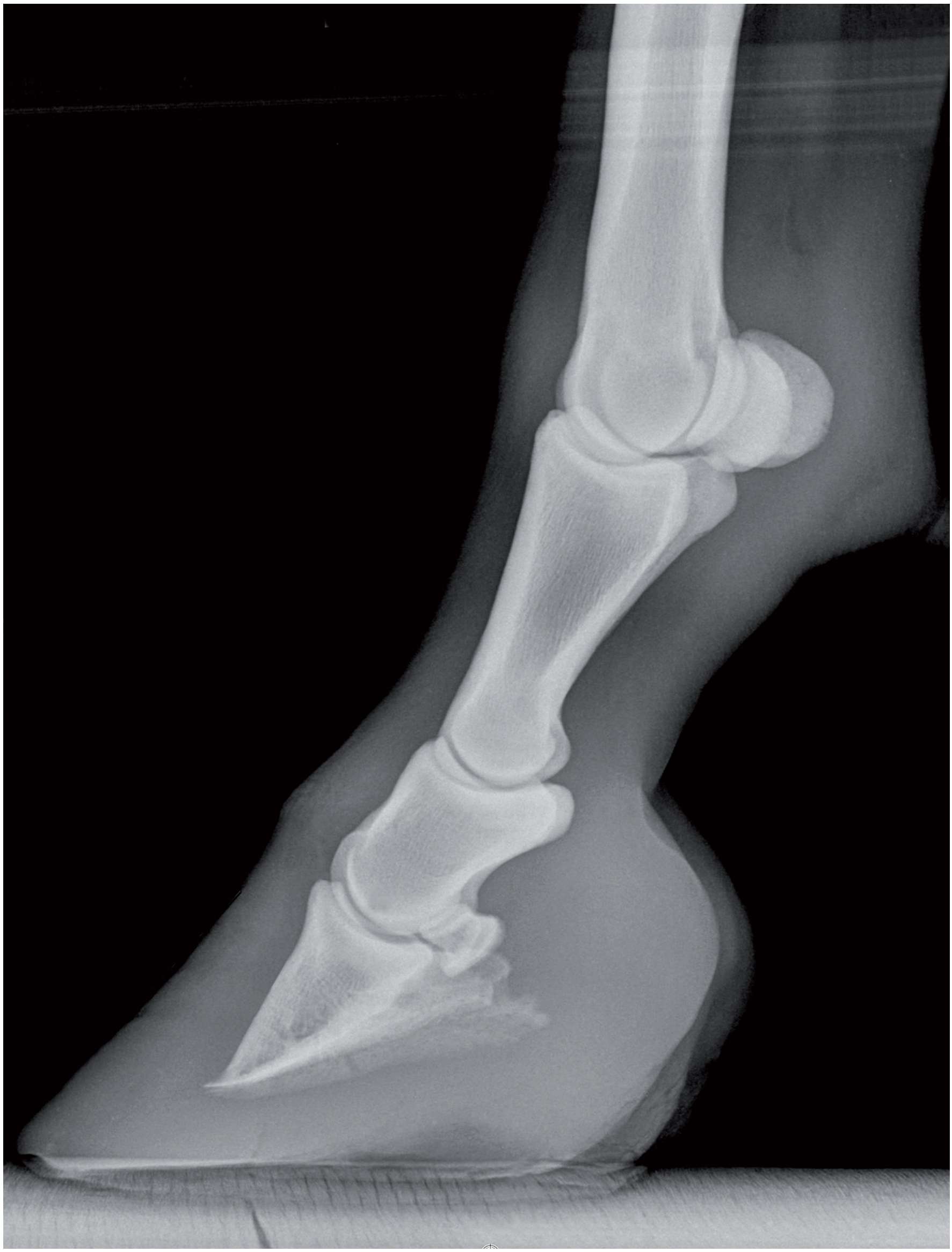
LAMINITIS

A HORSE-CENTRED APPROACH

RICHARD VIALLS
BSc, MSc, PhD, MEPA (UK)

LAMINITIS

A HORSE-CENTRED APPROACH



LAMINITIS

A HORSE-CENTRED APPROACH

RICHARD VIALLS

BSc, MSc, PhD, MEPA (UK)

J.A. ALLEN

First published in 2019 by
by JA Allen

JA Allen is an imprint of
The Crowood Press Ltd
Ramsbury, Marlborough
Wiltshire SN8 2HR

enquiries@crowood.com

www.crowood.com

This e-book first published in 2020

British Library Cataloguing-in-Publication Data
A catalogue record for this book is available from the British Library.

ISBN 978 1 90880 987 2

Acknowledgements
I would like to thank Sally Bell, Vikki Fear, Gina Foulkes-Halbard, Jayne Hunt, Oakdale Equine Rescue, Stuart Smith, Debra Taylor and Yvonne Thomas for supplying some of the photographs in this book. Special thanks go to my partner Clare Burrows for the countless hours of intelligent debate that have helped shape many of the ideas in this book, and also to my children Jacob and Lucas for putting up with me during its creation. Finally, I would like to thank all my clients (both equine and human) for contributing so much to my knowledge over many years.

Contents

Introduction

Laminitis is a condition that every horse owner dreads. And yet many owners (and many horse professionals for that matter) know little about either the causes of the condition or what actually happens inside the horse's foot as a result. The internet, text books and magazines are full of advice and comment, and yet much of this information is contradictory or inaccurate. This book is intended to help guide the horse owner through the process of understanding what laminitis is, what causes it, and what can be done about it.

I first became involved in hoofcare when my partner's horse Esmé went lame, and no one could find a solution for her problem. Faced with losing a promising young horse to a problem that none of the professionals we had engaged could explain, my scientific curiosity got the better of me and I started reading up about hooves. Having come from a background of physics and electronic engineering, I was shocked by the overall poor quality and paucity of research into the equine foot (although some individual pieces of research are outstanding). Many of my simplest questions about how the foot functioned appeared to be unanswered.

Traditional farriery failed to provide the answers that Esmé needed, and in desperation we turned to the newly emerging discipline of equine podiatry. As I studied the subject more and more, and eventually trained to become an equine podiatrist myself, I began to become excited about a topic of science where some of the basic research was yet to be done. In most branches of science today, little progress can be made without millions of pounds of funding. And yet here was a topic where even some of the basics were yet to be investigated, and there were still opportunities for the kind of low-key science that has historically underpinned so many scientific disciplines. That excitement has only grown over the years as I've found myself playing my own very small part in advancing our understanding of the equine foot.

One reason why equine podiatry has had, and continues to have, such an important role to play in this area is that equine podiatrists are typically people entering a second career, often bringing with them a wealth of transferable skills from their previous lives. This has created a culture that is significantly different from that within the profession of farriery. Farriers are craftspeople who are highly skilled in the art of making and fitting shoes, but they do not typically come to their profession with scientific training. In contrast, equine podiatrists in the UK, especially those who form the membership of the Equine Podiatry Association, have taken a far more evidence-based approach to hoofcare. A critical part of this is the practice of taking detailed notes and photographs, which includes data not only on the hooves and the soundness of the horse, but also on its environment, including diet. This has created a vast library of data on how hooves respond to different interventions, and has allowed patterns to be identified, links to be made between the health of the foot and a range of environmental factors, and, arising from this, the development of new approaches to the management of common foot problems such as laminitis. This research has yet to be formally published, but is being used very successfully in daily practice in the UK by the twenty plus members of the Equine Podiatry Association, working with around 2,000 horses. It has also been assimilated into the work of many other hoof-trimming groups around the world, again with good levels of success.

Most cases under the care of an equine podiatrist are unshod. This typically represents two separate subgroups of horses. At one end of the spectrum are the horses with good feet who, often with minor tweaks to their environment, are capable of significant levels of work without shoes. The common myth that horses without shoes cannot do much road work does not stand up in the face of the many cases that are routinely doing tens of miles per week on challenging terrain. Not every horse

is capable of working at this level without shoes, but with appropriate management of their environment (and especially their diet), many more can do so than has been traditionally believed.

At the other end of the spectrum are the horses with major foot problems. The traditional approach to these cases has often been ever more complex remedial farriery. This has evolved from the historical position where horses were typically working animals and needed to be kept sound and in work or they had no value. Traditional remedial farriery has understandably been biased towards masking and delaying problems, rather than taking the horse out of work for the significant length of time often needed to resolve them. Today, many more horses are considered to be pets, and even working horses are typically considered more as long-term investments, and hence this approach is less of a good match to need.

A horse shoe supports the horse's weight primarily via the hoof wall and the laminae, ignoring other structures that the wild horse has evolved to use as well. Given the huge degree of redundancy of structure in the equine foot, this is not generally a significant problem for the healthy horse. But when considering the long-term health of a horse with problem feet, an approach that only utilizes a part of the natural strength of the foot risks being a poor solution. That is not to say that equine podiatrists are somehow anti-shoe – far from it. But a shoe is a tool that can be used to fix certain problems and shouldn't be seen as the correct solution to every problem. If your only tool is a hammer, then you are going to struggle when faced with a screw. In my experience, shoes are great for otherwise healthy horses that over-wear their feet when worked unshod, but are often less useful as a means of supporting a foot that is failing structurally. It is common for vets and farriers to recommend shoeing such feet to 'provide support', but when questioned on exactly how the shoe provides such support, most struggle to give meaningful answers.

In Esmé's case, remedial farriery failed to resolve her mystery lameness, repeated quarter cracks and frequent abscesses. Indeed, as we employed ever more respected farriers, her problems only got worse. We eventually reached the point where we had nothing to lose, and so in desperation took her shoes off. To everyone's surprise she showed almost immediate signs of improvement. That was the start of a long road of discovery. A key milestone on that road was my gradual recognition of the role that low-grade laminitis played in her case – but the journey continues.

What has become apparent to me over the years is that the feet are just one part of a complex biological system which is interconnected in a myriad of ways. It is very clear to me now that tackling foot problems in isolation is no more than tilting at windmills. The word 'holistic' has been much misused and maligned over the years, but it is the right word to use. Equine hoofcare *has* to be done within a holistic framework if we are to achieve any degree of success. Every part of the horse's environment has the potential to impact on the hooves, from the bedding in the stable, to the exercise regime and the surfaces the horse works on, through to (especially) the horse's diet.

The other thing we have to change if we are to be successful with our horses is the human habit of looking at everything from our own point of view. I hope to show in later chapters that this human-centric viewpoint clouds our thinking and obscures more helpful views of the problem. Reframing the problem of laminitis to put the horse at the centre of it unlocks a viewpoint that is far more useful, whether that relates to the mechanical effects of laminitic rotation in the foot, or to the role the horse's central nervous system has on posture, gait, and hence the distribution of mechanical forces in the foot. That is the reason why this book is subtitled 'a horse-centred approach'.

Finally, I must add a few disclaimers…

The information that follows relates to laminitis in the horse. Laminitis in the donkey, mule or hinny (or zebra for that matter) is outside the scope of this book. That said, most of the contents of this book can be made to apply to other species of equid with only minor tweaking.

This book contains many photos, including some of dissected feet, which some people may find distressing.

And perhaps most importantly, laminitis is a condition that can cause significant pain to the horse and should be considered a veterinary emergency, especially in severe or acute cases. This book is not

a substitute for professional veterinary involvement, but is rather an attempt to inform readers so that they can have a more productive interaction with the professionals involved in the care of their horses.

Part 1
What is Laminitis?

1 Essential Anatomy

It is not possible to discuss the subject of laminitis without first having a reasonable understanding of the anatomy of the equine foot. This chapter attempts, without going into excessive detail, to provide a quick guide to those aspects of basic anatomy that are necessary to understand the remainder of the book. As well as covering the names for the key structures, this chapter also looks at what functions these structures have. This helps to make the rather dry subject of anatomy a little more interesting.

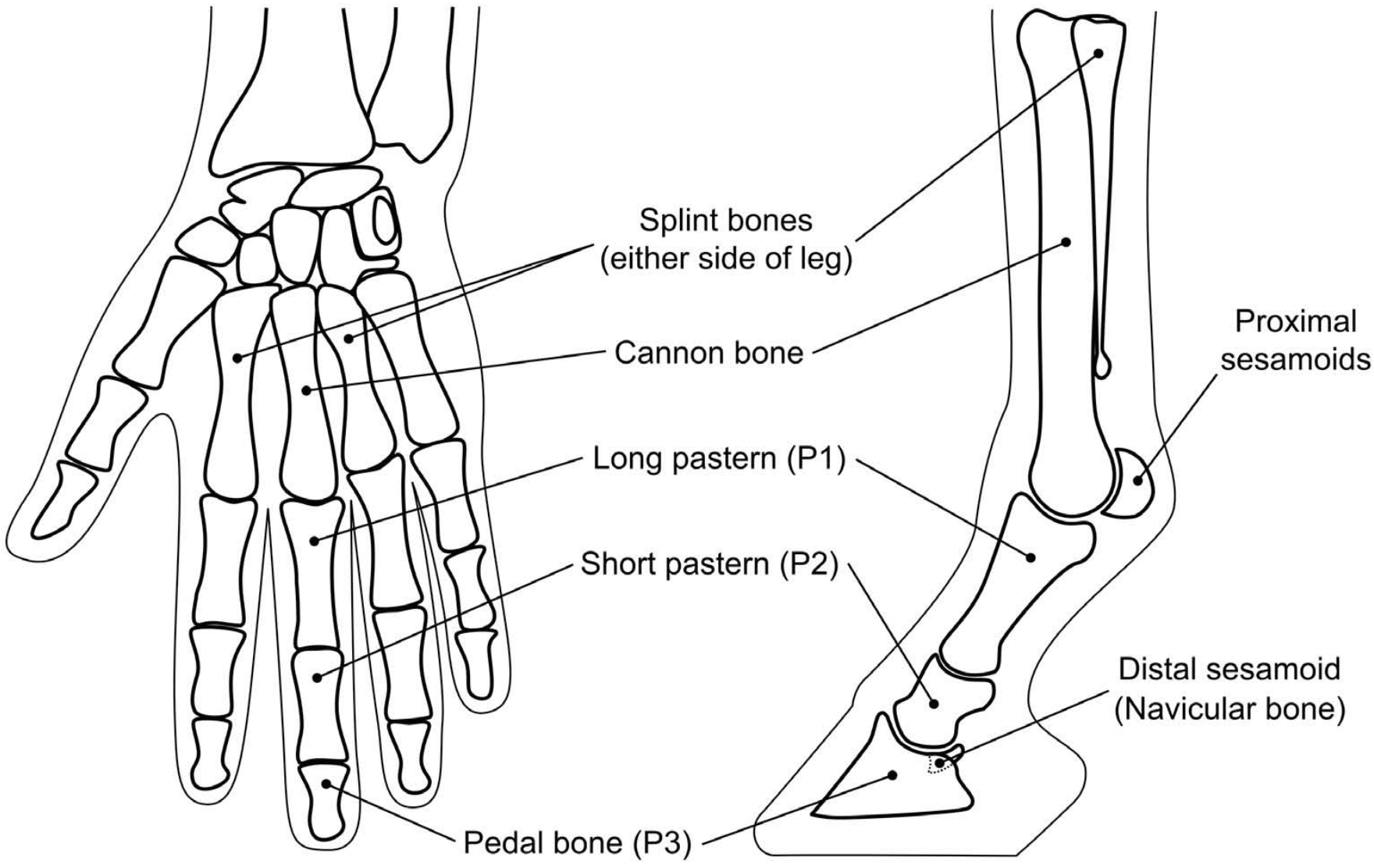


Fig. 1 The bones of the lower foreleg of the horse compared to the equivalent bones in the human hand. The proximal and distal sesamoids are not present in the human hand.

BONES

Horses and humans have evolved from common ancestors and share similar bone structures. The bones of the horse's lower leg (from the knee/hock downwards) closely map to the bones of the human hand/foot, with the horse having largely lost all but the middle finger/toe. The horse's knee is equivalent

to our wrist, and the horse's hock is equivalent to our ankle. For that reason the lowest part of the horse's leg is often referred to as the digit (just as our fingers and toes are digits).

The bones most relevant in the discussion of laminitis are the three phalanges (singular phalanx) – these are the finger and toe bones in a human, but in a horse they provide the underlying bone structure to the pastern and foot.

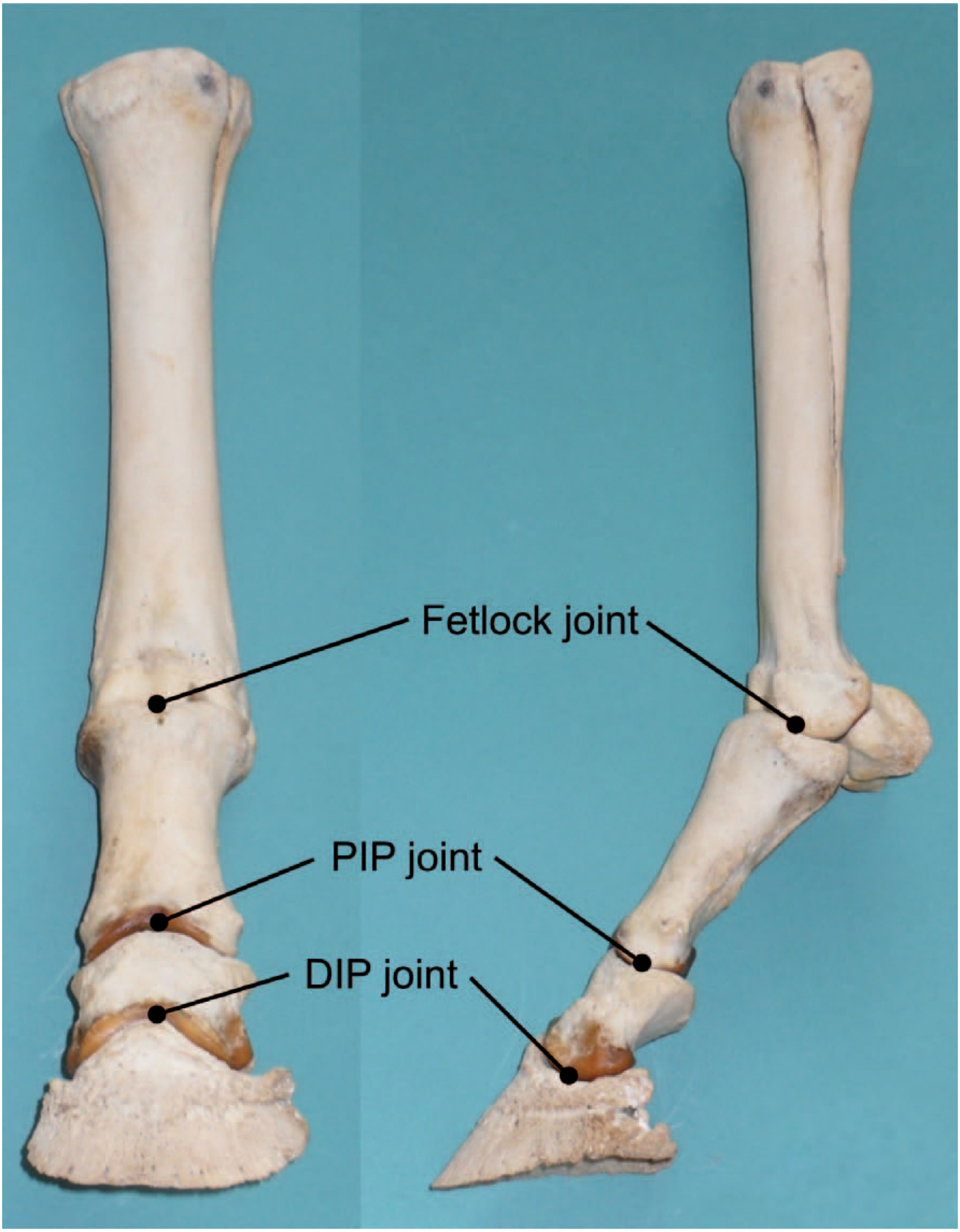


Fig. 2 A set of bones of a lower foreleg wired into their correct positions.

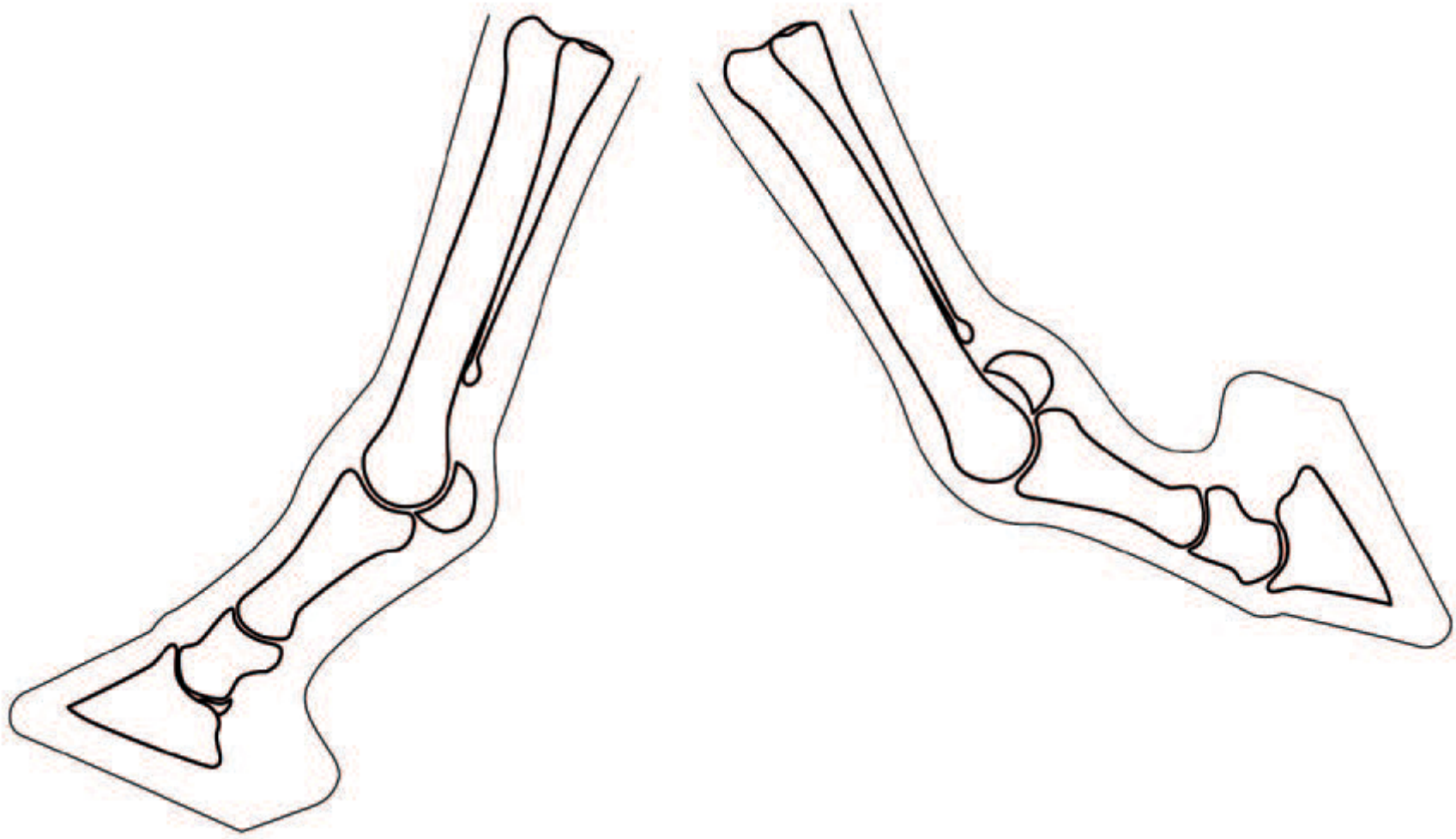


Fig. 3 The bones of the lower leg in extension (left) and flexion (right).

JOINTS

Between each adjacent pair of bones in the lower leg there is a hinge joint – a joint which can extend (curl forwards) or flex (curl backwards). Those parts of each bone that are involved in a joint are covered with articular cartilage, which is smooth and shiny. This allows the two bones to move in relation to each other. To further minimize friction, the joint is lubricated with an oily liquid called synovial fluid. The entire joint is then encased in a membranous sac called a bursa so as to keep the synovial fluid in place. Where two or more joints are very close together, they sometimes share a single bursa.

The pastern is made up of the long and short pastern bones (often abbreviated to P1 and P2 for first and second phalanges). Between these two bones sits the pastern joint. The technical term for this joint is the ‘proximal interphalangeal joint’ – often abbreviated to PIP joint. The pastern joint can extend and flex but only by very small amounts, which is why most horse owners are unaware that the pastern has a joint in the middle of it. The main role of this joint is to allow absorption of some of the impact of the foot hitting the ground by extending very slightly.

The third phalanx is the pedal bone (sometimes also called the distal phalanx or coffin bone, and often abbreviated to P3). Between P2 and P3 is the distal interphalangeal joint, or DIP joint (sometimes also referred to as the coffin joint). Unlike the PIP joint, the DIP joint can extend and flex through quite a large range of movement.

At the back of the DIP joint, between P2 and P3, sits the navicular bone. This bone is tightly attached to the back of P3 by the impar ligament and so for the most part it moves with P3 as the DIP joint flexes and extends. What little movement there is between P3 and the navicular bone allows the flexor tendon (see below) to pass into the foot at the most advantageous angle as the foot lands on uneven ground.

TENDONS AND LIGAMENTS

Bones alone are not enough to allow a horse to move. To start with, something has to keep the joints from dislocating, and this is the task of ligaments. Ligaments are connective tissues that, in most cases,

join bone to bone. They can be thought of as like strong rubber bands. Each of the joints in the lower leg has collateral (on each side) ligaments that hold the joint together whilst allowing it to move. Another important ligament is the suspensory ligament, which passes down the back of the cannon bone, under the fetlock and attaches to the back of the pastern, as well as splitting to form two branches that pass either side of the pastern and attach at the front. The role of this ligament is to support the weight of the horse at rest and prevent the fetlock from dropping to the ground. The horse uses the suspensory ligament rather like a spring, and much of the efficiency of movement in a horse comes from its ability to bounce off the lower legs like a pogo stick. The arrangement of the pastern, fetlock and suspensory ligament works in rather the same way as the running blades used by human amputees.

There are many other ligaments in the lower leg that hold joints together and act as support bandages, keeping important structures where they should be. The end result of all these ligaments, as well the joints being held together, is that the bone column has a ‘neutral’ position that it will adopt when no muscles are brought into play.

For the horse to move around, it needs to be able to flex and extend the lower limb under the control of muscles. But muscles are heavy structures, and having them in the lower leg would make the leg heavier and hence reduce the horse’s ability to run away quickly from predators. So instead, the muscles are located higher up the leg and pull on the relevant bones using tendons. Tendons are similar to ligaments but act more like strings than rubber bands. Each tendon is pulled by a muscle at one end and is attached to a bone at the other, allowing the muscle to move the bone from a distance.

There are three main tendons in the lower leg. The extensor tendon (its full name is ‘common digital extensor tendon’ or ‘CDET’ on a front leg, and ‘long digital extensor tendon’ or ‘LDET’ on the hind leg) sits at the front of the leg and acts to extend the lower leg. The horse typically pulls on the extensor tendon just before the foot hits the ground to lift the toe. Its main role is to ensure that the foot hits the ground with the heel first, with no risk of the horse tripping over the toe.

At the back of the leg are two flexor tendons. The deep digital flexor tendon (DDFT) connects to the back of the pedal bone and has the effect of flexing the DIP joint. The superficial digital flexor tendon (SDFT) – so named because it is closest to the surface at the back of the cannon bone – acts largely to flex the fetlock joint. These two tendons between them have two roles. The first is to fold the foot and pastern up behind the leg while the limb is in flight, so minimizing the risk of catching the foot on uneven ground, vegetation and suchlike. The second is to assist in propelling the horse forwards during breakover (the point at which the heels have left the ground but the toe is still in contact). Much of this propulsion comes from the release of energy stored during the previous impact in the suspensory ligament, but the flexor tendons play an important role in controlling the release of this energy.

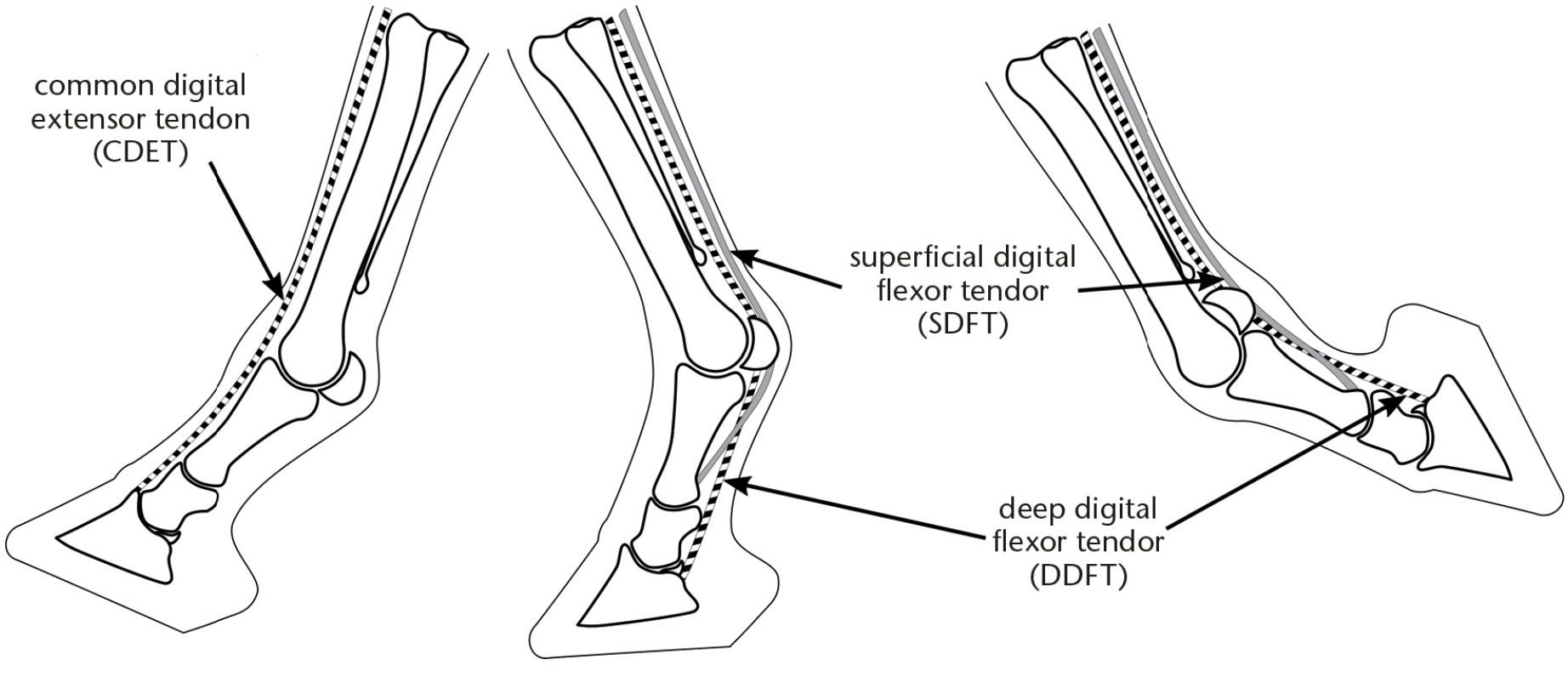


Fig. 4 The role of the tendons. LEFT: ***The limb extended prior to impact as a result of tension in the CDET.*** CENTRE: ***The limb at the point of breakover, with the SDFT and the DDFT contributing to propulsion.*** RIGHT: ***The limb flexed during flight as a result of tension in the SDFT and the DDFT.***

MAJOR FOOT STRUCTURES

Of the bones of the lower limb, two and a half sit within the foot: the pedal bone, the navicular bone, and the lower half of the short pastern bone. The pedal bone in particular gives the front portion of the foot its shape. There is, however, no bone in the back of the foot. Given that a sound horse on hard ground lands heel first, this makes sense – the horse does not want to jar bones by landing directly on to them. Instead, the shape of the back of the foot is formed by two lateral cartilages (each of which controls the shape of one heel), between which lies the digital cushion.

The shape and position of the lateral cartilages in a healthy foot supports the pedal bone such that it is tipped slightly forwards (the optimal angle is believed to be between 3 and 5 degrees), rather than being oriented flat to the ground. The result is that, with the outer horny structures removed, the internal structures of the foot form an arch similar to that seen in a human foot. As the foot hits the ground (heel first), the lateral cartilages flex slightly, which allows the arch to flatten somewhat, so absorbing and storing some of the energy of impact. As the foot leaves the ground again, the arch springs back to its normal shape, releasing much of that stored energy in a way that helps to propel the horse forwards. You can think of the internal arch as a form of spring on the bottom of the foot, with which the horse can bounce off the ground. Although this is a much smaller effect than the bounce the horse gets from the suspensory ligament, it nevertheless contributes to saving the horse energy and making movement more efficient.

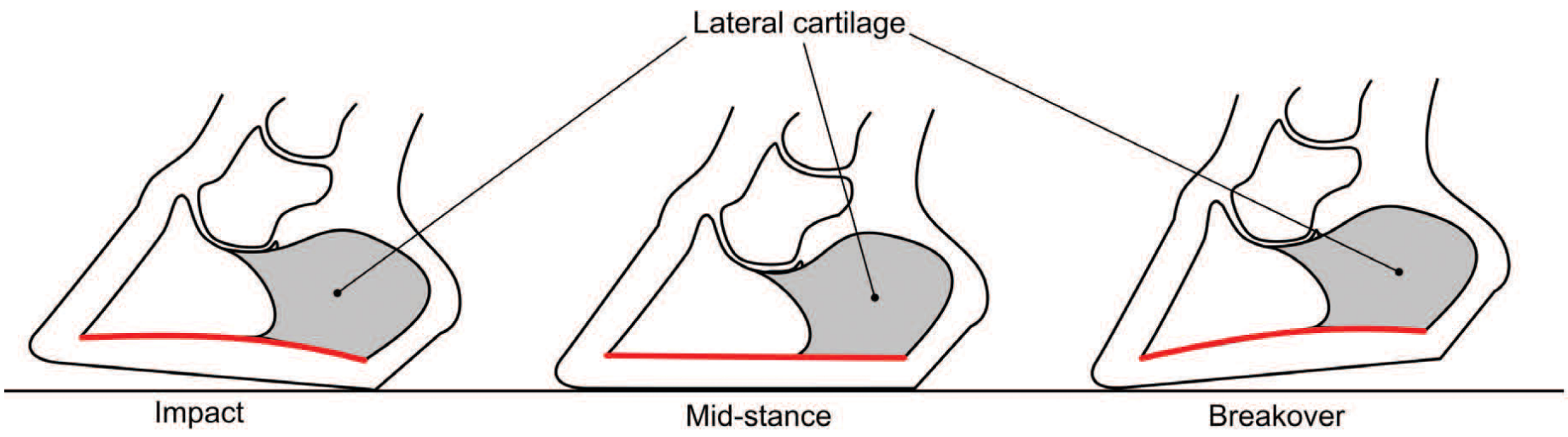


Fig. 5 The role of the lateral cartilages at impact, mid-stance and breakover. The internal arch is indicated in red.

