributing significance unless other causes have been excluded.
- Likewise, significance of lameness should be determined on individual basis: most horses that exhibit lameness do not underperform.
- Underperformance in one or several races/high-speed training sessions prior to detection of tendon (superficial digital flexor tendon [SDFT]) strain is a common occurrence and implies that horses may sometimes be aware of (and protect against) SDFT injury before it becomes clinically evident.

Uncommon causes

Exertional rhabdomyolysis

- Exertional rhabdomyolysis (p. 265) is a very common condition that in most horses is controllable with good management and does not cause poor performance.
- In small subset of affected horses control of condition may be difficult and poor performance (single/recurrent) can occur if episode(s) of 'tying up' occur during a race.
- Not always clinically obvious; blood testing for muscle-derived enzymes aspartate aminotransferase (AST) and creatine kinase (CK) often necessary (see also p. 460).

Gastric ulcer syndrome

- Presence of gastric ulcers has been linked to poor performance and speculated to arise at least in part from discomfort caused by 'splashing' of acidic stomach contents on the non-glandular lining of the stomach.
- As with some other conditions mentioned earlier, gastric ulceration is very common in horses in training and interpretation of significance may rely on elimination of other potential causes as well as response to treatment.

Cardiac abnormality

- Heart arrhythmias or valvular insufficiency (Chapter 10, p. 313) are rarely-encountered causes of poor performance.
- Transient nature of most arrhythmias may make diagnosis difficult; investigation is a medical specialism.

'Overtraining'

- Poorly-defined syndrome characterized by long-term underperformance or reduced exercise capacity, with or without other physical or behavioural signs. Comparable condition in humans is described as 'burnout', 'staleness' or chronic fatigue and is considered a severe manifestation of 'non-functional overreaching'.
- Better described as 'unexplained underperformance syndrome', as 'overtraining' implies causal relationship with training loads.
- Limited understanding of the underlying cause(s) and why certain individuals are affected, although it is considered to represent a mismatch between training load and recovery and oxidative stress or hormonal alterations may be implicated.
- Red blood cell hypervolaemia is not implicated in poor racehorse performance (and presumptive treatment through phlebotomy/blood-letting therefore not supported by science).
- Clinical features include loss of condition/bodyweight (with or without a drop in feed intake) and changes in behaviour such as irritability and unwillingness to train; these may persist even after horse is allowed a recovery period.
- Diagnosis difficult and frequently inconclusive due to the current absence of any single effective diagnostic test, although heart rate variability (p. 18) considered useful in human athletes.

Other medical conditions

- Range of other uncommon/rare medical conditions may cause ill-thrift or poor performance.
- Include rare medical conditions (e.g., neoplasia) and poorly defined syndromes such as 'post-viral' underperformance/'fatigue'.
- Investigations in these cases are typically lengthy and frequently unrewarding and should not be commenced before more common/likely causes of poor performance have been ruled out.

INVESTIGATION

As the potential causes of poor performance are so diverse and encompass most body systems, it is important for investigation to be methodical to maximise the likelihood of successful/timely diagnosis and minimise cost to client

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arising from diagnostic procedures undertaken. History, signalment and initial clinical evaluation can give early direction to the investigation: for instance it may be considered that initial focus should be on upper airway function or on musculoskeletal health. Procedures/tests that have a high 'yield' (more likely to furnish a diagnosis) should be utilised first, reserving complex/expensive diagnostic procedures for those cases that remain unresolved. Many of the most common conditions responsible for poor performance can be ruled in or out quite rapidly through undertaking a clinical evaluation (trot-up in hand, limb/topline palpation and heart auscultation), resting endoscopy +/– wind-test, and routine blood sampling. Beyond these preliminaries the diagnostic workup is likely to become wide-ranging. Economic considerations will typically guide how far the 'atypical' poor performance case is investigated; in many cases once obvious upper airway obstruction or musculoskeletal pathology is ruled out the trainer/owner may direct that further diagnostic costs are not warranted.

History

History is a vital part of any poor performance investigation and obtaining a thorough background to the case from the trainer and training staff can strongly influence the success or otherwise of the initial workup. Details such as racing form, length of time trainer has been concerned about horse, previous veterinary interventions and prior use of tack to aid upper airway function are important. Whether poor performance has been restricted to racing or also encompasses training gallops, has been associated with other signs of interest (poor recovery, inappetence, weight loss) or jockey concerns about respiratory noise or action all contribute to direction of any subsequent investigation. It should also be established whether indeed the trainer has a genuine concern about performance or whether veterinary input has been requested to satisfy an owner's or jockey's concern.

Clinical evaluation

Initial clinical evaluation need not be detailed or lengthy. Observation at walk and trot (+/– under tack at faster paces) is sufficient to detect any 'significant' (or uncharacteristic) lameness; in combination with palpation of limbs and topline the likelihood of a musculoskeletal cause can usually be established. While lameness investigation and diagnostic analgesia may have a role in some poor performance investigations, background knowledge of the horse's 'baseline' action and previous interventions are just as important (as presence of lameness is per se is not necessarily relevant to performance). Auscultation of heart and assessment of body condition also form part of clinical examination.

Diagnostic procedures

Blood testing

Routine haematological and biochemical screening can be used to identify whether a horse has a current or recent (past few days/week) infectious or inflammatory condition, whether they have suffered an episode of exertional rhabdomyolysis ('tying up') in the past few days or whether some key markers of potentially important medical conditions (e.g., elevated gamma-glutamyl transferase [GGT] syndrome; protein malabsorption) are present. Biochemistry analysis should include acute phase proteins (fibrinogen, serum amyloid A [SAA]) otherwise detection of inflammation/infection is compromised. Haematology cannot be used to judge fitness or athletic potential of a racehorse and red and white blood cell figures, even if outside reference range, should be interpreted with caution.

Assessing the airways

Resting endoscopic examination of the upper airway and trachea is preferably undertaken immediately following exercise, to permit full assessment of the possible presence of lower airway inflammation/infection or EIPH. Most upper airway obstructions are dynamic; however, resting endoscopy can give a good initial indication of whether overt laryngeal dysfunction is present. 'Wind' testing (detection of audible abnormal respiratory sound) at exercise is also a useful and low/no-cost procedure during the initial examination. Definitive diagnosis of dynamic upper airway obstructions (and their severity/complexity) necessitates overground or treadmill endoscopy.

Sampling of lower airway secretions may have a place in poor performance investigation. Tracheal and bronchoalveaolar lavage each have strengths and weaknesses; however, in general it can be stated that the more a diagnosis relies on microscopic/histological (rather than macroscopic) evidence of lower airway disease or EIPH the less likely it is to fully explain the poor performance being investigated. Respiratory samples from most 'normal' racehorses in full training have histological evidence of lower airway inflammation +/– EIPH.

Assessing the heart

While auscultation of the heart is readily performed and should be part of any initial evaluation, when arrhythmias are linked to poor performance they are usually transient or intermittent and may not be detectable at rest. Use of electrocardiogram (ECG) during training session (and for variable periods at rest) is preferred. Interpretation

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requires caution: intermittent premature complexes during and immediately after exercise are common in normal horses and may not always be significant unless frequent or associated with tachycardia (Table 10.2, p. 314).

Bone scan

Musculoskeletal conditions significant enough to cause poor performance are usually clinically apparent at lameness assessment. Even when lameness is present, the plausibility of association with poor performance should be judged on factors such as anatomical origin of pathology, chronicity and recent trajectory of lameness and whether exacerbated by high-speed exercise. It may sometimes be warranted to perform a screening bone scan on a horse free from overt lameness to determine whether any significant subclinical injury may be present or developing; injuries such as pelvic or lumbar stress fractures are not always associated with lameness yet can be linked to unexpected poor race outcomes.

Gastroscopy

Gastroscopy and other medical diagnostic procedures may be considered if initial musculoskeletal and respiratory assessments prove unrewarding. Detection of moderate to severe degrees of gastric/duodenal ulceration warrants commencement of anti-ulcer medical therapy. As ulceration is commonplace in horses in training, it is generally necessary to assess response to treatment (over initial weeks) before it can be confidently held responsible for the reported poor performance.

Assessing nutrition

Assessing diet and nutrient intake (p. 449) may be warranted for horses that have poor condition or fail to thrive. When undertaking a nutritional review the current or prior use of any supplements should be noted; overuse of supplements is common (particularly in horses that the trainer is unhappy with – whether condition or performance) and may lead to over fortification or competitive malabsorption of some nutrients.

MANAGEMENT

If investigation has resulted in definitive or presumptive diagnosis of a particular condition, management advice/treatment will be determined by that diagnosis. As strength of diagnosis diminishes, it becomes more important to follow up on whether any trial intervention(s) are resulting in clinical or athletic recovery; depending on the suspected condition, this may take the form of either observing subsequent racetrack performances or repeating the diagnostic test(s) (blood analysis, gastroscopy, etc.) that led to provisional diagnosis.

The greatest challenges lie with the management of underperforming horses for which it has not been possible to arrive at a diagnosis. In these cases it is generally best that the trainer/owner determines whether to pursue any trial interventions, rest or continue training the horse or dispose through public auction. All of these options have potential costs and uncertainty of outcome, ranging from lost training time, financial cost of continued training, diminished residual value with further unsatisfactory performances and even risk of horse regaining form after moving training yard.

When reason for poor performance remains unknown, expectations regarding likelihood of success with any intervention should be managed. Common recommendations include a period of time either out of training or out of full exercise; this is a rational approach for horses thought to be experiencing an ‘overtraining’ syndrome; however, there are currently no validated guidelines regarding optimal length or type of rest period and it is more likely that individual circumstances (stage of season, further racing options) will be the primary influence on such an approach.

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HERD HEALTH

BIOSECURITY

Although the racehorse population is highly mobile, continually mixing and housed intensively, the risk of serious disease transmission is generally considered to be low. This can be attributed to several factors: that horses attending races are by definition healthy enough for competition; that standards of husbandry in racing yards (including veterinary attendance and routine vaccination) are usually high; and that health testing and certification implemented by studs, public sales companies and border control authorities limit the spread of economically important diseases. When serious infectious disease does occur, however, the financial impact can be severe, and it is advisable for racing yard management to incorporate some basic biosecurity measures. These may include:

- Regular disinfection (steam cleaning followed by application of approved virucidal/bactericidal disinfectant) of horse transport and communal facilities.
- New arrivals from high-risk populations housed in isolation area (and exercised separately to core group) until health status established: period of 2–3 weeks is optimal (or until test results available).
- Vigilance for signs of disease (fever/nasal discharge/enlarged lymph nodes) in new arrivals.
- In the absence of an isolation facility, consider testing horses prior to/upon arrival to detect animals with subclinical disease.

The risk of disease transmission (including respiratory disease and fungal skin infection) is greatest at times of year when high levels of population mixing occur, such as during and following autumn public sales. Vaccination programmes should take this into account and biosecurity measures (separation of new arrivals, disinfection of tack) used to minimize spread to resident horses nearing race targets.

Quarantine

Quarantine is the strict isolation of a group of horses in order to establish or maintain their health status prior to integration with a larger population. The two main circumstances in which racing yards may encounter quarantine are pre-export isolation prior to international shipping and quarantine within a yard in response to a suspected or confirmed infectious disease outbreak. Pre-export quarantine requirements are set out in the statutory regulations of the receiving country; typically, this form of isolation takes place on dedicated premises away from the normal operation of the training yard.

Quarantine procedures instigated within a yard due to a disease situation vary depending on the disease involved, extent of spread and practicalities determined by location and setup of the yard. The most common diseases prompting quarantine in yards are neurological herpesvirus, strangles and infectious diarrhoea. Length of time required for quarantine, testing procedures and the rigour with which isolation is enforced are dictated by local codes of practice applicable to the disease in question (see Chapter 17). The goals are to limit spread to other horses or premises and to determine whether recovered horses are free from disease before release into the wider population. General principles relating to quarantine are discussed next.

Population at risk

- Initial action: divide yard population into risk groups; (1) horses with (or suspected to have) disease, (2) direct or close contacts of infected horses (physical contact or sharing facilities) and (3) low-risk group with no known contacts.
- Separate management of these groups with immediate effect, the goal being to move horses from in-contact group to low-risk group as they are cleared through testing.
- Testing procedures (blood sampling, nasopharyngeal or faecal swabbing) appropriate to the specific disease, to determine exposure and infection status of all contacts. Testing may typically be conducted at weekly/bi-weekly intervals to allow for seroconversion and incubation periods.
- High vigilance to signs of disease within at-risk population (daily/twice-daily monitoring of rectal temperature, feed intake, etc.).

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Movements

- Depending on disease: all movements onto or off yard should cease from the time that disease is confirmed or strongly suspected, with a movement ban remaining in place until the disease status is confirmed. May have major impact on racing yard operations; however, ramifications of (and liability for) not implementing a movement ban is far greater if infection spreads through racetracks or auction ring.
- In certain situations (particularly where there are few or no in-contacts) it is permissible to consider moving infected individual immediately off-site to a separate isolation facility, providing there is full disclosure to the receiving party. This may allow for more appropriate (and lower risk) management of an infected individual; most relevant for horses requiring hospital-based care (e.g., severe colitis). Important to follow through with movement ban, disease surveillance and testing in remainder of yard population.

Communication

- Communication with neighbouring yards and local trainers'/breeders' associations essential to enhance vigilance for disease and limit spread in locality, particularly when public training grounds are involved.
- In many jurisdictions there is a statutory requirement to notify national or state racing authority of suspected or confirmed infectious disease in training yard.
- Communication with yard staff and riders to combat misinformation and limit risk of spread to other horse populations.

Isolation procedures

- Inevitably involves some adaptation of 'textbook' procedures to the particular circumstances of yard.
- Infected horses should be housed >25 m (100 m for pre-export quarantine facilities) from other horses to minimize risk of airborne spread of disease.
- Isolation procedures should apply to exercise routes as well as stabling; if exercised on grounds accessible to other horses, a distance of at least 100 m should be maintained.
- Isolation area should ideally have a separate water supply and waste bedding destroyed/removed in manner that poses no risk of disease spread
- Perimeter of isolation area should be clearly marked so that accidental entry by unauthorized personnel does not occur (may necessitate locking up after-hours).
- Designated staff to attend to horses in isolation area. These should be staff that have no other duties on remainder of premises for the duration of quarantine. If contact is required with non-isolation horses, this should take place before duties in isolation area are commenced (with no return later in day).
- Protective clothing/boots kept at and worn in isolation area; disinfectant footbaths (continually replenished) and hand-wash at isolation perimeter to be used on entry and exit.
- Dedicated equipment (grooming, handling, riding) for isolation area, with no crossover of equipment to remainder of premises.
- Logbook to track entry of personnel and approved visitors (vets, farriers) to isolation area.

HOUSING AND AIR HYGIENE

Air quality

Air quality, measured by respirable particulate matter, can influence respiratory health. Inhalation of high concentrations of dust particles of a certain size (<5 μm diameter) has potential to cause respiratory inflammation or slow recovery from respiratory tract infections. In the stable environment, fungal spores are the major contributor to respirable dust; however, other organic particles and endotoxins may also have a significant impact on lower airway health. The concentration of these particles at any one time is determined by release of dust from feed and bedding, and its rate of clearance is determined by ventilation. In yards with good management and adequate ventilation, there is little difference in dust release rates between straw, wood shavings or paper bedding (although quality of straw and shavings can vary between suppliers). Airborne particulate concentrations in the stable environment can vary markedly both throughout the day in response to horse and human activity (mucking out, feeding, horse movement), and between seasons (higher in winter than summer). Baseline levels of respirable particle concentrations may exceed recommended ambient air quality guidelines for humans with consequent implications for workplace safety. Air quality of indoor (or covered) rides where horses undertake warm-up exercise may also be a factor in respiratory health and attention to track maintenance and watering is important.

Ventilation and housing

Stable and yard design have a major influence on air quality. Most racehorses are stabled either in loose boxes or in barns with shared airspace. There is some evidence that racehorses housed in loose boxes with sufficient ventilation are less prone to lower airway disease than those stabled in barns. Optimum ventilation rates when dust release from

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feed/bedding is not excessive should be ≥4 air changes/hour. In calm conditions loose boxes achieve good ventilation rates (6–7 air changes/hour) providing the top door is open (ventilation rates usually unsatisfactory with door closed). Barn ventilation is satisfactory even in still conditions with doors open at each end, although air quality varies with location within the barn (worse in middle and adjacent to doors). With the end doors shut, traditional barn ventilation is poor. Ventilation of barns is assisted by air movement caused by convection, with warm air exiting through ventilation spaces high in the walls or ceiling. Convection is less of a factor than in the more intensively managed housing of farm animals; however, insulation of walls and roof can improve its effectiveness and reduce the need for large ventilation inlets. Assisted/mechanical ventilation of barns may permit greater control and improvement of air quality. Proximity of (and relationship of wind direction to) muck heaps and hay storage areas should also be considered when assessing a yard for respiratory hazards.

Minimizing respirable dust

Although adequate ventilation is an important factor in respiratory health, it does not always reflect air quality in the 'breathing zone' of the stabled horse. A large proportion of inhaled dust particles (including fungal spores) comes from hay/forage. Immersion of hay in water immediately prior to feeding can reduce this load by more than 90%; brief immersion ('dunking') is preferred, as nutrients are leached if forage is soaked for too long. Alternatively, haylage or steamed hay can be fed.

Yard management may be used to minimize inhaled dust loads. Dampening passageways before sweeping can reduce dust levels. When bedding is disturbed during cleaning out of a stable, the concentration of dust particulate matter in the air rises markedly. This effect is greatest with straw; however, regardless of bedding type the period of time around stable cleaning and laying down of new bedding is associated with poor air quality. Failure to remove soiled and wet bedding leads to rapid fungal growth and increased ammonia levels, which may cause respiratory irritation.

LOWER AIRWAY DISEASE

See also Chapter 9, p. 303.

The following points are relevant to the management of respiratory disease in racing yards:

- Lower airway inflammation/infection (characterized by increased tracheal mucus and coughing) is common and generally self-limiting.
- Causes are multifactorial and incompletely understood.
- Bacterial infection (either primary or secondary) is thought to play a large role in development and persistence of excessive tracheal mucus.
- Bacterial species involved are endemic in the normal population, therefore elimination of exposure is impractical.
- Viral infection is generally less important than bacterial infection.

Investigation

Onset of concern about a wider health problem in a yard is often insidious and may arise from increased frequency of coughing, 'dirty' endoscopy results or poor racetrack performances over a period of weeks. Successful management should aim to minimize lost training days and economic cost; appropriate treatment relies on the quality of information regarding prevalence and severity of respiratory disease:

- Background information: frequency of coughing/fevers/poor performers in recent weeks.
- 'Batch' scoping: best way to assess yard prevalence of lower airway disease; random sampling or entire group(s) (coughing is an insensitive indicator of disease).
- Determine whether particular groups (e.g., 2 YOs) are most affected.
- Clinical, endoscopic and laboratory findings: generally (but not always) possible to determine whether respiratory disease has an infectious component or is primarily related to air hygiene.
- Blood sampling is of little use for the detection of respiratory disease.

Management

- Approach to treatment dictated by stage of season/extent and severity of disease/economic cost.
- Respiratory disease is common in yearlings/2 YOs during first months in training environment; at these times it is often preferable to restrict treatment to cases that do not resolve naturally (or to limit wider spread within yard).
- Treatment: either targeted at individual horses confirmed with respiratory disease (through endoscopy), or medication of target groups with high levels of disease.
- Group medication: may be appropriate if guided by diagnostic work and administered responsibly; however, compliance with antimicrobial stewardship guidelines is of primary importance. May reduce

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shedding/transmission of bacterial pathogens and speed up yard recovery.
- 'Re-treats' (horses that fail to respond to initial course of medication) targeted for further investigation.
- Influence of continued exercise on persistence of lower airway disease has been poorly investigated: common advice is to refrain from fast work/racing while moderate-marked levels of excessive mucus are present in trachea. Continued light cantering is not considered to be detrimental to recovery.
- Routine endoscopy prior to race confirmation/declaration during period of concern, to establish respiratory health status of horses with race targets.
- Air hygiene problems should be addressed where practicable or if respiratory disease becomes a persistent concern.
- Immunostimulants: some evidence to support use of non-specific immunostimulants (inactivated *Propionibacterium acnes*/inactivated *Parapox ovis* virus) as adjuncts to conventional therapies; may assist recovery/reduce severity.

MUSCULOSKELETAL INJURIES

Unusually high rates or patterns of injury may prompt investigation of the overall musculoskeletal health of a yard. For meaningful conclusions to be drawn, assessment should be based on objective information rather than reaction to a recent injury cluster.

Quantifying injury rates
- Number and type of injuries over a set time period has to be determined and then assessed in the context of number of horses at risk.
- Minimum time period examined should be months/years, as prevalence of injury can fluctuate considerably through racing season.
- Injury incidence rate is best measured against the number of 'horse-training-days at risk'; however, calculating this figure (number of days training at different paces for each horse) is rarely possible in practice.
- More workable approach: analyse number of injuries in relation to average number of horses in work or yard.

Comparison of injury rates
- 'Normal' injury rates vary between training centres/types of racing and published research (to utilise as comparator) is limited. Differences in study design and diagnostic criteria mean that it is currently not possible to reliably/accurately compare injury rates between training centres/racing jurisdictions. A guide to expected annual number/type of injuries in an average UK flat racing yard extrapolated from existing publications and clinical practice is found in Table 21.1.
- Disregarding sore shins, splints and accidental injury, approximately 20–25% of a yard population can be expected to sustain some form of musculoskeletal injury during a calendar year.
- Fracture incidence rate in training is approximately 1/100 horse-months (i.e., on average 1 fracture/month can be expected for every 100 horses in training). Accounting for irregular distribution throughout the year, this may equate to several fractures/month during high-risk periods.
- Availability of good records may allow comparison with previous seasons.

Analysis of injury types
- Patterns of injury vary between trainers/training centres and reflect many factors including training regime track, and horse and jockey variables.
- Level of yard vigilance for early orthopaedic injury may be indirectly assessed by severity of stress injuries at time of diagnosis/detection: high prevalence of 'end-stage' stress fractures (those requiring surgical repair or euthanasia) is indicative of poor detection or failure to react to early signs of injury.

Analysis of risk factors
- Many factors can influence risk of orthopaedic injury (see Chapter 3, p. 31) and horses may be exposed to these many months before a clinical problem is recognised; assessing underlying causes of injury clusters is frequently complex and dependent on availability of detailed exercise history.
- Trend towards greater prevalence of hindlimb stress injuries with heavy use of uphill tracks (greater relative loading of hindlimbs).
- Risk of particular injuries varies with age profile; yard with a large proportion of 2 YOs might expect to have a greater prevalence of sore shins, tibial stress fractures or proximal metacarpal lameness than a yard with predominantly older horses.
- Firm ground (turf) consistently associated with higher risk of fractures during training high-speed exercise.

Minimizing injury rates
See also Chapter 19.
- Reduction (but not elimination) of injury risk may be possible in many circumstances.

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Table 21.1 **Estimated* incidence of clinically significant musculoskeletal injuries (training + racing) for an average Newmarket (UK) flat racing yard of 100 horses†**

INJURY	ESTIMATED INCIDENCE
Tibial stress fracture	3–4 per year
Carpal fracture/fragmentation	2–3 per year
Clinically evident POD:	
• bilateral hindlimb lameness (uninterrupted training; intervention optional)	4–10 per year
• unilateral forelimb/hindlimb lameness (uninterrupted training possible, but may require imaging/intervention/rehabilitation)	2–3 per year
• unilateral end-stage (untrainable: collapsed subchondral bone plate and articular involvement)	1 every 8–10 years
Pelvic stress fracture	4–5 every 2 years
P1 fracture ('split' pastern)	4–5 every 2 years
MC3/MT3 condylar fracture (including prodromal)	2 per year
Suspensory ligament branch desmopathy	2 per year
SDFT strain	1–2 per year
Suspensory ligament (proximal or mid-body) desmopathy	1–2 per year
Forelimb proximal limb stress fracture (humerus, radius)	1 per year
Tarsal fracture (T3/CTB/proximal MT3)	1–2 every 2 years
Diaphyseal cannon fracture (fatigue proximal MC3/dorsal cortical/transverse)	1–2 every 2 years
Proximal sesamoid bone fracture	1 every 2–3 years

* Extrapolated from Ramzan PH, Palmer L (2011) Musculoskeletal injuries in Thoroughbred racehorses: a study of three large training yards in Newmarket, UK (2005–2007) *Vet J* 187(3) 325–329; Verheyen KL, Wood JL (2004) Descriptive epidemiology of fractures occurring in British Thoroughbred racehorses in training. *Equine Vet J* 36(2) 167–73; and unpublished data.

† Excluding dorsal metacarpal disease (sore/bucked shins) and accidental/traumatic injuries.

- Conditioning early in life (foal/yearling), nutrition and conformation may be associated with risk of particular injuries and should guide selection of yearlings.
- Chronic nutritional imbalances (particularly of calcium:phosphorous ratio) can lead to skeletal weakness and increase injury risk; this is usually readily determined through analysis of the yard diet.
- Inappropriate training goals/methods applied to late-maturing horses or those of particular physical types should be avoided.
- Optimal training strategies to minimise injury risk have not been validated; however, it is known that stress fractures result from failure of skeletal repair processes to keep pace with workload. High stress injury rates may warrant consideration of longer intervals between fast exercise days or strategic use of low-intensity training periods to coincide with the peak risk (early fast-exercise) phase of training. Introduction of small cumulative training loads of fast exercise early in career, followed by a 'spell' rather than unbroken progression through to racing, may also be beneficial.

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CHAPTER 22

NUTRITION

OVERVIEW

Digestive anatomy

The horse is a grazing animal with a gastrointestinal tract that has evolved to digest fibrous plant material (principally grasses) consumed continuously throughout the day. Ingested roughage is first broken down through the powerful cutting and grinding action of the cheek teeth, with the swallowed bolus moving down the oesophagus assisted by the production of large volumes of saliva (up to 40 L/day in a grazing horse), which acts both as a lubricant and an alkaline buffer to the acidity of the stomach. The stomach of the horse is relatively small (8–15 L capacity) and here food is mixed with acidic gastric secretions and then discharged into the small intestine, where digestion begins. The rate of gastric emptying is determined by the type of ingesta and meal size: liquids pass through the stomach faster than solids, as do larger meals. Passage of food through the small intestine is also rapid, but it is here that exposure to enzymes produced by the pancreas and small intestine permit the digestion of non-structural carbohydrates (such as starch), proteins and fats; the small intestine is also the site of absorption of most trace elements, minerals and vitamins.

The hindgut of the horse comprises the caecum, large and small colons and rectum and has a very large capacity, containing up to 135 L of liquid. Unlike the small intestine, digestion in the hindgut does not come about through the action of mucosal enzymes; rather, it is through the activity of a large resident population of microbes ('microbiome': primarily bacteria, but also fungi and protozoa), which ferment fibre leading to the production of volatile fatty acids.

Principles of feeding

The diet of the modern athletic horse has diverged markedly from the natural grazing state for which the digestive tract has evolved. High energy demands and permanent stabling whilst in training necessarily means that grazing has been replaced by smaller volumes of preserved forage (hay/haylage) and the majority of nutrients are instead derived from energy-dense concentrates.

Concentrates are primarily cereal based. The starch and fats that comprise the majority of energy in these feeds are largely absorbed in the foregut (small intestine), while the hindgut is the site for digestion of fibre. The speed of transit of food through the small intestine and the relatively low levels of amylase (the enzyme required for starch breakdown) in pancreatic secretions mean that the capacity to digest starch before it reaches the hindgut is limited: diets high in concentrates may therefore result in 'spillover' of starch into the hindgut and subsequent changes in the microbial population of the hindgut may cause diarrhoea or colic. It is therefore necessary to split the daily concentrate ration into three or four separate feeds to avoid overlarge meal size. Because a large part of the horse's digestive function relies on the hindgut microbial flora, dietary changes should be made gradually to allow adaptation to occur.

Feeding strategies vary widely between trainers and derive from tradition, personal preference, local availability of feed and economic factors. When a diet is grain based ('straights'), a 'balancer' (concentrated source of protein/vitamins/minerals) is usually fed to ensure that nutritional imbalances do not arise. Alternatively, complete feeds offer the advantage of being formulated to provide all major nutrients in a single, uniform product. It matters little how the ration is fed providing the diet is balanced (not over- or underfortified in key nutrients) and feed intakes and health/performance outcomes are satisfactory. Energy requirements to maintain body condition are directly correlated to expenditure through exercise and, while small adjustments in training intensity have a limited effect on energy needs, a horse in active racing competition will generally require 10–15% more energy intake than a horse in full training that is not racing.

KEY NUTRIENTS

Carbohydrates

Carbohydrates utilized by the horse derive primarily from starch (from concentrates) and digestible complex carbohydrates found in forage. Starch is broken down by enzymes in the small intestine into sugars (glucose) that are readily absorbed across the gut wall into the bloodstream.

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Proteins

Proteins are made up of amino acids and form the building blocks of the body; they are needed for maintenance and growth. The horse can synthesize some but not all of its required amino acids and must source the remainder from dietary protein. Proteins are broken down by enzymes in the small intestine into their constituent amino acids. Protein quality and digestibility are important factors in the value of a protein source: higher quality protein is that which conforms closest in amino acid makeup to that required by the tissues and is digestible in the small intestine. Protein content is usually reported as crude protein (CP): this is an indirect estimation of protein content based on the amount of nitrogen present and is not a measure of digestibility, quality or amino acid composition.

Protein requirements increase only by a moderate amount with full work; it is rarely necessary to supplement protein as increased ration intake with workload usually covers losses. High protein does not equal high energy, and feeding too much protein may be detrimental to performance.

Fats

Although fats/oils make up a very small proportion of the horse's 'natural' diet, their digestion is surprisingly efficient. The small intestine is the main site of fat digestion and absorption.

Fibre

Much of the horse's natural diet is made up of fibre: substances that give structural support to the cell walls of grasses and forage include complex carbohydrates (cellulose and hemicellulose) and lignin. Lignin is an indigestible component and as plants mature lignin content increases. As with ruminants, the horse does not have enough innate enzymatic activity in the gut to break down all the insoluble fibre that passes through it into useable molecules. For this it relies on a large population of microbes in the hindgut to ferment dietary fibre into volatile fatty acids, which are then absorbed and used as an energy source. Fibre content of forages influences digestibility and feed intake, and higher fibre fraction generally equates to lower available energy. Fibre and its fermentation draw water into the hindgut: high-fibre diets lead to greater fluid content of the digestive tract and consequently greater bodyweight (BWT), a potential influence on athletic performance.

Electrolytes

Electrolytes are water-soluble salts and minerals that control the fluid balance of the body and have key roles in nerve and muscle activity, energy metabolism and many cellular functions. The primary electrolytes are sodium, chloride, potassium, calcium and magnesium. Sweat contains salt and as a consequence the exercising horse loses sodium, chloride and potassium. Other than calcium, the body has little in the way of stores of electrolytes and must replenish losses on a daily basis.

Minerals

Minerals are inorganic nutrients and include the previously mentioned electrolytes as well as phosphorus, sulphur and trace minerals (iron, zinc, copper, selenium, iodine, manganese, cobalt) that are needed for normal growth and development. Mineral requirements are typically small and vary with workload and age. Absorption and utilization of trace minerals is complex, with dietary source and interactions between certain minerals being as important as absolute intake. One of the key interactions relevant to the racehorse is that between calcium and phosphorus. The body's reservoir for both minerals is bone, and if the dietary ratio is inappropriate calcium can be lost from bone to maintain blood levels, resulting in reduced skeletal strength and increased injury rates. Cereals are high in phosphorus, and use of an appropriate balancer +/– forage (frequently high in calcium) is required to ensure that the calcium:phosphorus ratio is within optimal range (1.2–2:1). Most commercial complete feeds are formulated to have a balanced calcium:phosphorus ratio.

Vitamins

Vitamins are organic compounds that are essential for many cellular processes. Vitamins are required in very small quantities for maintenance of health; however, little is known of the ideal dietary requirements of the racehorse. Many diets and supplements are fortified with vitamins based on the premise that if particular vitamin deficiencies are detrimental, then supplementation must be beneficial, without accounting for normal daily intakes. The body is able to store fat-soluble vitamins (A, D, E and K), allowing some flexibility with regard to daily intakes, but overfortification can occasionally lead to toxicity. Water-soluble vitamins by contrast are not stored but many are produced within the body (B vitamins by microbes in the hindgut, vitamin C from glucose in the liver), and overfortification simply results in excretion through the kidneys rather than toxicity. B vitamins have important roles in energy metabolism and horses in full work may have increased requirements, meriting some supplementation. The benefits of vitamin C and, to a lesser extent, vitamin E supplementation are controversial, with mixed evidence of antioxidant effects; bioavailability of nutritional sources may also vary considerably.

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FEED TYPES

Cereals

Cereals are rich in starch and contain more digestible energy (DE) than forage. They are relatively deficient in calcium. Cereal types differ in their energy and nutrient content and digestibility. One of the primary goals when feeding cereals is to optimize digestibility in the small intestine and limit potential spillover of starch into the hindgut. Processing achieves this by increasing digestibility:

- Oats: grain size, palatability, digestibility and good-quality protein make oats a safe cereal to feed unprocessed.
- Naked oats: varieties that lose husk readily so therefore have higher energy and oil content than normal oats. 30% higher DE than oats.
- Barley: hull is more closely adhered than that of oats and therefore requires rolling/crimping.
- Wheat: requires processing. Very high starch content so less safety margin than oats.
- Maize (corn): double the energy density of oats but less readily digestible in foregut, therefore greater fermentation in hindgut (less desirable); processing (cooking/extrusion) improves digestibility and is preferred.

Other common feedstuffs

- Wheat bran: produced from husk of wheat grain. CP 14–16%. Rich in phosphorus and deficient in calcium. Palatability and ability to absorb water make it a common choice as mash.
- Rice bran: fat supplement. Should be heat-stabilized to prevent spoiling.
- Sugar beet pulp: highly digestible fibre. Similar protein level and quality to cereals.
- Linseed meal: protein supplement (CP 20–35%) with high omega-3 fatty acid content.
- Soybean meal: protein supplement (CP 45–50%).
- Lupins: protein supplement (CP 28–34%); digested predominantly in hindgut. Usually fed in processed form.