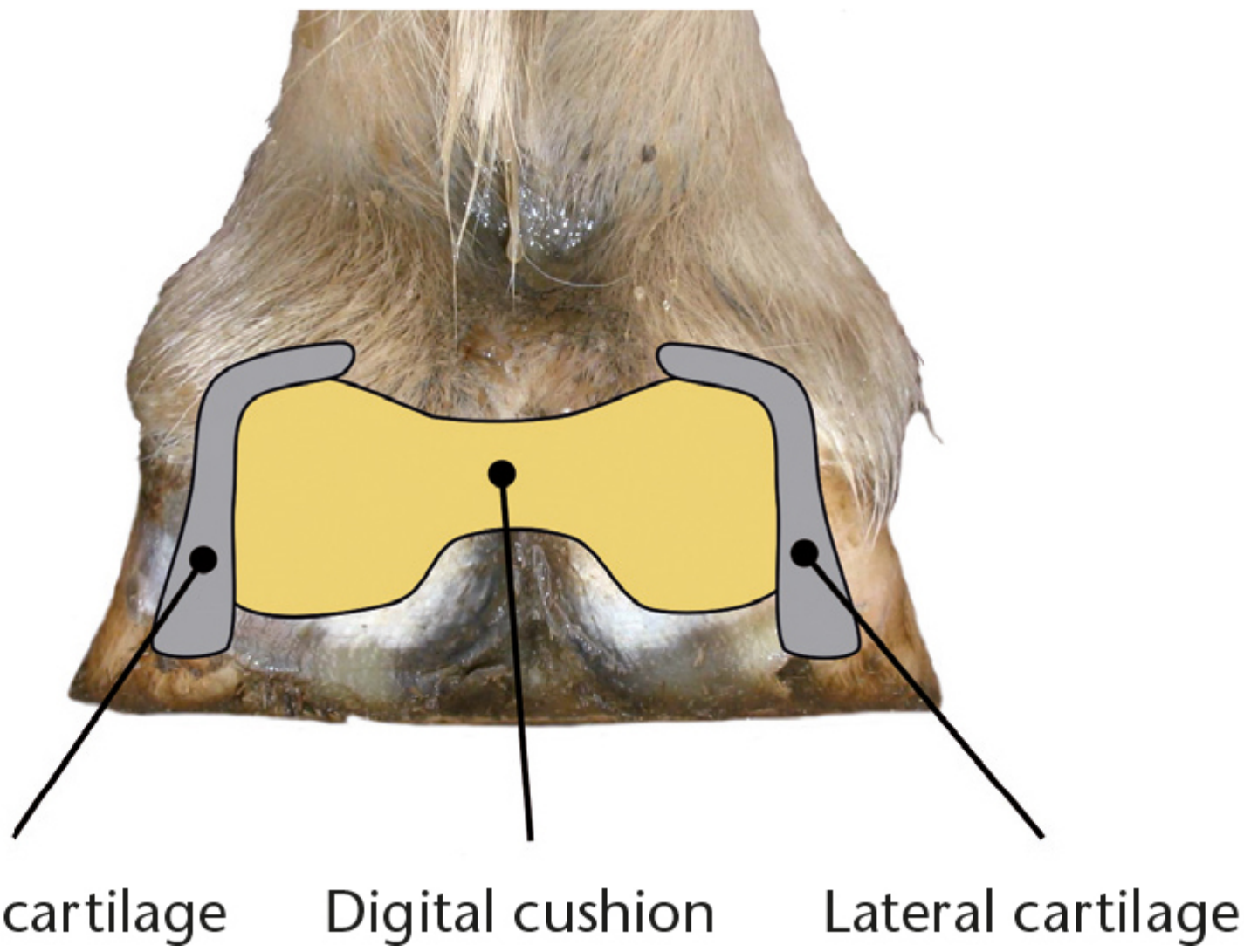


Fig. 6 The position of the digital cushion and lateral cartilages (viewed from the back of the foot).

The digital cushion sits between the lateral cartilages, and the top part of it can be felt by pressing down on the foot above the heel bulbs and between the lateral cartilages. Its main role is to absorb the vibration associated with the impact of the foot on hard ground so that this vibration doesn't reach the bone column (where it could cause inflammation and damage, typically in the form of arthritis in places such as the navicular bone, coffin joint and so on). In a healthy foot, the digital cushion should fill the space between the lateral cartilages such that there is little or no depression in the centre of the back part of the foot. It should ideally also feel like a block of hard rubber. Where it is small and feels more like chicken fat, this suggests that it has atrophied and that it won't work so well as a shock absorber.

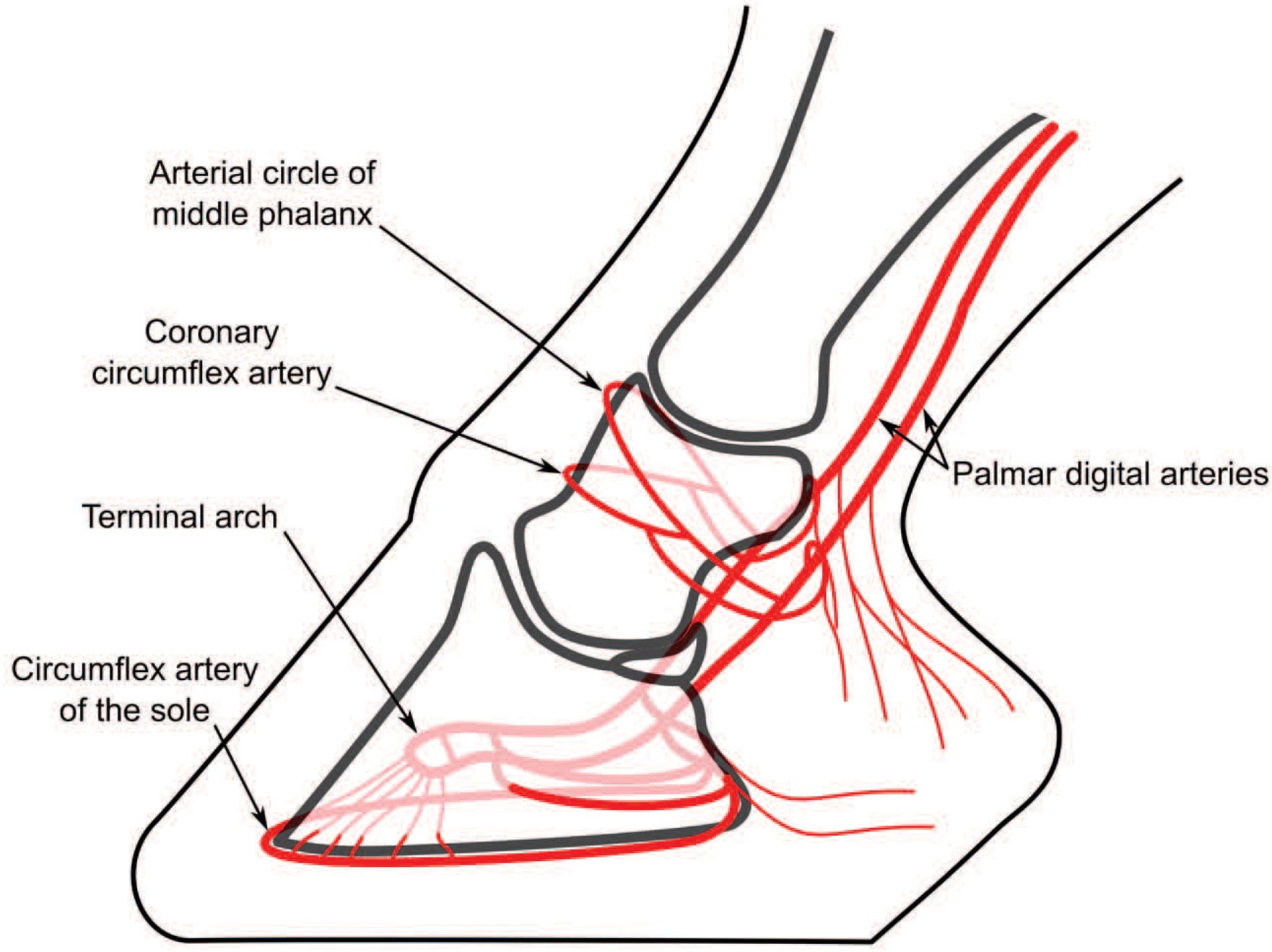


Fig. 7 The major arteries of the foot.

THE BLOOD SUPPLY

The details of the blood vessels in the foot are beyond the scope of this book, but two arteries within the foot are worth mentioning. The terminal arch circles through the middle of the pedal bone approximately half way up, and is the key arterial blood supply to the pedal bone. The circumflex artery of the sole (usually just referred to as the circumflex artery) passes around the rim of the pedal bone just outside and just below the bottom of the bone. The circumflex artery supplies both the lower portions of the pedal bone and the sole. It is important to note that the main way in which blood reaches the sole is via the circumflex artery, as there are no significant channels allowing blood to pass through the bottom surface of the pedal bone to supply the sole directly. As such, any damage to the circumflex artery will have a direct impact on the growth of the sole. The relevance of these two arteries in laminitis will become clear in later chapters.

The other arteries relevant to laminitis are the two palmar digital arteries, which provide the main blood supply to the foot and sit one on each side of the leg towards the back.

THE HOOF

The foot is defined as everything (including the hoof) below the hairline, in addition to the part of the lateral cartilages that protrudes above the hairline. The hoof (sometimes called the hoof capsule because it encapsulates the foot) is defined as all the avascular (not containing blood vessels) structures of the foot – which essentially means everything that is made of horn. The pedal bone, lateral cartilages and digital cushion form the shape of the foot. The external hoof then forms a hard casing around these internal structures. The hoof has a number of important roles, including the following:

- Protection of internal structures from mechanical trauma
- Prevention of infection
- Durability (the foot must not wear away too fast)
- Traction (so the horse doesn’t slip)
- Adaptation to environment (a horse living in a desert needs a different foot from one living in a bog)

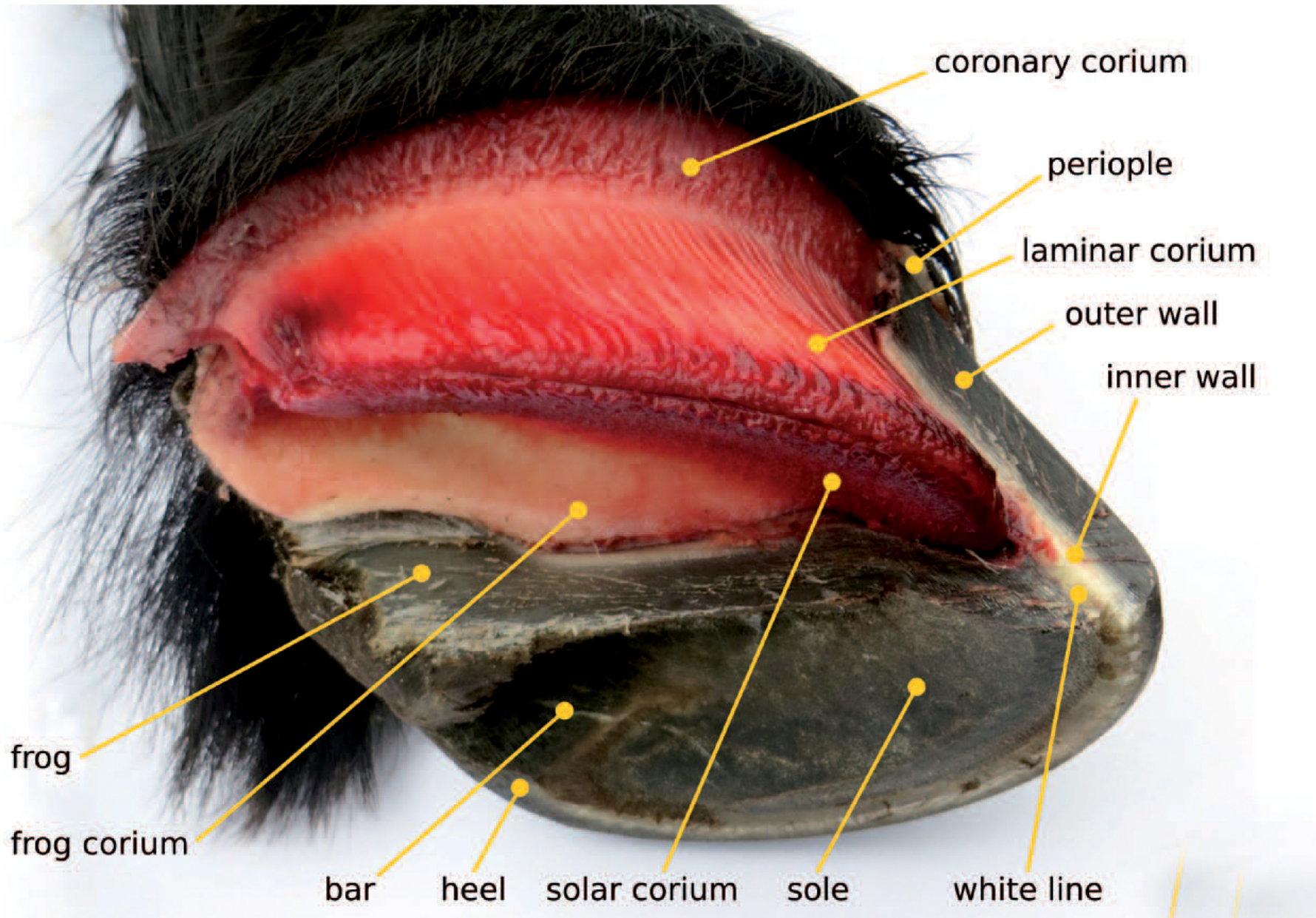


Fig. 8 A cut-away dissection showing the structures of the hoof. All hoof horn has been removed on one side of the foot so as to expose the underlying coria that produce the hoof (see below). The damage to the coronary band at the heel and the knife cut along the join between the solar and laminar coria are artefacts of the way the dissection was performed.

The horny hoof is divided into four separate structures: the sole, the frog, the hoof wall and the periople.

The sole: Sits under the pedal bone and the lateral cartilages. In an unshod horse, its main roles are to support the weight of the horse and to provide durability and traction.

The frog: A softer structure that sits between the heels and under the digital cushion. Again, it provides weight support, durability and traction in an unshod horse. It also allows (again where no shoe is applied) the heels to move independently of each other to an extent. This is similar to the independent suspension of a car, and allows the foot to deform to cope with uneven and sloping ground. Finally the frog allows the heels to move apart from each other slightly on impact, which helps the digital cushion absorb the vibration of impact.

The hoof wall: Encases the front and sides of the foot, and turns a corner at each heel to form the bars on each side of the frog. The wall is important in protecting the foot from knocks and scratches, and it also provides additional horn to wear away on the ground, hence contributing to the durability of the foot. The addition of the bars results in a greater amount of wall on the ground at the heels (where impact creates the greatest wear, and hence the need for the greatest surface area of hoof), and also helps to create a strong structure in the heel area whilst contributing to traction.

The periople: The equivalent of the cuticle on a human finger nail. The periople is a covering of skin that protects the hoof wall until it has had time to harden. As the hoof wall is produced, it is initially soft and only hardens after a few days.

In addition to the four sections of the hoof, the white line (which is actually a yellowy grey colour in a healthy hoof) serves as a joint between the rim of the sole and the inside surface of the hoof wall. This joint has to allow the wall to move past the sole as it grows down from the coronary band (as the wall grows faster than the sole) whilst preventing dirt, moisture and infection from getting into the foot. Strictly the white line is not a structure in its own right as it is formed from the combination of the edge of the sole and the inner surface of the hoof wall, but it is often treated as a discrete structure.

THE CORIA

Each of the four horny structures in the foot is produced by a corium. Corium is the vascular tissue that lies directly beneath the hoof. To understand how the hoof is produced, it is helpful to understand that hoof horn is just a modified form of skin. The hoof has no sweat glands or hair follicles, and the horn has a lower moisture content than normal skin, but otherwise it is more or less the same tissue.

Skin is formed of two layers. The inner layer (the dermis or corium) provides the underlying structure of the skin, including nerves and blood vessels. This layer is sometimes called the sensitive layer because it contains nerves. The outer layer (the epidermis) is the external skin we see. Between the two layers is a thin membrane called the basement membrane. Sitting at the bottom of the epidermis is a layer of cells one cell thick called the basal cell layer. These basal cells are responsible for producing the epidermal layer of the skin and exist in small groups of cells centred on a stem cell. The stem cells are responsible for producing more basal cells when required – these are very important when the skin is damaged as they allow any area of basal cells missing due to injury to be recreated from the stem basal cells surrounding the hole.

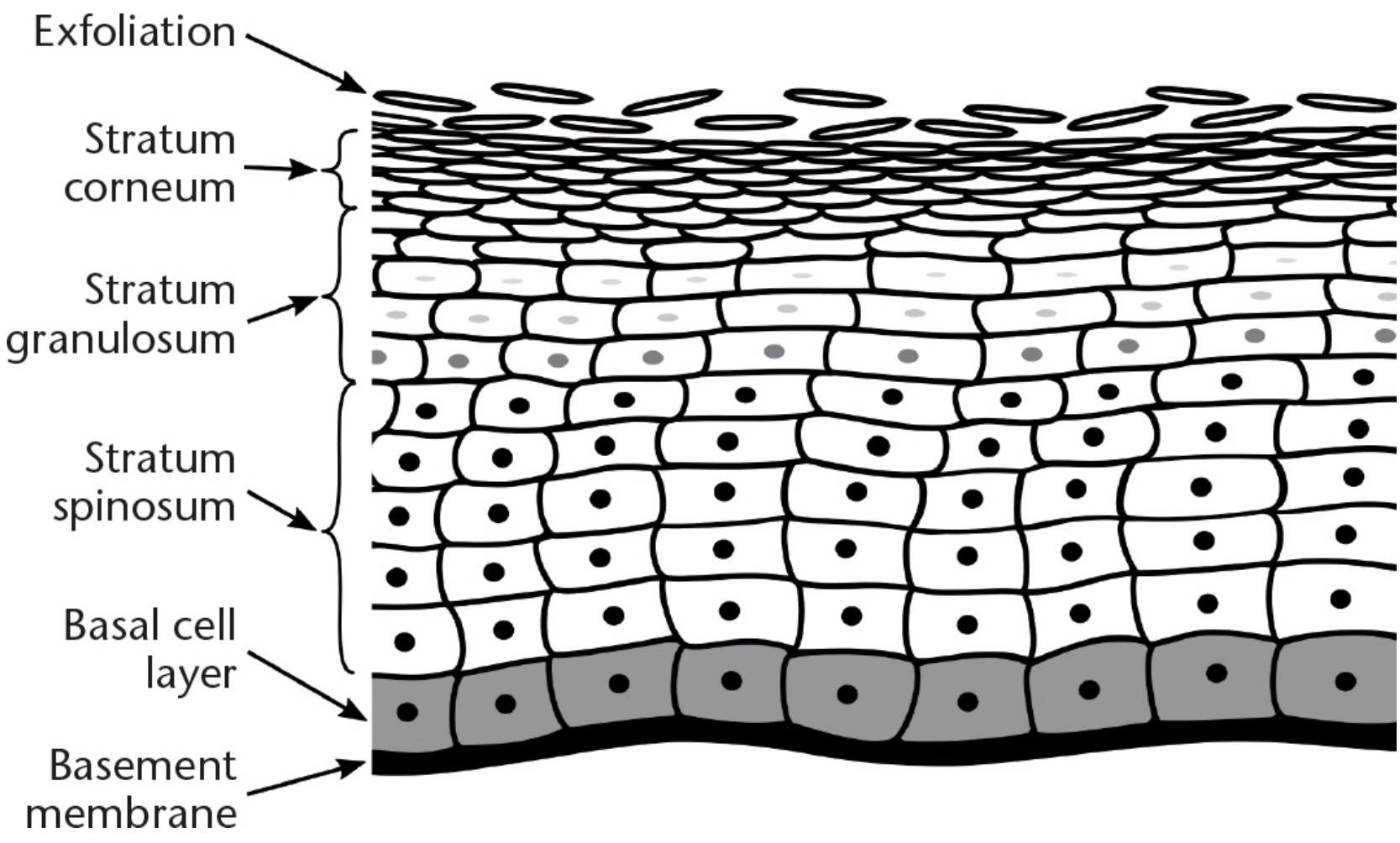


Fig. 9 A stylized representation of the structure of skin. In reality, with the exception of the basal cell layer, which is always one cell thick, each layer would have more cells in it than shown here.

The basal cells are responsible for creating the epidermal cells that form the layer of external skin that protects the body from infection, moisture, mechanical damage and so on. Each basal cell periodically produces a keratinocyte or skin cell by a process called mitosis – one basal cell divides into

two cells, one of which remains as a basal cell, the other becomes a keratinocyte. These keratinocytes gradually migrate outwards towards the outer surface of the skin, pushed there by the production of more keratinocytes below them.

Keratinocytes are so called because they are cells that gradually harden as they migrate towards the surface of the skin through various layers. They do this by manufacturing keratin, a protein which is extremely tough. As each cell keratinizes, it gradually loses its cell nucleus and also flattens until it forms a cell that is more scale-like in shape. These flattened cells are called squamous cells (from the Latin for scale) and they form the outermost and toughest layer of the skin, the stratum corneum (Latin for horny layer). In most cases the stratum corneum is very thin – it is only fifteen to twenty cells thick in human skin, for example. In a horse's hoof, however, the stratum corneum is much thicker – around 10 to 12mm in a healthy sole.

DESMOSOMES AND HEMIDESMOSOMES

Because skin needs to be tough, infection proof and moisture proof, it is important that the cells of the epidermis are tightly bonded together. A key mechanism for this is the presence of desmosomes between epidermal cells. These desmosomes act like rivets or spot welds, tightly attaching the cells together. The basal cells also need to be tightly attached to the basement membrane, and this is achieved by similar structures called hemidesmosomes (so called because they look like half desmosomes).

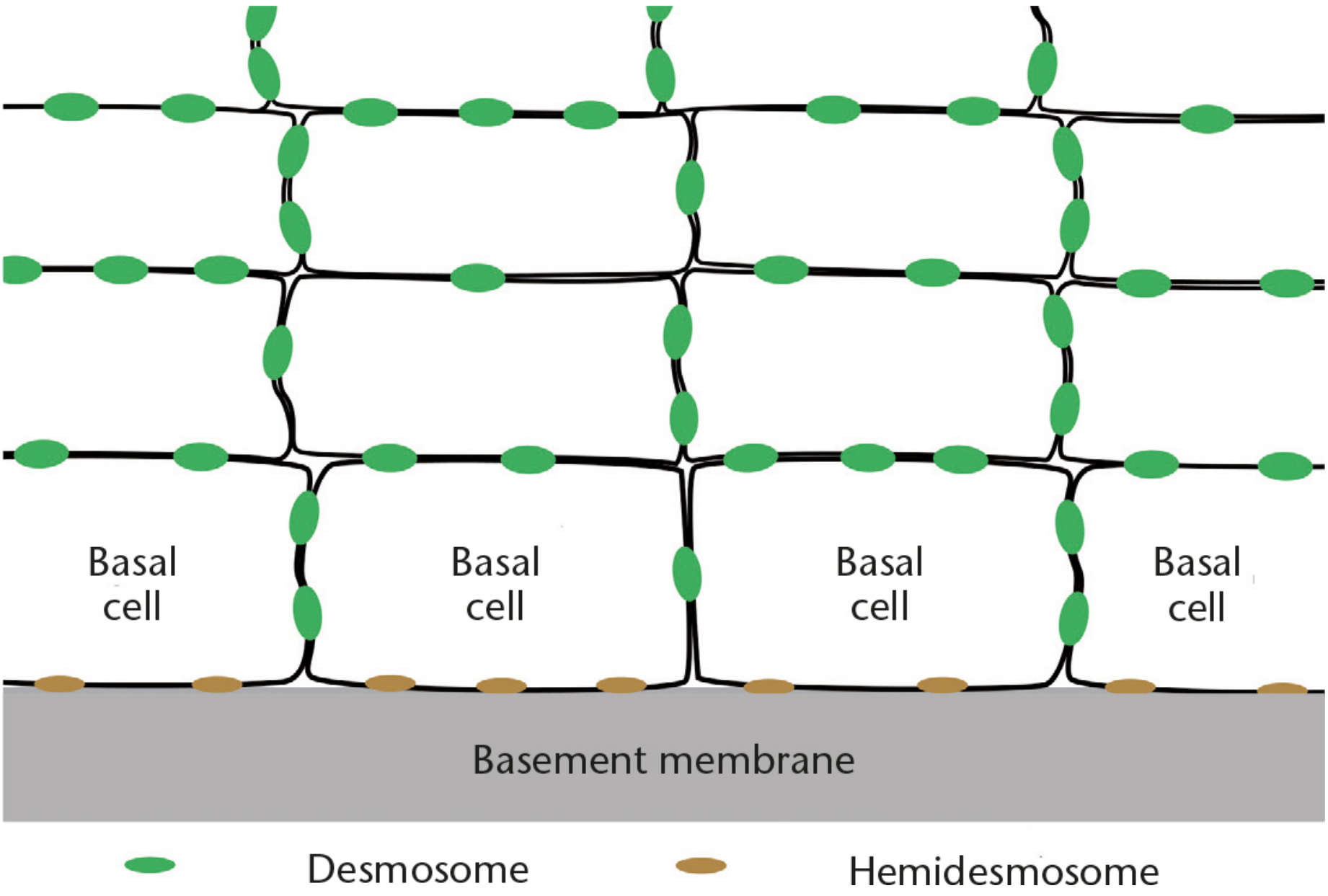


Fig. 10 The positions of desmosomes and hemidesmosomes in the deepest portion of the epidermal layer.

THE ARRANGEMENT OF THE EPIDERMIS

If skin cells were constantly produced without being lost, the skin would gradually get thicker and thicker. In reality some skin cells are naturally worn off the surface as a result of day-to-day activity. But

where the rate of wear is less than the rate of production (which is usually the case except in high-wear areas of the body), the outermost layer is continuously shed or exfoliated. The exact mechanism by which exfoliation occurs is not yet known, but it is thought that each skin cell activates some form of timer when it is produced, which results in the desmosomes that attach it to adjacent cells self-destructing a set number of days after production.

Of course if the rate of wear is greater than the rate of production then the skin is gradually worn thinner. The skin needs a mechanism to cope with this, or we'd end up with blood everywhere. In fact the skin has two such mechanisms. The first is that repeated pressure on the skin stimulates an increased rate of production of keratinocytes at the basal layer. As a by-product of increased keratinocyte production, the affected area of skin also becomes thicker, so providing more protection to the underlying dermis. This is what is happening when a callus forms. The other mechanism is that when wear becomes temporarily excessive, the epidermal layer becomes thinner and this also stimulates a more rapid production of keratinocytes at the basal layer. Once the skin has achieved a more normal thickness, this increased keratinocyte production tails off again.

Some areas of the skin are subject to dramatically greater wear than normal and need mechanisms that allow for both much thicker skin (to provide protection from damage) and a significantly increased rate of skin production (to compensate for the increased wear). The soles of the feet and palms of the hands in humans are good examples of this. The reason we have finger prints is that the basement membrane (and hence the basal cell layer) is folded into deep ridges and furrows in this part of our skin. This means that, for a given area of skin, there is a much higher number of basal cells than normal. If these basal cells produce keratinocytes at the normal rate, then the increased density means that keratinocytes are produced at a higher rate for a given area of skin. This allows for both an increased rate of production of skin to compensate for increased wear, and the required resources to produce a thicker skin than normal.

The horse's foot is an example of the above mechanisms taken to extremes. The area of foot in contact with the ground is tiny when you consider the weight of the horse. The total surface area of the bottom of all four feet of a typical thoroughbred (assuming that the whole of the bottom of the foot is in contact with the ground as would be the case on a soft surface) is only about one and a half times that of the two feet of a typical adult male human. And yet the horse weighs six to seven times more. The end result is that the pressure on the feet at rest is around four times higher in a horse than in a human. The horse's foot has to be tough enough to withstand that pressure. The horse has also evolved to be able to travel at speed on fairly challenging terrain (arid plains strewn with stones and rocks, for example) without injuring its feet, so not only does it need to be able to support a lot of weight, but it needs to be able to do so while running on uneven or even sharp surfaces.

To achieve this, the solar, frog, coronary and perioplic coria in a horse all share the same adaptation. The basal cell layer is formed into myriads of tiny spikes called papillae. These papillae are typically around 2–3mm long, and are spaced every quarter millimetre or so (the exact spacing varies depending on where you look in the hoof, and also on how the hoof is adapting to the horse's environment). This formation creates a massive surface area of basal cells, roughly twenty times as much for a given area compared to a flat arrangement of basal cells, and hence the ability to produce horn quickly and in a thick layer. In addition, this arrangement of basal cells allows for the production of a non-uniform epidermis. Each papilla in the corium produces keratinocytes in a helical fashion so as to form tubes (more commonly referred to as tubules as they are very small). The basal cells between the papillae produce a region of more homogeneous keratinocytes (referred to as inter-tubular horn), which serves to glue the tubules together. The end result is a horny structure that has hard, fibrous tubules glued together in a softer substrate. This is not unlike glass-reinforced plastic (or fibreglass) and has immense strength. The outermost layer of tubules in the hoof wall can clearly be seen as vertical lines running down the outside of the hoof wall. The tubules of the sole can be seen under a magnifying glass as little dots all over the sole. The tubules of the frog are more difficult to see because they are not as hard and dense as those of the sole and wall, but they can be seen under magnification on a well prepared sample.

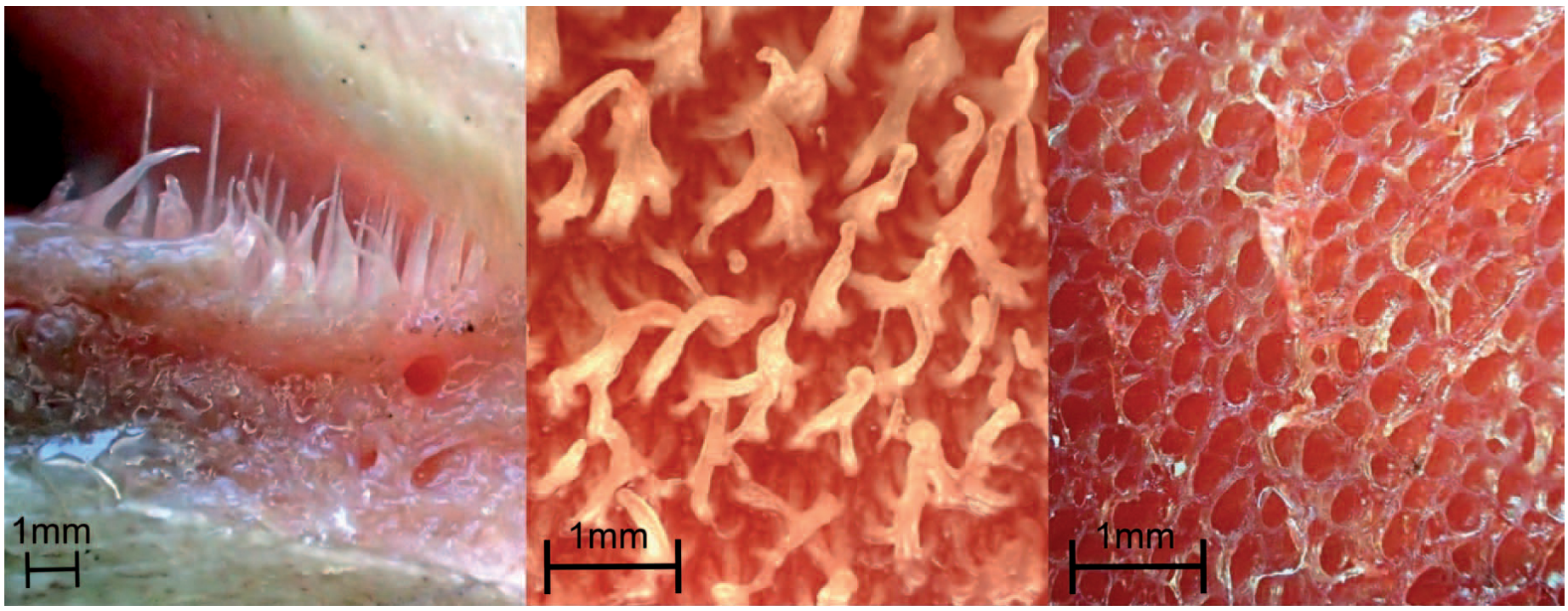


Fig. 11 The papillae of the solar corium. LEFT: ***Solar papillae being exposed as the solar horn is pulled off a section taken from a cadaver foot (the solar horn is at the top, the pedal bone at the bottom).*** CENTRE: ***Solar papillae with the horn removed. With the horn removed, the papillae tend to stick together in clumps, but the individual papillae can be seen at the base of each clump.*** RIGHT: ***A close-up of the solar horn after removal from the corium showing the holes that are normally filled by papillae.***

The laminar corium sits between the inner surface of the hoof wall and the underlying structures of the foot, and is somewhat different from the other coria. This difference is driven by its very different function. Whereas the solar, frog and coronary coria are there to produce horn in large quantities, the laminar corium produces virtually no horn in a healthy foot. The laminar corium's main purpose is to allow the hoof wall to slide down past the pedal bone as it is replaced by more horn at the coronary corium. It does this by letting go and reattaching the hoof wall in tiny ripples, in rather the same way that a caterpillar moves.

Given that the horn is being constantly detached and reattached, it is important to have in this corium a large area of basal cells for a given area of hoof wall so that there are always enough attached cells to make the attachment strong overall. Papillae would not work in this situation, as spikes pushing horizontally into the hoof wall would prevent it from moving downwards. Instead the corium is folded into vertical fins (primary dermal laminae), which the hoof wall can slide down. To increase the surface area even further, each fin is itself covered with even smaller fins (secondary dermal laminae). The primary and secondary laminae of the dermal layer interlock with mirror-image structures in the epidermal layer (on the inside of the hoof wall) to form an extremely strong bond.

In a healthy horse, the primary dermal laminae are spaced around the hoof roughly every third to half a millimetre, and are typically around 2–3mm long. Each primary dermal lamina has around 60 to 120 secondary dermal laminae, and each of these is 0.1– 0.15mm long. This means the laminar corium has around fifty times as many basal cells for a given area as a flat corium would have.

Fig. 12 A close-up of the underside of a dried hoof sample near the toe showing wall tubules, white line and sole tubules.

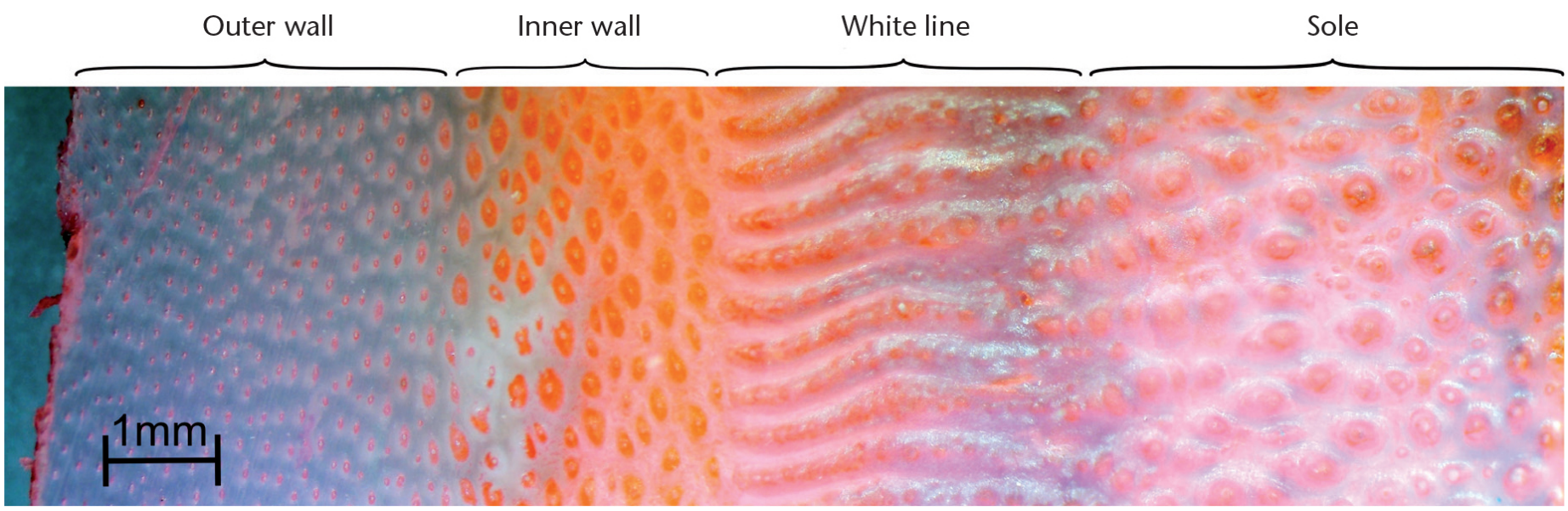


Fig. 13 A microscopic view of a horizontal section through the hoof wall, the white line and sole just above ground level, stained with methylene blue and eosin to make the various structures more visible.

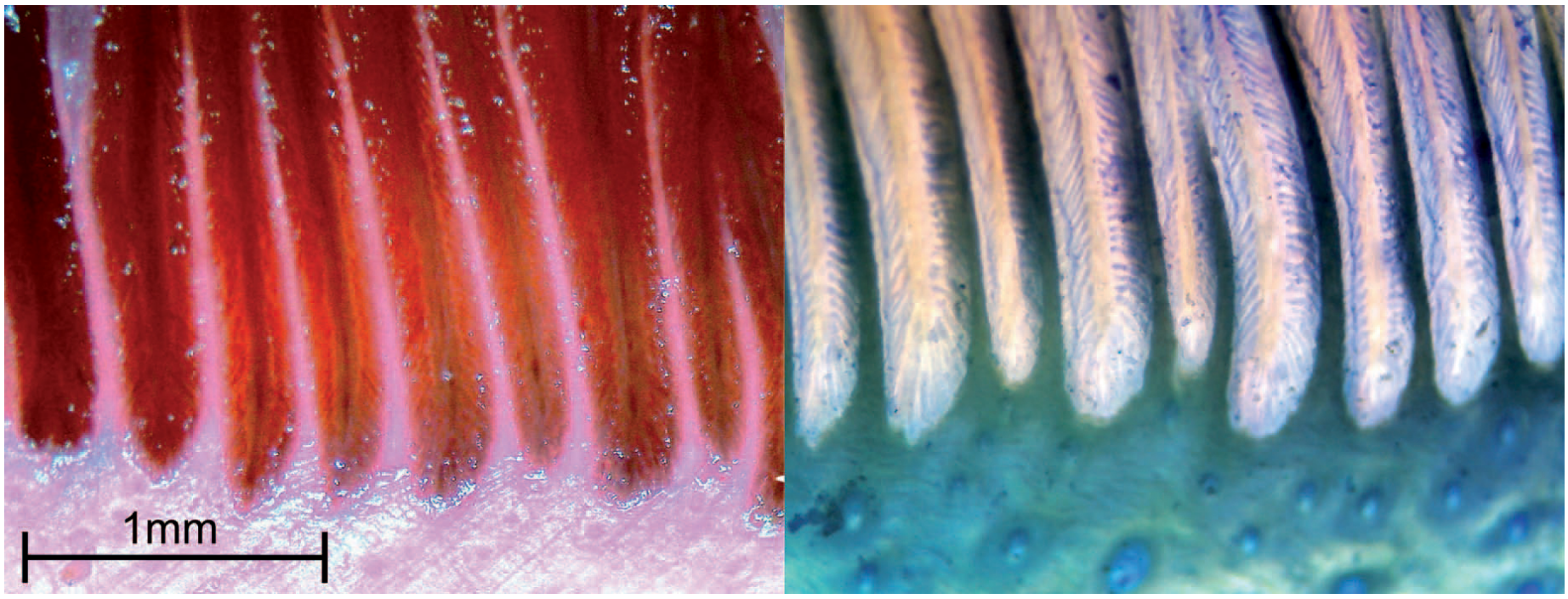


Fig. 14 LEFT: *A microscopic view of a horizontal section through the laminae. The inner hoof wall is at the bottom – the front surface of the pedal bone is just off the top of the picture. Nine primary dermal laminae can be seen (dark red), interspersed by eight primary epidermal laminae (light pink). Each dermal lamina is surrounded by approximately sixty secondary laminae, which can just be seen.* RIGHT: *A similar section at the same scale stained with eosin and methylene blue, making the secondary laminae and wall tubules more visible.*

As well as providing for a strong attachment as the hoof wall slides down the pedal bone, the laminae provide a huge amount of resources that can be brought to bear in the healing of injuries. If the sole gets damaged, say by an abscess, the solar corium can start producing new solar material fairly quickly (within days), and typically the original solar horn is still in place providing mechanical protection. In contrast, serious injury to the hoof wall can result in the wall being broken or cut away, revealing the laminae. For the laminae then to recover with horn produced in the normal fashion at the coronary band might take as much as twelve months – but the horse cannot afford to wait that long. Instead, the laminae are able to switch on horn production, producing large amounts in a very short space of time. Within a few days, exposed laminae are able to cover themselves with a millimetre or two of horn, and within a few weeks, a hoof wall of almost normal thickness can be produced in this way. This temporary but functional repair then gets pushed down the hoof by more normal wall over time. For obvious reasons, the horn produced in this situation is sometimes termed ‘repair horn’.

Fig. 15 An example of ‘repair horn’ produced by the laminae in response to injury, creating a temporary wall. (Photo: Yvonne Thomas)

ANATOMY OF THE WHITE LINE

The white line is a critical structure because it provides a joint between the sole and the hoof wall that allows for the wall to grow down faster than the sole. As such, it needs to be a flexible joint but a strong one. One half of the white line consists of the epidermal laminae, which remain attached to the inside surface of the hoof wall as it passes off the bottom of the dermal laminae. In order to interlock with that half, the papillae at the edge of the sole (called terminal papillae) need to produce horn that is an exact mirror image (and hence a copy of the shape of the dermal laminae). Detail on exactly how this occurs is sparse in text books and scientific papers – it appears to be an important area of the functional anatomy of the hoof that has been largely ignored.

STIMULUS FOR GROWTH AND PAIN

An important but often neglected area of functional anatomy when considering the feet is the role of pain. Pain exists to tell us that we are at risk of damaging ourselves. If it hurts, the chances are that damage is occurring. Conversely, if it doesn’t hurt, the likelihood is that no damage is being done. Unfortunately we can’t ask a horse whether it is feeling pain, but we can observe its behaviour and deduce a lot about what pain it is experiencing. For example, if a horse is carefully avoiding putting weight into one particular foot, it is quite likely that the foot in question, or some part of the leg above it, is sore. Similarly, if a horse is loading the outside heel of the foot significantly more than the inside one, this might suggest that there is pain associated with the inside heel.

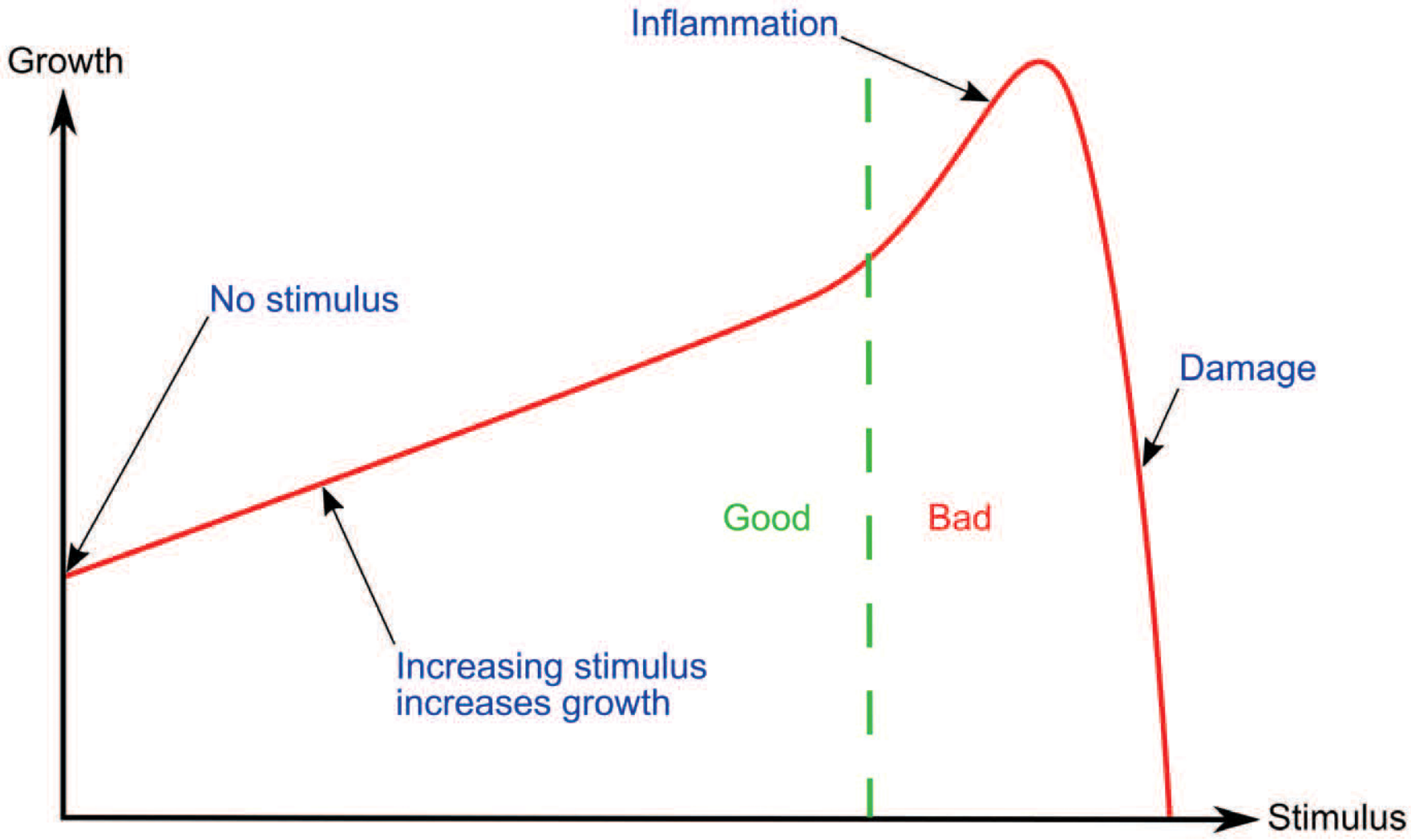


Fig. 16 The relationship between stimulus (pressure and/ or impact) and hoof growth rate. With no stimulus, there is a minimum growth rate. Increasing stimulus increases growth rate. At excessive levels of stimulus, the corium starts to become inflamed and will produce horn at an accelerated rate. Further stimulus damages the corium to the point where horn production tails off rapidly. Once the corium is badly damaged, the stimulus needs to be reduced dramatically before any significant horn production will return.

The description above of how skin is produced touched on the role of pressure in stimulating growth. This is a very important concept when it comes to understanding the equine hoof. Each individual area of corium is separately responsive to pressure. This means that the hoof can adapt by creating increased growth in any part of the hoof that is getting used more.

The opposite side of this is that too much pressure causes damage (and hence pain). There is therefore a range within which increased pressure creates a calloused hoof that is capable of doing more, but also a range beyond which damage is done. Once the corium is damaged, the production of horn tails off fairly dramatically. As such, working ‘through the pain barrier’ when it comes to horse’s feet is largely counterproductive.

Inflamed corium is also more sensitive to vibration than pressure. Anyone who has ever hammered a thumb will know that squeezing it reduces the throbbing sensation (because it reduces the circulation to the area), but that catching the affected area on a hard surface is excruciating. The same is true in the equine foot. For that reason, pain from underlying inflammation is usually more obvious when the horse is working on a hard surface such as tarmac or concrete, as these surfaces create more vibration.

Once the corium becomes damaged, it becomes more easily damaged until such time as it manages to heal again. This means that not only is inflamed tissue more sensitive to pain, but also that any failure to remove the excessive stimulus results in a fairly rapid and ultimately catastrophic escalation in the rate of damage.

THE APPEARANCE OF THE FOOT ON AN X-RAY

An x-ray (more accurately termed a radiograph) is a picture of the internal structures of the body taken by passing x-ray radiation through it. A photographic plate is used to capture the image (or more recently a digital sensor), which turns black when exposed to x-rays. As a result, areas of the body that

x-rays pass through easily (such as soft tissues) appear black, and areas that don't easily pass x-rays (such as bone) appear white. The most common shot used in laminitis is the latero-medial (from the side) shot. This helpfully shows the position of the bones within the foot. Unfortunately, the hoof capsule (and in particular the very outside surface of the hoof capsule) doesn't usually show up well on an x-ray, so it's important for markers (anything that won't pass x-rays and hence will show up) to be placed in key positions on the foot to mark the extent of the hoof capsule.

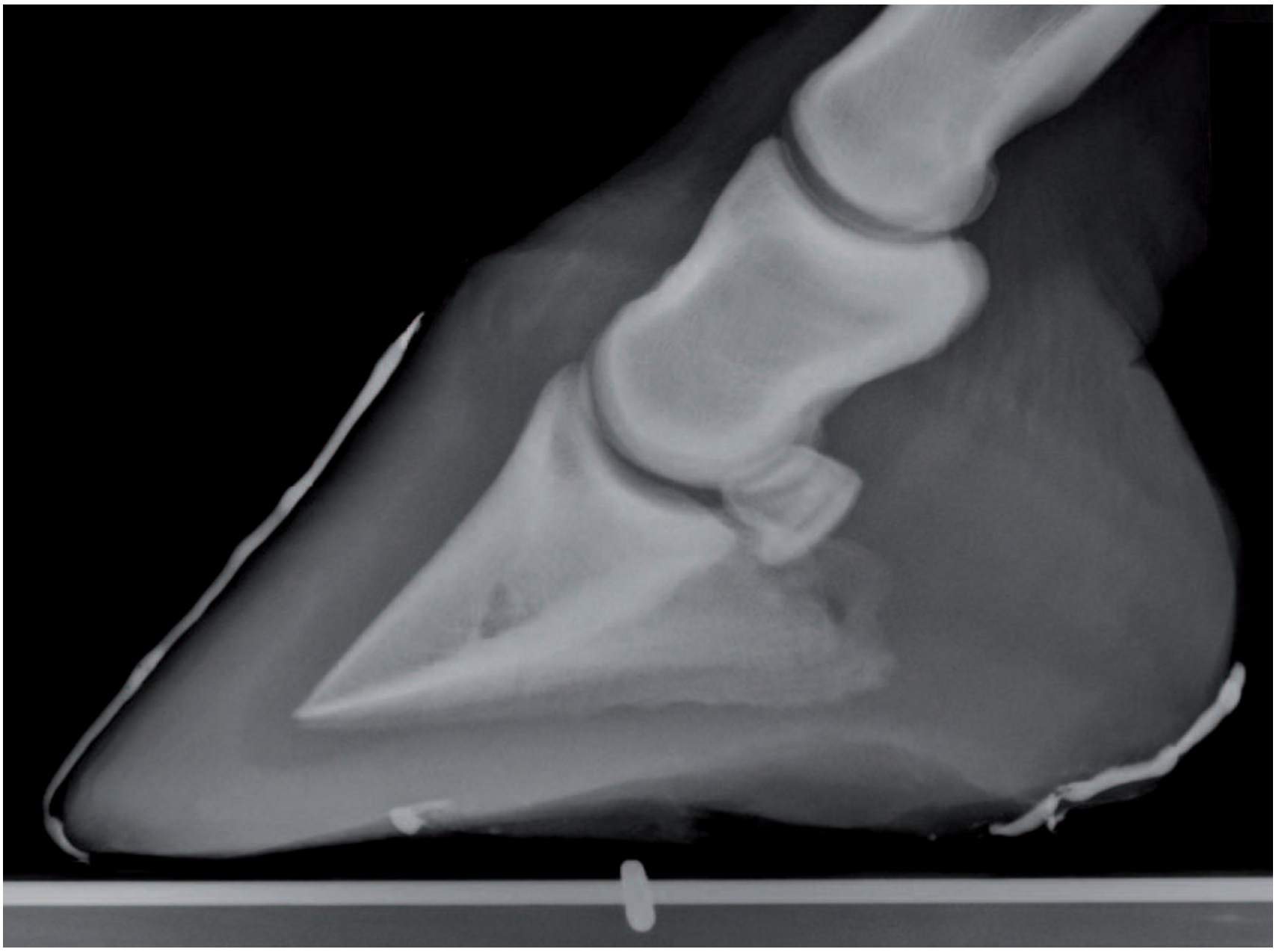


Fig. 17 A latero-medial radiograph of a healthy equine foot with markers placed on the front surface of the hoof wall and the back of the heel. The ground surface is also marked. (Radiograph: Debra Taylor, Auburn University)

THE DIGESTIVE SYSTEM

As described in later chapters, the digestive tract is an important part of the jigsaw that is laminitis. As such, it is useful to have a brief understanding of the anatomy of the digestive system.

Food is initially chewed by the teeth and mixed with saliva, which is somewhat alkaline. The horse does not salivate in anticipation of food, but relies on chewing to stimulate saliva production. Food then moves down the oesophagus to the stomach, where it is mixed with stomach acid. The acidity of the stomach is reduced to an extent by the alkalinity of the saliva, which is why it is important for horses to eat fibre-based foods that take a lot of chewing so that sufficient saliva is produced.

Stomach acid is important for two main reasons. Firstly, it is strong enough to kill almost all microorganisms in the food, hence sterilizing the food and preventing unwanted infections making it through to the rest of the digestive system. Secondly, the acid helps in the breakdown of foods, especially proteins. In addition to the action of the acid, enzymes are secreted in the stomach to aid digestion, such as pepsin (which aids the breakdown of proteins) and lipase (which starts the process of breaking down fats).

From the stomach, food is passed into the small intestine. This is around 25m long, but is fairly narrow and is coiled up within the abdomen. It is divided into three sections. In the first, the duodenum, further enzymes are secreted that help with the breakdown of fats, starches and sugars. Brunner's glands in the duodenum also secrete bicarbonate, which helps to neutralize the stomach acid (which has already done its job) so that the rest of the gut isn't subjected to strong acid. Iron is also largely absorbed in the duodenum. In the middle section, the jejunum, the various enzymes gradually do their work and break up carbohydrates into ever smaller molecules.

Carbohydrates are molecules made of carbon, hydrogen and oxygen atoms. The smallest carbohydrates (which cannot be broken down in the gut to form smaller ones) are called monosaccharides (also called simple sugars), and examples include glucose and fructose. Two monosaccharides can be combined to form a disaccharide. For example sucrose (table sugar) consists of a fructose molecule attached to a glucose molecule. You can make ever bigger carbohydrate molecules by joining multiple monosaccharides together to form polymers called polysaccharides. For example, starch consists of multiple glucose molecules joined together.

Only monosaccharides can be directly absorbed through the gut wall and used by the body, so various enzymes are used to cut up the polysaccharides into ever smaller units until as much as possible of the carbohydrate has been broken down into monosaccharides.

As well as breaking down and absorbing carbohydrates, most minerals and vitamins are absorbed in the jejunum.

The last part of the small intestine is the ileum. More minerals are absorbed here, as well as any remaining vitamins and simple sugars. Further enzymes are secreted in the ileum to break down any remaining proteins and carbohydrates. Fats and oils are also absorbed in the ileum. In order to be absorbed, these fats and oils need to be broken down by bile. Humans are able to store bile in the gall bladder and release it in bulk when a fatty meal is eaten. Horses, however, don't have a gall bladder and so can only secrete bile at a fairly steady rate. This means that the horse's gut doesn't cope well with sudden influxes of fats and oils. For this reason the quantity of oils and fats in any bucket feed should be kept small.

From the small intestine, food makes its way into the large intestine or hindgut. This is made up of the caecum, a large blind-ended fermentation chamber, and the colon. The large intestine is only around 5m long in total but is much wider than the small intestine, and, as a result, has around double the volume.

The horse has evolved to eat a diet containing large amounts of plant fibre (complex polysaccharides) that is not directly digestible. Indeed the majority of the carbohydrates in the horse's natural diet cannot be broken down by enzymes, and without another way to utilize those carbohydrates, the horse wouldn't have survived. The hindgut is full of microbes (more than 500 species, mostly bacteria with some fungi and protozoa) which are able to ferment dietary fibre to form useful nutrients such as volatile fatty acids (which the horse can use for energy), and vitamins B and K. There are around ten times as many microbe cells in the hindgut as there are cells in the body of the horse. As a useful by-product, the fermentation process also generates heat, which helps to keep the horse warm. The horse needs a constant supply of fibre to keep these microbes healthy, which is why a fibre-based diet is so important for a horse.

As well as fermenting fibre, the hindgut is responsible for the absorption of water as well as the remaining minerals: sodium, potassium, chloride and phosphate. From the large intestine, food makes its way into the rectum before being excreted.

The entire length of the intestines is covered in microscopic spikes called villi. These increase the surface area of the gut wall and so increase the rate at which nutrients can be absorbed from the gut into the bloodstream. Although it is not immediately obvious, the contents of the gut are technically outside the body. The body can be thought of as a hollow tube with the mouth at one end and the anus at the other. The outside and inside surfaces of the tube are covered by skin. On the outer surface, the skin is thick to protect the animal from injury. On the inside surface, the skin is a single cell thick and covered in villi to aid the absorption of nutrients. As food passes from the mouth to the anus along this

tube, it is gradually broken down and useful nutrients are absorbed into the body. Anything that can't be used is excreted from the anus.

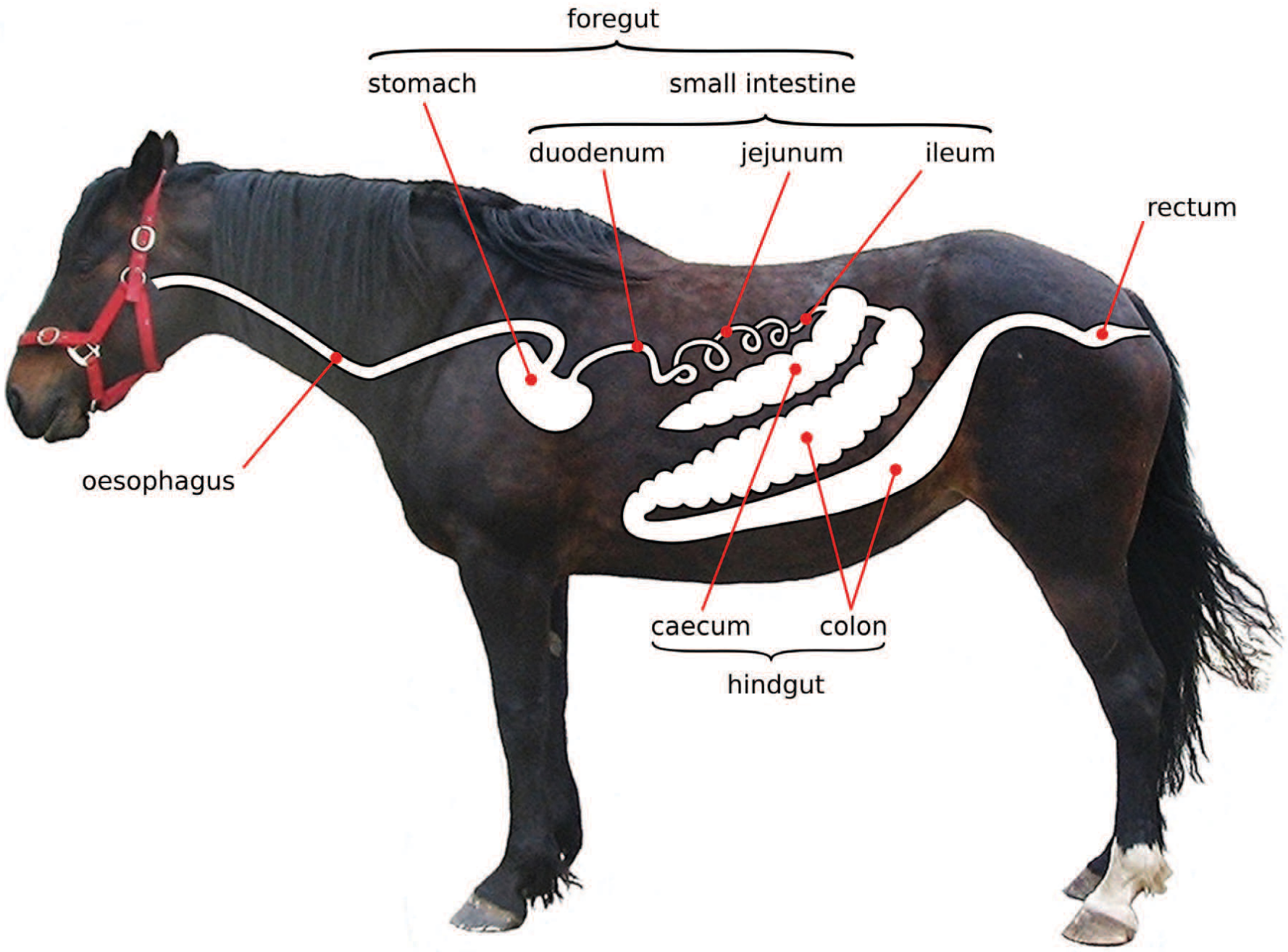


Fig. 18 A stylized representation of the digestive tract of the horse.

2 The Established View of Laminitis

This chapter describes the established view of what happens in the equine foot as a result of laminitis, and how to recognize it. The causes of laminitis will be covered in later chapters.

RECOGNITION OF LAMINITIS

Laminitis is defined as inflammation of the laminar corium. It is typically associated with a loss of circulation to the foot. The inflammation (and hence any loss of circulation) tends to be strongest in the toe area. This can be clearly demonstrated by taking a venogram (an x-ray of the foot taken after a dye that blocks the x-rays is injected into a vein, hence making the veins of the foot visible).

Raised Pulses

The heart creates a pressure wave with each beat, which propels blood through the arterial system. As this pressure wave passes through arteries, the artery walls bulge very slightly, and this is what we feel when taking a pulse. If the arterial pressure wave hits a blockage, such as a foot where the blood vessels have closed down due to laminitis, the wave is reflected back, causing pressure to build up locally and the artery to expand more than usual. This can be felt as a stronger than normal pulse. This is an extremely useful diagnostic technique that is used by professionals dealing with laminitis cases, but which can also be used by owners to pick up the early warning signs of a laminitis attack. It is important to note that the pulse rate is not important here (although a faster pulse rate can indicate distress, often combined with rapid breathing): rather it is the strength of the pulse that indicates the severity of the problem.

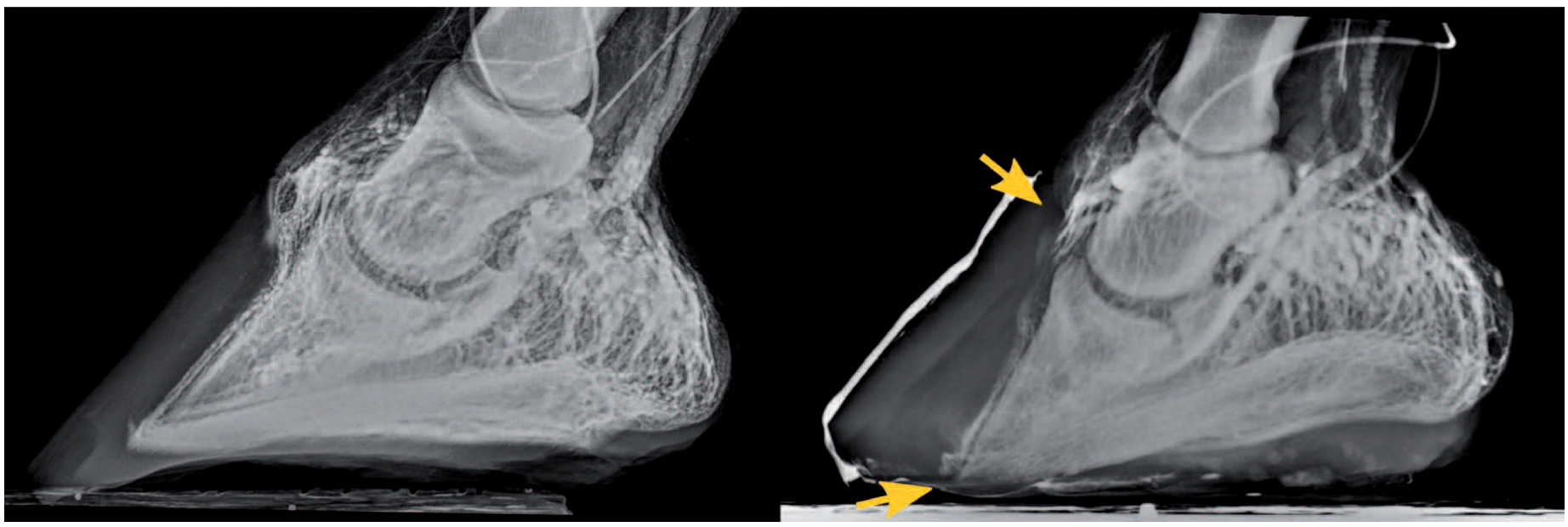


Fig. 19 Two venograms. LEFT: ***A healthy foot with the contrast dye filling all the veins.*** RIGHT: ***A laminitic foot with loss of circulation in the toe area. Note the lack of contrast dye (and hence the lack of circulation) in the veins at the coronary band and under the tip of the pedal bone, as well as the poor circulation to the laminar corium. (Radiographs: Debra Taylor, Auburn University)***

In most horses, the easiest place to take the pulse is on either side of the leg just above the fetlock, between the suspensory ligament and the flexor tendons. There is an obvious groove between these two structures when the leg is weight bearing, and the best place to check pulses is just at the bottom of this groove. The main arteries of the leg are close to the surface in this location and there are

convenient hard structures beneath the arteries against which to press them to detect the pulse. Taking the pulse involves applying light pressure to the area and waiting for a few seconds. A light, regular pulse should be felt. There is a corresponding location just below the fetlock (a depression between the flexor tendons and the extensor branch of the suspensory ligament), but this is more difficult to use in horses with feathers. A raised pulse in a leg indicates a problem below where the pulse is taken, so taking the pulse above the fetlock does not rule out a problem with the fetlock itself. Hence the location below the fetlock can be useful when narrowing down the causes of a raised pulse.

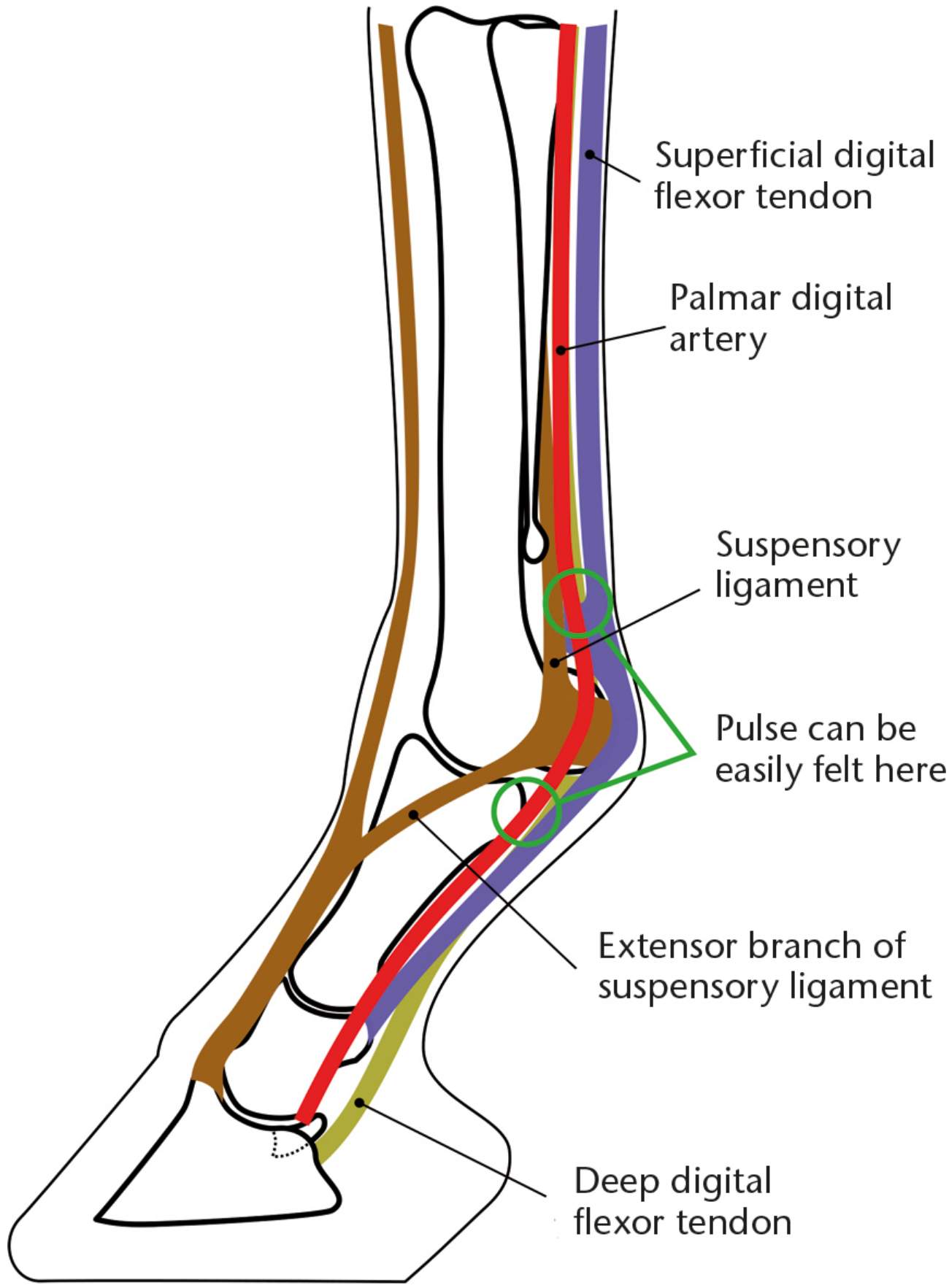


Fig. 20 Locations for taking a digital pulse.

The pulse should be taken at rest (preferably at least thirty minutes after any exercise, as exercise can create a raised pulse in a healthy horse) with the leg bearing weight. If necessary, the opposite leg should be lifted to ensure that the leg being checked remains weight bearing. It is helpful to know what is normal for an individual horse, and hence it is good practice for owners to check their horses' resting pulse regularly. A normal pulse should be almost undetectable (some people can't find a pulse in a healthy horse), although some people are better at detecting pulses than others. The resting heart rate of a horse (twenty-five to forty beats per minute) is somewhat slower than a human's (sixty to seventy

beats per minute) – which helps to identify if the person taking the pulse is accidentally feeling their own!