Oils

Digestion of oil/fats is very efficient and takes place entirely in the small intestine. Vegetable oils are energy dense, containing approximately three times the DE of oats. It is therefore possible to increase the energy density of the diet without increasing the volume of feed intake; substitution of oil for starch may also be beneficial to gut function. Oils should be introduced gradually, but total dietary content of 10% oil usually well tolerated. The typical quantity top-dressed to racing diets is 50–150 mL/day.

The balance between levels of two types of dietary fats (omega-3 and omega-6 fatty acids) in the diet has potential importance for the horse: the natural grazing diet is relatively higher in omega-3 fatty acids, whereas omega-6 usually dominates in concentrate-based diets. Oils found in most cereals are rich in omega-6 fatty acids, whereas a few cereals (linseed, rapeseed and soya) algal and fish oils are rich in omega-3 fatty acids. There are some possible anti-inflammatory benefits from a diet relatively high in omega-3 fatty acids; although optimal levels have not been determined, algal and fish oils, linseed (flaxseed) meals or use of linseed/soya oil are therefore preferred to other sources of fat.

Electrolytes

As electrolytes are not stored in the body, losses through sweat and urine should be replenished on a daily basis. Most electrolyte requirements are met by feeding a balanced diet. Sodium and chloride requirements are influenced by workload and temperature/humidity, but are usually covered by addition of salt to the diet or provision of salt licks/rock salt. Contents of commercial electrolyte products should be scrutinised as many contain large proportions of sugars and relatively low levels of electrolytes.

OTHER SUPPLEMENTS

Dietary supplements other than those required for normal nutritional needs are commonly fed to racehorses in training, for a variety of reasons that may include support of particular aspects of health, behaviour or athletic performance. The rationale for adding supplements to any diet may range from provision of micro- and macronutrients to correct an underfortified normal ration (or meet enhanced needs arising from intense training), supporting direct and indirect pathways to enhanced athletic performance, and assisting recovery. A significant influence on trainers is also peer pressure and marketing; many supplements are introduced into a yard based simply on knowledge that other trainers are using them and without veterinary advice or critique of actual potential for benefit. It is rare for the use of such supplements to be supported by solid scientific data specific to the horse, and in most cases the rationale for use and dosing advice is extrapolated from other species. Assessing usefulness is challenging, as even for common nutrients there is no clear scientific agreement on what threshold level is considered optimal for performance. In both human and equine sports it is generally considered that among the potential factors involved in successful athletic

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performance the contribution of dietary supplements is minor, and the strength of evidence to support marketing claims is usually poor.

As most supplements are non-essential and have limited scope for significantly enhancing performance, a pragmatic risk-benefit approach should be taken when determining whether to use them. Important considerations include current dietary intake, legality, safety, likely efficacy, cost, appropriateness for stage of training/maturation, country of origin and risk of contamination with prohibited substances.

Probiotics

Probiotics are live microbes (bacteria and yeasts) used as dietary additives to provide possible beneficial effects beyond that of their nutritional value, principally through influence on microbial population of hindgut. Possible roles include inhibiting growth of intestinal pathogens and enhancing digestion. Bacterial families that produce lactic acid (lactobacilli), and *Saccharomyces* (yeasts) are commonly used. Although used widely and generally considered to be harmless, most products do not deliver the claimed type or quantity of viable organisms and their efficacy is doubtful. Overall evidence to support use (any product, in any situation) is weak.

Prebiotics

Prebiotics differ from probiotics by being composed of nutrients that assist the growth of 'beneficial' microorganisms, rather than containing the microorganisms themselves. They consist mostly of non-digestible sugars (oligosaccharides) and fibre. Efficacy uncertain and generally considered safe although some evidence that they may potentiate the development of gastric ulcers.

Enzyme-rich malt extract

The intestinal wall of the horse cannot absorb complex carbohydrates. In order that starch (a complex carbohydrate) is digested before arriving in the hindgut (where it can lead to deleterious microflora changes) it must be broken down into simple sugars (monosaccharides) as it passes through the small intestine. This occurs through the action of an important salivary and pancreatic enzyme, amylase; however, levels of amylase in the small intestine are low and easily overwhelmed by the high starch content of concentrate diets fed to racehorses. Enzyme-rich malt extract (ERME) comprises a high concentration of amylase along with other potentially beneficial enzymes, is highly palatable and when used as a daily dietary supplement appears to have beneficial effects both on efficiency of pre-caecal starch digestion and on hindgut digestion.

Antioxidants

Intense exercise (among other internal and external challenges) generates unstable molecules termed 'free radicals', which react readily with other molecules in the body through the process of oxidation. A balance between free radical production and the body's antioxidant defences is necessary for normal physiological function, and if these regulatory processes are overwhelmed then cell damage may ensue ('oxidative stress'). Antioxidants are molecules that utilise a variety of mechanisms to interfere with free-radicals' ability to cause cellular 'chain reactions', through inhibition of their production, 'scavenging' of unstable molecules or repair of the damage caused by them. Widely recognised antioxidants include micronutrients such as vitamins E, C and beta-carotene, and the use of supplements containing them (either as fortified single/multi-vitamins, or in a range of extracts and juices) is common in human and equine sports. Bioavailability of some micronutrients strongly depends on source (including natural/synthetic) making direct comparison of supplements/feedstuffs difficult in many cases. Turmeric (curcumin) is also widely incorporated in human and equine supplements for a range of purported antioxidant and anti-inflammatory effects, but bioavailability is poor and efficacy unlikely and there is no current scientific rationale for use in racehorses.

While oxidative stress can potentially impair athletic performance through a range of effects such as muscular damage and fatigue, the case for supplementation with antioxidants to counteract these is not straightforward. Oxidative stress is known to be an important signalling mechanism to stimulate training adaptations, and chronic antioxidant supplementation can therefore interfere with the 'anabolic' effects of training and recovery from exercise. High-dose antioxidant supplementation has also been found not to reduce muscle soreness after exercise.

Biotin

Biotin is a water-soluble B vitamin. It can improve the speed and quality of hoof growth with long-term supplementation. Supplementation at 15–25 g/day is recommended for horses with poor hoof quality or foot balance; the effect is on new hoof growth and positive changes therefore take months to be noticeable.

FORAGE

Although much attention is given to concentrates, forage (in the form of hay or haylage) typically contributes between 20% and 35% of the total energy in a racehorse's

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diet. Quality and daily consumption of forages can vary widely with season and geographical location, and variability in forage supply even for the same racing yard can have consequences for nutrient intake that may go undetected until secondary problems arise much later. An understanding of nutrient makeup of forages and how to assess quality is therefore an important component of racehorse nutrition.

Types of forage

Hay

Grasses (timothy/ryegrass/orchardgrass/bluegrass) and legumes (alfalfa) are cut and allowed to air dry/wilt to produce hay. Energy density and cleanliness (from dust and microbial spores) are largely determined by maturity and weather conditions at the time of cutting. Leaves are more nutrient rich than stems, and forage cut at a later stage of maturity (when it has greater stem) has a lower energy and protein content and more indigestible fibre. Dry matter (DM) content of hay should be approximately 85–90%; moisture levels greater than this (<85% DM) may permit fungal spoiling. The nutritional value of most hay does not diminish significantly with storage.

- Grass hay (timothy/ryegrass/fescue): CP levels 8–10% for average quality hay.
- Legume hay (alfalfa): CP levels 15–18% for mid-bloom; greater DE and calcium content than grass hays. Leaves may shatter more readily than grass hay, causing reduction in nutrient density and greater respirable dust.

Chaff

The main purpose of feeding chaff is to slow down the intake of concentrates. Chaff made from alfalfa or good-quality forage is preferable to that made from straw.

Haylage

Grasses cut for haylage are baled when wilted (rather than completely dry) and have greater moisture content than hay (55–70% DM). Bales are compressed and sealed to allow natural fermentation to occur; this process limits mould contamination. Haylage can markedly reduce levels of exposure to dust and is more palatable than hay, although digestibility does not differ substantially (if comparing hay and haylage made from the same crop).

HAY OR HAYLAGE?

- Neither hay nor haylage are inherently superior as the forage component of a racehorse diet: each has advantages and disadvantages.
- Choice of which to feed depends on many factors particularly quality, consistency and relative cost of available supplies.
- Lower dust content of haylage may be beneficial for respiratory health, although steaming hay can be used to same effect.
- If made from same/similar grasses, levels of key nutrients (CP, inorganic mineral/ash, fibre) are broadly similar.
- Main nutritional difference is that water-soluble carbohydrate (WSC) levels in haylage are lower than in hay; fermentation that occurs during storage process uses up sugars.
- Main practical difference is that for a horse to get the same quantity of nutrients from a haylage ration as from comparable hay, it must be fed 25–50% more haylage by weight (due to higher moisture content).
- Often presumed that haylage is 'richer' and can be fed in smaller quantities; opposite is true.

Feeding forage

The value of a particular forage to an individual horse is influenced not only by the basic nutritional content of the forage but by its palatability, digestibility and intake.

Palatability

Palatability of a forage refers to how well it is accepted by the horse and has a direct impact on intake. Palatability generally correlates well with forage quality, but can be influenced by moisture content, texture/harshness and odour.

Digestibility

Digestibility is the proportion of forage intake that is not excreted and relates primarily to fibre content. Because digestion is a complex biological process, digestibility of a forage can be affected (either positively or negatively) by a variety of factors relating to both the diet (through interaction with other dietary components) and the horse itself. Horse-related factors include exercise/activity level and poorly defined intrinsic factors relating to individual physiology and metabolism.

Intake

The hindgut requires a certain amount of forage to function well; a minimum daily intake of 1% BWT is recommended. Feeding less than this increases the risk of intestinal upsets, such as colic, or of deficiencies of

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nutrients found predominantly in forage (potassium deficiency: may lead to exertional rhabdomyolysis). Intake of forages varies between trainers and geographical locations and is influenced by many factors such as tradition, and availability and quality of supply. Average daily grass hay intake by racehorses typically falls in the range of 2.5–4 kg/day.

An attribute of forage that can only be estimated indirectly but which is of importance to its value in the diet is intake potential. Although there are exceptions, it is generally the case that good digestibility leads to high intake; poorly digestible, fibrous forages take longer to pass through the gastrointestinal system and hindgut fill limits further intake. If sufficient quantities are fed, horses will preferentially eat the higher value (high protein/low fibre) part of the ration.

Ideally changeover between batches of forage that differ substantially in key nutrient levels should be slow and gradual to allow for adaptation of the hindgut microflora to occur (although the overall impact of forage changes on hindgut microflora balance is considerably less than that of concentrates). Practical circumstances rarely permit this to happen, but consideration should be given to dietary transitions for those individuals in a yard identified as being prone to colic or digestive upsets. Changing between feeding of hay and haylage (providing substrate plant is of similar type/maturity) is considered to have only mild effects on hindgut microflora and does not generally necessitate gradual transition.

Haylage has a lower DM compared with hay, and more haylage (25–50%) should therefore be fed by weight per day if fibre and potassium deficiencies are to be avoided.

Allocating forage

When a range of forage qualities is available in a yard, the horses with the highest nutritional needs should be fed the highest quality forage. This is generally taken to be horses in the high-intensity exercise phase of their training and those that are actively racing: nutritional demands are higher and intakes are generally lower making energy density and digestibility important factors in dietary management. In practice it is typical for horses in this category to be fed legume (alfalfa) forage in addition to grass forage, particularly if the nutritional quality of the latter is below average.

Factors that affect forage quality

Plant species

Legumes (e.g., alfalfa) are generally higher in protein and calcium than grasses. Nutritive value of cool-season/temperate grasses (e.g., timothy, bluegrass, ryegrass, fescue) is generally higher than that of warm-season/tropical grasses (e.g., bermudagrass).

Stage of maturity at harvest

The goal of hay or haylage production is the preservation of the harvested plant in the best possible condition for later consumption. The stage of maturity of the plant at the point of harvest is a major contributing factor to the final quality of forage: the nutritive value of hay or haylage can never be greater than that of the harvested plant. As grasses mature, greater stem length and strength equate to a rise in fibre content: the more fibrous a forage is the lower its energy value to the horse, the longer it takes to digest and the lower the amount consumed. Digestibility of spring grasses can be as high as 80%, but as growth progresses this can decline to < 50%. Levels of CP and sugars are highest during rapid growth in spring and regrowth in autumn, lower during mid-summer flowering (bloom) and lowest in winter. In general, the stage of maturity at the time of harvesting is a more important determinant of the nutritive value than whether hay is first or second cut.

Leaf-to-stem ratio

Leaves have higher nutritive value than stems, with approximately half the fibrous fraction and double the CP. Relative ratio of leaves:stem declines as a plant matures.

Harvesting and storage

When and how harvesting is conducted and weather conditions between harvest and baling can influence forage quality. Levels of soluble sugars build up in the plant during the day (particularly in sunshine) such that levels are significantly higher at end of day than in the morning. Once the plant is cut, rainfall during field drying can leach out some nutrients such that a greater proportion of what remains is fibre, with consequent impact on digestibility. Legumes appear to be more susceptible to this type of damage than grasses. Handling of forage that results in leaf loss, and storage conditions (protected/unprotected, and humidity) can also influence rate of nutrient loss and mould growth.

Mould contamination

Mould contamination arises from inadequate drying at baling and therefore is more of a problem in countries with wet/unpredictable summers. Fungi may affect health through the production of mycotoxins (such as aflatoxin, ergot alkaloids), with diminished appetite/compromised immunity being possible consequences. Mycotoxins are resistant to high temperatures and are not destroyed by hay steamers.

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HAY: SOAK OR STEAM?

- Soaking and steaming hay are commonly used strategies to reduce inhalation of dust particles (implicated in inflammatory small airway disease).
- Soaking for as little as 15 minutes significantly reduces levels of wide range of nutrients including WSCs, protein and trace elements.
- Because of the leaching of soluble nutrients, soaked hay has relatively greater proportion of insoluble fibre and lower metabolizable energy.
- Losses vary with hay type and maturity (and temperature of water: leaching less efficient in cold water).
- Feed intakes of soaked hay lower than dry or steamed hay (also DM much lower: 30% compared with >85% for dry/steamed).
- Steaming does not generally have any negative effects on nutrient content of hay.

Physical evaluation of quality

Physical/sensory appraisal of forage is useful as an adjunct to (but not replacement for) laboratory evaluation of nutrient content. Appearance, texture and odour all contribute to give an overall impression of quality and likely energy value. Leaf-to-stem ratio and presence of seed heads or blooms can give a good indication of maturity at harvest, which is a determinant of digestibility and feed intake; as is plant species (grass or mixed grass/legume or meadow species). Texture: whether brittle, prickly/stemmy or 'soft' (where leaves and stems are similarly pliable) to handle is another characteristic used to judge maturity at harvest and handling/storage following harvest. Colour can be misleading as it does not necessarily correlate to nutritional value; retention of green may simply indicate rapid curing or protection from sunshine. Hays that smell musty indicate the presence of mould and exposure to moisture during harvest or storage.

Chemical evaluation of quality

Nutrient content of forages can vary greatly between batches, geographical locations and producers and periodic submission of samples for laboratory analysis can assist greatly in management of yard nutrition. Where physical evaluation is subjective and at best only loosely indicative of forage quality, nutrient analysis can provide objective information that can be used to modify diets, allocate forage to different groups within the yard according to nutritional needs and make suppliers aware that the trainer is monitoring quality of forage. See Table 22.1.

Sampling forage

Given the large volume and number of bales in a delivered batch of hay, it is important that samples taken for laboratory analysis are as representative as possible and that errors are not introduced to the analytical process through sampling technique. Multiple samples should be taken from several randomly selected bales and pooled into a single sample for submission weighing between 100 and 200 g. It is preferable to obtain samples using a core sampler/hay probe, accessing each bale through the end of the bale (for square bales). If samples are collected manually, they should be obtained from within bales with care taken not to dislodge leaf during acquisition. Samples should be placed in a sealed, airtight plastic bag for postage to an accredited laboratory with proficiency in the analysis of equine forages.

Table 22.1 **Guideline measures of forage quality**

	MEASURE	SUBOPTIMAL QUALITY	SATISFACTORY QUALITY	ABOVE AVERAGE QUALITY
Grass hay (cold-climate)	Crude protein	<8%	8–10%	>10%
	Dry matter	<84% or >92%	85–90%	-
	ADF	>50%	35–45%	<35%
	NDF	>60%	45–55%	<45%
Legume hay (alfalfa)	Crude protein	≤14%	15–18%	>18%
	Dry matter	<84% or >92%	85–90%	-
	ADF	>45%	30–40%	<30%
	NDF	>55%	40–50%	<40%

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Interpreting a forage analysis report

The main measured/calculated parameters found on a forage analysis report are detailed in Table 22.2. When interpreting an analysis report it is important to remain aware that none of the reported values are absolute and some are dependent upon opaque calculations and may be significantly influenced by laboratory proficiency. Laboratories providing a forage analysis service are primarily focused on production animals and establishing in advance whether formulae and reference ranges applied relate to ruminant or equine nutrition can be important when interpreting results. It is also the case that quality of a forage is determined by many more factors (such as palatability, texture, amino acid profile) than can be deduced from a nutrient analysis report alone.