A raised pulse does not automatically signal the presence of laminitis. Other conditions such as abscesses, bad sole bruising and bone fractures, can cause a raised pulse. However, these other conditions usually only affect a single foot, whereas laminitis typically affects at least both front feet. As such, a raised pulse in both front feet (or both hinds for that matter) should raise suspicions of laminitis. Of course, bad sole bruising or abscessing can happen on both front feet simultaneously, but in such cases there is almost always some degree of laminitis involved, increasing the risk of the bruising or abscessing (this will be covered in the chapter on low-grade laminitis). It is also possible for a horse to have a raised digital pulse because it is overheating, perhaps due to the use of an overly thick rug, or to standing in the sun on a hot day, so this needs to be ruled out as a cause if a raised pulse is detected.

The typical description in laminitis is of a ‘bounding’ pulse, although less obvious pulses may be significant if they are above the normal level for that horse. In severe cases, especially in thin-skinned breeds such as the thoroughbred, the arteries can be seen pulsing with just the naked eye.

The Presence of Heat

Laminitic feet are typically hotter than normal. This can be difficult to feel because the hoof is a fairly good insulator, but with practice, the temperature of the foot can be gauged, and a raised temperature detected. It’s important to note that measuring the surface temperature of the hoof with an infra-red thermometer isn’t in itself useful in detecting laminitis, because the external temperature of the hoof is so highly affected by factors such as the ambient air temperature, and the temperature of the ground. The temperature also varies significantly over the different parts of the hoof. However, significant differences in temperature between the front and hind feet, or between the mid-wall in the toe and the heel, can be useful indicators of a problem. However, as with raised pulses, it’s important to note that the external temperature of the feet is often raised for some time after exercise, as the horse uses its legs and feet as radiators to ‘dump’ excess heat.

An Abnormal Stance

A horse with severe laminitis tends to stand with a very specific posture. Because the front feet are typically worst affected, and the toes more affected than the heels, the horse modifies its posture so as to take the weight off the front toes. In the classic laminitis stance, the front legs are stretched out in front of the horse with the toes either lightly touching the ground or lifted slightly off the ground. By the time the horse has adopted this stance, it is also usually looking quite distressed. The horse will often shake and shuffle from foot to foot, and may have a pained expression to the face.

Fig. 21 The typical stance of a severely laminitic horse.

Whilst the classic laminitis stance makes recognizing laminitis very easy, it only shows up in more serious cases. The lack of a laminitis stance does not preclude the possibility of laminitis, and some milder laminitics can have counter-intuitive posture problems, as described in later chapters.

An Abnormal Gait

A horse with laminitis will modify the way it moves to avoid pain. Vibration typically causes more pain to the horse than pressure. As a result, gait abnormalities are most pronounced when the horse is moving on hard ground, such as tarmac or concrete. Because the inflammation is concentrated in the toe area, the horse will try to move in a way that minimizes vibration to the toe. A healthy horse working on hard ground lands just heel first so as to make use of the shock-absorbing structures in the back part of the foot. A laminitic horse exaggerates this heel-first landing so as to absorb fully any vibration before the toe hits the ground. The result is an exaggerated heel-first landing that is sometimes described as a ‘toe flick’. It is useful to watch healthy horses in walk and trot to develop an eye for what a normal heel-first landing looks like. This makes it easier to pick up any subtle change towards an excessive heel-first landing.

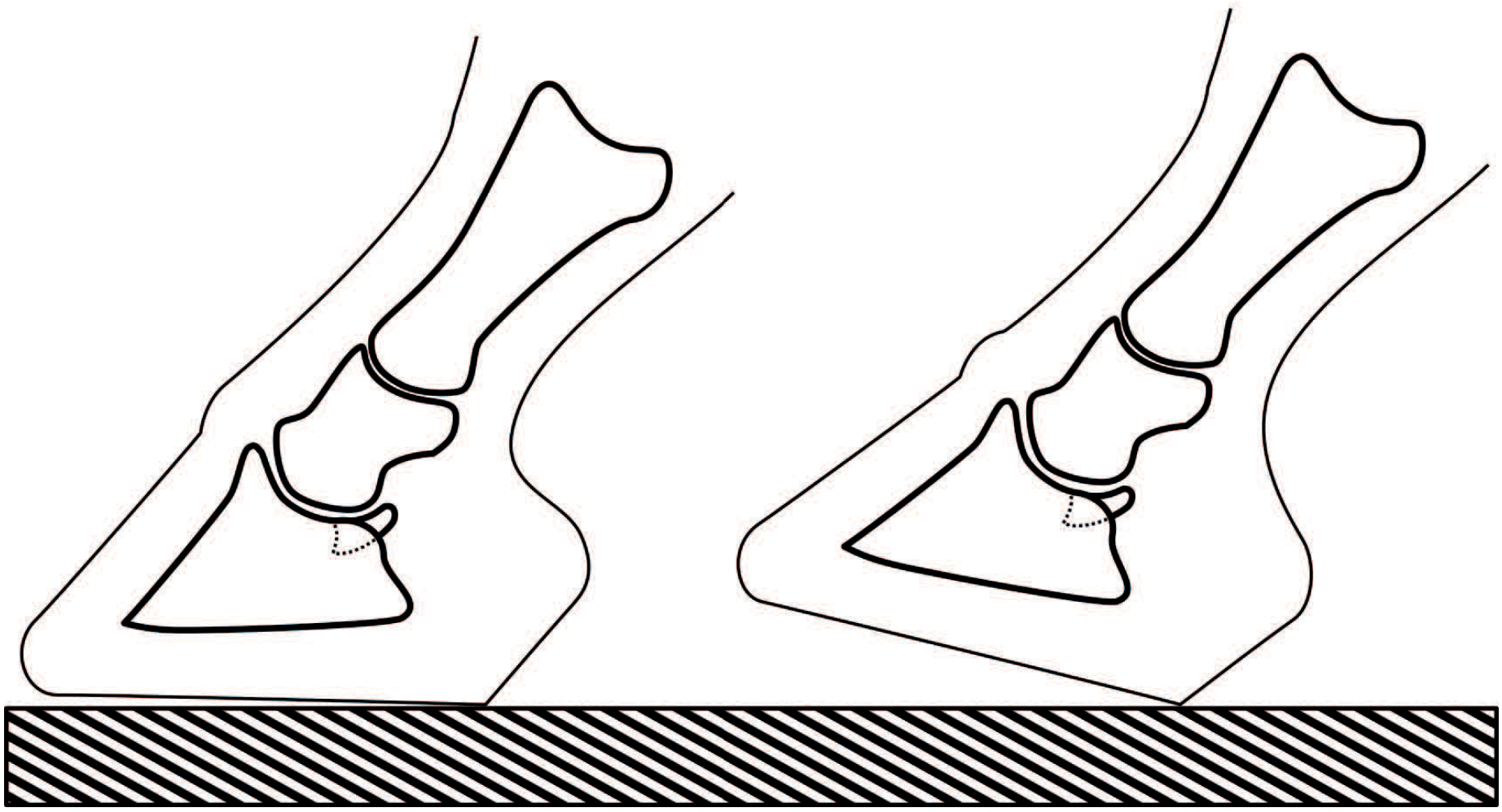


Fig. 22 The foot at the point of impact. LEFT: ***A healthy horse with a normal heel-first landing.*** RIGHT: ***A laminitic horse with an excessive heel first landing.***

Any horse with significant laminitis will tend not to want to trot, and if forced to, will do so in a way that minimizes impact on the feet. The horse typically appears to flatten the trot or even shuffle. In more severe cases, the horse may be reluctant to move at all, and may appear stiff throughout the body as it attempts to hold itself in an unnatural posture to reduce pain.

Other Diagnostic Techniques

Professionals involved in a suspected laminitis case may also use techniques such as hoof testers and nerve blocks to try to determine if the lameness is focused on the feet. Hoof testers apply a known amount of pressure to different parts of the foot, which allows detection of any tender areas. This is not a technique that should be attempted by anyone without appropriate training as it is difficult to be accurate and it is easy to inflict unnecessary pain on the horse if used incorrectly.

Nerve blocks involve injecting anaesthetic into the nerve just above the foot so as to numb the entire foot. If the horse then becomes sound, this indicates that the pain is somewhere in the foot, rather than higher up the leg.

X-rays are frequently used to image the internal structures of the foot, especially the bones. Whilst the main use of x-rays in laminitis is to assess the position of the pedal bone in the foot, other useful information can be read from a radiograph, such as the degree to which circulation to the tip of the pedal bone has been lost.

ROTATION

When the laminae become inflamed during a laminitis attack, they become weaker and less able to support the weight of the horse. If the inflammation is sufficient, they can tear apart, allowing the weight of the horse to push the pedal bone lower down in the hoof capsule. Laminitis most commonly affects the toe area more than the heel area, so in all but the most serious cases the majority of tearing tends to happen at the front of the foot. This allows the front of the pedal bone to drop, while the back portion remains more or less in place. The pedal bone is then described as having rotated. The degree of rotation is measured as the angle by which the front surface of the pedal bone has deviated from the

hoof wall. This cannot be seen externally in the early stages of laminitis, but shows up well on an x-ray so long as the front wall of the hoof has been suitably marked. The degree of rotation gives some indication of the severity of the damage caused by the laminitis, and hence the likelihood of recovery, and the time that any recovery is likely to take.

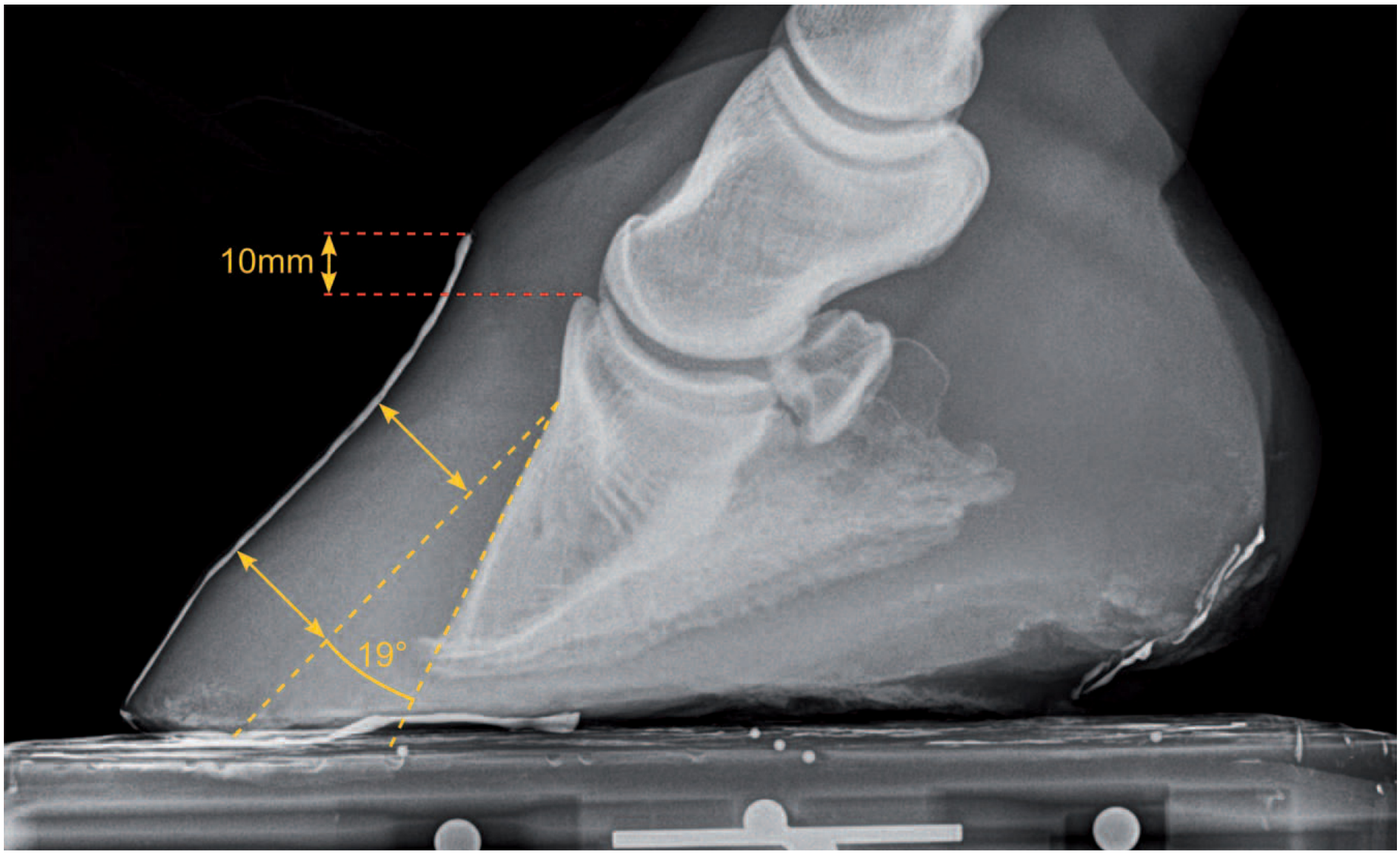


Fig. 23 A radiograph showing rotation as a result of laminar failure. The front surface of the pedal bone deviates by 19 degrees from its normal position (parallel to the front surface of the hoof wall). The founder distance is 10mm. (Radiograph: Debra Taylor, Auburn University)

SINKER

If the degree of inflammation is severe enough, the area of laminae towards the heels will also become detached, even though it will typically be less inflamed than the toe. The end result is that the pedal bone drops vertically without rotating. This scenario is described as ‘sinker’. The term ‘founder’ is also sometimes used as an umbrella term covering both rotation and sinker (particularly in the USA).

In reality, most rotation cases show some degree of sinker, and most sinker cases show some degree of rotation. It is unusual to see pure sinker or pure rotation.

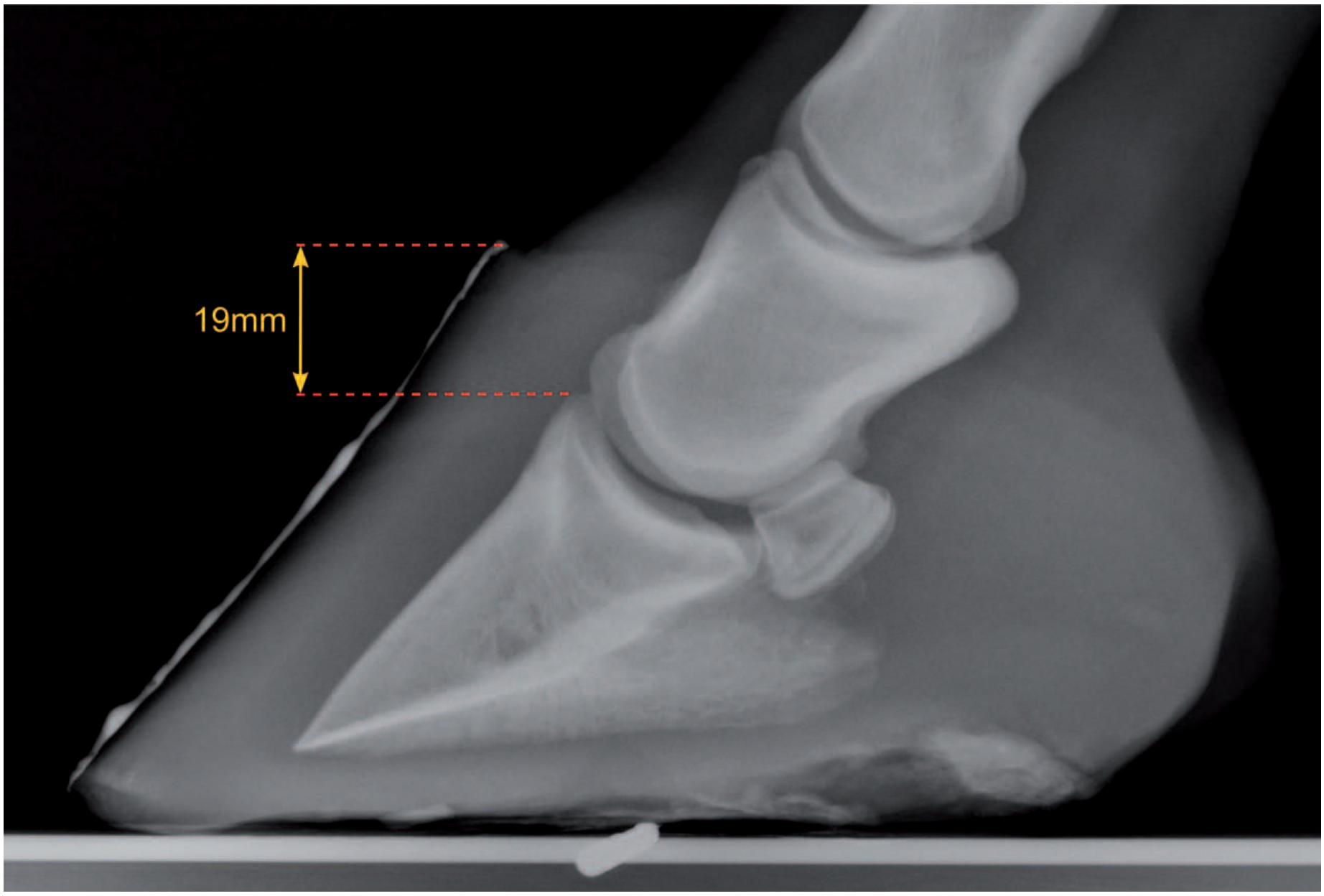


Fig. 24 A radiograph showing sinker as a result of laminar failure. In this example, the pedal bone has dropped without any rotation (which is unusual), and hence the front surface of the pedal bone is parallel to the front surface of the hoof wall. The founder distance is 19mm, making this a severe sinker case. Without the marker on the front surface of the hoof wall (the top of which indicates the position of the hairline), the severity of this case would likely be missed. (Radiograph: Debra Taylor, Auburn University)

Founder Distance

The extent to which the front portion of the pedal bone is lower than normal in the foot can be determined using a measurement called the ‘founder distance’ (also known as the ‘coronary extensor distance’). This is measured on an x-ray as the vertical distance from the hairline externally (which has to be marked with a marker to be visible on the x-ray) and the top of the pedal bone – specifically the top of the extensor process (the prominence to which the extensor tendon attaches). What is deemed to be a healthy founder distance varies somewhat depending on breed, but healthy values are typically less than 6mm. The founder distance will be increased in both rotation and sinker cases. In sinker cases, however, the rotation angle will be small despite a large founder distance. If the founder distance is not measured, the presence of sinker can easily be missed. For example, a severe sinker case is sometimes misclassified as a mild rotation case because the founder distance has not been measured and hence the significant vertical displacement of the pedal bone is missed.

Although the founder distance can’t be accurately measured without taking an x-ray, it is possible to get some idea of the degree of rotation/sinker externally. This is because the extensor process of the pedal bone can be felt through the skin just above the coronary band. Where the pedal bone has rotated or sunk, this bony prominence drops, leaving a depression above the coronary band that can easily be felt. The depth of the depression gives an indication of the severity of the laminitis. However, it is not possible to tell the difference between sinker and rotation by this technique. As with all the other diagnostic techniques, it helps to practise on healthy horses to get a feel for what is normal.

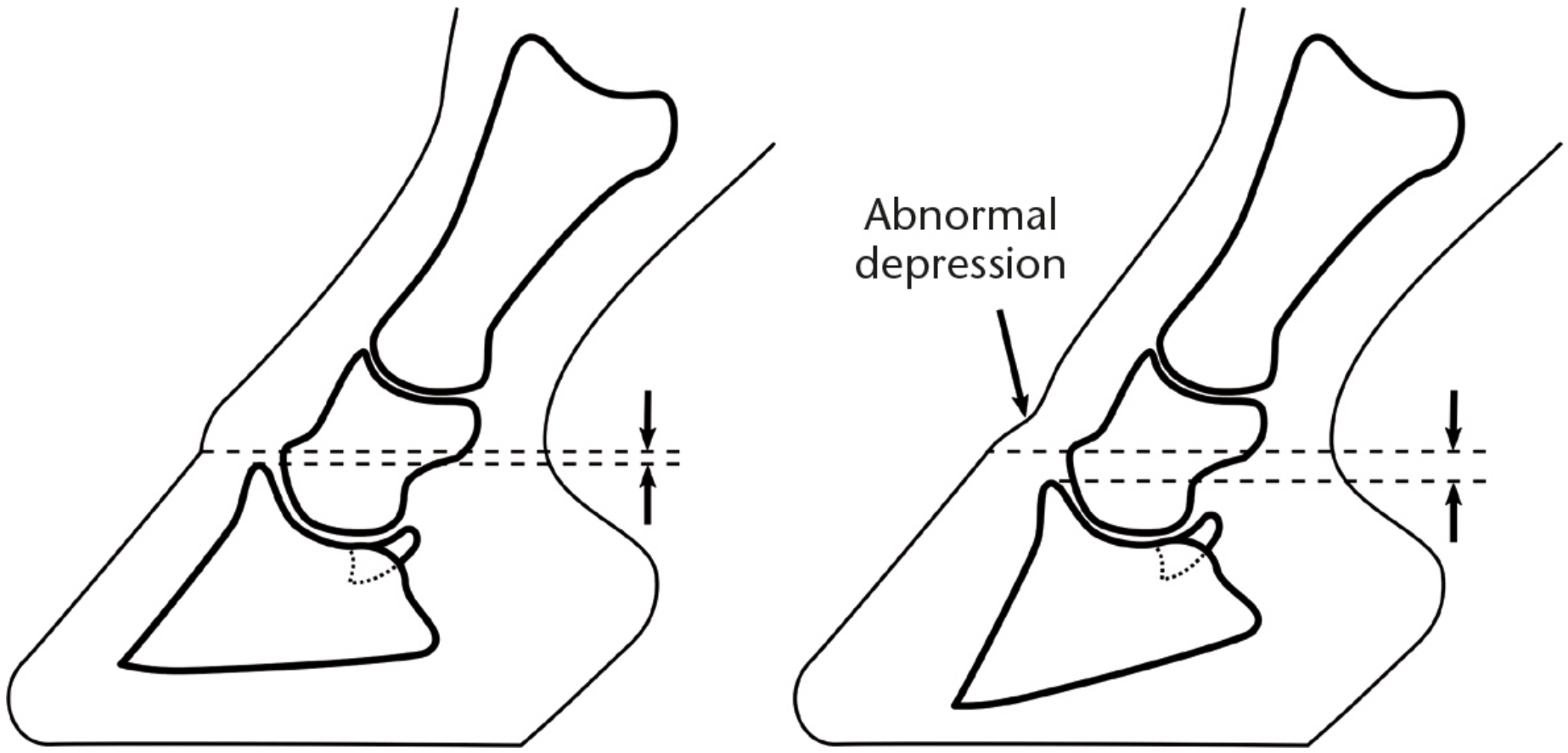


Fig. 25 LEFT: ***A normal foot showing the vertical distance between the hairline at the toe and the top of the pedal bone.*** RIGHT: ***A rotated foot showing the top of the pedal bone lower in the foot relative to the hairline, which can be felt as an abnormal depression just above the coronary band.***

ACUTE VERSUS CHRONIC LAMINITIS

Much of the above discussion relates to acute laminitis. Although the word ‘acute’ is often misused to mean ‘severe’, the definition of acute is ‘something that happens over a short period of time’. The opposite is ‘chronic’, meaning ‘something that happens over an extended period of time’. Acute laminitis refers to a single attack of short duration (a matter of days), from which the horse then recovers. The recovery may take many months, but the underlying laminitis is short lived. Chronic laminitis refers to underlying laminitis that continues for long periods. It is often episodic, with the horse having several smaller attacks per year, although some horses seem to have low levels of laminitis nearly all the time. The effect of many smaller laminitis attacks may do significant cumulative damage to the foot and cause just as many issues as a more severe acute attack. Many acute cases also go on to develop chronic laminitis – usually because the underlying causes of the initial attack have not been identified and corrected. The result of chronic laminitis is often described as a ‘slipper foot’, where the front of the hoof wall curves forwards away from the pedal bone.

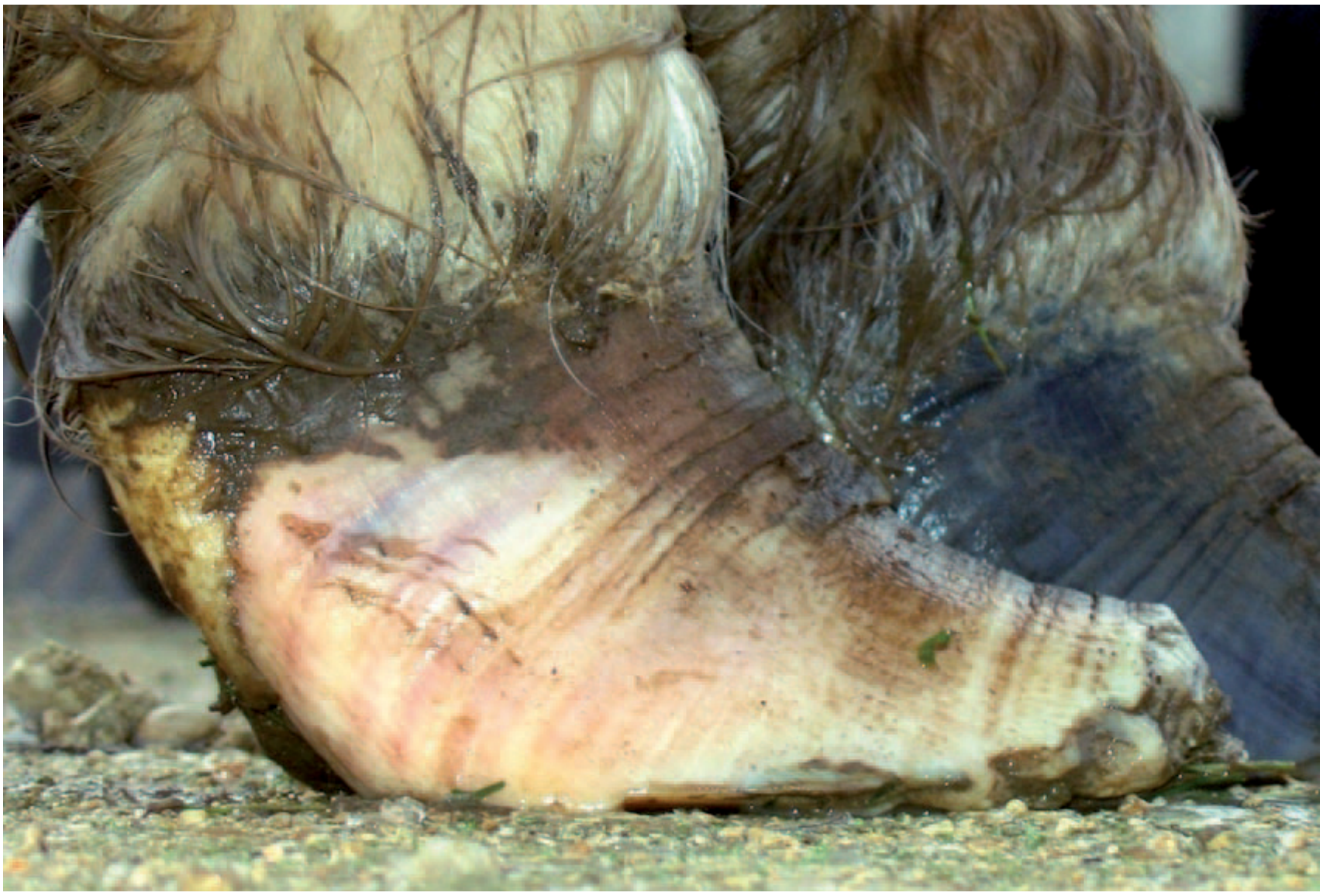


Fig. 26 A chronic laminitis case showing a slipper foot. (Photo: Jayne Hunt)

THE LAMINAR WEDGE

As the laminae are pulled apart during rotation or sinker, varying degrees of damage can occur. The precise nature of that damage depends on the rate and severity of the stretch. As we saw in Chapter 1, the dermal laminae take the form of fingers that reach from the soft tissue adjacent to the bone into the inside surface of the hoof wall. Like all tissues, the dermal laminae will remodel to a different shape in response to excess loading. If the dermal laminae are put under constant tension (as happens when the pedal bone rotates), then they will elongate. The epidermal laminae will also typically elongate as a result (the dermal laminae producing the additional horn needed for this to happen). If the stretching is slow and even, this elongation can happen with very little damage to the laminae – they get longer but function fairly normally. The stretched epidermal laminae then merge with the edge of the sole to form a stretched white line. Whilst a slight stretch to the white line is commonly seen in mild laminitis cases, it is rare for the white line to stretch evenly to any great extent; most cases tend to occur in bursts, with stronger attacks interspersed with periods of remission.

If, on the other hand, the hoof wall is pulled suddenly and dramatically away from the bone (as can happen in a severe acute attack), the dermal laminae can break, completely disconnecting the hoof wall from the dermal laminae. In the process of breaking, the blood vessels within the dermal laminae will burst, releasing blood into the newly formed gap between the hoof wall and the dermal laminae. This pocket of blood (a laminar haematoma) can be seen on an x-ray as a dark region immediately behind the hoof wall. This dark region is often described as a ‘gas pocket’ (an unhelpful term in this case, but this is because it’s not easy to distinguish gas from fluid on an x-ray). Once the wall has been torn away, the dermal laminae (assuming the underlying laminitis process has largely ceased) will rapidly start to heal. With no connection to the wall and hence no tension on them, they will heal back to fairly normal proportions within quite a short timescale.

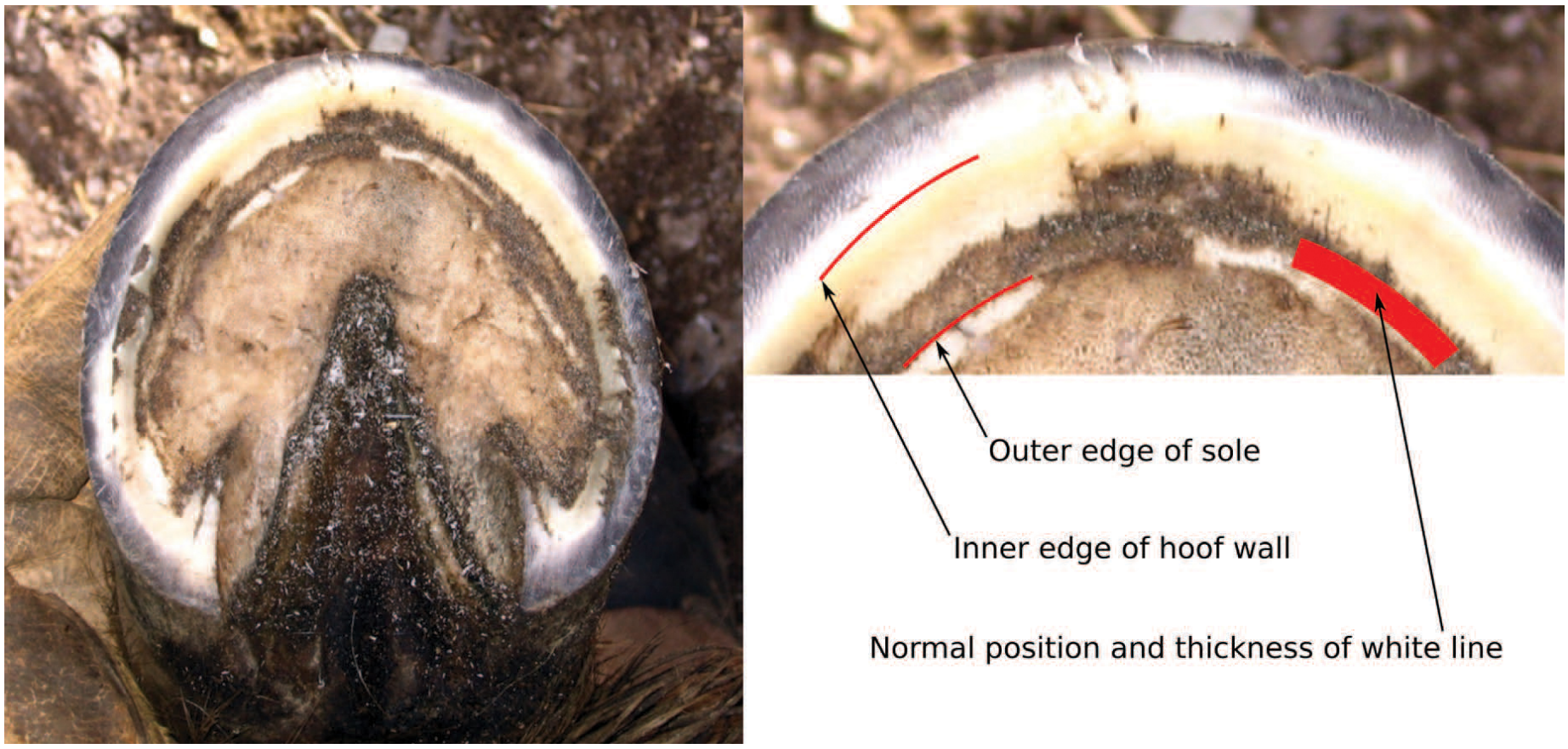


Fig. 27 Stretched white line in a chronic laminitis case, reflecting stretching of the epidermal laminae. Some damage from laminar tearing can be seen in the central toe area, but the laminae on each side of the toe have stretched without obvious damage. It is unusual to see such a dramatic stretch without significant damage to the laminae.

The two scenarios described above represent the extremes, and most cases fall somewhere in between. The degree of damage being done at any time ranges from mild stretching, where the laminae remain fairly intact, through moderate tearing where some of the laminae may break and others stretch, to the severe case where all the laminae break. Whatever happens to the epidermal laminae will be reflected in the white line in the following weeks: hence you can often get an idea of what has happened by examining the white line.

The mechanism of rotation means that the laminae are torn apart the most at the bottom of the wall, and the least at the top. The result is a wedge-shaped region between the wall and the bone, which is filled with anything from mostly blood to mostly horn with some blood. This is termed the laminar wedge.

In the aftermath of an acute laminitis attack, as the underlying laminitis subsides, the dermal laminae start to heal. The healing process involves inflammation, and the response of the laminae to inflammation is to produce horn very rapidly (remember that the dermal laminae normally produce virtually no horn). Those dermal laminae that are still partially attached to the wall will be under tension, and will respond by producing even more horn. The quality of this horn tends to be poor as it is being produced by damaged laminae. It is soft (as a result of rapid production), and contaminated with remnants of broken dermal laminae, blood products and so on. Because some laminae fail and others don’t, the resulting material appears stringy, and is patchily red (from whole blood) or orange (from serum).

Initially the laminar wedge is not visible as it is contained entirely within the foot. However, as the hoof wall continues to grow down in the following weeks and months, the laminar wedge (which is typically attached to the inside of the hoof wall) displaces the normal white line. You then have an externally visible portion of the laminar wedge that consists of badly damaged epidermal laminae mixed with blood products and remnants of dermal laminae, and into which the terminal papillae attempt to inject horn to form some kind of white line.

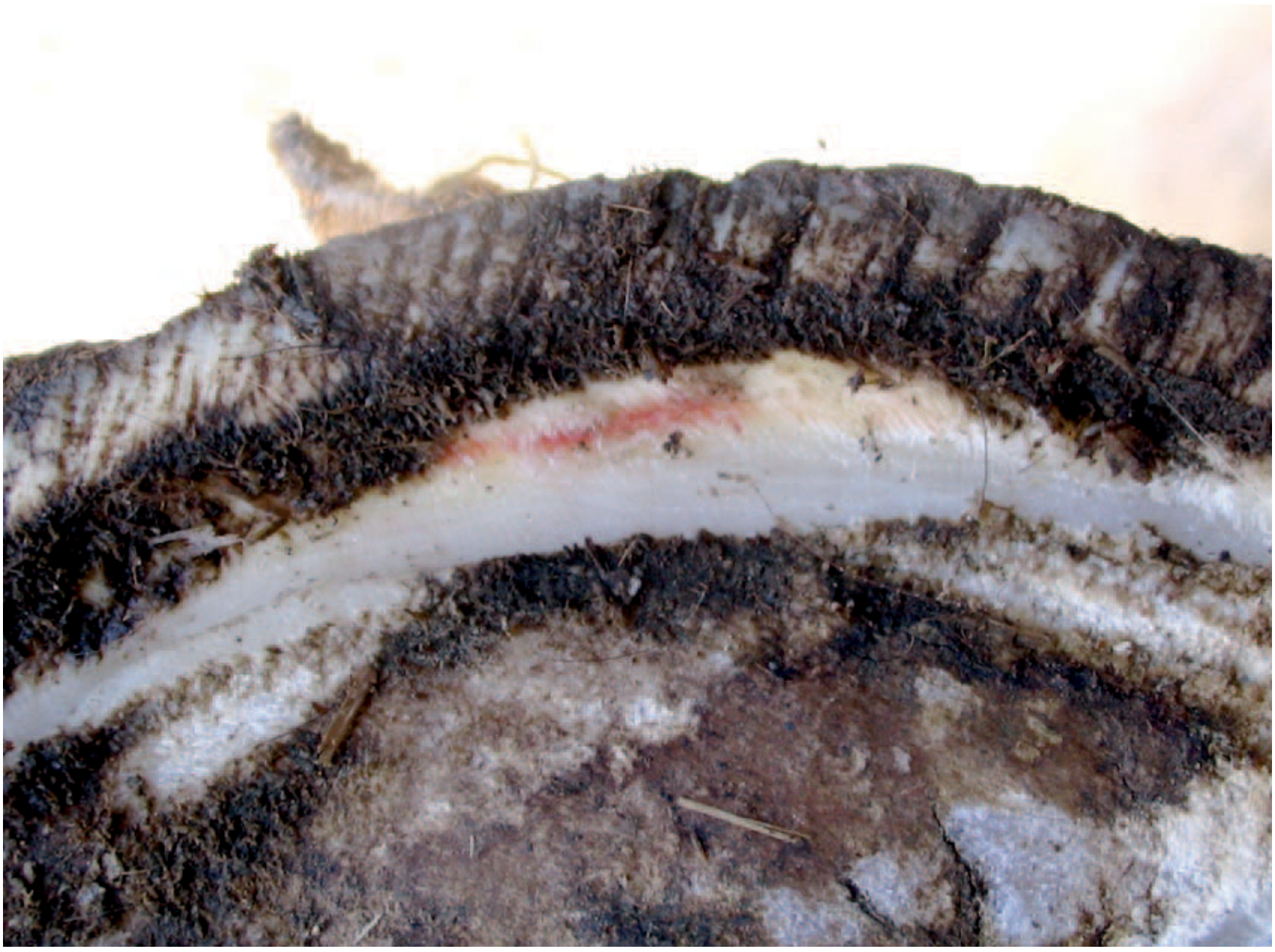


Fig. 28 Blood in the white line as a result of a mild laminitis attack a few weeks previously. The attack was very short lived and resulted in bruised dermal laminae (and hence blood products being incorporated in the epidermal laminae), without significant stretch or tearing.

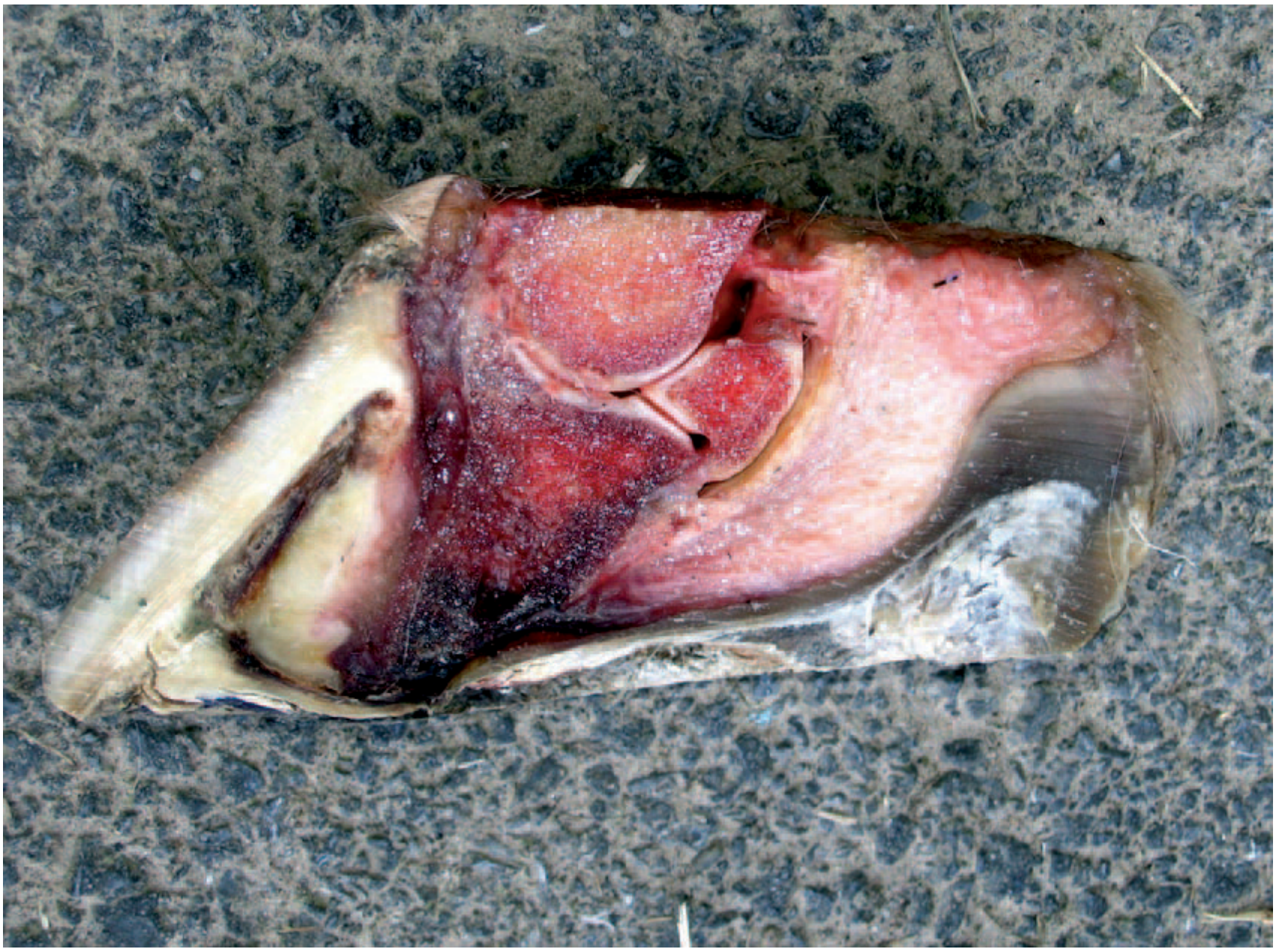


Fig.29 A section through an acute laminitis case showing the initial formation of a haematoma behind the hoof wall, followed by production of a laminar wedge as the dermal laminae have healed. The nature of the damage to the top of the wall indicates that this entire process lasted a matter of days to weeks, rather than weeks to months, showing just how much laminar wedge can be produced in a short time.

It's important to bear in mind that any blood products incorporated into the hoof horn will rot as soon as they get near the surface of the hoof. Blood products disrupt the tight matrix of horn cells so allowing moisture and infection in, but they also form a good source of food for bacteria and fungi, resulting in a characteristic smell. The normal smell of rotting horn is present (as you would get in a bad case of thrush), but this is combined with the smell of rotting flesh as a result of the blood products and fragments of dermal tissue that are incorporated into the laminar wedge. The resulting sweet, sickly smell can sometimes be identified at a distance – especially in the worst laminitis cases.

As a result of rot, blood products in the hoof horn tend to appear black rather than red, unless the hoof has been recently trimmed to reveal them. Fig. 30 clearly shows how some laminae are damaged to the point where they can produce no horn, whereas others manage to produce horn. The result is a laminar wedge which looks like stringy cheese interspersed with blood.

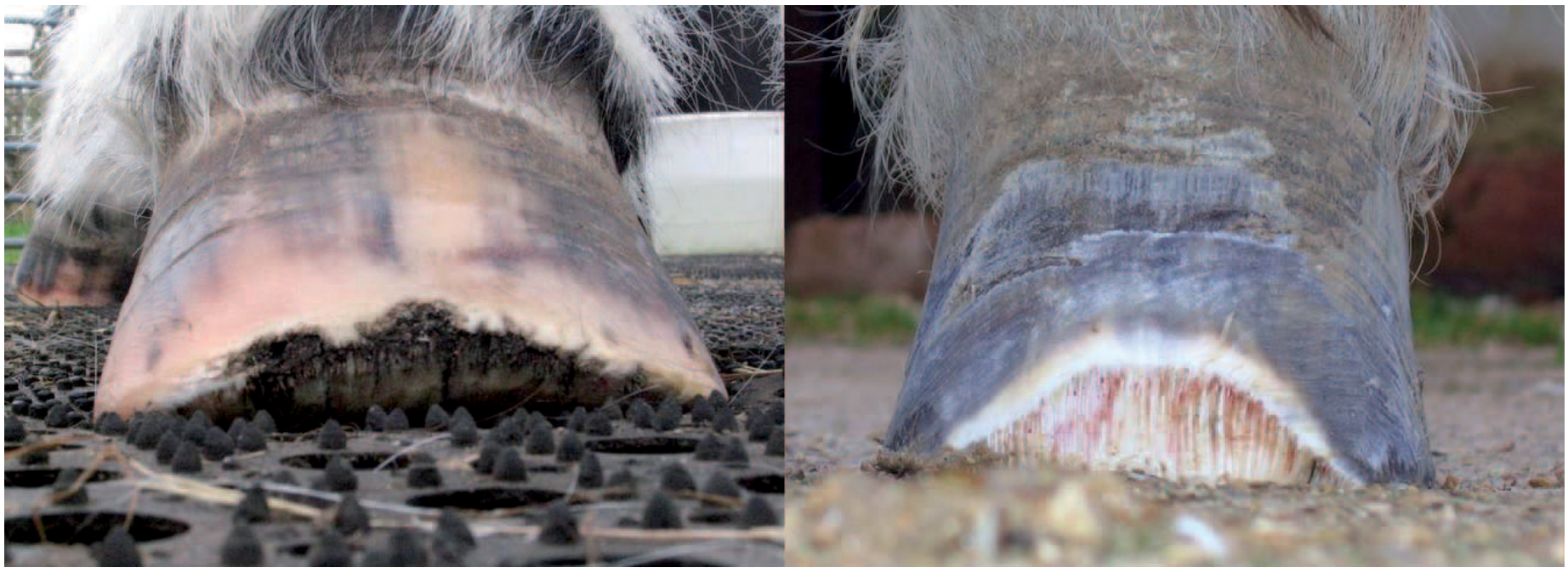


Fig. 30 LEFT: ***Rotten laminar wedge exposed at the bottom of the hoof wall.*** RIGHT: ***Laminar wedge trimmed to the point where most rotten material has been removed – blood products can clearly be seen. (Photo: Jayne Hunt)***

The externally visible portion of the laminar wedge represents a problem for the recovering horse. It does not form a water-tight seal in the way that healthy white line does. As a result, there is a dramatically increased risk of moisture and infection getting into the non-visible portion of the laminar wedge. If the underlying causes of the laminitis were short lived and the horse is making a good recovery, by the time the laminar wedge becomes externally visible, the dermal laminae will typically have healed. The last part of the healing process involves producing a thin layer of normal healthy horn that covers the dermal laminae. This layer will effectively seal the dermal laminae away from the remaining laminar wedge, ensuring that any infectious material present in the laminar wedge does not pose a risk to the dermal laminae.

If, however, the dermal laminae continue to be damaged, or a subsequent laminitis attack does more damage before the white line has fully closed again, then there is a high risk of infection tracking from the laminar wedge into the dermal laminae. The combination of a source of infection and badly damaged dermal tissue creates the perfect environment for a foot abscess to form. For that reason, horses with ongoing laminitis problems often suffer repeated and severe abscesses to the affected feet.

It is not uncommon for a severe laminitic to abscess in both front feet simultaneously. This shouldn't be surprising, as the presence of laminar wedge means that infectious material is present close to the dermal laminae, and it only takes a further attack of laminitis to damage the protective layer of horn over the recovering dermal laminae on both front feet, and allow the infection to become established. Whereas an abscess in a single foot in a laminitis case is fairly easy to spot, the lameness that results from a combination of laminitis and abscessing in both front feet is very difficult to distinguish from laminitis alone, and is often misinterpreted as a much more severe recurrence of laminitis. Although the degree of lameness involved is often dramatic and can do as much damage to the foot as the most serious laminitis attack, it is a mistake to see this as an indication that the horse is doomed to further severe laminitis attacks. The decision as to whether to go for euthanasia or not in such cases should be based on whether it is ethical to continue, given the immediate crisis, rather than on an attempt to predict the likelihood of further laminitis down the line. If the horse has repeated bouts of laminitis on all four feet and abscesses on all four feet simultaneously, this then becomes an extremely serious situation.

DROPPED SOLE

As the pedal bone rotates and/or drops in relation to the hoof capsule, it starts to press on the solar corium, particularly around the tip of the pedal bone. The initial response of the solar corium to increased pressure is to produce more horn. The result is false sole (an artificially thick layer of normal solar horn) in the front portion of the foot. This build-up of sole is typically greater than would be seen in

normal callusing, and tends to have a different shape from a normal callus. Ultimately it can result in part of the sole becoming proud of the rest of the bottom of the foot. In shod horses, it is not unheard of for the sole to protrude enough that it actually hits the ground despite the thickness of the shoe – which then causes bruising. This form of extreme callusing is termed a ‘dropped sole’.

Fig. 31 An example of dropped sole, with the sole under the tip of the pedal bone proud of the rest of the ground surface of the foot.

SOLE PENETRATION

If the pressure on the solar corium becomes more extreme, it can start to crush blood vessels and prevent blood from reaching the corium. The circumflex artery is particularly vulnerable to this form of damage. The majority of the damage is typically focused in a crescent-shaped area under the front rim of the pedal bone. Once the blood supply is compromised, the ability of the front portion of the solar corium to produce horn is severely impaired. Now there is a risk that external wear and exfoliation of the sole exceeds the ability of the solar corium to replace it with new horn. In this situation, the sole becomes progressively thinner locally under the tip of the pedal bone. If not corrected, this process has only one possible outcome: the affected area of solar corium loses its covering of horn and is exposed to the outside world. The resulting risks of damage and infection to both the corium and the bone immediately beneath (not to mention the pain involved) are then so severe that the horse is usually euthanized on humane grounds.

Fig. 32 Solar penetration. The solar horn in this case became so thin as a result of loss of circulation to the solar corium that infection got through and caused a serious abscess. The abscess then blew out through the sole, destroying what little solar horn was left. The resulting release of pressure allowed the solar corium to produce a covering of 'repair horn' within a matter of hours. Through the hole in the sole, yellow repair horn can be seen covering the rim of the solar corium. The remainder of the solar repair horn is covered in dried blood that has leaked from badly damaged arteries. (The green discoloration is antiseptic that has been sprayed on by the vet to prevent further infection.) Unusually, this horse survived and made a full recovery.

SUB-SOLAR HAEMATOMAS

Thankfully solar penetration is rare, but it is not uncommon to see significant damage to the circumflex artery. Where this artery is badly damaged, blood leaks into the surrounding tissues, creating a haematoma (blood blister). If the artery is able to recover, then solar horn production gradually resumes, and the haematoma (sometimes with fragments of damaged corium) typically gets incorporated into the solar horn. The presence of this blood softens the solar horn immediately around it, creating a pocket of blood with a layer of soft, flexible horn around it. Encapsulated in this way, there is no mechanism for the blood to dry out or for the immune system to break it down – it remains as liquid arterial blood that looks convincingly fresh.

Fig. 33 Two examples of sub-solar haematomas, one with pus present (right).

Over a period of weeks, any haematoma will move to the surface of the sole, pushed out by new horn produced at the corium. The unwary trimmer or farrier cleaning exfoliating material from the surface of the sole may unexpectedly hit soft horn, and will then see what appears to be fresh blood oozing from the sole. When fully revealed, the haematoma is typically crescent-shaped – a reflection of the shape of the circumflex artery. The volume of blood involved can be as much as 1ml – enough to worry anyone not aware of the true cause. However, unlike a nick in the solar corium, the 'bleeding' stops almost immediately.

Sub-solar haematomas are most commonly uncovered when the sole is exfoliating a thick layer of false sole that it no longer needs as healing progresses. As the exfoliating material is removed, the haematoma appears between the exfoliating material and the remaining sole. It is also not uncommon to find haematomas that have a mixture of blood and pus in them – these are the result of abscessing in the aftermath of damage to the circumflex artery.

LOSS OF PEDAL BONE

Whilst loss of the circumflex artery can result in pedal bone penetration, there is another possible outcome. In some situations it would appear that the circumflex artery can become severely damaged and yet the sole can retain a degree of circulation, allowing it to grow enough to prevent penetration. This typically happens where the damage to the circumflex artery happens slowly over an extended period of time, rather than a matter of days or weeks. In this situation, the network of arteries and veins within the foot has time to adapt to the damage. Blood vessels are remarkably good at repairing themselves, and are also remarkably good at rerouting where one route becomes non-viable. Whilst the main arterial supply of blood to the solar corium is via the circumflex artery, there is a secondary supply from the terminal arch (*see* Fig. 7 in Chapter 1). With the circumflex artery severely compromised, this secondary supply from the terminal arch gradually becomes more prominent. Whilst this allows blood to reach the solar corium and hence allows sole to continue to grow, it appears that this route does not provide sufficient circulation for the bottom portion of the pedal bone to survive. It seems that the blood vessels of this alternative supply pass through the pedal bone to the solar corium, but do not actually supply the bone itself.

Without sufficient blood supply, the tip of the pedal bone dissolves away, leaving a smaller pedal bone. The exact mechanism by which this process occurs is not well understood, but the end result is that the lowest portion of the pedal bone shrinks until the terminal arch is close to the bottom of what is left of the bone. At this point, some kind of equilibrium is reached whereby the remaining bone has sufficient blood supply, as does the solar corium.

One would expect that such serious loss of pedal bone would cause permanent, severe lameness, but in reality horses often manage to cope quite well with a significantly smaller pedal bone so long as the underlying laminitis is brought under control. The foot appears shorter, but still functions as a fairly

normal foot. Sole growth is usually less than ideal with the rerouted blood supply, and such horses typically won't cope with significant amounts of wear. With the use of shoes or removable hoof boots, a degree of light work is often achievable. If, however, the underlying laminitis continues, then the terminal arch may also become compromised, resulting in the loss of the majority of the pedal bone. This causes sufficient problems that it becomes unethical to continue to try to keep the horse alive.

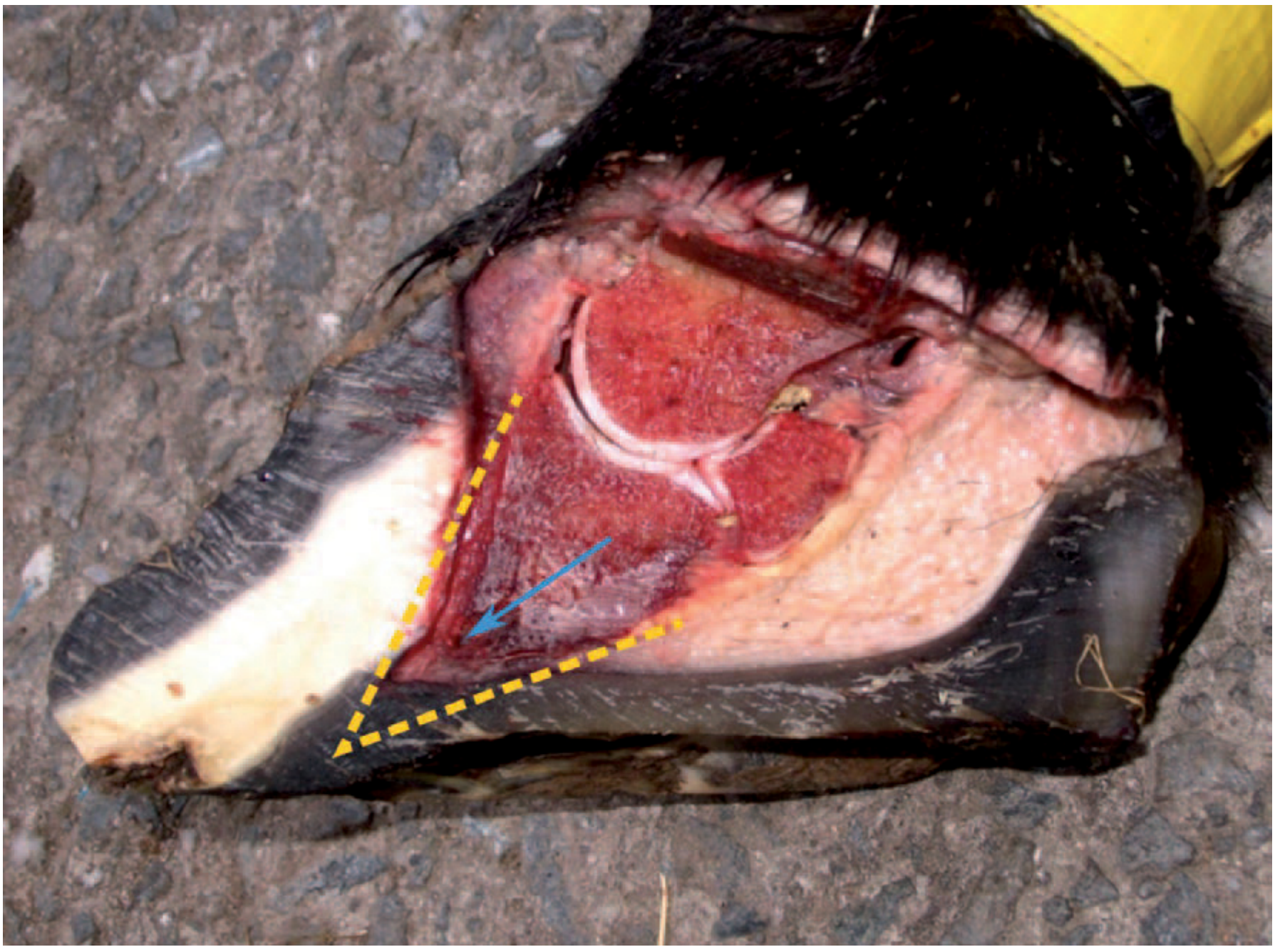


Fig. 34 Dissection of a foot showing total loss of the circumflex artery. The approximate original extent of the pedal bone is marked (dotted yellow line), as is the position of the terminal arch (blue arrow). The presence of large amounts of recently formed laminar wedge indicates that the underlying laminitis in this case was not under control. However, where the underlying laminitis is well controlled, this degree of pedal bone loss is survivable.

Fig. 35 Frontal sections through two similar-sized feet showing the shape of the pedal bone just in front of the apex of the frog. LEFT: *A healthy foot with the underside of the pedal bone normally concave and a fairly thick sole.* RIGHT: *A long-term laminitic foot with significant loss of bone mass around the rim of the pedal bone. The underside of the pedal bone is nearly flat, and the sole is thin. The position of the arterial channels in both pedal bones helps with comparison.*

PEDAL BONE REMODELLING

Whilst total and permanent loss of the circumflex artery is thankfully extremely rare, temporary loss of circulation via the circumflex artery is very common. We have already seen how this can affect the sole, resulting in poor growth and haematomas. However, it can also have a significant effect on the pedal bone. The scenario described above, where a substantial portion of the pedal bone is lost, is just the extreme example. In milder cases, some degree of pedal bone loss can occur at the tip of the pedal bone before circulation returns to the circumflex artery, so halting the loss of bone. Initially, the pedal bone loses density right at the tip. This makes this part of the bone weaker than normal. This situation is usually associated with high heels and the pedal bone being at a steep angle. This means that the tip of the pedal bone is not only weaker than normal, but is subjected to abnormal forces attempting to push it upwards. The tip of the pedal bone will typically respond by remodelling into a shape described as a ‘ski tip’. This is very commonly seen on x-rays of horses with a significant history of laminitis.

In some cases, rather than remodelling, the weakened tip of the pedal bone may fracture away from the rest of the bone, leaving a fragment of bone floating about adjacent to the pedal bone. These fragments usually heal back on to the main part of the pedal bone in time, but often at an incorrect angle. As healing of the fracture progresses, the pedal bone attains a ‘ski-tip’ shape just as with remodelling, but usually more exaggerated.

At first sight, the presence of a ski tip would appear to be a major problem for the horse, as this could present an obstruction to the laminae as the wall grows down the front of the bone. In reality, the horse deals with this situation admirably by creating additional connective tissue between the front surface of the pedal bone and the dermal laminae, spacing the dermal laminae far enough away from the bone so that they navigate safely past the ski tip. This may result in the dermal laminae no longer being quite parallel to the front surface of the pedal bone, although this effect is usually very small. The solar corium also stretches forwards slightly such that the front portion of the corium is adjacent to the newly positioned white line. Occasionally, a horse may appear to have a slightly lower quality white line as a result of the presence of a ski tip, but in most cases, the presence of a ski tip cannot be detected without x-rays.

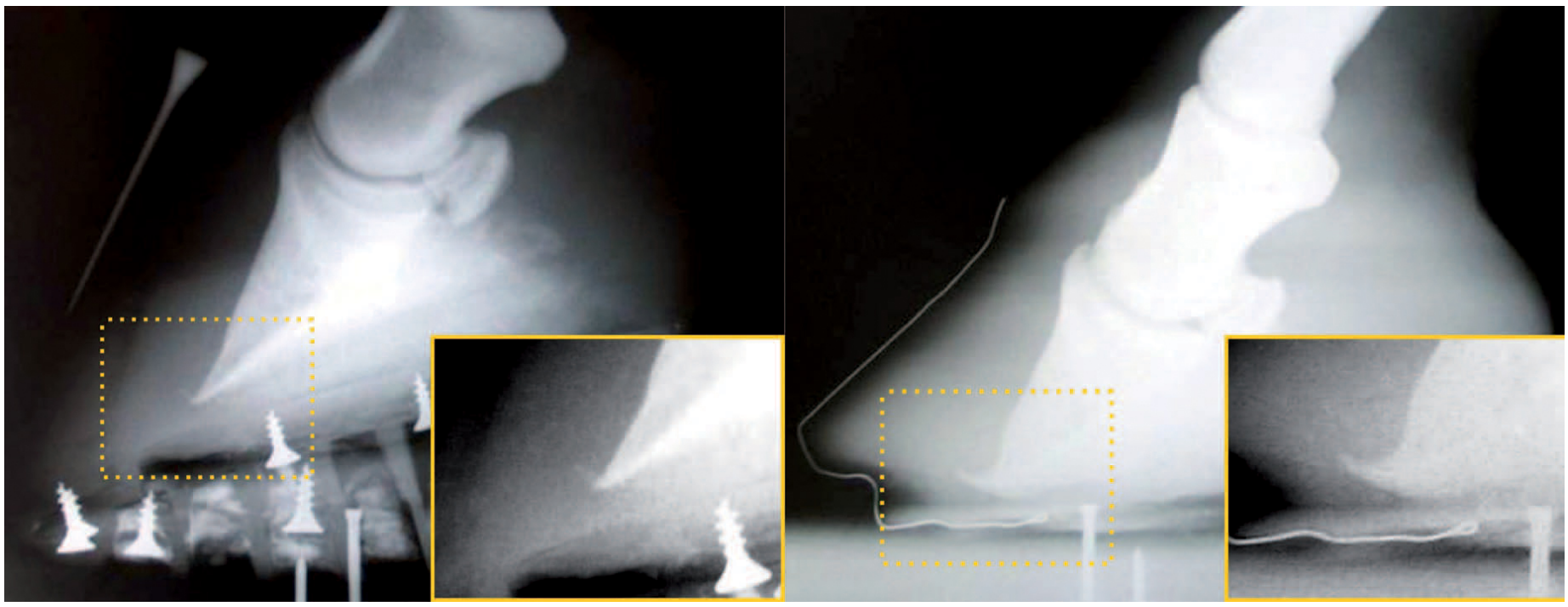


Fig. 36 LEFT: *A fracture to the tip of the pedal bone, with the free fragment at 90 degrees to its original position. A wedge shoe, combined with ongoing laminitis, has created the perfect conditions for the fracture to occur.* RIGHT: *The same foot after nine months, the first two months of which was with the wedge shoe fitted. The fragment of bone has reattached to the pedal bone, creating a classic ski-tip shape. Damage to the circumflex artery means that the sole is barely growing, resulting in a paper-thin sole. The sole in this foot recovered extremely well with appropriate management, but there was permanent damage to the tip of the pedal bone (despite which the horse was sound).*

FLEXOR TENDON INVOLVEMENT

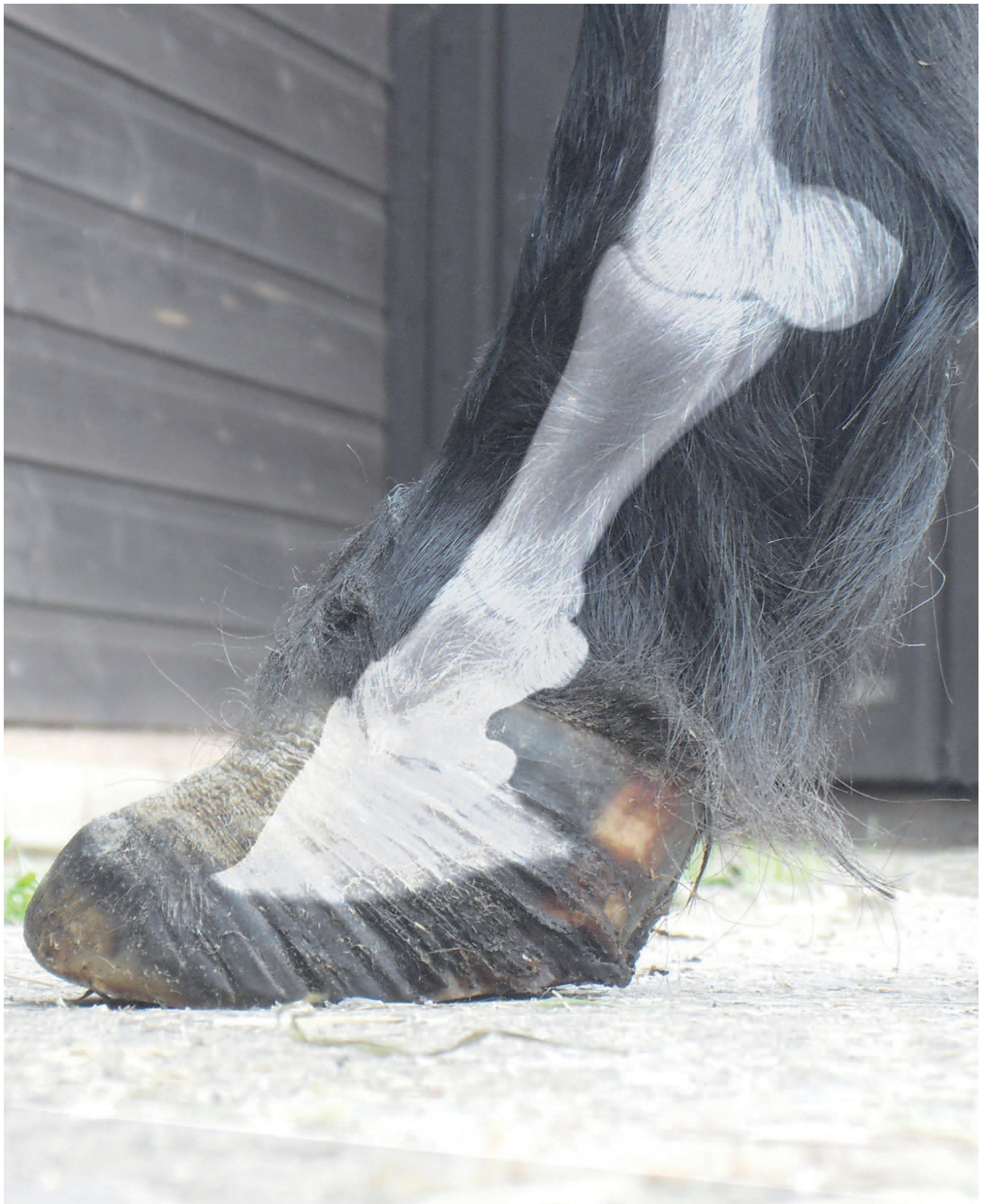
Given that the pedal bone typically rotates in laminitis, anything that acts to force the pedal bone further in that direction is likely to be a problem. For this reason the deep flexor tendon is often seen as being involved in the rotation process. This tendon acts to flex the coffin joint and hence moves P3 in the same direction as the rotation that happens in laminitis. As such, any tension in the deep flexor tendon will tend to pull damaged laminae further apart and increase rotation. For this reason, vets will sometimes recommend fitting a wedge between the hoof and the shoe so as to raise the heels significantly. This has the effect of flexing the coffin joint and hence relaxing the tendon so as to reduce the degree to which it pulls on P3 and causes more rotation. This approach is, in the vast majority of cases, deeply flawed, as will be seen in the next chapter.

3 A Horse-Centred View of Laminitis

Chapter 2 described how the established view of laminitis sees the pedal bone as rotating and sinking within the hoof capsule. However, there is a way of reframing the problem that can help to create a better understanding of the processes involved and give us more tools to fix the feet. The trick is to look at things from the horse's point of view – something humans aren't always that good at.

REFRAMING ROTATION

The typical view of laminitis takes our world, or more specifically the ground under the horse, as the frame of reference. When the term 'rotation' is used, what is usually understood is that the pedal bone has rotated in relation to the hoof capsule (specifically the front section of the hoof wall). But that hoof capsule is sat on the ground that the horse is stood on, so effectively we tend to visualize the rotation in relation to the ground (Fig. 37). While there is nothing technically incorrect with this, taking the horse's view can result in a more helpful visualization of the problem.



A laminitic pony foot, trimmed to correct physiological balance. An x-ray of the same foot is superimposed to illustrate that the pedal bone is correctly oriented to the ground. The toe has been trimmed conservatively.

From the horse's viewpoint, the pedal bone, far from being incorrectly positioned, remains exactly where it is supposed to be – on the end of the leg. Rotation of the pedal bone around the coffin joint is completely normal and is necessary to allow for extension and flexion of the coffin joint. From the horse's perspective, it is the hoof capsule that has rotated away from the bone, rather than the other way around. If we modify Fig. 37 to keep the pedal bone at the same orientation to the rest of the leg, the concept of the hoof capsule rotating away from the bone becomes apparent (Fig. 38).