Some nutritional components are measured directly through physical (such as DM) or chemical processes (fibre content: acid detergent fibre [ADF] and neutral detergent fibre [NDF]; soluble carbohydrates: WSCs, ethanol-soluble carbohydrates [ESC]), whereas others (such as digestible and metabolizable energy [DE and ME] and digestibility: D-value) are derived from predictive formulae applied to one or more constituents of the forage. To measure true digestibility of a feed component is too difficult and time-consuming to undertake in anything other than a research setting, as it involves controlled (and invasive) experiments measuring all energy intake and excretion. Instead, laboratories use calculations based on recommendations published by several different nutritional bodies worldwide. Given that the foundation

Table 22.2 **Interpreting a forage analysis report**

ABBREVIATION	MEASURE	DESCRIPTION	WHAT IT MEANS
DM	Dry matter	Proportion of forage that is not water.	Measure of moisture content of forage. Forages with lower DM content (such as haylage) need to be consumed in greater quantities to account for lower concentration of nutrients.
CP	Crude protein	Indirect estimation of total protein in forage based on nitrogen content (nitrogen content × 6.25, as protein is approximately 16% nitrogen).	Useful parameter but is not a measure of protein quality, amino acid composition or digestibility.
ADF MAD fibre	Acid detergent fibre Modified acid detergent fibre	Measure of cellulose, lignin and insoluble minerals. Is not a measure of total fibre.	Is an estimation of the indigestible component of the forage and therefore reflects forage quality and digestibility. Higher ADF/MAD = lower digestibility and energy value.
NDF	Neutral detergent fibre	Measure of cell wall components and equates to ADF + hemicelluloses. Best measure of total fibre content of forage.	Differs from ADF by including 'digestible' cell wall components and is therefore not a useful indicator of forage quality or digestibility. Used as measure of forage intake potential: as NDF increases, expect forage intake to decrease.
Ash	Ash	Measure of total mineral/inorganic component of forage.	Does not contribute to energy value of feed.
WSC	Water-soluble carbohydrates	Simple sugars + fructan; digestible in the foregut.	Higher simple sugar content raises glycaemic index of forage.
ESC	Ethanol-soluble carbohydrates	Simple sugars (without fructan); digestible in the foregut.	Higher simple sugar content raises glycaemic index of forage.
D-value	Digestibility value	Equation-derived (rather than measured) figure.	Calculation is typically based on ruminant (cattle) data therefore of limited relevance to the horse, other than as factor used to compare multiple samples.
DE	Digestible energy	Estimate of energy utilised by animal based on gross energy intake and faecal losses.	Derived figure and of limited value. More useful in comparison of analyses of multiple batches of forage over time than as 'one-off' assessment of forage quality.
ME	Metabolizable energy	Estimate of energy utilised that takes into account faecal, renal and gaseous losses of energy. ME = DE – [urinary energy + gaseous energy]	Derived figure and of limited value. More useful in comparison of analyses of multiple batches of forage over time than as 'one-off' assessment of forage quality. Better estimate of true energy value of forage than DE.

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research for these recommendations varies widely in terms of methodology (and is usually not racehorse specific), results relating to digestibility and available energy are probably best used as an indirect measure to monitor quality of forage supplies over time (rather than to formulate diet per se). Caution is also advised when comparing results between different laboratories unless the underlying formulae used are known.

Because the calculations used to arrive at ME take account of energy losses through the three major excretion pathways (urine, faecal and gaseous), it is considered a better estimate of true energy value of a forage than DE (which just accounts for the faecal pathway).

ASSESSING NUTRITIONAL PROBLEMS

An objective analysis is required when assessing any change in feeding practice or possible nutritional influence underlying a yard health problem. Formulation or detailed analysis of diet should be performed by a qualified nutritionist; however, basic investigations are useful to guide or monitor feeding practices.

History

- Establish trainer's main concerns: is it physical condition, faecal consistency, feed intake or racetrack performance? Is the perceived problem yard-wide or limited to individuals or certain sub-groups?
- Determine whether the diet has changed in any respect (constituents/quantities) over the preceding months in response to perceived problems; previous diet may be more relevant to current clinical query.
- Is there any additional evidence of conditions in the yard with possible underlying nutritional basis, such as excessive incidence of rhabdomyolysis, colic, diarrhoea?

Conducting a 'weigh-out'

- Measurement of total daily intake of all components of diet (concentrates, forage and supplements) as fed to an individual horse.
- Energy and nutritional values rely on weight (not volume).
- Observation of feeding practices: ensure measured samples are 'as fed' by regular feeding staff. Feeding staff are the most reliable source of information on variations in dietary management practices in yard: trainer frequently unaware.
- Subjective assessment of feed quality: discolouration of grains (possible fungal contamination)/rancidity of oil/spoiling of hay.
- Analysis (nutrients/dust/spores) of representative forage samples.
- Comparison with previous yard data useful to identify any 'creep' in feeding practices.
- May permit simplification of diet or removal of components that are duplicated or have better quality/value alternatives

Testing for nutritional imbalances

Homeostatic mechanisms generally maintain blood levels of electrolytes and nutrients within a tightly defined range so that important cellular functions continue uninterrupted regardless of fluctuations in nutritional status. Interpretation of blood analysis is therefore rarely straightforward. Testing for nutritional deficiencies is only useful if plasma concentration reflects total body store of a particular nutrient:

- Calcium/iron: plasma levels not affected by dietary intake.
- Potassium: plasma levels are a good measure of status but affected by dietary cation–anion balance.
- Copper: plasma levels only useful for long-term deficiency.
- Magnesium: plasma levels of little use to establish status.
- Selenium: blood selenium good measure of status.
- Vitamin E: assessment is problematic; measure serum α-tocopherol over several days.

POOR APPETITE/CONDITION

Optimal BWT in training varies between individuals. Horses that do not eat their full ration or fail to hold condition despite eating well are commonly encountered. Loss of appetite, or failure of appetite to keep pace with workload, may be a transient or persistent feature and can have several causes. Contributory factors may involve different stages of the digestive/metabolic process and encompass inadequate availability, consumption or absorption of nutrients, inadequate utilization of absorbed nutrients or increased nutritional demands not met by digested nutrients. Poor condition may be a diagnostic challenge and the causes in the following sections should be considered.

Causes

'Over-reaching'/'overtraining'

Application of training/racing loads in excess of physical or temperamental capacity may lead to poor appetite and weight loss.

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Teeth
A common cause of transient (few days) inappetence is shedding of deciduous premolar teeth ('caps') in late 2 to early 4 YOs. Loose caps are easily dislodged with forceps and immediate improvement in appetite should result. Rarely, laceration to tongue or cheek may cause oral pain: healing is rapid. Fractured cheek teeth may cause dysphagia but are rarely encountered in young racehorses; when they occur management is determined by individual circumstances.

Gastric ulcers
Gastric ulcers are a common cause of reduced feed intake. The onset of clinical signs is typically concurrent with increased workload. Presence/severity can only be determined by gastroscopy. Treatment usually results in improved appetite/condition within days/weeks (p. 335).

Parasites
Parasites are rarely the cause of poor condition in horses in training (p. 337) but should be considered in new arrivals/young stock.

Feed quality
Palatability may be affected by feed quality and, to some extent, by individual appetite for salt. Grains do not spoil under normal storage conditions, but oils may suffer from rancidity and additives to processed feeds or hay may affect palatability. Oil rancidity can be determined by peroxide value. Mycotoxins can diminish appetite and laboratory analysis of grain/concentrate and hay rations may be considered.

Fillies
Feed intake of fillies is sometimes depressed during oestrus, particularly during the transitional phase in late winter/early spring when workload is also increasing. Control of oestrus behaviour may be beneficial (Chapter 13, p. 341).

Management
It is important to determine whether weight loss is absolute or relative to increased workload/energy expenditure, and if reduced/inadequate feed intake is responsible. Uneaten feed may represent overfeeding of energy-dense ration (rather than necessarily reflecting inadequate appetite). Some horses are simply unable to balance the rigours of training with a normal racing diet. The equine gastrointestinal system has evolved to accommodate many small grazing 'meals' of roughage and the small stomach size limits the ability to deal with large infrequent meals. Strategies to improve feed intake include:

- Smaller meals at more frequent intervals.
- Increase energy density through addition of vegetable oil (may permit reduction of weight/volume of meals required).
- Maximise forage quality: poorly digestible fibrous forage is retained for longer in hindgut and may depress appetite.
- Consider addition of highly digestible fibre to diet (beet pulp, soy hulls).
- Consider supplementation with amylase-enriched product (e.g., ERME, p. 444) to assist pre-caecal starch digestion.
- B-vitamin supplementation: although it is common for horses displaying poor appetite to be supplemented with B vitamins (either orally or injectable), true deficiency is actually rare.

Once common causes of poor appetite have been ruled out, medical investigation may be warranted (infection/inflammatory bowel disease/tumours). Tests include blood analysis, ultrasonography and biopsy. Low serum albumin (hypoalbuminaemia) +/– low total protein may be consistent with intestinal malabsorption or increased loss of nutrients, and blood analysis can be used to monitor response to medical therapy, although rapid changes should not be expected. Persistent and uncontrollable weight loss should prompt referral for detailed medical investigation.

NUTRITION FOR SPECIFIC CONDITIONS

Gastric ulceration
The primary factors that predispose racehorses to gastric ulceration relate to feeding and management practices: the restricted access to forage, and provision of a small daily number of large carbohydrate-dense concentrate meals lead to greater acidity in the stomach and greater contact time of the non-glandular mucosa of the stomach with those acidic contents. These feeding practices exist for a reason, however: most racehorses are permanently stabled and cannot graze, and provision of ad lib forage and concentrate-free diets is not compatible with maximising athletic potential. While management of gastric ulceration in racehorses therefore rests largely on medication, the following dietary aspects may be considered:

- Provision of as much forage as practicable, with access throughout the day being more important than at night.

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- A small forage meal prior to exercise can mitigate the 'acid splashing' that occurs when the stomach is empty.
- The high calcium content of alfalfa hay may assist acid buffering in the stomach.
- Limit the regular use of electrolyte paste syringes.

Recurrent colic

Colic may have many non-dietary causes and recurrent or persistent colic warrants medical investigation. Colic risk in individuals and across a yard may also have an underlying dietary basis and when other causes of colic have been ruled out, appraisal of feeding practices should be undertaken to identify any contributory factors. The following dietary modifications may be considered:

- Attention to feeding practices: horses that recurrently colic or get anxious at evening feeding times may benefit from being fed first, or being kept occupied on the horse-walker until feeding time.
- Slow gut transit times and reduce nutritional pressure on the small intestine by feeding smaller concentrate rations (3–4 meals/day).
- Improve overall protein quality of forage.
- Aim to feed forage at 1.5% of BWT/day.

Nutritional support following injury

See Chapter 5, p. 62.

Diarrhoea/loose faeces

Most cases of diarrhoea or loose faeces in racing yards are self-resolving. When diarrhoea is associated with other signs of illness, a medical investigation is warranted; however, cases of chronic loose faeces in otherwise healthy racehorses are sometimes raised for veterinary attention. Diarrhoea is a symptom rather than a disease and in the athletic horse it may have many causes including dietary carbohydrate overload, inadequate dietary fibre, inflammatory bowel disease or secondary to antibiotic or non-steroidal anti-inflammatory drug use. Diarrhoea may result from disturbances in the balance of the microbial population of the hindgut, which has negative impacts on digestive efficiency and absorption of nutrients. Dietary management aiming to re-establish equilibrium and slow gut transit times may be beneficial; recommendations include:

- Assess diet to determine whether excessive levels of starch/concentrates are being fed: if so, reduce amounts fed. Ensure concentrate meal sizes are small (feed small meals more frequently i.e., >3 meals/day).
- Ensure feeding of adequate amounts of fibre: 1.5% BWT/per day of forage +/– other fibre-rich feeds to increase faecal bulk.
- Feed grass hay (greater water-holding capacity of fibre) in preference to alfalfa.
- Short fibre (pellets/cubes/chaff) may be beneficial during episodes of diarrhoea as more readily digested in the hindgut.
- Short-term (2–5 days) supplementation (or oral/intra-gastric dosing) with additives known to bind/adsorb pathogenic bacteria and toxins when diarrhoea is excessive (smectite clay).
- Hindgut 'buffers': supplementation with antacids/nutritional products/prebiotics that are proposed to reduce the acidity of the hindgut may be beneficial, although evidence of clinical efficacy limited at present.
- Some empirical evidence to support the use of ERME (p. 444) if starch 'overload' is thought to be a contributory factor.

Rhabdomyolysis

See also p. 265.

Most episodes of rhabdomyolysis ('setfast'/'tying-up') are sporadic and related to either easily remedied management lapses or transient mismatch in energy consumption/expenditure that can occur when a susceptible horse is not fully fit. For the more problematic cases of recurrent rhabdomyolysis it is frequently recommended that dietary changes are made to reduce reliance on starch and increase energy derived from oils/fats +/– highly digestible fibre. Some relevant points relating to nutritional management of recurrent cases are:

- Partial replacement of cereal-based carbohydrates with high oil/fat and highly digestible fibre may be beneficial, although wholesale replacement of starch in diet usually incompatible with maintenance of satisfactory body condition in full work.
- High-fat (15–20% of daily DE intake) diet well tolerated and may act by reducing excitability.
- Most commercial complete feeds formulated and marketed for rhabdomyolysis cases are low starch but not starch free.
- Utilising dietary changes alone (i.e., without exercise management +/– dantrolene sodium) to manage the condition is usually unrewarding.
- Assessment of diet to ensure adequate potassium intake: >1.5% BWT/day good-quality forage advisable (or alternatively supplementation with electrolytes containing sufficient potassium).

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- Some empirical evidence to support supplementation with branched-chain amino acids (BCAAs) +/or beta-alanine.
- Little/no benefit of supplementation with sodium bicarbonate or vitamin E/selenium.
- Increases in dietary provision of concentrates should follow (rather than precede) any anticipated increase in workload; similarly, energy intake should be reduced in anticipation of any planned reduction of workload.

Temperament/behaviour modification

Dietary supplementation or oral administration of a range of nutrients to modulate difficult behaviour or anxiety is commonplace. Most commercial 'calming' products contain a variety of substances that have purported or demonstrable effects on a variety of behavioural conditions in humans or laboratory animals: these include magnesium (or magnesium-boron), L-tryptophan or 5-hydroxytryptophan (5-HTP); B-complex vitamins; anxiolytic peptides (such as alpha-casozepine, derived from the milk protein casein) and gamma-aminobutyric acid (GABA). Potential effects are thought to be the result of direct or indirect mediation of neurotransmitters or neural pathways. While there is some evidence that magnesium supplementation may improve clinical signs of neuropathic headshaking, efficacy for other claimed benefits of calming products in horses has not been established and validated basic information regarding bioavailability and administration dose does not exist at present. Current evidence favours the conclusion that L-tryptophan/5-HTP, B-complex vitamins and GABA do not significantly modify behaviour in horses.

FEEDING FOR PERFORMANCE

Carbohydrate loading

Unlike in people, carbohydrate 'loading' offers no advantages to racehorse performance and indeed carbohydrate overload is more likely to cause detrimental effects such as diarrhoea, colic or (rarely) laminitis. Feed intake should be determined by immediate needs rather than attempting to build up 'reserves' for future use.

Pre-race feeding

The weight of a racehorse, and therefore the energy needed to propel it throughout a race, can be influenced by pre-race diet. The hindgut (colon and caecum), where digestion of fibre occurs, also acts as a large reservoir of fluid; tapered reduction of forage intake (to ≤1% BWT) over the 3 days leading into a race may therefore be beneficial to reduce gut fill/water retention (thereby reducing BWT). Hays with greater digestibility are preferable in this pre-race period. Withholding forage (or limiting it to small amounts) on race day is recommended, although up to 1–2 kg of forage >3 hours pre-race is unlikely to be detrimental to performance.

The body's response to a meal is for blood insulin levels to rise to assist with uptake of glucose from blood to muscle. High insulin levels have a detrimental effect on performance; however, this 'glycaemic response' varies between feeds and is influenced by many factors. Some dehydration also occurs in the immediate period after feeding. The optimum timing of pre-race meals is a poorly researched area; the general advice is that large glycaemic meals should be avoided. A small meal of concentrate/grain may be fed 4–5 hours pre-race, but nothing closer than 4 hours to the race. Hay can also elicit a rise in blood glucose, which peaks at around 3 hours following consumption.

Post-race feeding

Muscle glycogen stores (a key energy source for the racehorse) are depleted by racing and high-speed exercise, and need to be restored to normal levels before peak performance can again be expected. This occurs naturally over 2–3 days, providing the horse is permitted rest from strenuous activity and eats/drinks its normal ration during this period. Repletion of muscle glycogen stores does not appear to be hastened by any particular dietary strategy, although there is some evidence that it may be optimised by diets that directly or indirectly increase availability of alternative energy substrates (such as volatile fatty acids produced by hindgut microbial action).

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FLUID AND ELECTROLYTE THERAPY

OVERVIEW

Water makes up a large proportion (approximately two-thirds) of the horse's bodyweight (BWT), with this being split between water within cells (intracellular fluid) and that which exists outside of cells (extracellular fluid: primarily blood plasma, interstitial fluid and lymph). The volume of the intracellular fluid 'compartment' is approximately double that of extracellular fluid. Dissolved in this water are charged particles that assist movement of fluid across cellular barriers and homeostasis (of acid-base status, hydration and energy balance) necessary for essential body functions such as muscle contraction and nerve conduction. These electrolytes are principally minerals: sodium (Na), chloride (Cl), potassium (K), calcium (Ca) and magnesium (Mg). Regulation of their losses, uptake and exchange with bodily stores is complex and dynamic.

Exercise in particular is associated with large and rapid shifts in the movement of fluid and electrolytes between body 'compartments'. At the commencement of athletic effort, fluid is redistributed from the interstitial space (and the splenic reserve) to boost circulatory volume; later on further electrolyte shifts facilitate sweat production and regulation of body temperature, and mitigate the effects of dehydration. While there is a large body of knowledge about how these processes occur in athletic horses, much of this has been derived under experimental conditions. Applying evidence-based fluid/electrolyte therapy to the great variety of situations commonly encountered in racehorse practice therefore relies heavily upon extrapolation; consequently this a field of medicine in which individual interpretation and practical considerations have a valid role.

BASIC PRINCIPLES

Definitions

The *osmolarity* of a fluid is the number of solute particles that it holds per litre, while the *osmolality* of a fluid is the concentration of dissolved particles that it holds, by weight. When considering body fluids (which are generally dilute solutions), the difference between these two measures is usually negligible.

Tonicity refers to the ability of a fluid to cause a cell with which it is in contact to gain or lose fluid. It is a measure of the fluid's osmotic pressure and is determined by the relative concentration of solutes (such as electrolytes), with sodium being the most important of these. *Isotonic* fluids are those which have the same osmotic pressure as the cell itself and therefore cause no net shift of water into or out of the cell (although water itself moves freely).

Sweat

Sweating serves an important role in thermoregulation by dispersing heat generated during exertional effort. The amount of sweat generated is influenced by ambient temperature and humidity, duration and level of exertion as well as less well-defined variables relating to the horse itself. Exercise losses in warm, humid conditions may be 2–3 times those incurred in cool, dry conditions. Equine sweat is rich in electrolytes (hypertonic) with the electrolyte composition being highly dynamic and varying with intensity and duration of exercise as well as current body stores of electrolytes. In the immediate post-exercise period the concentration of electrolytes in sweat rises, presumably in response to changes in plasma electrolytes that happen at this time. Reported levels of electrolytes in sweat are therefore influenced by timing and method of collection; likely range of losses during exercise and in the immediate post-exercise period are found in Table 23.1.

FLUID/ELECTROLYTE THERAPY

Assessing fluid/electrolyte status

Determining the true magnitude of fluid and electrolyte deficits arising from exertion, travel or illness is challenging, and calculating how best to correct them in any given situation is neither simple nor exact. Clinical indicators of dehydration include tacky mucous membranes, slow capillary refill time, reduced skin turgor, elevated pulse rate and reduced appetite; however, variability and inconsistency makes them unreliable as measures of severity. Blood parameters such as packed cell volume (PCV)/haematocrit (HCT) and total plasma protein may give some indication of hemoconcentration; however, the large

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Table 23.1 **Electrolyte composition of plasma and sweat**

ELECTROLYTE	PLASMA (REFERENCE RANGE, RESTING) (mEq/L)	SWEAT (LIKELY RANGE, DURING/ AFTER EXERCISE) (mEq/L)
Sodium (Na)	130–160	125–160
Chloride (Cl)	96–108	140–180
Potassium (K)	3.4–4.3	25–40
Calcium (Ca)	2.9–3.3	5

shifts in blood volume and constituents (due to splenic reserve recruitment) that result from excitement and exertion make interpretation difficult. Finally, due to homeostatic mechanisms and the great mobility of electrolytes between body compartments, plasma concentration of key electrolytes is typically an unreliable indicator of total body reserves. In practical terms therefore, when considering whether and how fluid/electrolyte replacement should be undertaken, it is more useful to base judgements upon the likely losses as detailed in the following sections and in Table 23.2. In the healthy racehorse calculations for fluids/electrolytes required are based on replacement of existing deficits (i.e., dehydration) rather than counteracting ongoing maintenance needs; the latter is usually only a consideration for horses receiving longer-term fluid therapy for medical conditions.

Table 23.2 **Estimated fluid/electrolyte losses with common training challenges, and best-practice recommendations for management**

CAUSE	ESTIMATED LOSSES (BWT)	ESTIMATED ELECTROLYTE LOSSES (UPPER LIMIT)	INTERVENTION RECOMMENDED?	PRACTICAL RECOMMENDATIONS
High-speed exercise/racing (>90% $V0_{2max}$)	1–1.5% BWT [5–8 kg]	Na: 1300 mEq (30 g) Cl: 1500 mEq (53 g) K: 350 mEq (14 g)	*Pre-race*: NO *Post-race*: generally NO, but dependent on losses, natural recovery and proximity to next race.	Interventions to assist post-race recovery generally unnecessary. Salt replenishment important but readily achieved through dietary supplementation. Glycogen repletion may be assisted by early restoration of fluid/electrolyte balance (intra-gastric or intravenous administration of electrolyte solution).
Furosemide + high speed exercise	Total 3–5% BWT [14–20 kg] (1.5–2.5% BWT additional to 'normal' losses)	Na: 3200 mEq (74 g) Cl: 3600 mEq (128 g) K: 800 mEq (31 g)	*Pre-exercise*: NO *Post-exercise*: YES/ MAYBE (electrolyte supplementation)	Additional electrolyte supplementation (dietary and/ or paste) post-exercise on treatment/high speed exercise days is recommended.
Travel	0.45–0.55% BWT/hour travelling. 6 h: approx. 3% BWT [13–15 kg] 12 h: approx. 5% BWT [22–25 kg]		*Pre-travel*: NO *Post-travel*: • Short distance: generally NO • Long-distance: dependent on losses and natural recovery.	Long-distance travel: weigh pre- and post-travel to estimate losses. Correction through stimulation of drinking (administration of electrolyte paste[s]) preferred. Major deficits +/– poor natural recovery +/– time-sensitive racing goals: consider intra-gastric or intravenous fluid therapy.

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When to intervene?

In most routine situations in racehorse practice in which dehydration is encountered (e.g., post-race, or post-travel), correction of fluid/electrolyte deficits is not an emergency. This affords significant flexibility both in determining whether any intervention is needed at all (by observing how well a horse recovers naturally), and in the choice of replacement therapy route. As a general rule, normal status is re-established (without intervention) within a matter of hours providing the horse has free access to water and food. Larger fluid deficits or electrolyte imbalances may sometimes be associated with suppression of thirst or appetite, with consequent delayed recovery times. In these situations assisted correction of deficits may be desirable to speed up recovery and minimise any negative impacts on training, particularly when racing engagements are imminent. Determining whether and how to intervene should be based on estimates of the severity of any deficit, the likelihood and expected speed of natural recovery and any time-sensitive training/racing goals. More subjective influences on decision-making may relate to how a particular horse has previously responded to challenges arising from travel or exertion; individuals known to sweat freely or recover poorly from travel may justify precautionary interventions that are not necessary in other horses. Horses are considered to have a significant capacity to maintain fluid/electrolyte equilibrium in the face of overzealous administration of fluid therapy; however, potential negative consequences such as pulmonary oedema, impaired gaseous exchange and increased urine production as well as unnecessary economic cost should not be disregarded when planning interventions.

Route of administration

Replenishment (or loading) of fluid and electrolytes may be undertaken by several routes. Stimulating the voluntary intake of water is the simplest, and is generally achieved through the oral administration of concentrated electrolytes. Providing that electrolyte pastes contain sufficient regular salt (NaCl), a rise in plasma osmolality rapidly follows ingestion and provokes a reliable thirst response that is superior to relying on offered water alone to restore fluid and electrolyte balance. Thirst response following electrolyte paste administration peaks at approximately 2–3 hours. Providing water alone (without electrolyte supplementation) may be ineffective in restoring normal hydration status. There is some evidence that provision of flavoured (particularly sweet-feed flavoured) water may increase voluntary water intake, and horses can also become habituated to voluntary drinking of electrolyte-fortified water.

When direct or immediate administration of fluids/electrolytes is desired, either the enteric (intra-gastric) or intravenous routes are available. Choice of route will depend on individual circumstances and may include magnitude of estimated deficit, compliance of horse, cost and time pressures. Although it is commonly perceived that intravenous administration is a superior intervention, in most situations the gastrointestinal pathway is at least as effective but with lower risk of negative consequences. In general, if the horse's gastrointestinal tract is functioning normally and not obstructed then the enteric (intra-gastric) route is preferred, because more 'room for error' (with respect to over-/under-administration) is afforded by the enteric mucosal transport systems than is possible by the intravenous route.

Enteric route

Enteric administration (of fluids) is undertaken via naso-gastric intubation, and providing no gastric reflux is obtained and appropriate volumes are introduced it is preferable for fluid to be pumped in (rather than gravity-fed). The equine stomach is relatively small; however, it empties rapidly and volumes of 4–8 L given every 1–2 hours are readily tolerated. Fluid given this way arrives in the large intestine/caecum within 1–2 hours. Occasionally, abdominal discomfort develops during/immediately following intra-gastric administration (presumably due either to gastric distension or the presence of gastric ulcers) and should prompt cessation of the procedure.

Intravenous route

Intravenous administration of fluids is typically undertaken through jugular vein catheterisation and while easily performed requires time and sufficient facilities to hang fluid bags; in most routine applications it is also necessary to keep the horse stationary during the procedure. Risk of catheterisation causing thrombophlebitis is very low in healthy horses that are on fluids for less than a few hours. Fluid administration rates of 5–10 L/h (10–20 mL/kg BWT/h) are tolerated well.

Types of fluid/electrolytes

Oral electrolyte pastes

Electrolytes are rapidly taken up by the body when consumed or administered through the gastrointestinal system. Giving oral electrolyte paste(s) (or intra-gastric 'slurries') serves to both replenish lost electrolytes and stimulate thirst, which in turn raises water consumption

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Table 23.3 **Intravenous fluid types: Composition and osmolarity**

	Na (mEq/L)	Cl (mEq/L)	K (mEq/L)	Mg (mEq/L)	Ca (mEq/L)	OSMOLARITY (mOsm/L)
Hartmann's (lactated Ringer's) solution	131	111	5	0	2	273
Normosol-R	140	98	5	3	-	296
0.9% NaCl solution	154	154	0	0	-	308

and assists correction of fluid and acid-base imbalances. Care should be taken to select a product that has sufficient quantities of key electrolytes to be efficacious: a guide to estimated magnitude of deficits is found in Table 23.2. Many commonly used commercial products contain physiologically insignificant levels of the key electrolytes, and more effective electrolyte pastes can be readily made up from simple ingredients: a 1:1 or 2:1 mixture of regular (table) salt (NaCl) and 'lite'/'lo' salt (KCl) given in one or two doses of 0.2 g/kg BWT (100 g/500 kg horse) can largely replace likely exertional losses of Na and Cl.

Table 23.4 **Milli-equivalent (mEq)/millimole (mmol)/milligram (mg) conversion table**

	mEq	mmol	mg
Sodium (Na)	1	1	23
Chloride (Cl)	1	1	35.4
Potassium (K)	1	1	39
Calcium (Ca)	1	0.5	20
Magnesium (Mg)	1	0.5	12.15

Intra-gastric fluids

Tonicity is an important factor in how well intra-gastric fluids are taken up and distributed in the body. Isotonic electrolyte solutions are preferable to both plain water and hypertonic solutions. Near-isotonic solutions can be readily made up by adding 45 g NaCl + 45g KCl/10 L water. Water temperature (whether cold or at- or below-body temperature) does not appear to have a significant effect on absorption of fluids from the gastrointestinal system.

Intravenous fluids

When choosing the correct intravenous fluid for administration it is important to distinguish deficit replacement from ongoing maintenance requirements. Most racehorse applications involve replacement of losses arising from a single, recent challenge such as racing or travelling; therapy is intended to assist rapid return to 'normal' status. By contrast, longer-term fluid therapy to counter 'maintenance' losses is only applied to systemically unwell (typically hospitalized) horses such as those suffering from colitis or colic. 'Replacement' fluids are isotonic solutions of electrolytes; they should not generally be used beyond the initial replacement period, as excessive Na administration would potentially lead to greater urine production and subsequent dehydration; additionally, K levels are too low for maintenance requirements. Conversely, 'maintenance' fluids should not generally be used for 'replacement' use, as they are comparatively low in sodium and chloride.

Administered fluids are distributed rapidly throughout the body, such that within 1 hour of administration less than a third of the delivered volume remains within the vascular system. Several types of crystalloid replacement fluids with differing electrolyte compositions are in common use (Table 23.3); those that have a composition similar to extracellular fluid (i.e., relatively high in Na and Cl and low in K, Mg and Ca) are preferred. Non-lactated polyionic isotonic solutions, and lactated Ringer's solution (Hartmann's solution) are the fluids of choice for replacement therapy. 'Normal' saline (0.9% NaCl) is isotonic but in containing both Na and Cl at higher levels than found in plasma can potentially worsen the acid-base disturbances found in horses following intense exercise or that are dehydrated.

Conversions for milli-equivalent (mEq)/millimole (mmol)/milligram (mg) are found in Table 23.4.

SPECIFIC SITUATIONS

Exercise and racing

Short-duration, high-speed exercise such as racing or training gallops causes large and rapid shifts in electrolyte levels and fluids between body compartments in order to sustain the muscular and cardiovascular efforts involved in athletic exertion. A metabolic acidosis is also generated,

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with blood pH falling as low as 6.8 from resting levels of 7.4. These dramatic changes are rapidly reversed after exercise, with a return to resting values usually occurring within 1–2 hours.

Average loss of BWT during a race or high-speed exercise session is estimated at 1–1.5% BWT (5–8 kg); as this loss is primarily through sweating, it may be influenced by many factors including ambient temperature and humidity, temperament of horse, level of exertion and preceding electrolyte and fluid status. Most horses fully recover their pre-exercise fluid and electrolyte status within 1–3 days.

Pre-exercise treatments

Fluid/electrolyte/vitamin administration in the pre-race period is commonplace in racehorse practice, typically taking place >24 hours prior to start time due to regulatory and practical constraints. Whether this has any effect (positive or negative) upon subsequent race performance is largely untested; however, it is probable, given current understanding of the basic physiological processes involved, that it is an intervention of little value. Commercially available products in common use frequently contain physiologically insignificant quantities of the major electrolytes. Intra-gastric administration of low volume (1.5 L) salt solution (0.3–0.4 g/kg BWT NaCl) causes a significant short-term (<5 hours) expansion of plasma volume; however, the duration of effect limits its practical use and effect on performance is in any case uncertain. In general, 'pre-loading' with electrolytes simply results in more rapid excretion of electrolytes in urine, with little or no overall gain or improvement in body status; any possible ergogenic advantages are likely to be restricted to athletic disciplines involving prolonged periods of exertion/sweating (e.g., endurance rather than flat racing).

THE PRE-RACE ELECTROLYTE/VITAMIN/AMINO ACID 'JUG'

- Typically given on day before racing to assist performance or counteract electrolyte losses that may occur in pre-race period (travel/sweating).
- Strong homeostatic mechanisms maintain body's electrolyte balance, and excess electrolytes are rapidly excreted in urine.
- Most commercial products in common use contain physiologically insignificant levels of electrolytes, sugars and other micronutrients.
- Balanced racing diets are well fortified in all key nutrients, and a racehorse with good feed/water intake probably derives no ergogenic benefit from pre-race administration of additional electrolytes/vitamins.

Post-race recovery

Full recovery from intense exercise (training or racing) involves replenishment of muscle glycogen stores. This occurs naturally (with normal feed intake) over 2–3 days. In limited circumstances (such as if a rapid return to intense effort is anticipated) it may be considered advantageous to assist this process, although it should be noted that glycogen depletion is thought to be less important to onset of fatigue than acidosis over flat racing distances. Assisted glycogen replenishment is not straightforward and many factors may influence the effectiveness of interventions. Experimentally it has been shown that large doses of intravenous dextrose (3–6 g/kg given as a 20% solution slowly over 6–8 hours) can be administered safely and result in enhanced glycogen resynthesis; however, situations where such intervention is both desirable and practically implemented are limited. More rapid administration of intravenous dextrose solution exceeds uptake capacity of target tissues and is inadvisable due to resulting diuresis and risk of thrombophlebitis. Intra-gastric administration of dextrose/glucose is also not recommended as it appears to have little appreciable effect on glycogen repletion. Preferred strategies are those that focus instead on muscle rehydration (rather than glucose supplementation). Both intra-gastric administration of hypotonic electrolyte solution, and intravenous electrolyte therapy can enhance glycogen repletion. Supplements (such as acetate) that increase the availability of important 'glucose-sparing' alternative energy substrates may also have a role in assisting recovery.