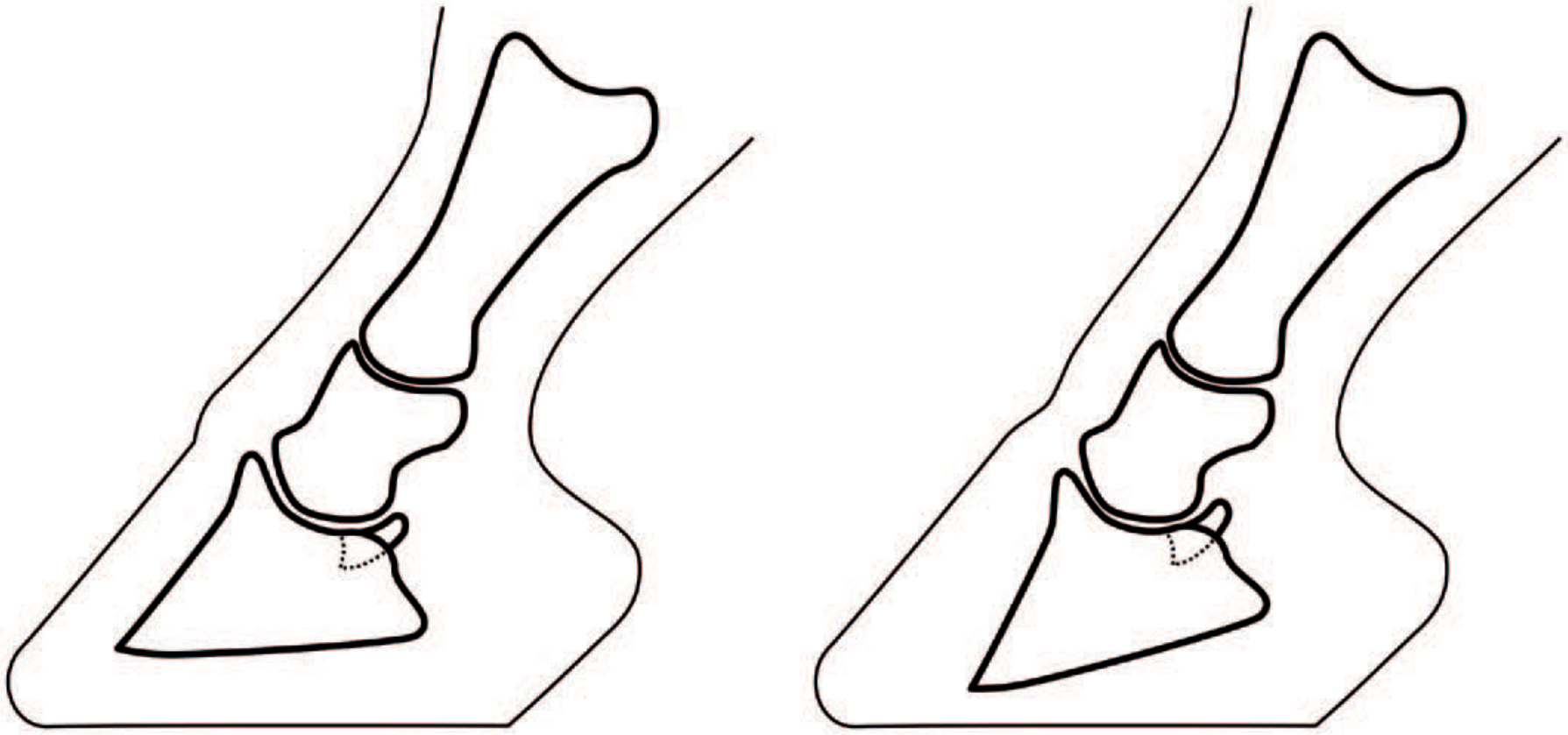


Fig. 37 The ground as the frame of reference. LEFT: *A healthy foot.* RIGHT: *A rotated foot.*

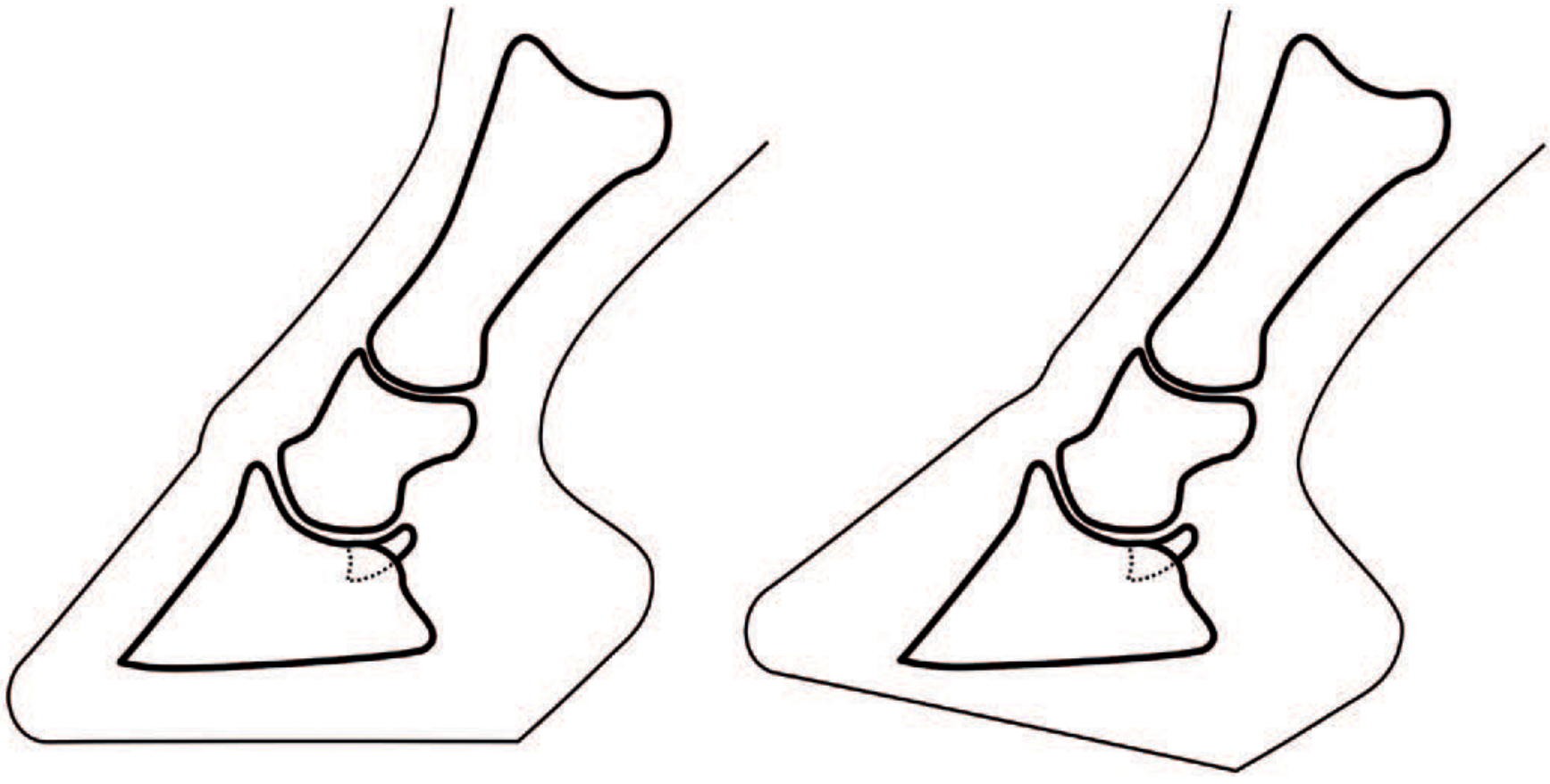


Fig. 38 The pedal bone as the frame of reference. LEFT: ***A healthy foot.*** RIGHT: ***A rotated foot. This view is an artificial construct as the hoof wouldn't sit on the ground at this angle, but repositioning the coffin joint in this way aids visualization of the process of rotation.***

This reframing of the problem doesn't change the relationship between pedal bone and hoof capsule at all, it only changes the way we see it. But that change in viewpoint helps to guide our thought processes when it comes to understanding what has happened and also what we can do about it. The conventional viewpoint leaves us wanting to try to push the pedal bone back into its 'correct' place, whereas the alternative viewpoint has us trying to realign the hoof capsule with the bone.

A useful (if somewhat gory) way to visualize this is to imagine your own fingernail being partially ripped off. If someone then suggested trying to push your finger back into place under the finger nail, you'd laugh at them. To you it is the finger nail that's in the wrong place and the old finger nail has clearly had it. You just need to grow a new finger nail that's correctly attached to your finger. This new nail will gradually push off the remains of the old, damaged nail. Exactly the same thinking applies to hoof capsule rotation in laminitis.

In the process of the hoof wall rotating away from the pedal bone, the laminae become damaged. The attachment between hoof wall and bone is destroyed, or at least badly damaged, and there is usually a significant amount of extra horn in the form of laminar wedge generated between the two. There is no way the old wall can be pushed back into its correct alignment with the bone, but there is equally no reason why new wall growth coming from the coronary band can't grow down correctly attached to the bone via the healing laminae.

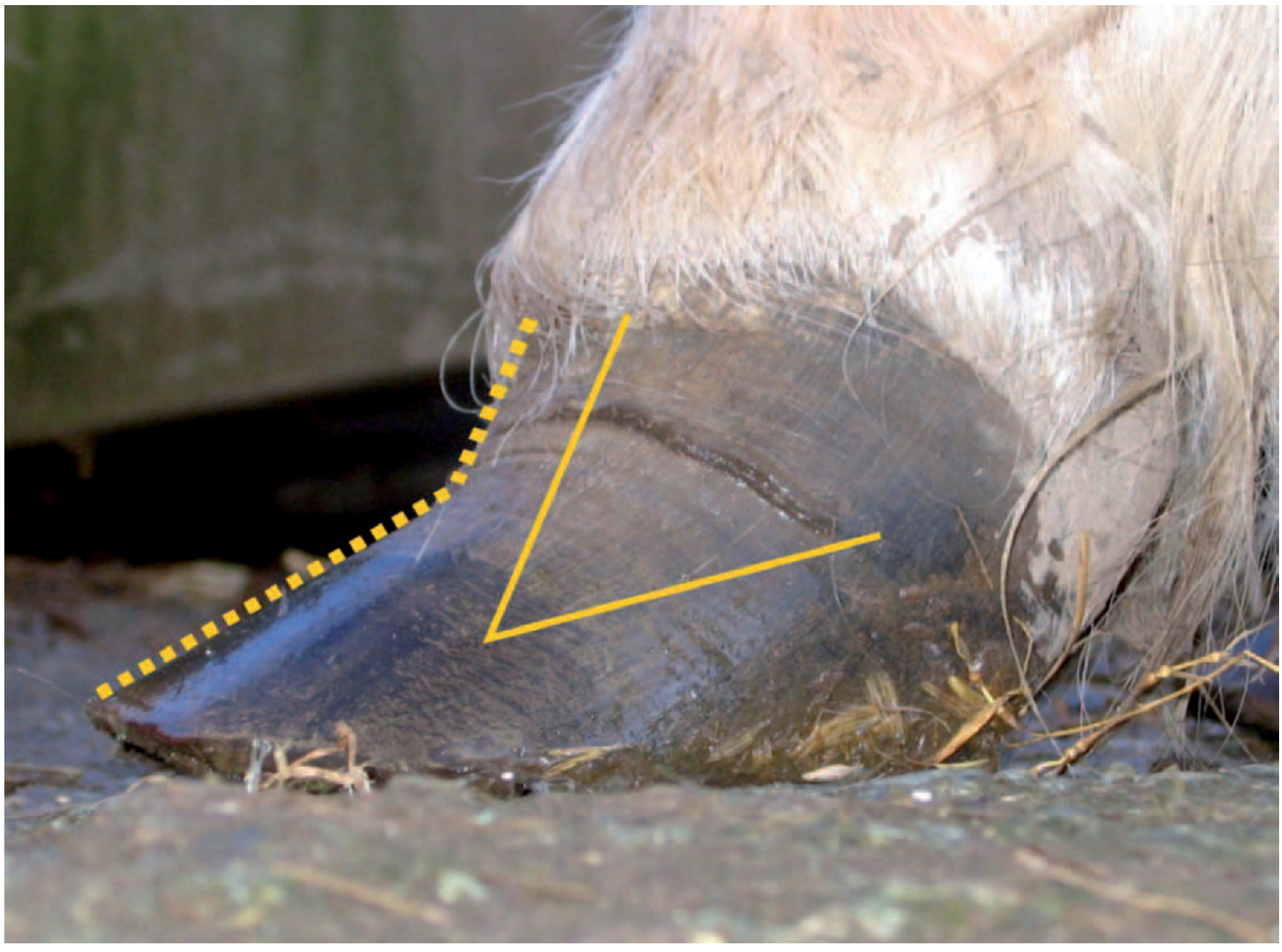


Fig. 39 A sudden change in wall angle in a recovering acute laminitis case, with the position of the front portion of the pedal bone indicated.

As long as the underlying causes of the original laminitis attack are removed, the dermal laminae are extremely good at healing and will almost always form a good attachment with any wall produced after the attack. We saw in Chapter 1 how the density of basal cells in the laminae is dramatically greater than in normal skin. This high density of basal cells is there for just such a situation. Sometimes recovery will happen fairly rapidly, but in more severe cases it can take time for the laminae to heal enough to form a good attachment with new wall growth. Fig. 39 shows a typical case a few months after an acute laminitis attack. You can clearly see that the post-attack wall growth is normally aligned with the bone whereas the pre-attack wall remains rotated away from the bone, just pushed down the hoof by the new growth.

THE DIFFERENCES BETWEEN ACUTE AND CHRONIC LAMINITIS

This reframing of the problem can help us to see the difference between an acute laminitis attack (one where the damage happens over a very short period of time) and a chronic case (where the rotation happens slowly and cumulatively over a matter of weeks or even months).

Rotation in Acute Laminitis

It is perhaps helpful to start by looking first at what happens in a severe acute attack. Such cases are usually related to an obvious and catastrophic cause. The laminitis might originate from a serious infection that has led to sepsis (blood poisoning), or might be as the result of a horse breaking into a feed store and eating a large amount of grain. Such trigger events tend to lead to catastrophic laminitis within a matter of hours. What appears to happen in these cases is a total loss of circulation to parts of the foot. Once the circulation has been lost, the soft tissues sandwiched between the bone and the hoof capsule start to degrade within a matter of hours, and lose their structural integrity as a result. The laminar attachment of the pedal bone to the hoof wall becomes very weak and can no longer suspend the pedal bone from the inside of the hoof wall. The solar corium also loses its strength and is crushed under the weight of the horse pressing down on the pedal bone.

With the various coria pulped, the bone effectively rattles around inside the hoof with nothing to space it away from the inside of the hoof capsule. If the entire foot loses circulation, the pedal bone drops vertically until it hits the solar horn. If the toe is the worst affected area (which is more common), then only the front of the pedal bone drops and hence the pedal bone rotates (Fig. 40). This rotation is the result of the solar corium crushing and the laminae simultaneously tearing, both as a result of loss of circulation seriously weakening these structures.

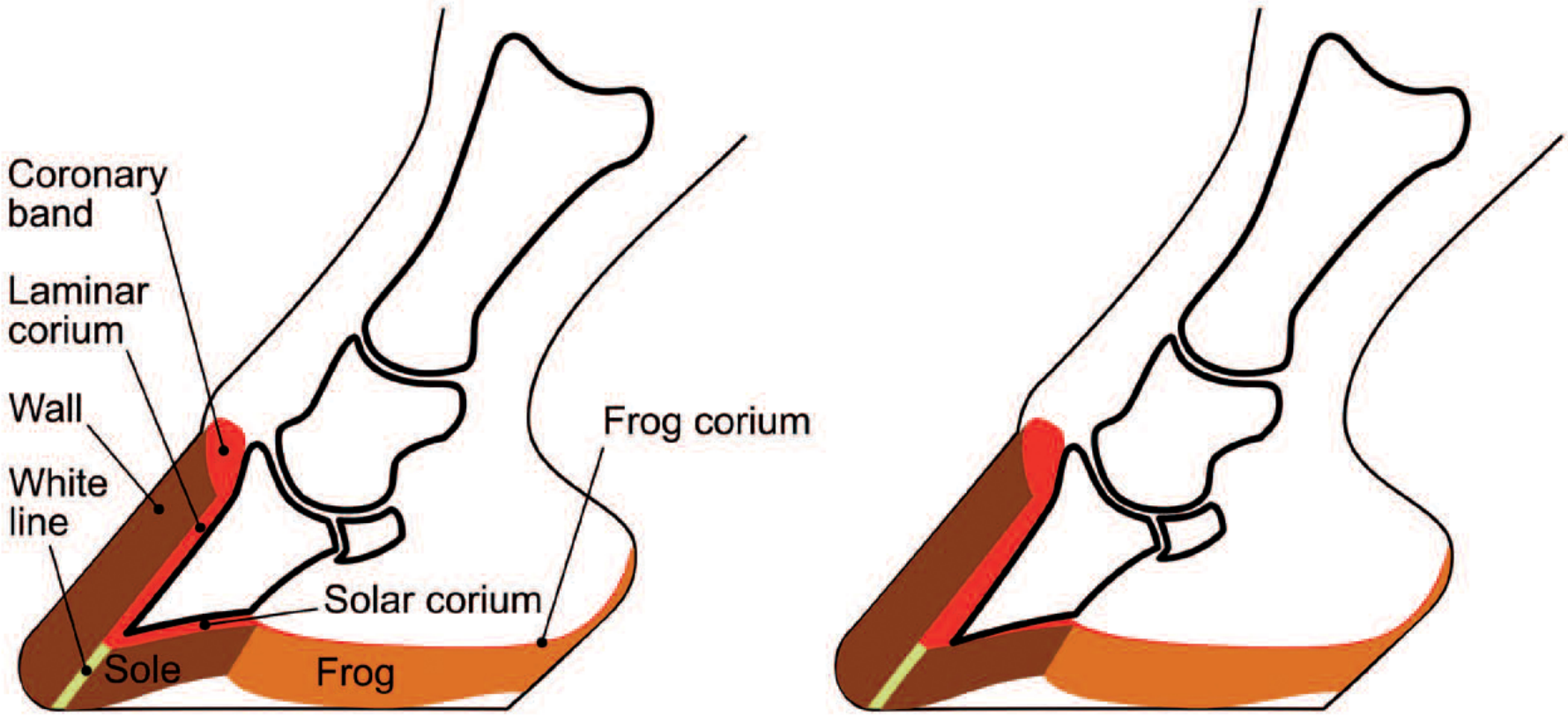


Fig. 40 LEFT: ***The positions of key structures when a healthy foot is cut down the centre line.*** RIGHT: ***In the first few hours of a severe acute laminitis attack the pedal bone rotates as far as it can before the tip of the bone hits the solar horn. As a result of this subtle movement the laminar corium is torn and the solar corium is crushed.***

But once the pedal bone hits the inside of the hoof capsule, it can go no further – at least in the short term. For the pedal bone to rotate further, something else has to change.

Rotation in Chronic Laminitis

To understand how further rotation can happen in an acute case and also how chronic cases rotate, we need to look at the whole horse rather than just the foot. Even in a severe sinker case, the worst of the damage is concentrated in the toe area. That means that the majority of the pain is also concentrated at the toe. An often overlooked part of bio-dynamics is the extremely important role the brain plays in posture and movement. When a particular part of the body hurts, the brain attempts to modify posture and gait so as to minimize that pain. In the case of a horse with toe pain, the obvious response is to

shift weight away from the toe and into the heel area. When the horse is standing still, this is manifested as an altered posture. Similarly, a horse with front foot pain will shift weight away from the front feet by placing the hind feet further forwards so as to move its centre of gravity more over the hind feet. Severe cases often exhibit the posture that is classically associated with laminitis (Figs 21 and 41). In practice, though, the majority of cases are not severe enough to stand like this (although there are usually more subtle changes in posture – *see* Chapter 4). This is because the foot only becomes very sensitive to static pressure when the level of inflammation is severe. Even without the classic laminitis stance, there will still be a tendency to shift weight from the toe to the heels to at least some degree, resulting in more pressure on the heels.

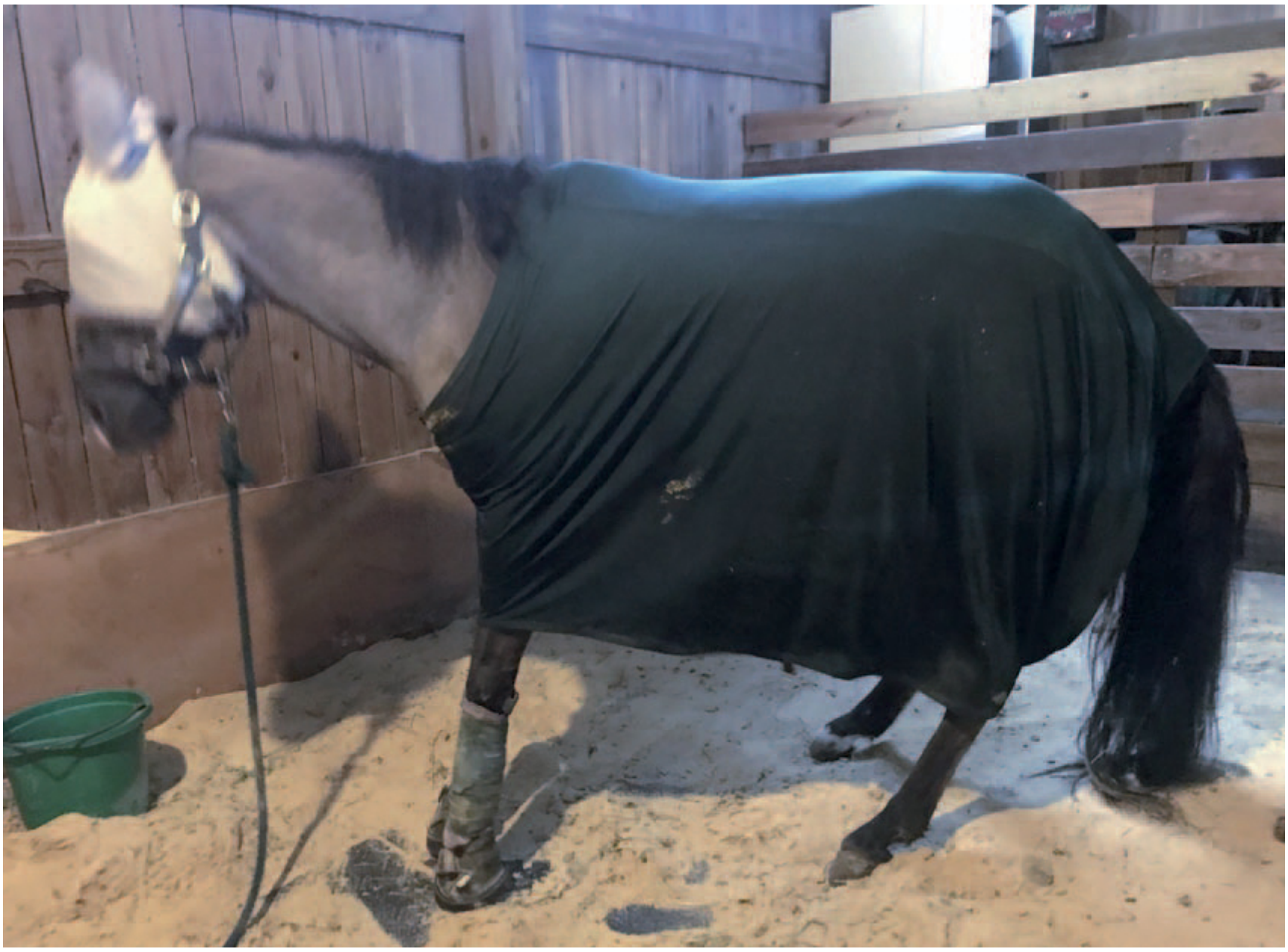


Fig. 41 A horse in a classic laminitis stance attempting to reduce the pressure on the front toes. (Photo: Debra Taylor, Auburn University)

As we saw in Chapter 1, inflamed tissues generate far more pain in response to vibration than to static pressure. As a result, we'd expect to see more pain as an inflamed toe hits the ground (especially on hard ground) than when the horse is stood still. The horse will do anything it can to avoid the pain of impact. It does this by landing excessively heel first, delaying the point at which the toe hits the ground until the majority of the impact has been absorbed by the soft tissues in the back half of the foot.

The knock-on effect of this change in gait is that the horse lands more heavily on the heel and hence the heel gets a greater stimulus. As we have seen in Chapter 1, an increased stimulus to any part of the hoof results in increased growth of that part. An easy way to see this is to look at the spacing of growth rings. In a healthy horse, the growth rings are evenly spaced – the distance between growth rings is the same at the toe and at the heel. But where a horse has toe pain, the growth rings tend to be further apart at the heels, showing that, over the same period of time, the heels have grown more than the toe (Fig. 42).

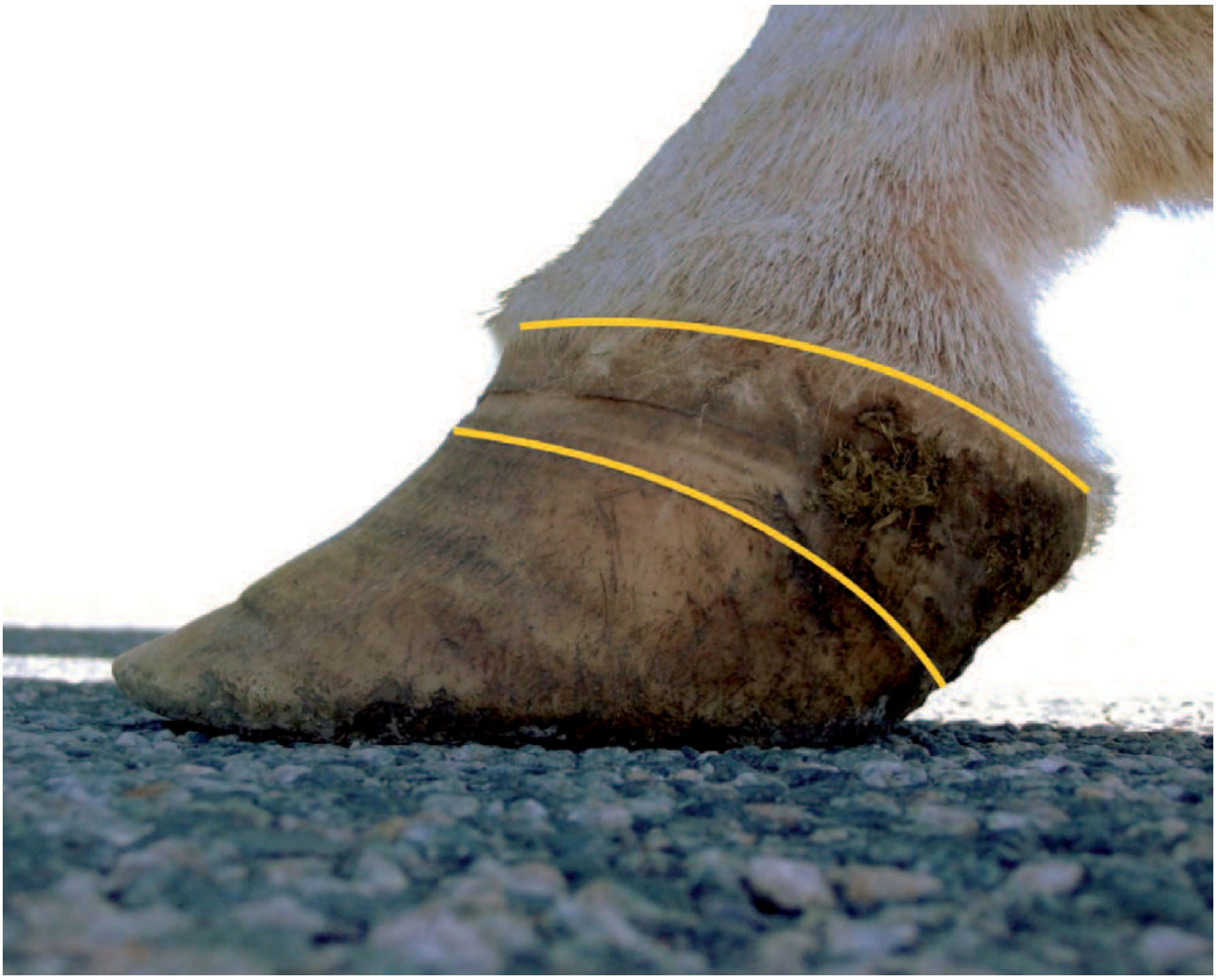


Fig. 42 The hairline and a growth ring marked on a chronic laminitic hoof, showing divergence of growth rings at the heels compared to the toe.

If this pattern of growth continues for any length of time, it results in a distortion in the shape of the hoof capsule. It helps to think of each month's new wall growth pushing the existing hoof wall downwards. If each month's growth is even between heel and toe, then a cylindrical hoof is created (or more accurately a slightly conical one, as the hoof wall gets slightly bigger in diameter as it grows down). But if each month's growth is a wedge that is thicker at the heel than the toe, then these wedges stack over time to create a hoof capsule that curls forwards in the classic 'Aladdin's slipper' shape (Fig. 43). The way in which the hoof wall curves away from the bone is called 'flaring'. Flaring can happen anywhere in the hoof wall, but in chronic laminitis, dramatic flaring is typically seen at the toe.

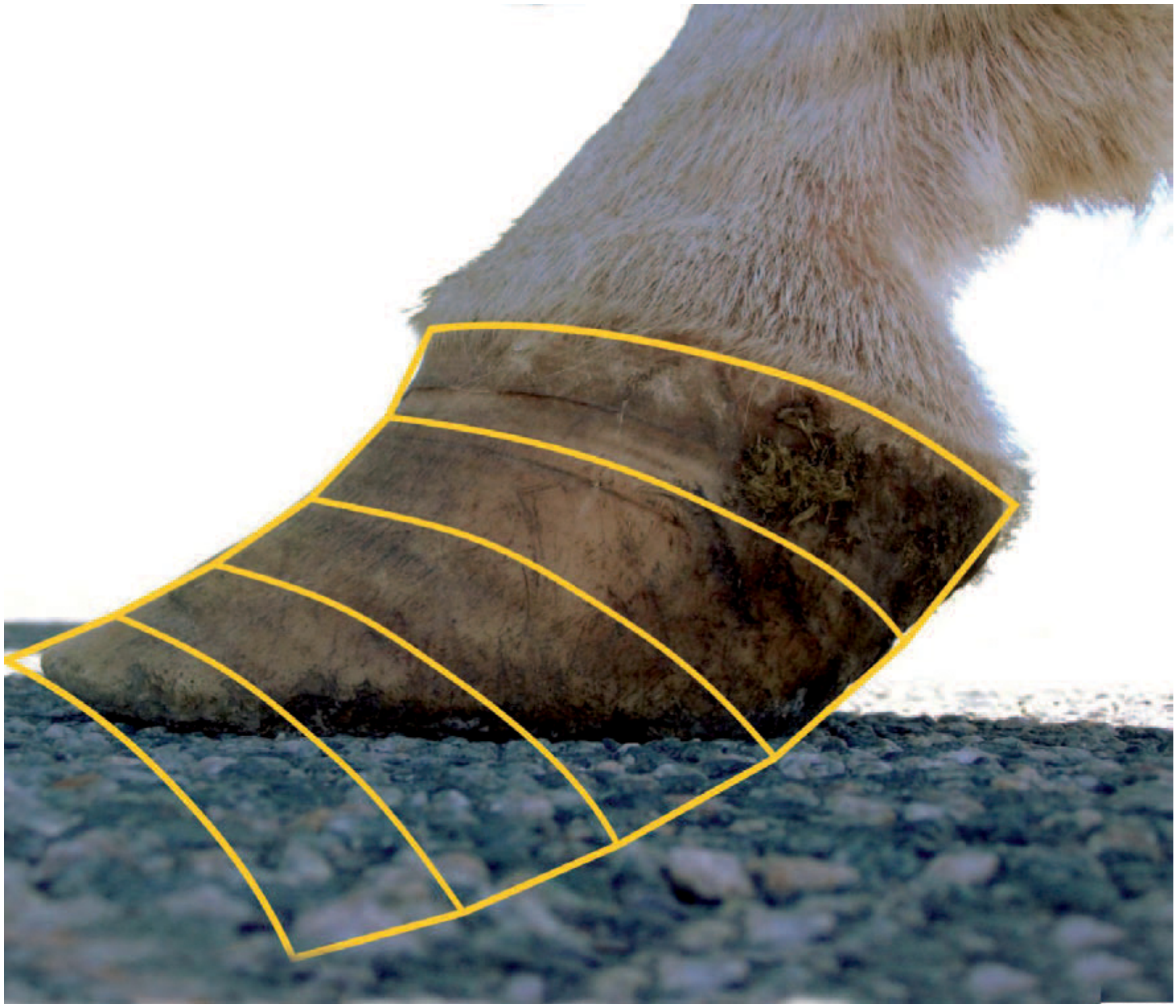


Fig. 43 The result of long-term excessive heel growth when compared to toe growth in chronic laminitis. The excess heel growth is often balanced to a significant extent by increased heel wear – hence some of the heel horn that would be expected given the length of the toe is not present.

Given that the horse is landing more on the heels than normal, it is reasonable to expect that there is also more wear at the heels than normal. Assuming that the horse has some access to hard ground, this means that much, or all, of the excessive heel growth is worn away again, leaving the foot in a fairly normal balance with the ground (as in Fig. 43) – the only outward sign of a problem is the curled shape of the hoof wall at the toe. Where there is less abrasion of the foot (for example when the horse is on soft pasture or on box rest), the heels tend to get longer and longer compared to the toe, which lifts the pedal bone into a more upright position. This can be seen externally when looking at the foot from the side. In chronic cases, the front half of the hairline tends to remain more or less attached to the top of the pedal bone and so can give a rough indication of the angle of the pedal bone to the ground. As the heels get longer and the pedal bone becomes more upright, the front half of the hairline becomes more horizontal (Fig. 44).