Furosemide

Furosemide is widely used in susceptible horses as a prophylactic against exercise-induced pulmonary haemorrhage (EIPH; p. 305) on high-speed exercise days. Although it has many pharmacological effects, it is principally a strong diuretic: it acts directly on renal tubular function by inhibiting the reabsorption of Na and Cl, thereby producing large volumes of isotonic urine. Average loss of BWT following routine administration at therapeutic doses (0.5–1.0 mg/kg BWT) followed by high-speed exercise is estimated at approximately 3–4% BWT (14–20 kg); as 'normal' exercise losses in untreated horses are estimated at 1–1.5% BWT (5–8 kg) then furosemide-attributed loss is likely around 8–12 kg. The important feature of furosemide administration is that Na is lost from the body along with fluid, and as such there may be no compensatory thirst response after exercise and natural recovery of fluid/electrolyte status to pre-exercise levels may take several days. There is a good rationale therefore to ensure that horses receive electrolyte supplementation (preferably through oral administration) post-exercise.

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Transport

Travelling, whether by road or air, may result in significant BWT/fluid losses. Magnitude of losses appears to be largely determined by duration of journey rather than mode of transport, and arises from reduced water (and to lesser extent forage) intake as well as energy expended in maintaining stance. Losses are estimated to be around 0.45–0.55% BWT per hour of travelling time, with up to 5% BWT (22–25 kg) lost on a 12-hour journey. As a general rule the dehydration resulting from reduced water intake while travelling is rapidly corrected through greater voluntary drinking when the horse arrives at its destination; however, feed intake may remain depressed for some time.

There is little scientific justification for the common practice of giving horses fluid/electrolyte therapy prior to long-haul air journeys; current understanding is that at best pre-travel fluid therapy has little beneficial effect and at worst may be detrimental, through overhydration and adverse effects on electrolyte balance. Instead of pre-travel fluid therapy, it is recommended that direct interventions (if required) should occur after the journey. Horses may be assessed for BWT losses after settling in at their destination, and any decision to correct such losses based upon the magnitude of the loss, demeanour of horse and how well it consumes water/feed after arrival. This is dependent on availability of accurate weights before and after travel.

Water deprivation

Partial or total water deprivation for a variable period of time pre-race is sometimes practiced in an attempt to minimise risk or severity of EIPH in affected horses. Efficacy or otherwise of this practice has not been investigated, and validated recommendations for individual circumstances (taking account of ambient temperature/humidity, distance travelled, race distance, likely sweat losses, etc.) do not exist. Additionally, the potential negative effect of dehydration on performance is a consideration. It is recognised, however, that horses tolerate sometimes long (days) periods of water deprivation well, and when dehydrated maintain adequate circulatory volumes by drawing fluid from intestinal stores. Small studies have demonstrated BWT losses after 24 hours of water deprivation to be in the range of 3–6%.

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BLOOD ANALYSIS

BLOOD CONSTITUENTS

The function of blood is largely to transport oxygen, nutrients, electrolytes and hormones to body organs and tissues, and to remove waste products from them. Blood has cellular (red and white blood cells, platelets) and plasma (water, electrolytes and plasma proteins) fractions. Measuring these constituents can allow assessment of factors that might influence performance, such as the oxygen-carrying capacity of blood, hydration status and immune challenge. Haematology refers to the study of blood (red blood cells [RBCs], white blood cells [WBC] and platelets), and biochemistry to the measurement of the enzyme, protein and electrolyte components of blood (Table 24.1).

Table 24.1 **Haematology and biochemistry overview**

CONSTITUENT	FUNCTION AND FEATURES	MEASURES AND INTERPRETATION
RBCs (erythrocytes)	Main cellular constituent of blood. Function: protein Hb binds and transports O_2. Produced in bone marrow. Circulation time: 155 days	**Red cell count**: number of circulating RBCs per unit blood. **PCV**: percentage of blood as cells (primarily composed of RBC). Both red cell count and PCV may be elevated with sampling stress/splenic contraction. **MCV**: average size of RBCs. Indirect indicator of blood loss/cell age (cells become smaller as they mature). **McHc**: amount of Hb in each RBC per unit size. Only relevant if decreased (anaemia); elevated levels indicate haemolysis of sample.
WBCs (leucocytes)	Cells with key roles in inflammation and immunity. Several types, all produced in bone marrow: • Neutrophils: first line of defence; ingest/destroy bacteria. Short (<24 hour) half-life. • Lymphocytes: B (plasma) cells produce antibodies; T cells involved in immune response. • Monocytes: precursors of macrophages. • Eosinophils: involved in allergic/hypersensitivity reactions	**White cell count**: total circulating WBC count per unit blood. Raised with stress or infection. **'Differential'**: relative proportions of different WBC types in the sample; may allow differentiation of disease states but can also be influenced by excitement/stress and other factors such as recent corticosteroid use. Absolute cell counts of more importance than differential 'split'/ratio. **Neutrophil count**: increases with stress, bacterial (acute/chronic phase) or viral (acute phase) infections; decreases with excessive demand for neutrophils (severe acute infection, endotoxaemia). Persistently low counts seen with post-viral challenge. **Lymphocyte count**: reduced with acute viral infection; increases with chronic viral infection. **Monocyte count**: non-specific; raised with acute/chronic inflammation (particularly viral). **Eosinophil count**: non-specific; may be raised with allergic response/intestinal parasite burden
Platelets	Main function: clotting; also have role in inflammatory/immune processes. Cell fragments produced in bone marrow from megakaryocytes. Circulation time: 4–7 days	Low platelet counts usually due to clumping during sample processing (rather than clotting disorders)
Plasma proteins	**Albumin**: produced in liver; main function is as a transport/binding protein. **Globulins**: role in immune response	**Total protein (albumin + globulin)**: elevated with dehydration or inflammation; decreased with severe protein loss (e.g., intestinal malabsorption). **Albumin:** mild decrease with chronic infection. **Globulins:** elevated with chronic infection

(Continued)

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Table 24.1 **Haematology and biochemistry overview** ***(Continued)***

CONSTITUENT	FUNCTION AND FEATURES	MEASURES AND INTERPRETATION
Inflammatory markers	**Plasma fibrinogen and serum amyloid A**: acute phase plasma proteins produced by liver. Have roles in inflammatory response and clotting	Acute phase protein levels allow interpretation of high/low WBC counts (inflammation or stress). **Plasma fibrinogen:** concentration increases with inflammation/dehydration. May take 1–2 days to rise in response to stimulus. Elevation may persist (>1 week) beyond resolution of pathology. **Serum amyloid A**: usually undetectable concentrations in healthy horses. Early rise (hours) in response to stimulus; peaks at ~48 hours and rapidly returns to normal after acute phase
Muscle enzymes	Enzymes released into circulation from damaged muscle cells. **CK**: largely muscle specific. **AST**: less so as found in other tissues (including liver)	Allow interpretation of episodes of exertional rhabdomyolysis (severity and timing): differences in peak levels and plasma half-lives permit assessment of whether episode occurred within 24 hours (both elevated; CK higher than AST) or several days previously (higher AST). **CK**: short plasma half-life. Plasma levels peak at 4–6 hours. **AST**: longer plasma half-life. Plasma levels peak at 12–24 hours
Liver function	**GGT**: enzyme found in bile ducts (also pancreas/kidney). Plasma half-life 3 days. **GLDH**: short half-life (14 hours). **ALP**: non-specific; found in liver, bone, intestine. **Bilirubin**: liver takes up, conjugates and excretes bilirubin. **Serum bile acids**: liver normally clears from circulation	Elevations may indicate liver disease or insufficiency but are rarely specific for type of disease. **GGT**: moderate increases (with other liver enzymes normal) of no concern. Large increases most commonly idiopathic or related to 'overtraining' syndrome (p. 370) rather than liver disease. Otherwise: raised with cholestasis (acute hepatitis and cirrhosis); also potentially with drug use and plant/fungal hepatotoxins. **GLDH**: specific for acute liver cell damage and indicator of ongoing damage. **ALP**: raised with chronic obstructive liver failure but non-specific. **Bilirubin**: rises in (unconjugated) bilirubin more likely to be due to inappetence or haemolysis than liver disease. **Serum bile acids**: raised with inappetence or hepatic insufficiency. Liver disease usually advanced before levels raised significantly, but good indicator of impaired function.
Kidney function	**Creatinine**: muscle breakdown product. **Blood urea nitrogen**: produced by liver as digestive product of protein. Both are waste products filtered out of the blood by kidneys	Moderate increases in urea and creatinine seen with renal insufficiency; diagnosis of kidney damage/failure also requires urinalysis to differentiate from pre-renal (e.g., dehydration/circulatory shock) causes

ALP, alkaline phosphatase; AST, aspartate aminotransferase; CK, creatine kinase; GGT, gamma-glutamyl transferase; GLDH, glutamate dehydrogenase; Hb, haemoglobin; McHc, mean corpuscular haemoglobin concentration; MCV, mean corpuscular volume; PCV, packed-cell volume; RBC, red blood cell; WBC, white blood cell.

SAMPLING AND HANDLING

- Technique and timing of blood collection, and handling/storage of sample have the potential to affect results of haematological and biochemical analysis.
- Preferable to sample early morning (pre-exercise/feeding) or late afternoon (pre-feeding) to minimize effect of exercise, excitement and feeding. Haematological values return to baseline ~2 hours post exercise.
- Excitement of horse at sampling may cause splenic contraction and a resulting 'stress' profile (as for exercise): takes approximately 20–30 seconds to mobilize RBCs from spleen.
- WBC counts may be altered for up to 36 hours following racing/travelling.
- Blood tubes used are determined by tests required and preferences of receiving laboratory, but in general:
  - Haematology: EDTA.
  - Biochemistry: clotted (serum).
  - Plasma fibrinogen: sodium citrate.
- Delays in analysis may result in deterioration of sample and can affect both haematology and

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biochemistry results; cell rupture may alter red cell, enzyme and electrolyte figures.
- Cooling (4–8°C) of sample advised; avoid freezing or direct contact with icepacks. If delay is expected, separate serum from clotted samples.

EFFECTS OF TRAINING

Red blood cells
- Training stimulates an increase in total RBC numbers and haemoglobin.
- Rises in resting RBC count, packed-cell volume (PCV) and haemoglobin concentration are modest and cannot be used to assess fitness; interpretation also confounded by the many other variables that affect circulating blood components.
- No link between RBC figures and racing performance.

White blood cells
- Training causes no alteration in total WBC counts or differential fractions.
- No link between WBC figures and fitness.
- 'Reversed' neutrophil:lymphocyte ratio in 2 YOs is normal finding: likely to represent maturation of immune system rather than effect of training.

Biochemistry
- Training causes few changes to biochemistry values.
- Modest increase in serum gamma-glutamyl transferase (GGT; in absence of other indicators of liver disease) is not uncommon in healthy horses; higher elevations may also occur with syndrome with possible link to poor performance/'overtraining' (p. 370).
- Transient increases in muscle-derived enzymes (creatine kinase [CK], aspartate aminotransferase [AST]) may occur in response to either racing or exertional rhabdomyolysis (p. 265).

INTERPRETATION

Because of the horse's readily mobilized splenic reserve of blood cells, a circulating venous blood sample is not necessarily representative of the whole-body status of many blood constituents. Relative proportions of circulating blood components may vary considerably with exercise, feeding and sometimes drug administration.

Haematology and biochemistry results are compared with recognized reference ranges of 'normal' values for age and stage of training. Reference ranges for some components may vary between laboratories depending on testing procedures and equipment. Reference ranges are population-based averages, and individuals with non-conforming blood values in the absence of disease may be encountered. The blood parameters of individuals typically show minimal variation over time when fit and in good health; reference ranges for the individual are usually much narrower than population or laboratory ranges. It is therefore advantageous to compare results with any previous samples (if available) from the individual (rather than against population/laboratory reference ranges) when determining the significance of values, particularly in relation to performance. Results of blood analyses should always be interpreted in the context of clinical presentation; the detection of a haematological or biochemical irregularity on routine sampling from an outwardly healthy animal may merit vigilance rather than intervention.

Health
- Blood analysis is most useful as a measure of health (not fitness).
- Permits detection of subclinical illness (through changes in inflammatory markers) and assessment of organ function.
- Inflammation/infection (bacterial or viral): typically raised WBC and neutrophil count plus raised acute phase proteins (viral challenge may lead to low WBC/neutrophil count in acute phase).
- Bacterial and viral infections may be differentiated by relative counts of neutrophils/lymphocytes in the post-acute phase.
- In the acute phase of severe infections (such as pleuropneumonia) WBC count may initially drop as white cells are sequestered to combat infection.
- Inflammatory profile is non-specific for location or type of disease but is a good measure of severity and current status.

Fitness
- Reliable assessment of fitness through analysis of RBC (or WBC) figures is not possible.
- Increase in RBC count and PCV occurs through early training; however, figures vary between and within individuals and are highly labile with exercise, excitement and feeding due to splenic release and fluid shifts.
- Sampling immediately after exercise may give better indication of total circulating RBC count.
- Measurement of plasma lactate concentration under controlled exercise conditions (treadmill) can be used

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to assess response to workload, but standardized protocol and horse-side testing essential.

Dehydration

- PCV and total protein are used as indicators of dehydration but may not reflect true status due to splenic contraction (RBCs) or gastrointestinal protein loss.
- In general: raised RBC count, PCV and total protein indicates dehydration.

Anaemia

- Anaemia is a decrease in the body's RBC mass, causing reduction in oxygen-carrying capacity of the blood.
- May be regenerative (due to blood loss or haemolysis) or non-regenerative (due to impaired production of red cells).
- True anaemia in horses in training very rare; may occasionally result from blood loss (such as following castration).
- May take >24 hours for PCV and RBC count to drop after haemorrhage (due to compensatory release of cells from spleen).
- Following acute blood loss, PCV usually fully recovered by 1 month.
- Iron deficiency not a factor with balanced diets.
- Equine infectious anaemia (EIA; p. 376) or piroplasmosis (p. 378) should be considered a possibility if anaemia is profound.

Musculoskeletal health

- CK/AST levels usually permit differentiation of muscular soreness/exertional rhabdomyolysis from potential skeletal injury (although concurrent exertional rhabdomyolysis may occur with some stress fractures).
- CK and AST blood levels 'peak' at different times; possible to determine approximate timing of episode of exertional rhabdomyolysis.
- Episode <24 hours prior to sampling: both enzymes elevated; CK higher than AST.
- Episode 1–3 days previously: both enzymes elevated, but AST higher than CK.
- Last episode > several days previously: AST elevated, CK normal.

Electrolytes

- Circulating levels of electrolytes usually a poor indicator of whole-body status, as most plasma concentrations are held within tight range by homeostatic mechanisms to protect body functions.
- Urinary fractional electrolyte clearance ratios frequently permit better assessment than single serum assay.
- Sodium (Na): plasma levels generally maintained regardless of whole-body status; if levels are outside reference range usually reflects hydration (rather than sodium) status.
- Chloride (Cl): plasma levels alter in response to exercise and sweat losses.
- Potassium (K): plasma levels vary widely with exercise/feeding and also are poor reflection of whole-body status.
- Calcium (Ca): plasma levels generally maintained regardless of whole-body status.

Renal function

- Urea and creatinine may be used to assess renal function.
- Urea produced in the liver from the metabolism of ammonia.
- Creatinine formed in muscles and excreted by kidneys; production/excretion typically very constant and used to determine excretion rates of other blood constituents.
- Alterations in both urea and creatinine are unusual and not always related to renal disease (can be affected by pre-renal factors, e.g., exercise, dehydration, protein metabolism).

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TRANSPORT

Long-distance transport of racehorses by both road and air is commonplace. National legislation and international guidelines provide a framework for the safe and humane movement of horses and preventive measures to limit the spread of exotic diseases across national borders. Travelling may have effects on health and athletic performance and understanding these is important particularly for horses that are expected to race/train upon arrival at destination.

EFFECTS OF TRANSPORT

Factors such as individual temperament, route planning, transit conditions and expertise of travelling grooms are fundamental to the well-being of a horse during and after the journey. Long-distance travel in particular has the potential to detrimentally affect health in several ways.

Stress

- Behaviour during and after travel differs between individuals.
- Horses generally settle well during air travel and in most cases are calmer/less stressed for the majority of a flight than during road transport.
- Stress during a flight is usually greatest during unloading/loading and landing/ascent.
- During road transport, heart rate (an indirect measure of stress) is generally higher when vehicle is moving than when stationary.

Respiratory tract

- Restraining the head in a raised position for lengthy periods prevents natural drainage of respiratory secretions and contributes to bacterial colonisation of lower airways.
- Quality of inhaled air (bacterial contamination, dust, humidity, exhaust gases) is often poor during transport and is poorest at times when air circulation is reduced, such as when vehicle is stationary.
- During flight, ventilation may not be uniform; airflow is from front to rear. Air temperature, humidity and bacterial counts rise rapidly when plane is on ground.
- Breathing dry, cool air during air travel can dehydrate the respiratory tract mucosa and reduce mucociliary clearance of secretions.
- Overall effect of long-distance travel (road or air) is a considerable bacterial challenge to the lower airways and impaired respiratory defences to deal with it.

Weight loss

- Regardless of temperament, most horses lose weight during transport.
- Weight loss results from reduced feed and water intake, as well as greater energy expenditure required to maintain postural balance.
- Horses eat and drink less when in a moving vehicle.
- Road transport typically results in losses of 0.45–0.55% bodyweight (BWT)/hour (around 3% for a 6-hour journey).
- Air transport losses typically total 3–5% BWT.
- Rehydration after a flight is often rapid, but BWT is slower to return to normal due to need to replenish gut fill.
- Speed of recovery is determined by temperament of horse (+/– previous travelling experience), amount of weight lost, duration of journey and environmental conditions at destination.
- Recovery time to normal BWT for air travel with 5% BWT loss can be >1 week.

Musculoskeletal system

- Considerable energy is expended in maintaining postural stability during both road and air transport. Postural corrections (both voluntary and involuntary) to regain balance or shift weight are common, and more frequent during periods such as landing and take-off.
- Wide-based stance with head and neck raised and more weight taken by hindlimbs for greater stability.
- Constant muscular effort can lead to very mild elevations in plasma concentrations of muscle enzymes (aspartate aminotransferase [AST]/creatine kinase [CK]).

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Gastrointestinal system
- Reduced food/water intake, disrupted feed patterns and change of diet on arrival can affect hindgut flora.
- Subsequent effects on digestibility of feed and gut motility.
- Risk of developing colic.
- Effect on BWT may persist for weeks.

Blood profile
- Common to get mild inflammatory/dehydration changes in haematology profile in first 3 days following long journey.
- White blood cell (WBC) count either normal or slightly raised (neutrophilia).
- +/– mild elevation in plasma fibrinogen concentration.
- Red blood cell (RBC) count and total protein figures reflect dehydration.

Jetlag and acclimatization
- Circadian rhythms are an evolutionary mechanism to optimise the functioning of physiological processes to the day/night cycle. They are mediated by photoperiod signals from the retina of the eye and secretion of the hormone melatonin.
- Circadian rhythms can be 'phase shifted' or reset by exposure to light as well as other factors such as exercise and feeding patterns.
- 'Jetlag' in people is an alteration in the normal balance of circadian rhythms associated with the crossing of time-zones: manifested by sleep disturbances and flatness as well as athletic underperformance.
- The horse's circadian rhythms are highly light sensitive and may be influenced by exercise and feeding routine; adjustment to local time-zone appears to be far more rapid than in humans.
- Altered circadian rhythm in horses is less likely to have a negative impact on athletic performance in horses than in humans, although scientific understanding is incomplete; some limited evidence that athletic performance under certain conditions may be improved, although magnitude of any such effect is uncertain.
- Acclimatization to hot/humid climates appears to be relatively rapid with good management.

Shipping fever
See also Pleuropneumonia, Chapter 9, p.309.

Shipping fever is an infrequent but regularly encountered condition in horses that have recently travelled long distances (greatest risk with journey times >20–24 hours), although can also be seen following shorter trips. It is a bacterial respiratory infection (most commonly *Streptococcus zooepidemicus*, *Pasteurella*) due to exposure to high bacterial loads in inspired air during transport and reduced clearance of respiratory secretions. Unless treated promptly and aggressively, it may progress to pleuropneumonia (life-threatening). Reported incidence rate after long-haul flights is approximately 6%.

Signs
- Reduced feed and water intake.
- +/– fever.
- Increased respiration rate +/– cough.
- +/– nasal discharge.
- Depression/lethargy, sometimes profound.

Diagnosis
- Blood analysis: inflammatory profile.
- Auscultation of chest: increased lung sounds dorsally, absent/muffled lung sounds ventral fields may indicate development of pleuropneumonia.
- Ultrasonography: detection of pleural fluid accumulation.

Management
- Aggressive broad-spectrum antibiotic and anti-inflammatory therapy.

Prognosis
- Good prognosis for full recovery with prompt and aggressive treatment.

VETERINARY MANAGEMENT FOR LONG-DISTANCE TRANSPORT

Pre-departure
- Pre-departure health and identification checks +/– disease testing protocols as required by receiving country/state.
- Important to ensure respiratory health during immediate period prior to travel; rectal temperature monitored daily and any cough/nasal discharge investigated/treated. Horses with current/recent viral respiratory disease may have compromised mucociliary clearance +/– immune status and are at increased risk of development of shipping fever.
- Pre-shipment weighing useful to allow determination of weight lost during transit.
- Normal diurnal BWT variation: lowest early morning before feeding or immediately post-exercise; highest in evening after feeding.

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- If a new diet (forage/concentrate ration) is to be used at destination, gradual introduction over >1 week prior to travel is advantageous.
- Maintenance of appetite during/after travel may be assisted by administration of anti-ulcer medication (omeprazole); should commence >1 week prior to departure.
- Prophylactic use of antibiotics does not reduce risk of bacterial colonization of the lower airways and is not recommended.
- Limited evidence that use of immunomodulators (*Parapox ovis* virus/*Propionibacterium acnes*-derived products) in period leading up to travel may be beneficial.
- Care required when planning orthopaedic medications in immediate pre-travel period: corticosteroids can depress appetite, suppress immune system and produce haematological changes similar to that seen with illness. Non-steroidal anti-inflammatory drug (NSAID) administration should be avoided for 2–3 days pre- and post-travel in order not to mask pyrexia or early signs of shipping fever.

During travel

- Good quality hay/haylage (ad lib) should be available during long journeys.
- Water should be offered every 3–4 hours.
- Horse should be allowed to/able to lower its head where possible.

Post-arrival

- Monitoring food/water intake during transit and after arrival guides the need for fluid replenishment. Horses should voluntarily eat and drink within 1–3 hours after arrival and if they do not should raise concerns about potential need for veterinary intervention.
- Weighing permits calculation of lost BWT and allows estimation of likely dehydration.
- Fluid administration if BWT loss is >5% (see Chapter 23, p. 455).
- Intra-gastric administration (nasogastric intubation) is recommended for mild-moderate deficits; intravenous isotonic fluids for larger deficits or more rapid replenishment.
- Twice-daily monitoring of rectal temperature for at least 1–2 weeks post-arrival.
- Blood analysis if any depression/inappetence/fever is observed.
- When considering resumption of training, it is recommended to allow for recovery periods of 1 day for journeys of duration 6–12 hours, and 2–3 days for journeys of duration >12 hours.

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APPENDICES

APPENDIX **1** Clinical parameters

APPENDIX **2** Blood reference ranges

APPENDIX **3** Drug administration reference table

APPENDIX **4** Radiographic techniques

APPENDIX **5** Guide to best practice for humane destruction in emergency situations

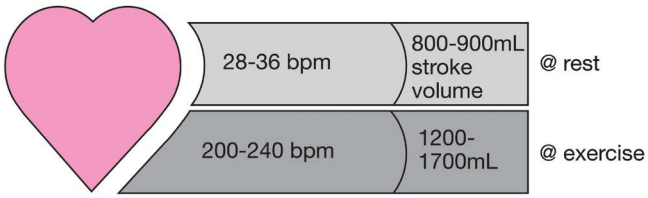
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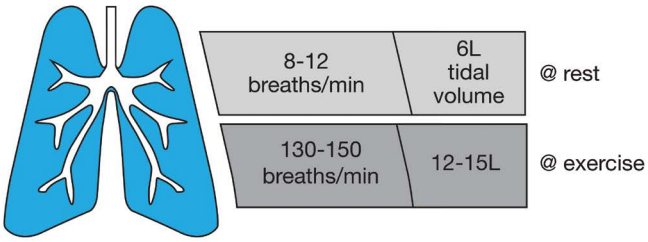


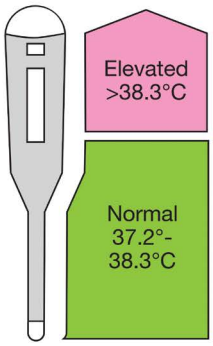
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CLINICAL PARAMETERS







Celsius–Fahrenheit conversion chart

°C	°F
36.9	98.5
37.2	99.0
37.5	99.5
37.8	100.0
38.1	100.5
38.3	101.0
38.6	101.5
38.9	102.0
39.2	102.5
39.4	103.0
39.7	103.5
40.0	104.0
40.3	104.5
40.6	105.0

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BLOOD REFERENCE RANGES

Table A2.1 **Blood reference ranges**

TEST	ABBREVIATION	RANGE	UNITS
Total red blood cells (erythrocytes)	RBCs	8.6–11.7	$\times 10^{12}$/l
Packed cell volume	PCV	0.34–0.45	l/l
Haemoglobin	Hb	12.8–16.6	g/dl
Mean cell volume	MCV	37–43	fl
Mean cell haemoglobin concentration	MCHC	35.9–38.8	g/dl
Mean cell haemoglobin	MCH	13.7–16.2	pg
Total white blood cells (leucocytes)	WBCs	5.5–11.0	$\times 10^{9}$/l
Segmented neutrophils	Segs	3.9–6.0 (42–64%)	$\times 10^{9}$/l
Lymphocytes	Lymphs	2.3–4.4 (28–46%)	$\times 10^{9}$/l
Monocytes	Monos	0.2–0.56 (2–6%)	$\times 10^{9}$/l
Eosinophils	Eos	0–0.3 (1–3%)	$\times 10^{9}$/l
Platelets	Plts	127–206	$\times 10^{9}$/l
Total protein	TP	58–67	g/l
Albumin	Alb	35–40	g/l
Globulin	Glob	21–29	g/l
Plasma fibrinogen	Fib	1.4–3.0	g/l
Serum amyloid A	SAA	0	mg/l
Aspartate aminotransferase	AST	<400	iu/l
Creatine kinase	CK	<400	iu/l
Lactate dehydrogenase	LD	430–917	iu/l
Gamma glutamyl transferase	GGT	12–47	iu/l
Glutamate dehydrogenase	GLDH	3–12	iu/l
Serum alkaline phosphatase	SAP	277–672	iu/l
Intestinal alkaline phosphatase	IAP	64–171	iu/l
Urea	Urea	3.8–6.4	mmol/l
Creatinine	Creat	110–167	µmol/l
Glucose	Glu	3.4–5.9	mmol/l
Total bilirubin	TBili	13–39	µmol/l
Direct bilirubin	DBili	4–15	µmol/l
Bile acids	BAcids	0–8	µmol/l

Modified from Rossdales Laboratories' reference ranges for 2- and 3-YO Thoroughbred racehorses.

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DRUG ADMINISTRATION REFERENCE TABLE

Table A3.1 **Drug administration reference table**

DRUG	DOSAGE (BWT)	ROUTE	FREQUENCY
Antibiotic drugs			
Cefquinome sulphate	1–2 mg/kg	IM or IV	q24h
Ceftiofur sodium	2.2 mg/kg	IM or IV	q24h (or12h)
Doxycycline	10 mg/kg	PO	q12h
Enrofloxacin	7.5 mg/kg 5 mg/kg	PO IV	q24h q24h
Gentamicin sulphate	6.6 mg/kg	IV or IM	q24h
Metronidazole	25 mg/kg	PO	q12h
Marbofloxacin	3.5–4 mg/kg	PO	q24h
Oxytetracycline hydrochloride	5 mg/kg	IV	q24h (or 12h)
Procaine benzyl penicillin	12 mg/kg	IM	q24h
Trimethoprim-sulphonamide	15–30 mg/kg	PO	q12h
Anti-inflammatory drugs			
Acetylsalicylic acid (aspirin)	10 mg/kg	PO	q12–24h
Flunixin meglumine	1.1 mg/kg 1.1 mg/kg	IV PO	q24h q24h
Ketoprofen	2.2 mg/kg	IV	q12–24h
Meloxicam	0.6 mg/kg 0.6 mg/kg	PO IV	q12–24h q12–24h
Phenylbutazone	2.2–4.4 mg/kg 2.2–4.4 mg/kg	PO IV	q12h q12h
Suxibuzone	3.3–6.6 mg/kg	PO	q12h
Morphine	0.02–0.2 mg/kg	IV	(single)
Pethidine	0.4–2.0 mg/kg	IM	q24h
Dexamethasone	0.06–0.1 mg/kg	IV	q24–48h
Prednisolone	0.2–4.4 mg/kg	PO	q12–24h
Respiratory drugs			
Furosemide	0.5–1.0 mg/kg	IV	Pre-exercise
Clenbuterol	0.8–1.6 µg/kg 0.8 µg/kg	PO IV	q12h q12h
Dembrexine	0.3–0.5 mg/kg	PO	q12h
Potassium iodide	10–40 mg/kg	PO	q24h
Sodium iodide	100 mg/kg	IV	q24–72h

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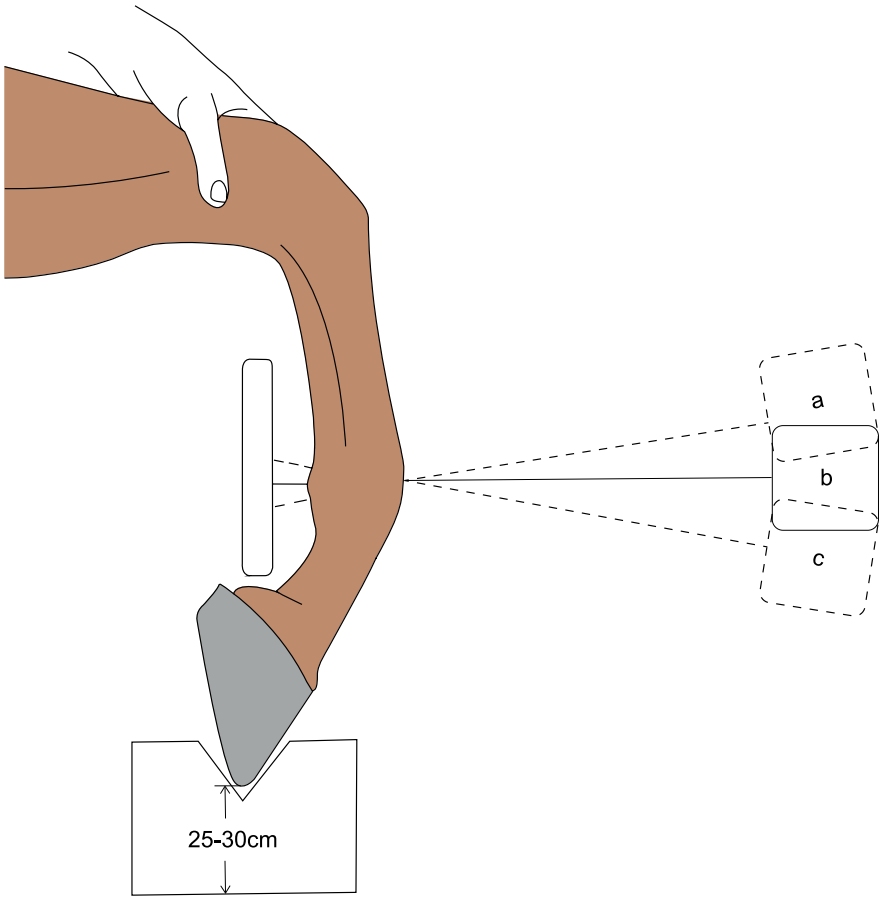
Table A3.1 **Drug administration reference table** ***(Continued)***

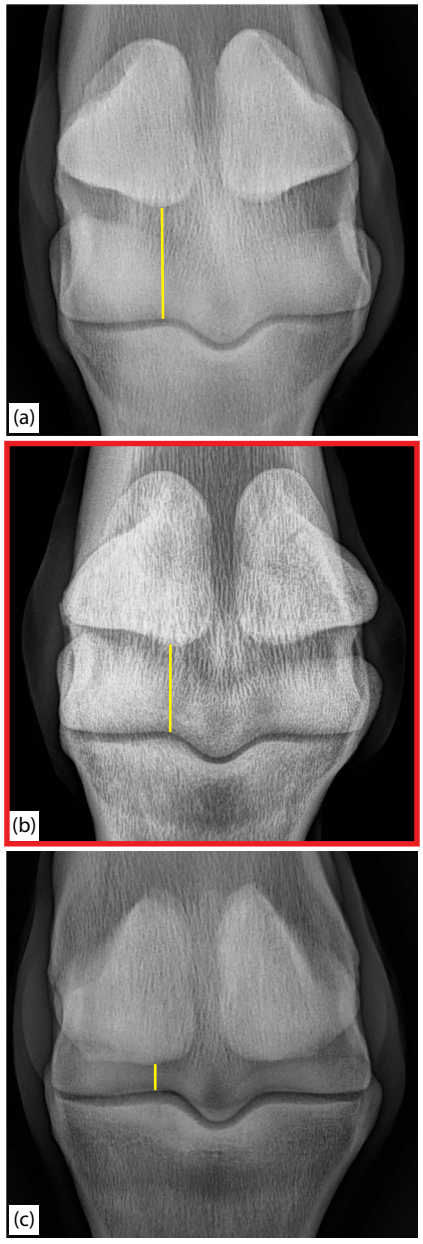
DRUG	DOSAGE (BWT)	ROUTE	FREQUENCY
Gastric ulcer drugs			
Omeprazole	2–4 mg/kg	PO	q24h
Misoprostol	5 µg/kg	PO	q12h
Sucralfate	12 mg/kg	PO	q12h
Miscellaneous orthopaedic drugs			
Dantrolene sodium	1–4 mg/kg	PO	Pre-exercise
Tiludronate	1 mg/kg	IV	(Single)
Sedative drugs			
Acepromazine	0.02–0.06 mg/kg	IV	As required
Detomidine	0.004–0.02 mg/kg	IV	As required
Xylazine	0.2–0.8 mg/kg	IV	As required
Butorphanol	0.01–0.04 mg/kg	IV	As required
Inhaled respiratory drugs			
Beclomethasone	500–3750 µg (total)	MDI	q12–24h
Fluticasone	2000 µg (total)	MDI	q12h
Ciclesonide	2700 µg (total)	MDI	q12h
Ipratropium bromide	2–4 µg/kg	MDI	q12h
Salbutamol	360–720 µg (total)	MDI	q12–24h

BWT, bodyweight; IM, intramuscular; IV, intravenous; PO, per os (by mouth); MDI, metered dose inhaler (inhaled); q, each/every.