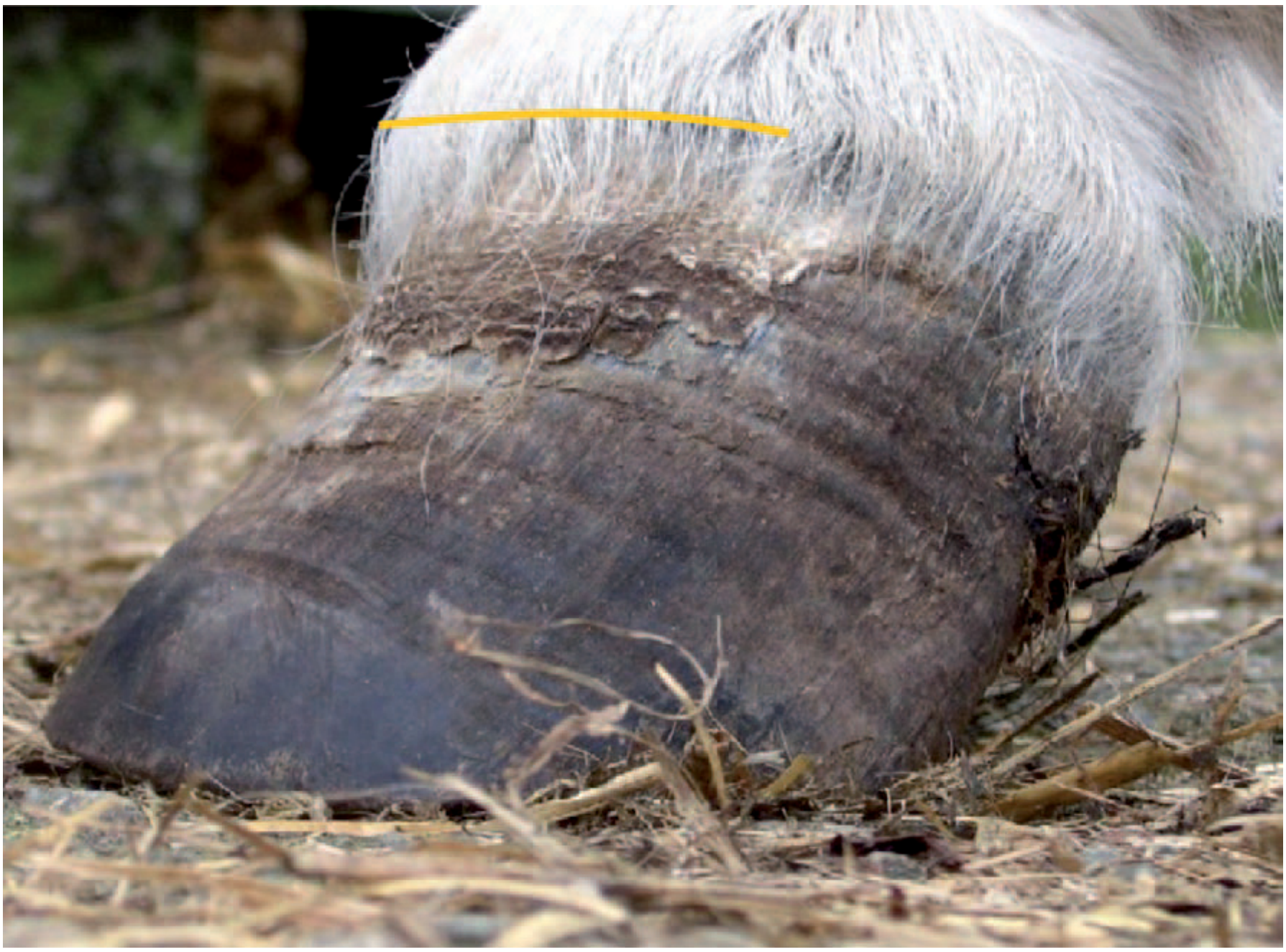


Fig. 44 An upright foot as a result of excessive heel growth without corresponding heel wear. Note that the front half of the hairline is horizontal

If this process is allowed to continue unchecked, the heels can become so long that the natural angle of their growth takes the ground-bearing surface of the heels forward of the centre of the coffin joint. At this point the foot is unstable, and unless the horse holds itself up by tensing the deep flexor tendon, the foot will tend to topple over backwards (Fig. 45).

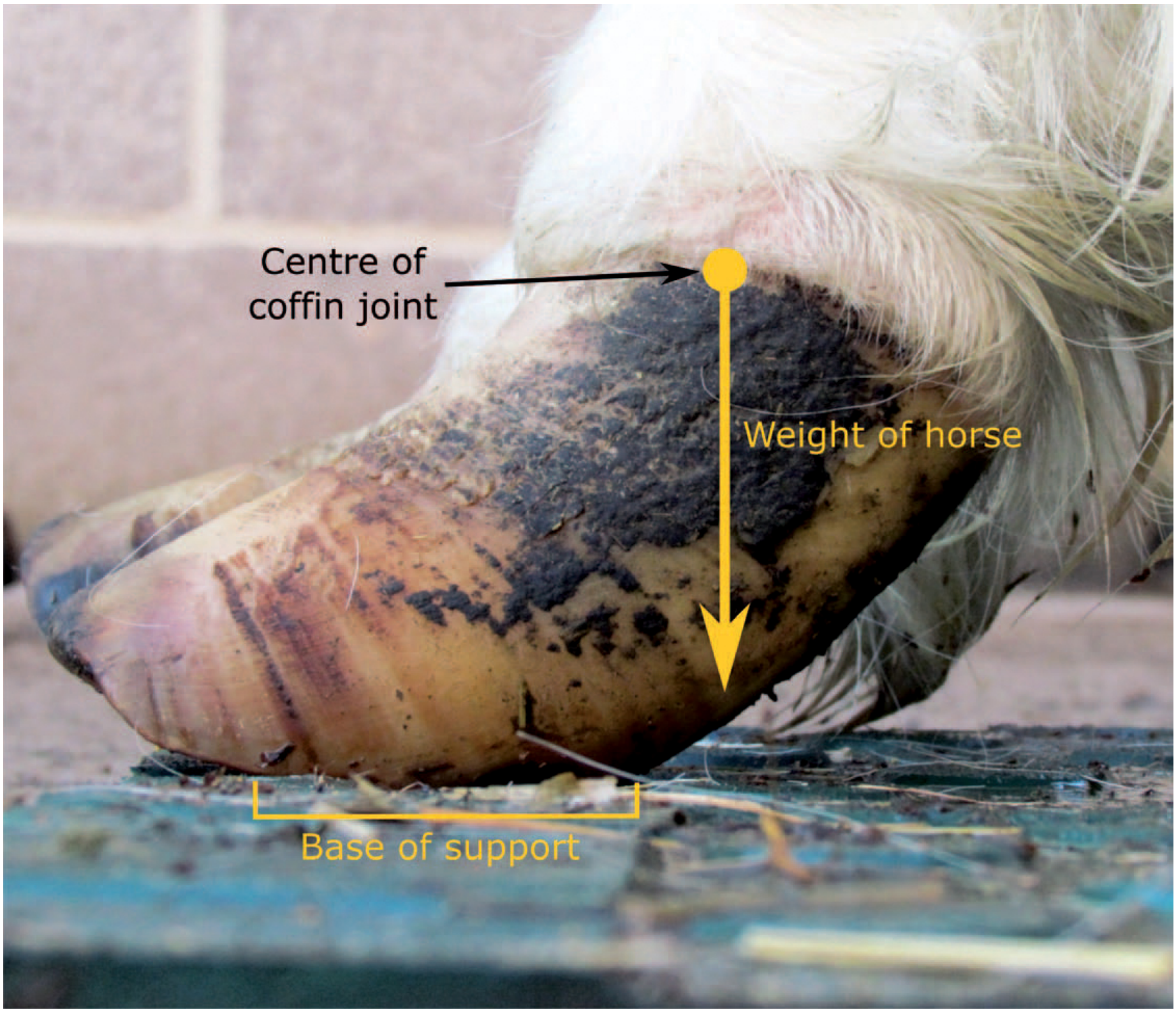


Fig. 45 An extremely long laminitic foot with high heels. This foot is unstable as a result of the base of support being forward of the centre of the coffin joint. The horse can only keep the toe on the ground by tensing the DDFT. (Photo: Sally Bell)

Of course, if the horse tenses the deep flexor tendon and the laminae are inflamed, the result will be more rotation. The horse has to choose between creating more pain and damage at the toe, or walking on the back of the heel bulbs. Eventually the heels become so long and are curled forwards so much that the horse no longer has any choice but to walk on the backs of the heels. Without wear or trimming, the hoof will then continue to curl around into the classic ‘Aladdin’s slipper’ shape (Fig. 46). In extreme cases the toe may hit the front of the leg and create an open sore.

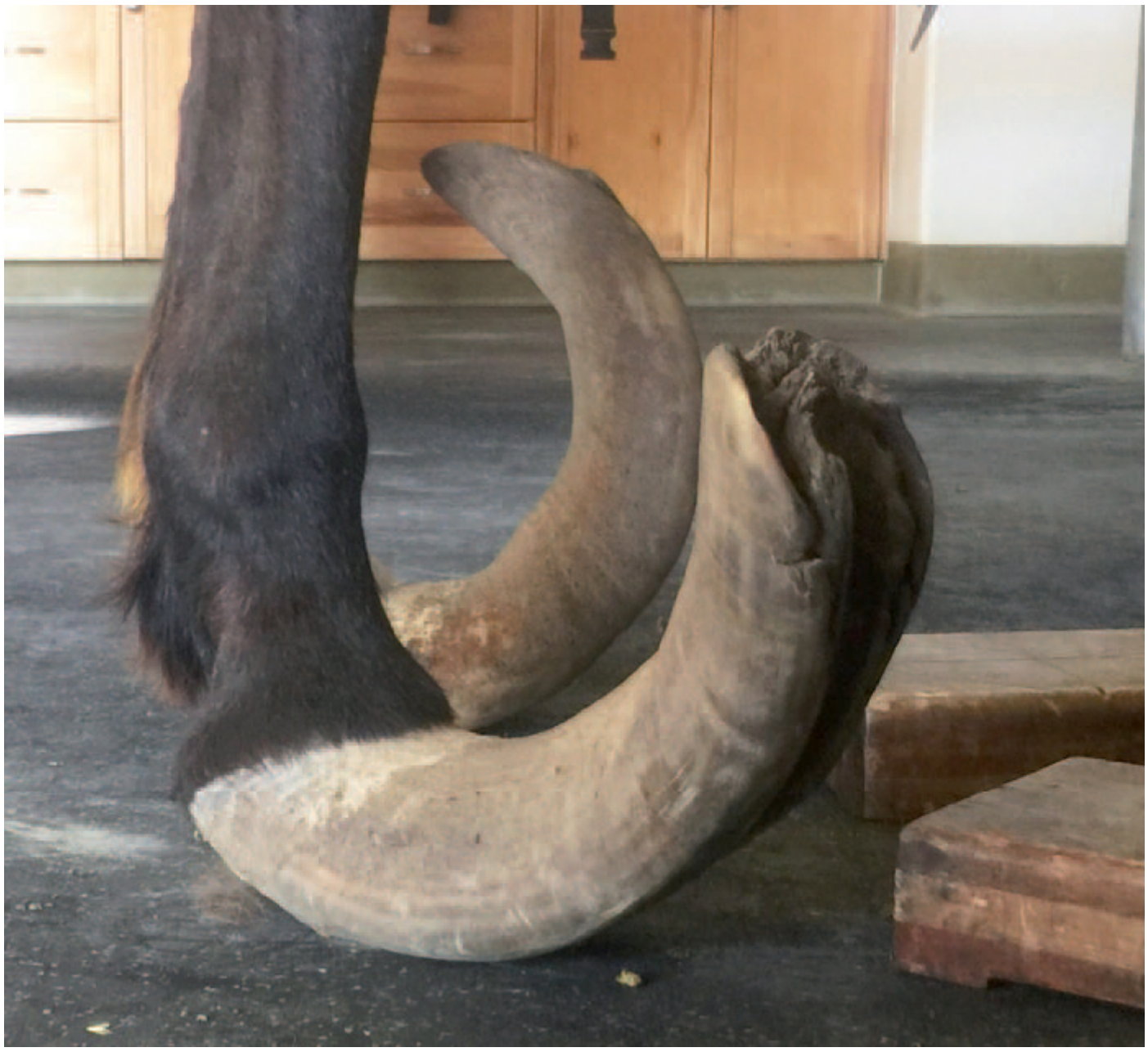


Fig. 46 The result of chronic laminitis over many months, with little wear and no trimming. (Photo: Oakdale Equine Rescue, California)

The Role of the Laminae in Rotation

For the hoof wall to curl forwards, something has to happen to the attachment between the pedal bone and the hoof wall. If this attachment remains strong, the hoof wall at the toe will remain parallel to the front surface of the pedal bone and hence straight from hairline to ground. The differential growth between heel and toe in this situation is absorbed by the hoof wall deforming. The heel still tries to curl forwards but the toe is prevented from doing so by the strong laminar attachment. The hoof wall between the heel and toe has to go somewhere and so bulges outwards just in front of the heel (which it can do because it is supported on lateral cartilage rather than bone in this area and hence can move). The typical end result is under-run heels and quarter flaring but no rotation (Fig. 47). Of course this begs the question of why you would have toe pain from laminitis and yet have laminae strong enough to resist the curling effect – this will be covered in Chapter 4.

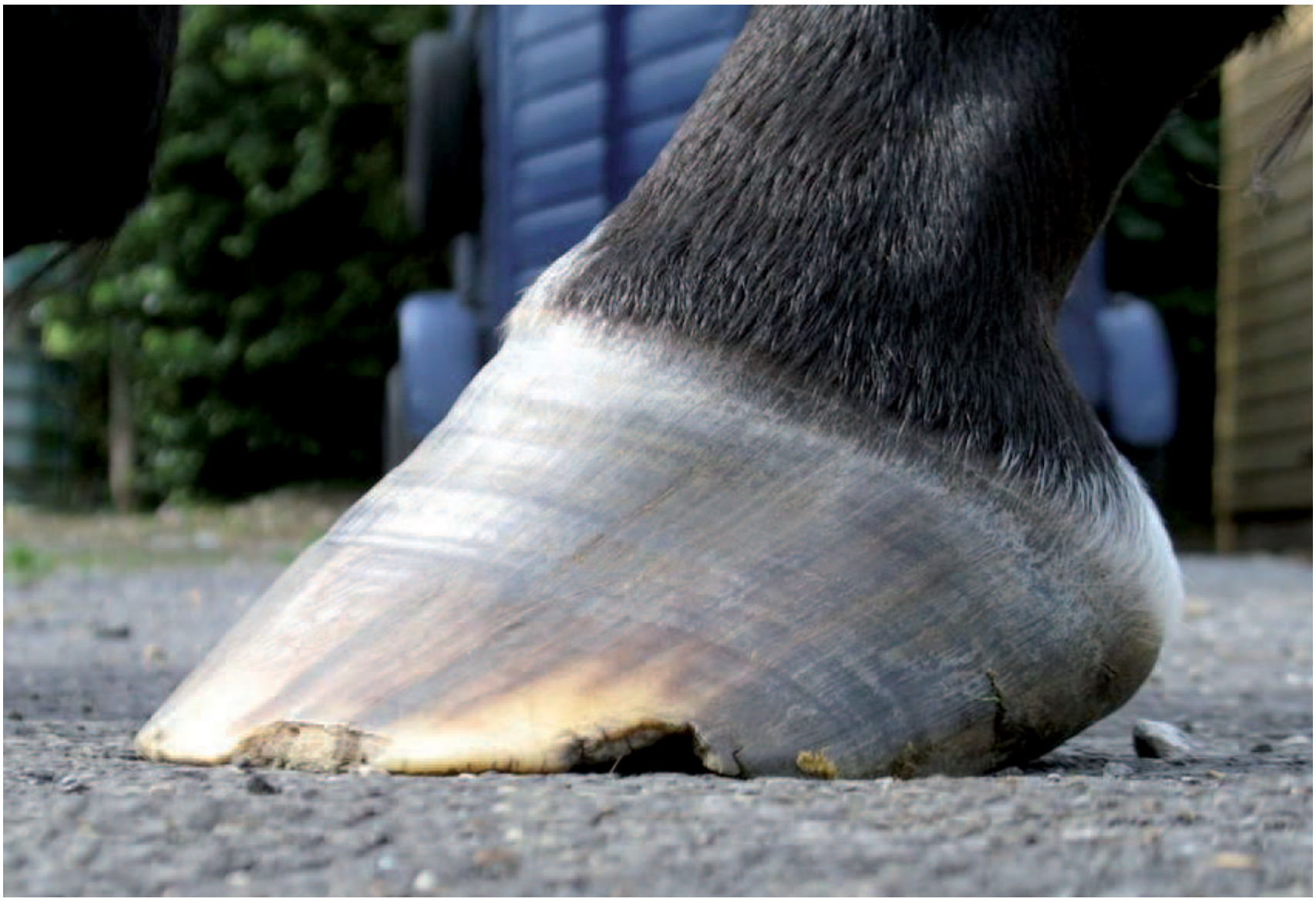


Fig. 47 An example of under-run heels associated with excessive heel growth but fairly strong laminae. The growth rings curl downwards and are spaced slightly further apart at the heel. The quarter flaring resulting from the under-run heels has been largely trimmed away, exposing inner wall in the quarters.

Where the laminae are weak – which is what you would expect in a typical laminitis case – the curling effect from the differential growth rate can pull the front of the hoof wall away from the bone by stretching and/or tearing the laminae. A redrawn version of Fig. 43 shows the approximate position of the pedal bone and the resulting stretch of the laminae to form laminar wedge (Fig. 48).

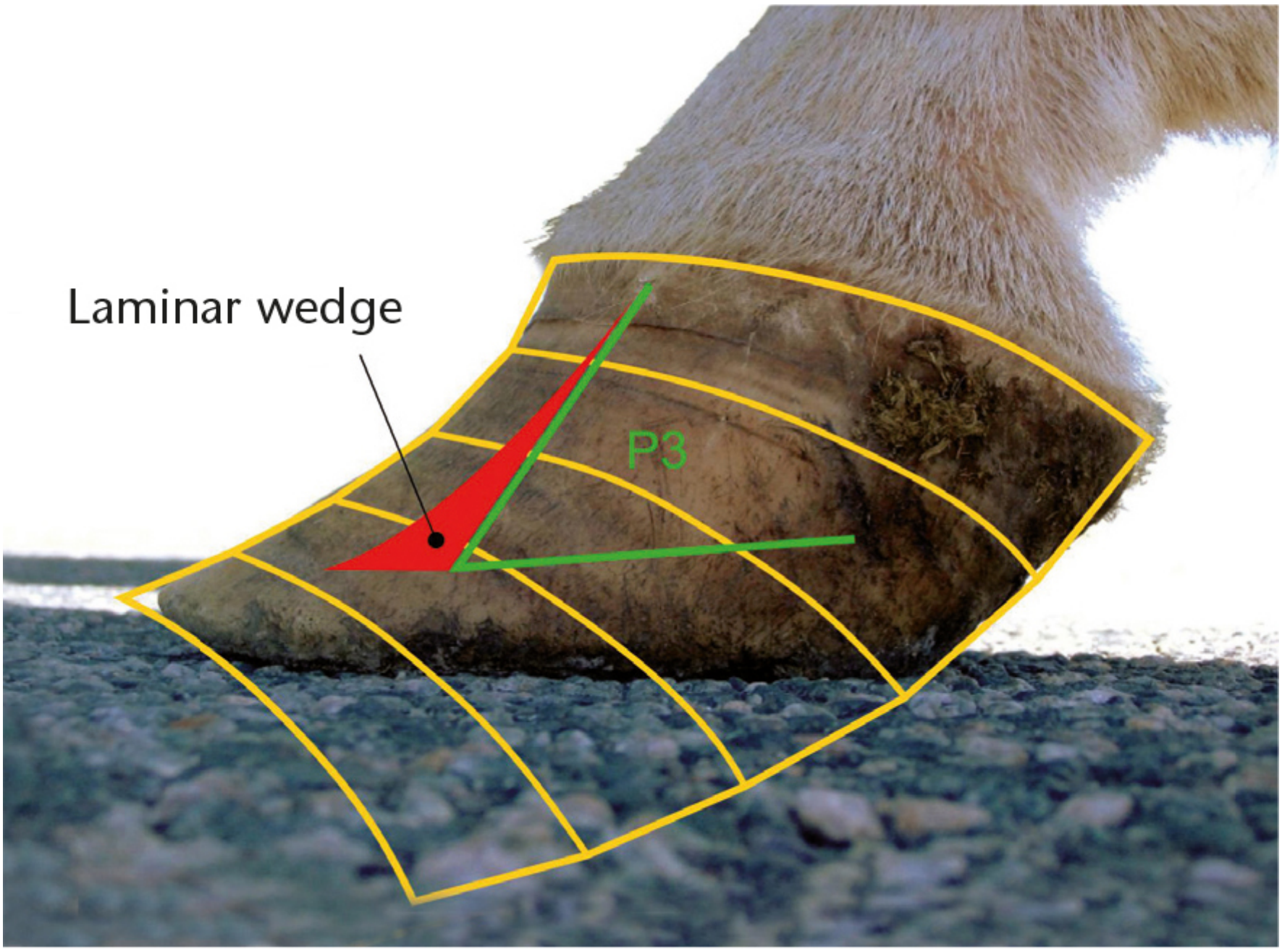


Fig. 48 Curling of the hoof capsule, with the position of the pedal bone and the formation of laminar wedge shown.

REFRAMING SINKER

Using the horse's frame of reference can help with understanding sinker as well as rotation. The laminae are traditionally seen as the most important structure in the foot for supporting the horse's weight. The pedal bone is often described as being suspended from the inside of the hoof wall by the laminae. In a shod horse there is a fair degree of truth in this assertion, although it is perhaps a touch simplistic. In an unshod foot with a correctly trimmed hoof, the rim of the sole (at least in the front half of the foot) is in contact with the ground and so the weight of the horse is supported by the solar, frog and coronary coria to a greater degree, with the laminae playing a significantly smaller role. Perhaps this bias in viewpoint reflects the fact that too much of the research into the equine foot is carried out on shod horses?

Healthy laminae are very good at resisting shearing forces – forces that try to slide the wall up or down past the bone. This allows the pedal bone in a healthy foot to be suspended from the inside of the hoof wall even where the sole is not contributing to supporting the horse's weight (as is the case in a shod horse). But laminitis (especially a more severe attack) weakens the laminae to the point where the wall and bone can slide past each other if enough force is applied.

In an unshod horse with the sole in contact with the ground the pedal bone cannot drop within the hoof capsule without first crushing the solar corium. In a very severe acute laminitis attack, this can happen – both the laminar and solar coria turn to pulp, and the pedal bone drops until it hits the sole, as discussed above. But in all but the most severe laminitis attacks, the sole will prevent the pedal bone from sinking.

If, however, the sole is not on the ground, either because of the thickness of a shoe or (in an unshod horse) because the hoof wall has been allowed to grow long, this solar support is less effective. Even

then, if the sole is thick and the white line strong, the sole will be well supported by the wall and will not bend, so providing support for the pedal bone. But if the sole is thin and hence flexible, and/or if the white line is weak and flexible, then the sole can flex downwards under the weight of the horse. In this situation, failure of the laminae due to laminitis can result in the pedal bone sinking within the hoof capsule (Fig. 49). It is likely to be for this reason that the pedal bone in shod horses appears to be more likely to sink as a result of laminitis than in well-trimmed unshod horses.

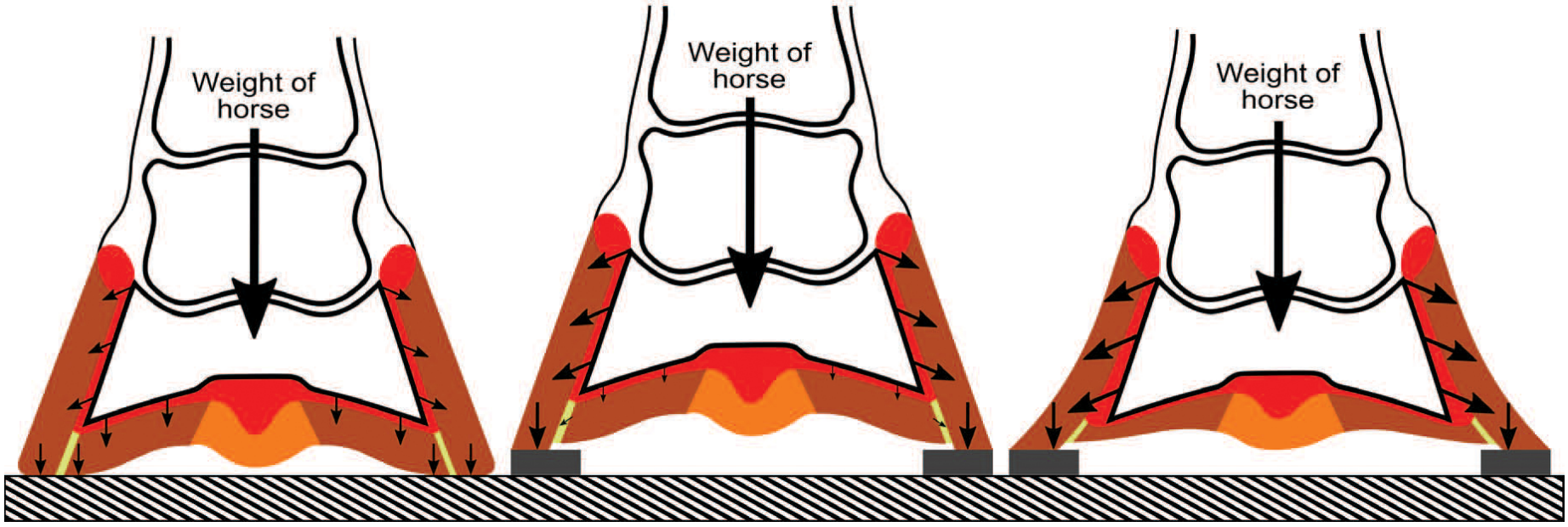


Fig. 49 Stylized representations of a section through the centre of the foot looking from the front and showing the distribution of forces through the hoof capsule as the horse's weight is supported. LEFT: ***An unshod horse with the weight supported equally via the coronary, laminar and solar coria.*** centre: ***A shod horse. More of the weight is supported by the laminar and coronary coria, but the stiffness of the sole and white line allows some weight to be supported via the solar corium.*** RIGHT: ***With a thin sole and a shoe fitted, support via the wall alone means that the sole can distort, providing little mechanical support for the foot. If the laminae become inflamed, the pedal bone can sink.***

As with rotation, the mainstream viewpoint uses the ground under the horse as the frame of reference. From this viewpoint, sinker is defined as when the pedal bone has dropped within the hoof capsule. However, from the horse's viewpoint the pedal bone is, as with rotation, where it should be, on the end of the leg, and it is the hoof capsule that has been shunted upwards in relation to the pedal bone. And, as with rotation, both viewpoints are equally correct but the horse-centred view can help with understanding the processes involved and the potential mechanisms for recovery.

In an initial acute laminitis attack, the hoof wall can only be shunted up the leg so far before the sole hits the bottom of the pedal bone (even allowing for the sole and white line distorting). It is not unheard of for the pedal bone to sink (or the hoof wall to be shunted upwards) as much as 15mm, so something else must happen for this to occur. The key to this process is the white line. The white line not only has to form a watertight and strong seal between the wall and the sole, it also has to accommodate different growth rates.

It is fairly easy to make rough estimates of the growth rate of the wall and sole. The wall at the toe typically takes a year to grow from hairline to ground (although this varies a lot depending on shoeing, workload and so on) – a distance of perhaps 10cm. This means that new wall is produced at a rate of around 10cm per year. In contrast the sole is typically around 1cm thick and takes around three months to grow down from corium to ground – a growth rate of around 4cm per year. Hence the white line has to join two parts of the hoof capsule that grow at dramatically different rates. It can only do this if it is capable of allowing the two structures to slide past each other, albeit at a very slow rate.

In sinker, the hoof wall gets shunted up the bone and can, over a period of days to weeks, continue to do so because the white line allows it to slide past the sole. This means that the distance from the bottom of the sole to the hairline at the toe actually gets longer. It is, for the most part at least, just the wall that gets shunted upwards, rather than the whole hoof capsule. The wall doesn't necessarily have to travel upwards with respect to the sole – it may just have to travel downwards more slowly than normal, breaking the balanced relationship between wall and sole growth.

With the laminae failing and the white line allowing the wall to slide in relation to the sole, the only structure left preventing the wall from being shunted upwards is the coronary band. Effectively the majority of the weight of the horse ends up being carried by the coronary band, and it just hasn't evolved to cope with those kinds of forces. The result is that the coronary band is sheared between the pedal bone and the hoof wall, and the normally oval coronary band becomes elongated and crushed. In bad cases, the coronary band may even be pulled out of the groove at the top of the pedal bone where it normally sits, stretching or tearing the ligament that holds it in place (Fig. 50).

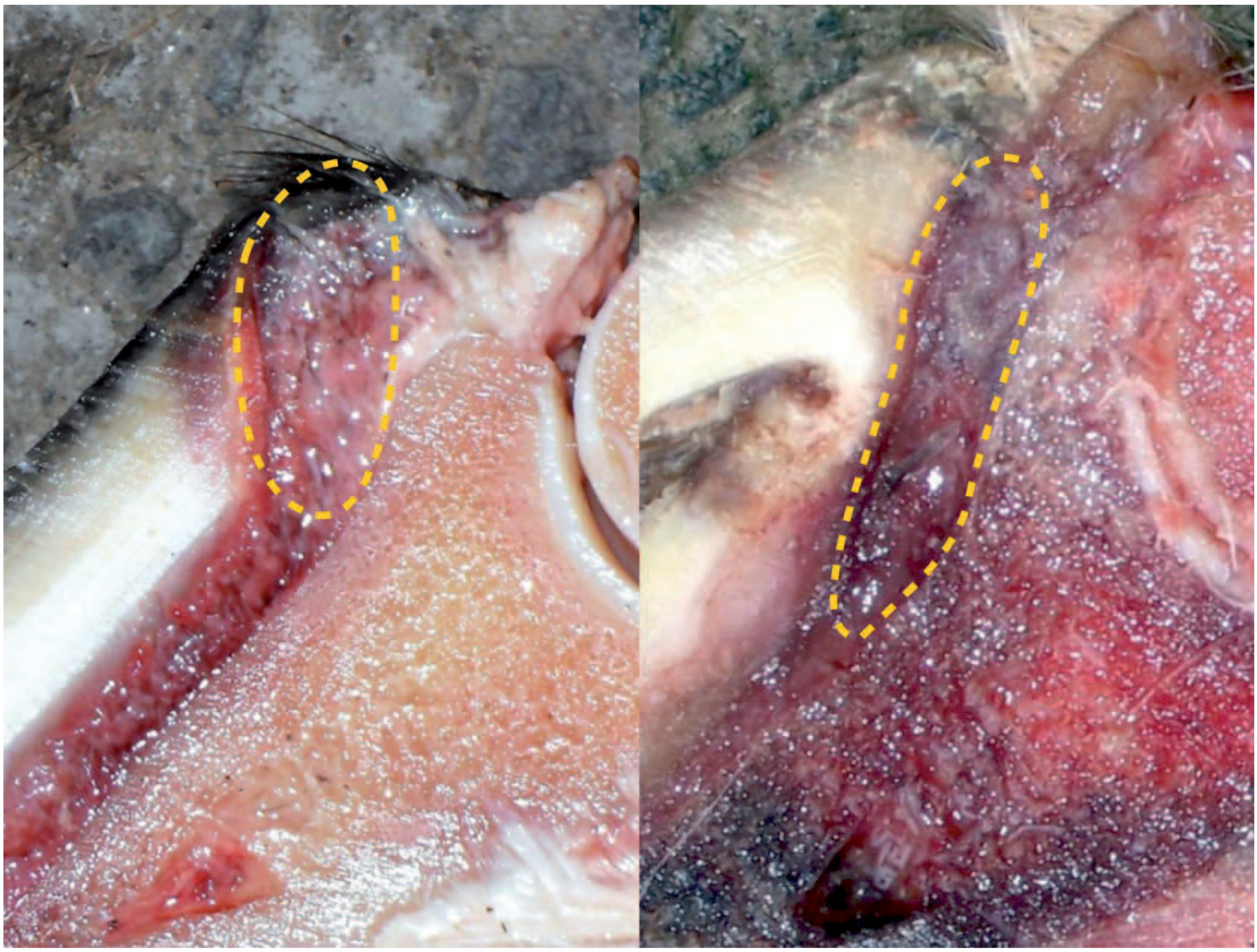


Fig. 50 Section through the coronary band of two laminitic feet (the approximate position of the coronary band is indicated). LEFT: ***Mild laminitis with no rotation or sinker.*** RIGHT: ***Severe laminitis with significant rotation and sinker.***

This crushing of the coronary band not only elongates it, but also re-orientates the surface that produces the hoof wall. The coronary papillae typically end up facing more horizontally, rather than down the front surface of the pedal bone. These papillae also end up spaced further apart as the coronary band stretches. Once this has happened, any hoof wall that is produced is thicker, with the horn tubules spaced further apart and oriented more horizontally. If the horse is able to recover from this scenario, the result is a marked step in the hoof wall.

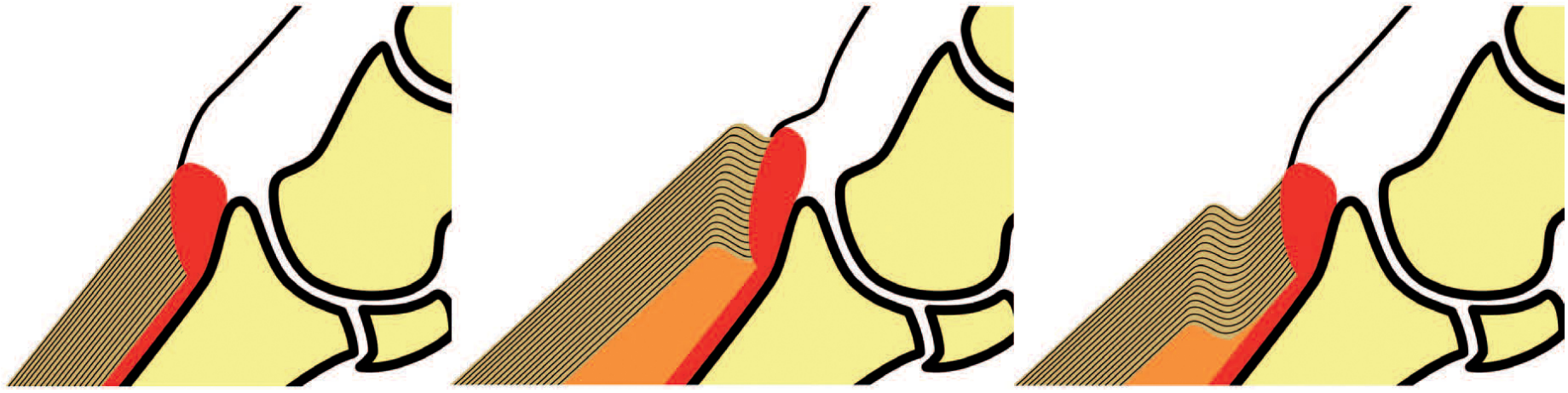


Fig. 51 A stylized view of the top of the hoof wall at various stages in a severe sinker case. LEFT: *A normal healthy foot.* CENTRE: *During sinker – this point can be reached in as little as two weeks. There is some rotation (laminar wedge is shown in orange) and the wall is shunted up the bone column. The coronary band is elongated and flattened, resulting in new wall production being horizontal with the tubules spaced further apart than normal.* RIGHT: *During recovery from sinker the coronary band has recovered its normal shape and new wall production is correctly oriented. The classic step appearance in the wall will be gradually pushed down the hoof by further wall growth.*

REFRAMING DROPPED SOLE

The same reframing techniques can also help in the understanding of dropped sole. It is very clear that, at least in some cases, the sole under the tip of P3 becomes artificially thick, as in the classic definition of dropped sole. However, when some cases are x-rayed, the degree of sole thickening isn't enough to explain the degree of dropped sole, suggesting that there is another effect coming into play.