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Guide for flexed dorsopalmar radiographic projection of forelimb fetlock.

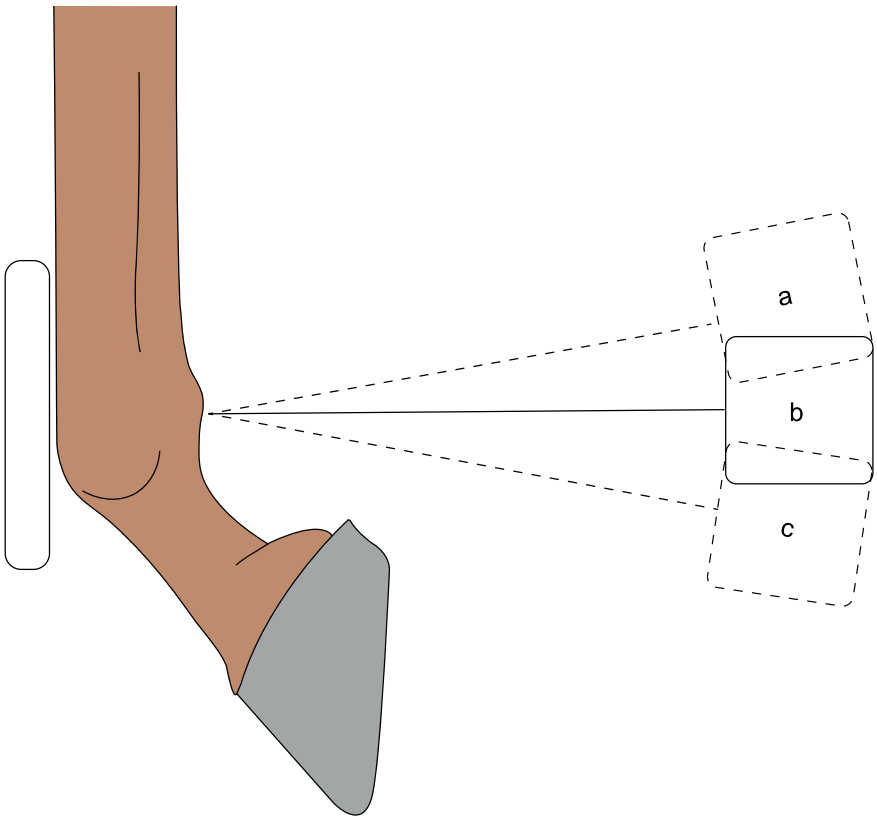


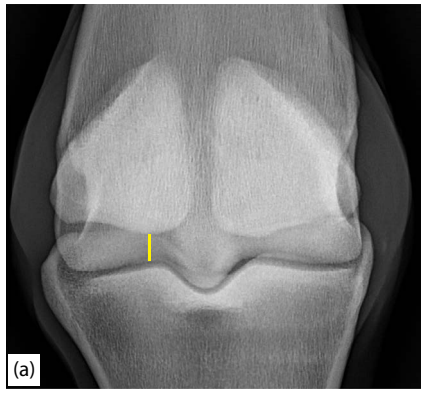


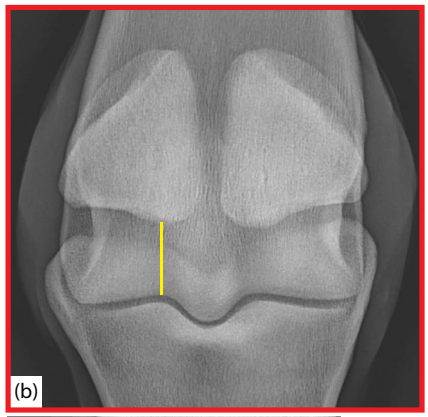
Tips: Bring the leg forward and place on a block with height of 25-30 cm/don't fully flex the fetlock/x-ray plate can rest on the heels or behind the foot/make initial projection horizontal and perpendicular to the fetlock/when screening for pathology, take multiple projections (proximodistal and distoproximal) to highlight different aspects of joint.

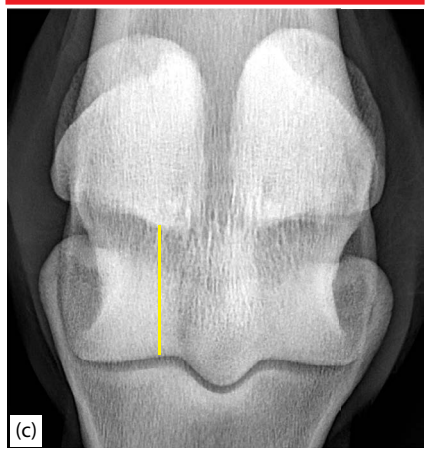
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Guide for flexed plantarodorsal radiographic projection of hindlimb fetlock.





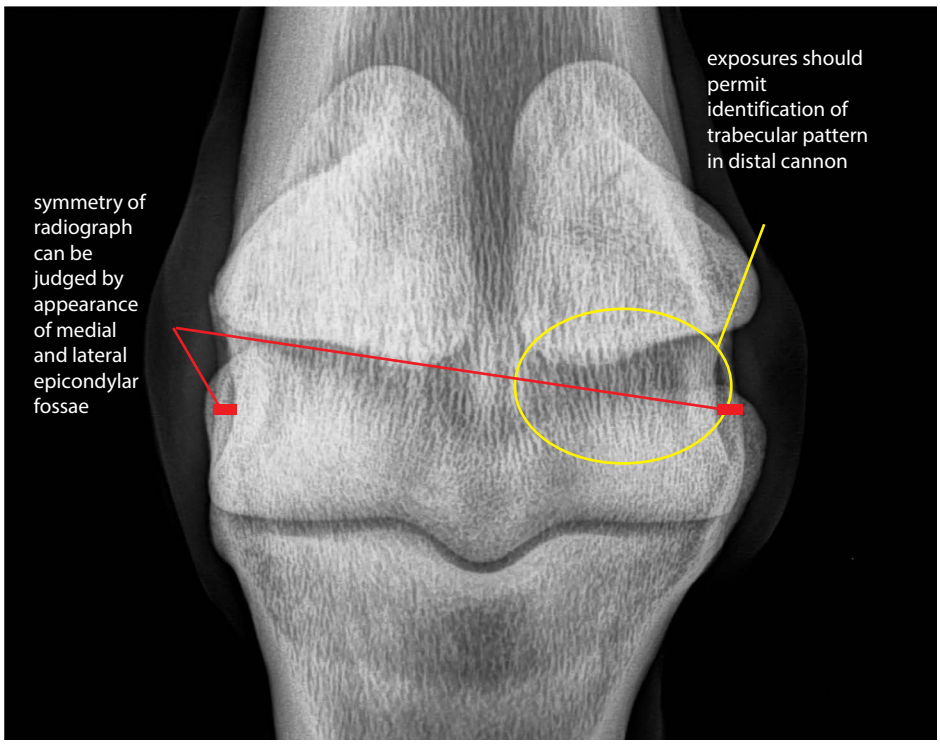


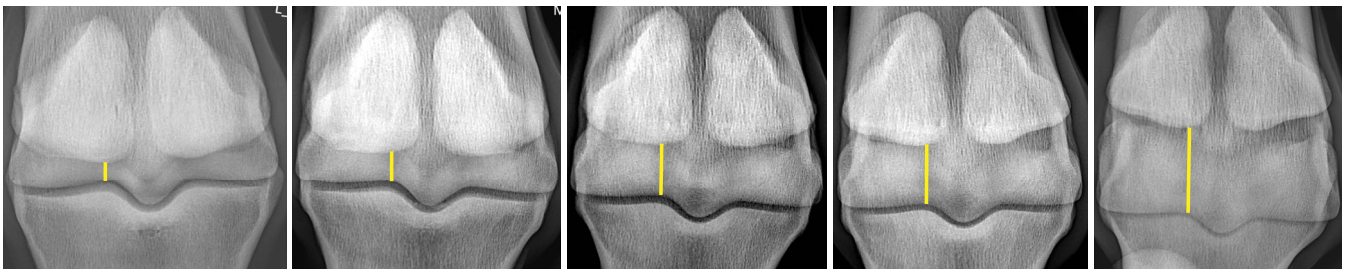


Tips: The assistant picks up the hindlimb and supports it with one arm under the hock, holding the X-ray plate flat against the dorsal aspect of the fetlock with the other/hold the limb so that the cannon is vertical

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Features of a diagnostic-quality image

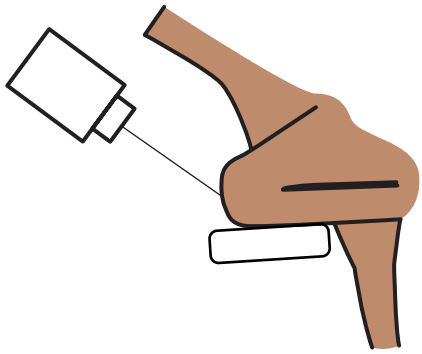




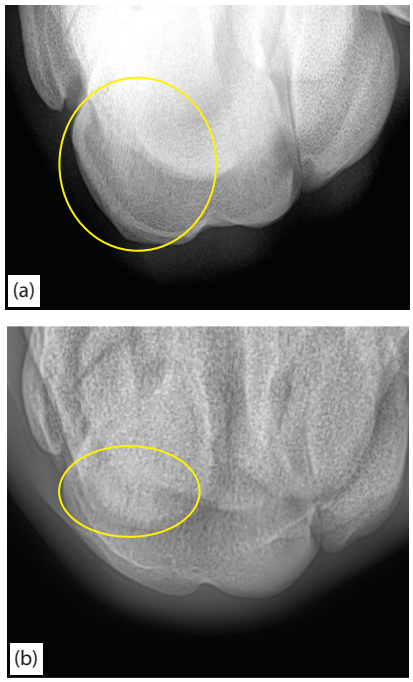
Multiple exposures of slightly varying proximodistal obliquity should be obtained if pathology is suspected, or to further define any radiological irregularities noted on initial screening.

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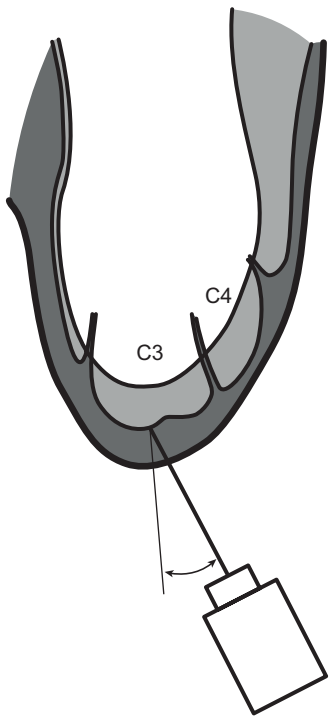
Guide for 'skyline' (DPr-DDiO) distal carpal row radiographic projection.

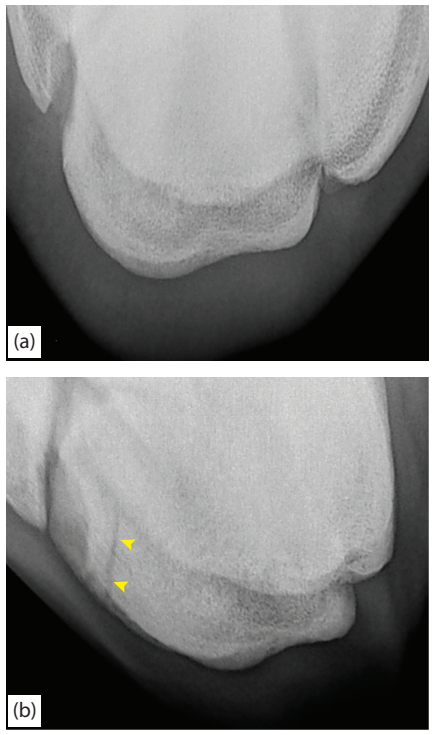


Tips: The limb is raised to full flexion by the assistant and supported by resting the cannon on the radiographic cassette./The cassette is held horizontally, preferably by an assistant on either side of the horse./The cannon and radius should be in sagittal alignment./Xray beam should be directed proximally to distally with a beam-cassette angle in the range 30-40°.



The ideal radiographic image permits assessment of the full dorsal portion of the third carpal bone, particularly the radial facet (circled, (a)); superimposition of the proximal row of carpal bones (b) is to be avoided.





If fracture pathology is suspected, 'skyline' projections with dorsolateral obliquity (DPrL-PDiMO) should be obtained. Range of lateral obliquity varies between individuals (15–35°).

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GUIDE TO BEST PRACTICE FOR HUMANE DESTRUCTION IN EMERGENCY SITUATIONS

In the case of suspected but not definite grounds for immediate euthanasia a second opinion should be sought before proceeding.

CONDITION	IMMEDIATE DESTRUCTION	PROGNOSIS: PASTURE	PROGNOSIS: ATHLETIC
Skeletal			
Pastern fracture: non-comminuted	No	Good	Good–Guarded
Pastern fracture: comminuted- one intact strut	No	Guarded	Guarded–Poor
Pastern fracture: comminuted- no intact strut	Yes	Poor–Hopeless	Poor–Hopeless
Condylar (MC3/MT3) fracture:	No	Good	Good–Guarded
Condylar (MC3/MT3) fracture: compound + comminuted	Yes	Poor–hopeless	Poor–hopeless
Compound long bone fracture	Yes	Poor–Hopeless	Poor–Hopeless
Humeral/radial/tibial/femoral fracture: displaced	Yes	Poor–Hopeless	Poor–Hopeless
Third carpal bone fracture	No	Good	Good–guarded
Multiple carpal/tarsal bone fractures (+ carpal/tarsal instability)	Yes	Poor–Hopeless	Poor–Hopeless
Pelvic fracture: standing	No	Good	Good–guarded
Pelvic fracture: recumbent	Yes	Poor–hopeless	Hopeless
Pedal bone fracture	No	Articular: good–guarded Non-articular: good	Articular: good–guarded Non-articular: good
Navicular bone fracture	No	Good–guarded	Guarded–Poor
Soft tissue			
SDFT tendonitis	No	Good	Good–guarded
SDFT rupture (below carpus/tarsus)	No	Good–guarded	FL: guarded–poor HL: good
SDFT rupture (musculotendinous junction)	Yes	Poor	Hopeless
SDFT rupture (bilateral)	Yes	Poor	Hopeless
Complete breakdown of suspensory apparatus	No	Guarded–poor	Hopeless
Partial laceration of SDFT/DDFT +/– suspensory ligament	No	Good	FL: guarded–poor HL: good
Complete laceration of SDFT	No	Good	FL: guarded–poor HL: good
Complete laceration of SDFT+ DDFT	No	Fair–good	Poor
Complete laceration of SDFT, DDFT + suspensory ligament	Yes	Very poor	Hopeless

(Continued)

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CONDITION	IMMEDIATE DESTRUCTION	PROGNOSIS: PASTURE	PROGNOSIS: ATHLETIC
'Slipped' tendon (displacement of SDFT from point of hock)	No	Good	Good–poor
Synovial sepsis (acute/chronic)	No	Acute: good Chronic: poor	Acute: good–guarded Chronic: poor
Neurological			
Spinal fracture + hindlimb paralysis/paresis	Yes	Hopeless	Hopeless
Recumbent non-responsive: post trauma	Yes (+ 2nd opinion)	Hopeless	Hopeless
Wobbler syndrome (grade 1–3)	No	Good–guarded	Good–guarded
Wobbler syndrome (grade 4)	No	Guarded–poor	Poor–hopeless
Wobbler syndrome (grade 5)	Yes	Poor–hopeless	Poor–hopeless

FL, forelimb; HL, hindlimb; MC3/MT3, third metacarpal/metatarsal; SDFT, superficial digital flexor tendon; DDFT, deep digital flexor tendon.

Modified from Appendix II of the BEVA / Equine Group of Veterinary Ireland document "A Guide to Best Practice for Veterinary Surgeons When Considering Euthanasia on Humane Grounds" (reprinted 2009).

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Note: Locators in *italics* represent figures and **bold** indicate tables in the text.

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