Where significant chronic rotation happens over a fairly short period (within a few weeks), the front of the hoof wall migrates forwards with respect to the bone before it has time to grow much longer. This new orientation takes the wall off the ground at the toe. The effect is compounded if there is any degree of sinker present, which in a rapid rotation case, there usually is. Because shoes are applied to the bottom of the hoof wall, it is natural to attach any shoe to the wall in its new position. But now the shoe is no longer correctly aligned with the bottom of the pedal bone. The end result is that the shoe is actually applied further up the foot than it should be, and at the wrong angle to the pedal bone, leaving the sole directly under the tip of the pedal bone dropping through the shoe (Fig. 52). A good example of this can be seen in Fig. 53. However, not every case of dropped sole can be explained in this way: some are truly caused by a massive thickening of the sole under the tip of the pedal bone. The important thing is not automatically to attribute every apparently bulging sole to thickening of the solar horn. Of course this mistake can be avoided if best practice is followed and x-rays are taken before trimming or shoeing.

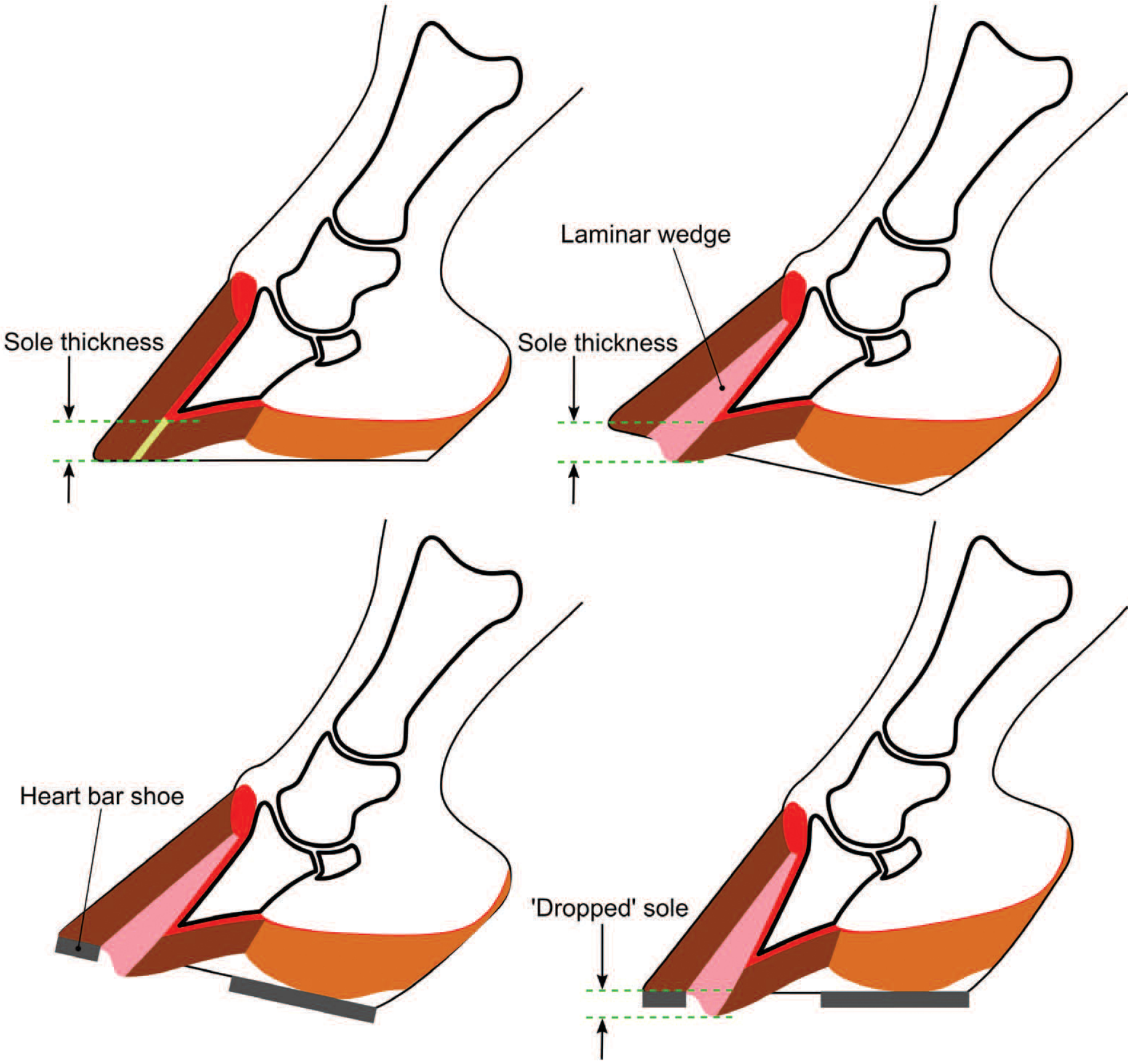


Fig. 52 The creation of 'dropped' sole. TOP LEFT: ***A normal foot.*** **top right:** ***After fairly rapid chronic rotation, the hoof capsule is rotated away from the pedal bone and shunted slightly upwards, and laminar wedge forms between the laminae and the hoof wall. The heels and frog grow rapidly, but the sole remains largely unchanged.*** BOTTOM LEFT: ***Application of a heartbar shoe to this foot without reference to x-rays results in the shoe being incorrectly placed.*** BOTTOM RIGHT: ***Reorientation of the shod foot to the ground creates the appearance of dropped sole.***

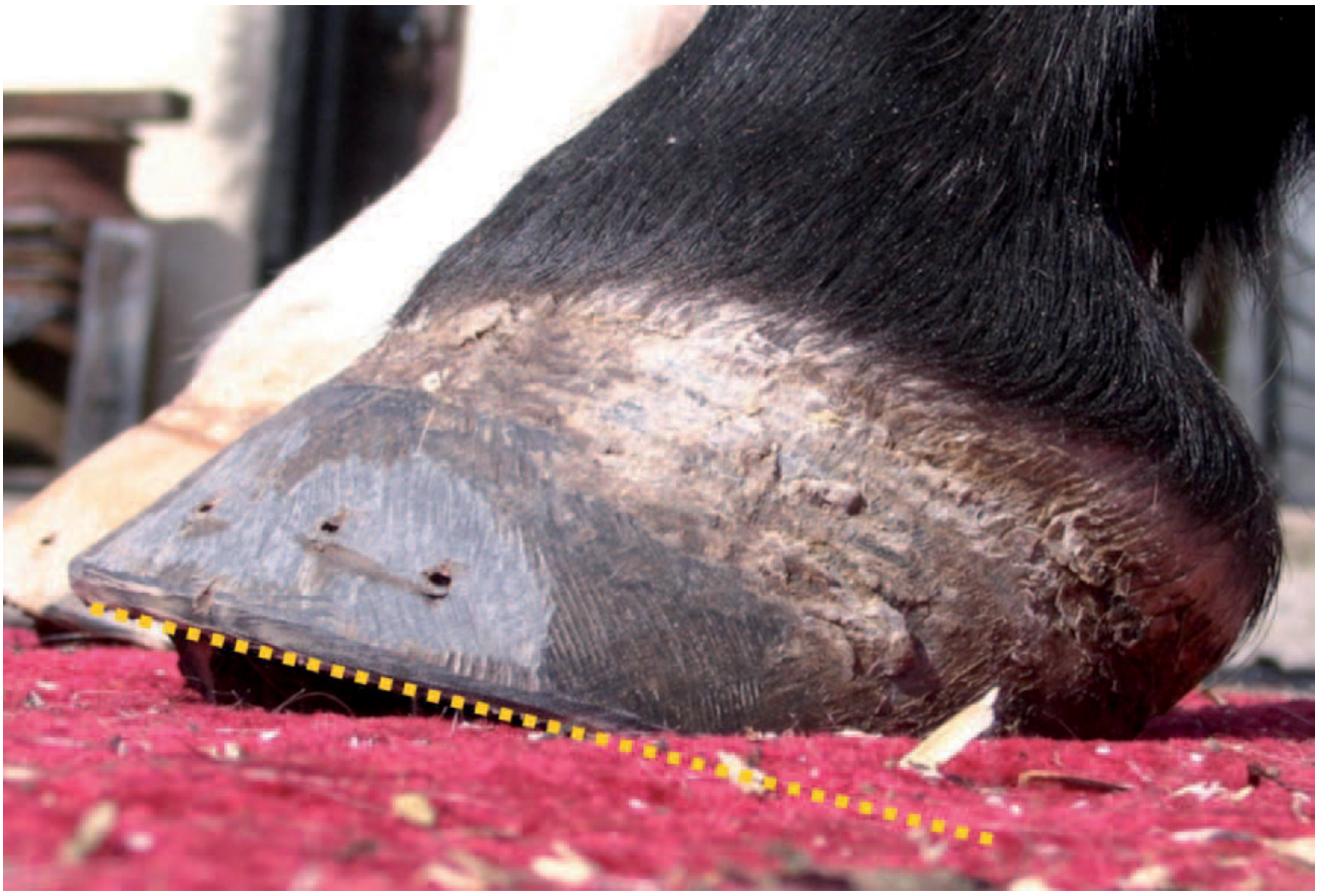


Fig. 53 A case of dropped sole with the shoe removed (the original position is shown) and the foot trimmed (with reference to x-rays) to bring the pedal bone into the correct orientation with the ground. In this case the sole was close to normal thickness but was clearly below the bearing surface of the original shoe. It would not be possible to shoe this foot conventionally and achieve correct balance because there is insufficient wall at the toe to which to attach the shoe.

THE RELEVANCE OF THE DEEP FLEXOR TENDON

As we saw in Chapter 2, anything that causes tension in the deep flexor tendon will tend to increase the rate of rotation by pulling the pedal bone away from the front wall of the hoof capsule. This effect is used to justify raising the heels either by trimming less off the heels, or by adding a wedge between the hoof and the shoe, supposedly to reduce the tension in the tendon and hence reduce rotation. However, this model of rotation ignores one very important factor: the involvement of the central nervous system.

The deep digital flexor tendon is attached to the deep digital flexor muscle, and that muscle is under the control of the brain. Except in some very specialist circumstances, tension in the deep digital flexor tendon can only result from a conscious or unconscious decision to tense the associated muscle. If this tension in the DDFT results in the laminae being pulled apart, then that in turn will result in pain messages being sent to the brain. The brain will then do what brains do in such situations: it will make every attempt to modify the use of muscles so as to reduce the pain. In this case, that means relaxing the deep digital flexor muscle.

Watching how a horse with laminitis moves, it is possible to see the modifications in gait that the horse makes to avoid pulling on the DDFT. As we saw in Chapter 1, the DDFT is normally tensed in two situations. The first is when the foot is flexed upwards out of the way during limb flight. In this situation there is no ground involved to create a resistance to the action of the DDFT, so the use of the DDFT will be normal and not associated with any pain. The other use is at breakover as part of the mechanism that propels the horse forwards. In this case, the tendon is used to pull the hoof capsule round, creating breakover and providing resistance at the toe, against which the horse can push to propel itself forwards.

If a laminitic horse tenses the DDFT during breakover, it experiences pain and risks creating further damage to the laminae. So instead, the horse avoids pushing itself forwards with the toes by breaking over early, just as the knee passes over the foot (rather than when the knee is well ahead of the foot, which puts the leg at a suitable angle to push forwards). With little or no propulsion coming from the front feet, the horse can only move itself forwards by pushing with the hind feet. The result is the classic stilted walk seen in severe laminitis cases. If the hind feet are also badly affected, the horse has an even bigger problem moving, and may just refuse to move at all. If it does move, it will barely shuffle, minimizing propulsion as far as possible so as to minimize tension in the DDFT.

Even when standing still, laminitic horses modify their posture to relieve tension in the DDFT. They do so by tensing the extensor tendon instead, standing with the toes almost lifted off the ground and the DDFT fully relaxed. The classic laminitis stance with the front feet placed further forward than normal also acts to relax the DDFT.

The only exceptions to this process come where there is a physical reason why the horse cannot let go of the DDFT. The first exception, as we have already seen, is where the hoof is so long that the heels are forward of the centre of the coffin joint (Fig. 45). In this situation, the horse can choose to relax the DDFT, but this results in the horse walking on the heel bulbs.

The second exception is where the DDFT is abnormal. Because horses with chronic laminitis tend to grow more heel than normal, the heels often become higher and higher over time compared to the toe. If this excess heel height isn't regularly removed, the pedal bone ends up in a more upright position long term. This in turn can result in the DDFT and its associated muscle shortening (the same effect can be seen in humans who wear high heels a lot). In this situation, trimming the foot back to a more normal balance may result in the DDFT being unable to relax fully because it is too short. For that reason, it is critically important to check tendon length in a laminitic before removing large amounts of heel height. Failure to do so may result in a dramatic worsening of the mechanical damage caused by the laminitis, and in some cases, more permanent damage to the DDFT. The DDFT can be gradually stretched back to its original length using either physiotherapy or by lowering the heels gradually, but this cannot be done safely whilst the laminae are inflamed because any attempt to stretch the DDFT will pull on the laminae.

More permanent damage to the DDFT may cause even more serious problems. For example, injury to the DDFT can result in adhesions forming between the tendon and its enclosing tendon sheath. Such adhesions then restrict the range of movement in the tendon, typically resulting in a condition called 'club foot' where the heels are permanently too high. If laminitis occurs in a horse with club foot, then keeping the heels high is necessary to prevent tension in the DDFT. Such damage to the DDFT is generally irreversible, and attempts to stretch the tendon will not only worsen the damage caused by any active laminitis, but will tend to strengthen the adhesions between the tendon and sheath. This possibility makes it even more imperative to assess the tendons correctly before lowering the heels of a laminitic horse. Not only must a short tendon be identified, but the two possible causes (reversible shortening or irreversible mechanical obstruction) need to be differentiated.

With the exception of the horse with damaged or shortened tendons, raising the heels in a laminitis case serves no useful purpose. Indeed it can be extremely counterproductive. With the pedal bone in its normal orientation to the ground, the weight of the horse is distributed over the large surface area of the bottom of the pedal bone. However, once the pedal bone is tipped forwards, the weight is shifted on to the tip of the pedal bone, which is a fairly sharp edge. With the pedal bone tipped forwards, this sharp edge is moved directly over the top of the circumflex artery of the sole. The result is that the pedal bone forms a chisel shape and can very effectively cut through or crush the circumflex artery, resulting in sub-solar haematomas, loss of sole growth and, ultimately, solar penetration. Laminitics with high heels (either due to being artificially raised, or that have ended up this way as a result of excess heel growth) often show immediate and fairly dramatic relief from pain once the pedal bone is returned to its normal orientation with the ground. Even a quite small reduction in heel height can, in some cases, bring dramatic relief from pain.

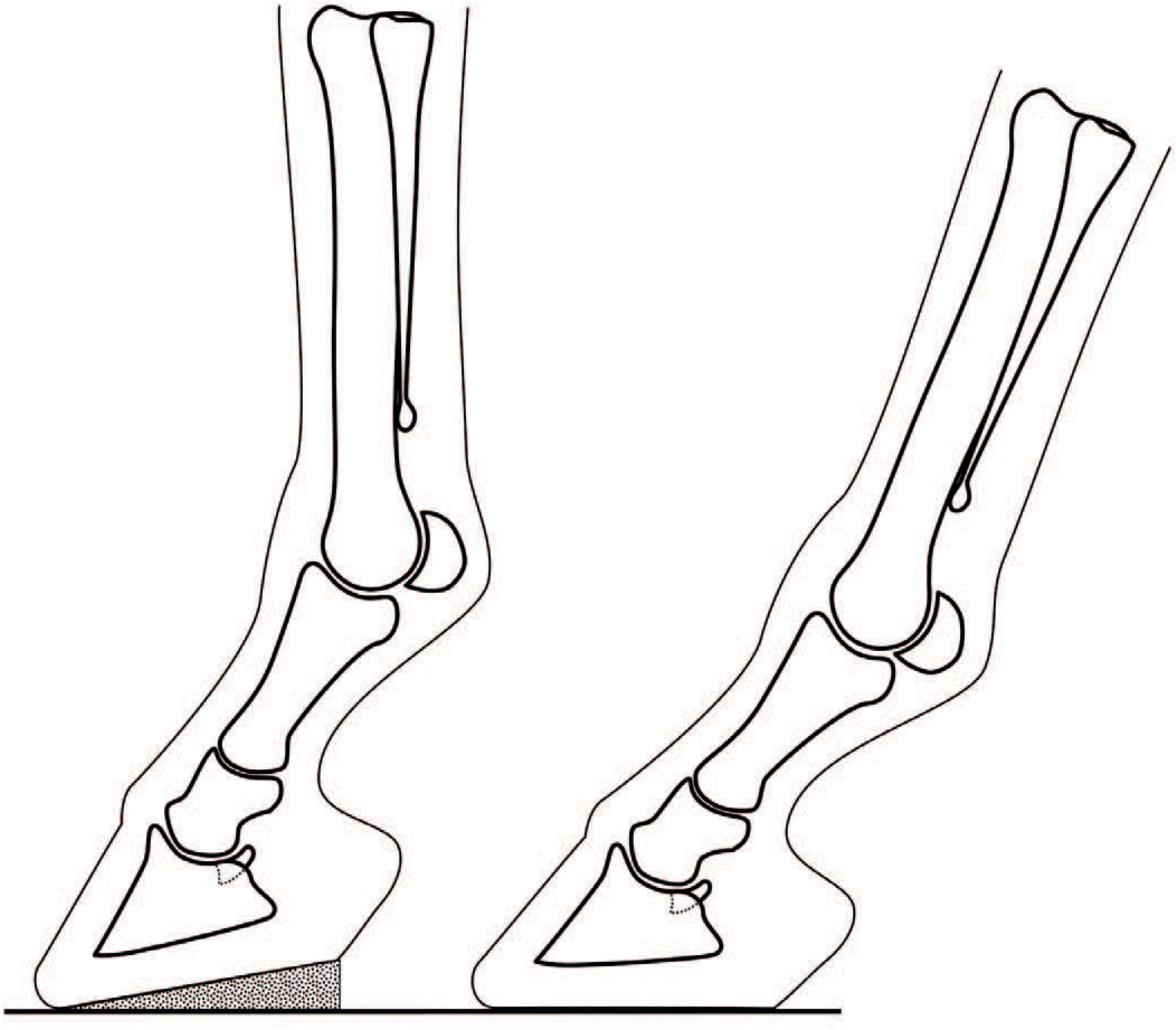


Fig. 54 LEFT: ***Heels raised by 10 degrees using a heel wedge (a common approach in a severe laminitis case). The coffin joint is flexed by 10 degrees from its neutral load-bearing position, reducing tension in the DDFT, but the fetlock joint is in its neutral position.*** RIGHT: ***A horse standing in a stance typical in severe laminitis. The coffin joint is again flexed by 10 degrees from its neutral position, but in addition the fetlock joint is flexed 10 degrees from its neutral position, providing additional release of tension in the DDFT. The natural response of the horse is therefore as, or more, effective at releasing tension in the DDFT as the heel wedge, and the horse has the ability to control this so as to minimize pain and hence damage. In addition, the heel wedge rotates P3 such that its tip is oriented downwards, creating undesirable point pressure on the solar corium and solar circumflex artery.***

UNDERSTANDING ABSCESSES

The hoof capsule normally forms a watertight seal around the live structures of the foot. Where this seal fails, either from a puncture wound or from laminitis damaging the white line, bacteria (and potentially other infections such as fungi) can penetrate the hoof capsule and infect the various coria. In an otherwise healthy corium, low levels of infection are efficiently dealt with by the immune system and are typically asymptomatic. Very high rates of infection are more of a problem. However, the most likely cause of an abscess is where the ingress of bacteria meets damaged corium. This damage might be caused directly by a puncture wound, or it might be the result of laminitis. Where infection meets damaged corium, the bacteria can take hold rapidly and the immune system is less able to deal with

this because of compromised circulation. The result is a runaway infection that, if left unchecked, could spread into the bloodstream and cause septicaemia and, potentially, death.

Like all mammals, the horse has a defence mechanism against rampant infection, and this involves partially or totally shutting off the blood supply to the affected area, effectively encapsulating the infection and preventing it from spreading via the bloodstream. This saves the horse's life, but effectively sacrifices the infected area of tissue. The infection will then run its course until it either runs out of tissue to feed on, or, more likely, waste products from the bacteria build up to the point that the area becomes too toxic for the bacteria to survive. The result is pus – a mixture of dead bacteria and dead tissue. If the abscess is close to the surface of the body, it will typically pop, draining the pus and allowing the immune system to move in and clear up the damage. Where that is not possible and the immune system is unable to dispose of the pus (and particularly if there is any form of foreign body present), the body will build a membrane around the infected area, so permanently encapsulating it in the form of a cyst.

All these mechanisms work in the hoof, but there are some added complications. In the wild, a foot abscess is a serious problem for a prey animal that needs to be able to flee predators (much more so than an abscess elsewhere on the body). The horse needs to get rid of the abscess as fast as possible, preferably leaving the hoof as functional as possible in the aftermath. The horse has evolved a useful mechanism for achieving these goals. One by-product of an abscess is that the bacteria produce gas, the pressure from which gradually builds up. In normal skin, this causes the area to bulge, but in the hoof capsule, the abscess (which usually starts in the solar corium) is sandwiched between bone and horn – there is no way the corium can expand, so the pressure builds up. The rising pressure causes pain but also presses on the adjacent corium, preventing it from producing horn and ultimately separating the horn from the corium. This means that the abscess can track along the corium, preventing new horn production and so opening up a void between the corium and the horn, which then fills with pus.

In the case of a puncture wound, the abscess will typically blow back out of the puncture hole, but in all other abscesses, the void will track along the sole to the white line. If the white line is sufficiently damaged, the pus can then blow out there. If not, the abscess will continue to track up the laminae, across the coronary corium and out at the hairline. Once the void reaches the outside world, the pus is expelled under what is now a fairly high degree of pressure, resulting in much of the pus being ejected. As the horse continues to move around, mechanical pressure helps to pump the majority of the remaining pus out of the exit hole over the following few days.

Once the pressure has been relieved, the horse generally looks much happier and the various affected areas of corium can start to produce new horn again (usually fairly rapidly as a response to the damage). In the sole, this results in the corium being sealed by a layer of new horn, and the abscess track being pushed out into the solar horn, where it gradually works its way to the surface over the following two to three months. Despite the abscess track buried within it, the sole is still the normal thickness in total and so functions normally, providing plenty of protection from bruising. The laminar corium, which normally produces virtually no horn, will suddenly produce a significant amount for a few days after the abscess has blown. This pushes the hoof wall outwards somewhat, sealing the pus-filled track away from the laminar corium. It also appears that in most cases this horn is able to flow back in amongst the epidermal laminae that were separated away from the corium as the abscess tracked past, effectively reattaching the laminar corium to the inside of the hoof wall (albeit with a slight bulge in the wall as a result of the temporary burst of horn production). So not only is the wall now glued back together, but the exit track at the coronary band is also backed with new horn and so effectively sealed from the various coria. That track will now grow down the hoof wall with normal wall growth, and eventually disappear off the bottom of the wall.

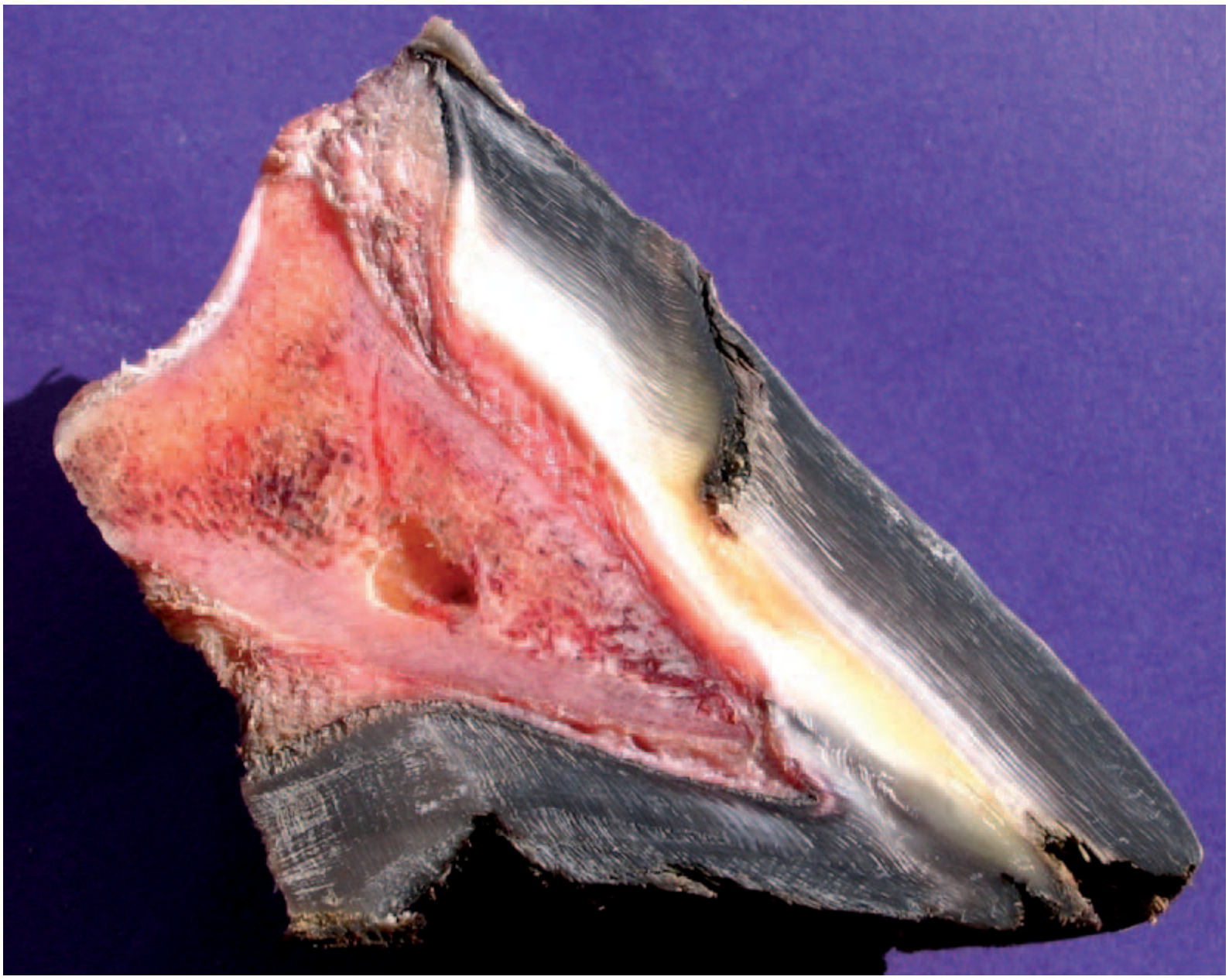


Fig. 55 The anatomy of an abscess. This section through the centre of the toe shows a foot that has abscessed at the toe around five months earlier. The part of the abscess track in the sole has already grown out. The exit track, which would originally have been at the top of the wall, has grown down. Below this, additional horn (stained yellow by blood products from the aftermath of the abscess) can be seen. This was produced by the laminae so as to reattach the hoof wall to the laminar corium and seal the corium away from the exit track.

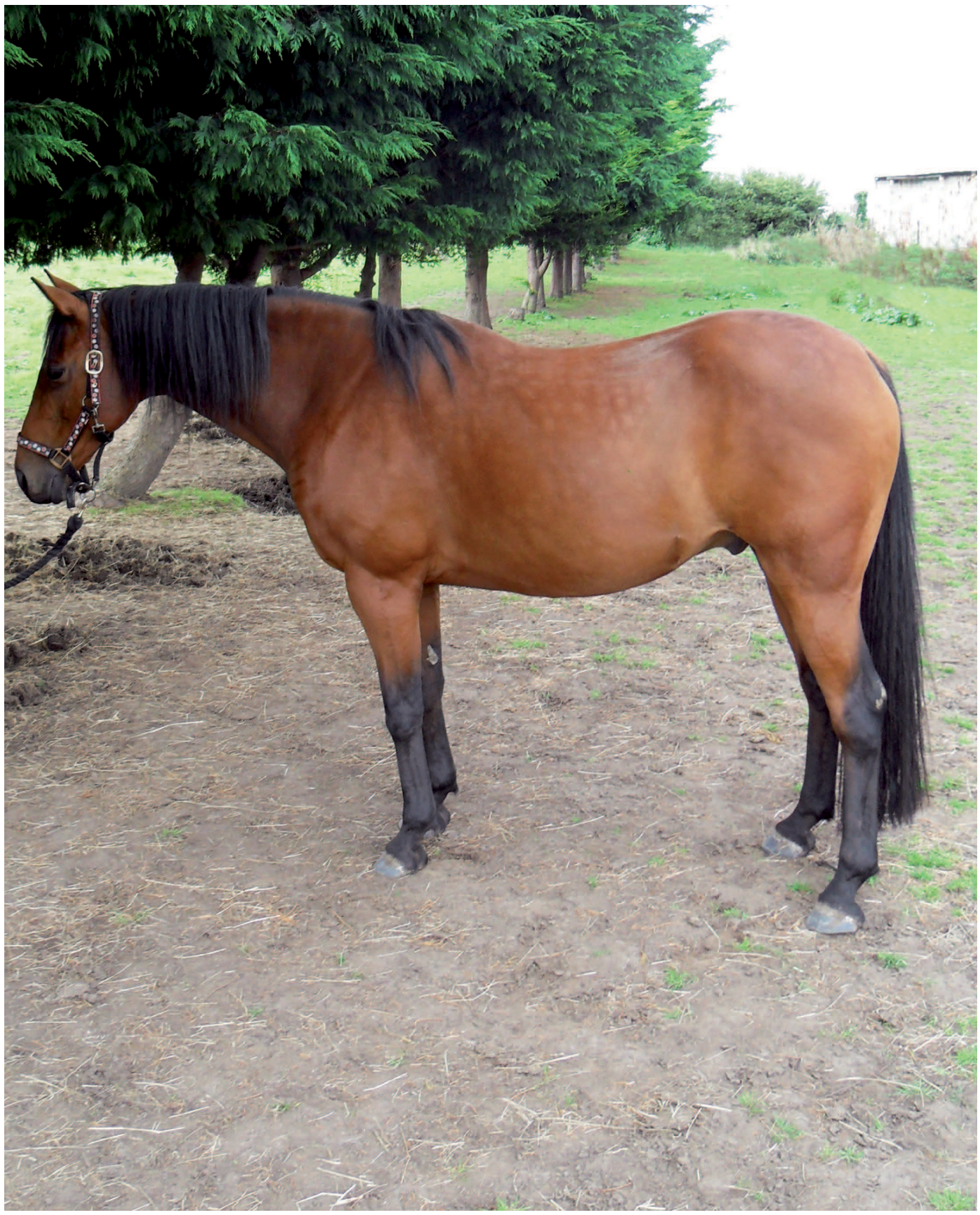
The combined result of these mechanisms is that, within a few days of the abscess blowing, the various affected coria are covered in new horn and so are sealed away from both the outside world and any remaining infection within the abscess track. Not only that, but the hoof capsule has recovered sufficient mechanical integrity to be able to function fairly normally, allowing the horse to keep up with the herd and run away from predators.

4 Low-Grade Laminitis

Laminitis has traditionally been seen as a binary event – a horse is either laminitic or it isn't. This makes sense in an acute laminitis case: the horse is fine one day but the next it is rocking back, sweating and shuffling from foot to foot. But with a milder, chronic laminitis case, it's harder to make the distinction between days when the horse is laminitic and days when it isn't.

It is clear that laminitis can vary from very slight to extreme. If the inflammatory event that we call laminitis can vary in severity, is it possible for there to be a level of inflammation that is insufficient to cause overt lameness? What would that look like? Could we even tell? These are questions that the author and his colleagues in the Equine Podiatry Association started to ask in 2006.

A clue to the answers came from the analysis of a series of photographs of one of the author's horse's feet, taken fortnightly over a four-year period. This horse showed a number of foot pathologies (flaring, quarter cracks, thrush, white line disease and suchlike) that varied seasonally. Traditional thinking would ascribe these pathologies to poor quality horn resulting from the direct effects of wet weather on the hooves. And yet the sequence of photos appeared to show the feet consistently improving over winter. In contrast, things consistently got worse both in spring and autumn – precisely the times of year when laminitis is typically most prevalent.



A horse standing ‘camped under’ as a result of mild toe discomfort caused by low-grade laminitis. The front canon bones are around 8° behind the normal vertical position. This posture is frequently misattributed to a conformation defect.

This one case raised the question of whether a range of pathologies that are not traditionally associated with laminitis might follow the same seasonal pattern as laminitis. As other EPA members started to look for this pattern in their own cases, it soon became clear that it was consistently observed over a large number of horses. With a group of horses identified as showing the same pattern, the next challenge was to try to determine whether these effects were the result of laminitis or some other seasonal process. EPA members started to use dietary interventions already known to help laminitis cases on this group of horses. Within months, it started to become clear that the same dietary interventions that helped in traditional laminitis cases also helped with the foot pathologies in this new group of horses.

Vets have long talked about ‘sub-clinical laminitis’ – the period during an acute laminitis attack where changes are happening in the foot but the horse has yet to go lame. It was initially suggested that what was being observed was sub-clinical laminitis. But central to the sub-clinical definition is that the horse shows no clinically observable signs of laminitis, whereas EPA members were very definitely seeing observable clinical evidence associated with what was now believed to be a very mild form of laminitis. A label was needed for this new phenomenon, and the term ‘low-grade laminitis’ was coined.

It turns out that this term is not ideal for describing the condition (as we'll see later in this chapter), but it has gained traction and we are probably stuck with it for the foreseeable future.

Subsequent work by the author and other EPA members has refined our understanding of low-grade laminitis (or LGL) over a number of years. It is now largely accepted by the majority of barefoot trimmers in the UK and abroad, and is starting to be discussed by vets and farriers.

DEFINITION OF LGL

Low-grade laminitis is defined as laminitis at a level that is sufficient to create observable clinical signs – such as changes in stance and gait, as well as changes in the structure and shape of the hoof capsule – but is insufficient to cause rotation or sinker (with the associated formation of laminar wedge) in the foot. This definition neatly splits laminitis into two camps: low-grade laminitis, where there is no rotation/ sinker, and full-blown laminitis where there is rotation/sinker. Of course in real life there tends to be a grey area between the two extremes.

As with full-blown laminitis, low-grade laminitis can be acute or chronic. Acute LGL tends to be short-lived (a few days at most) and causes significant damage to the hoof capsule, often accompanied by short-term lameness (most commonly in the form of tender footedness). Chronic LGL tends to be the result of repeated, smaller acute attacks, which individually might be difficult to spot, but cumulatively result in significant damage to the hoof capsule. Chronic LGL cases are often less obviously lame, but may still show tender footedness. Where repeated larger attacks occur (similar in strength to acute LGL attacks), this tends to lead to sufficient cumulative damage for rotation and/or sinker to occur, and hence this would no longer be classed as LGL.

LGL AFFECTS THE ENTIRE FOOT

The traditional view of full-blown laminitis suggests that it is the laminae that are primarily inflamed, and that other structures only become inflamed because mechanical failure of the laminae causes increased pressure to be placed on those structures. However, it is very clear in LGL that *all* the coria in the foot are affected. Given that the definition of LGL precludes the mechanical failure of the laminae, this cannot be a secondary effect of inflamed laminae. At first sight, this appears to be a significant difference between LGL and full-blown laminitis, which might suggest that they are two fundamentally different conditions. However, there is another possibility: that all the coria are affected in full-blown laminitis as well, but that this has been incorrectly attributed to secondary effects because the most obvious damage is seen in the laminae.

In full-blown laminitis, the mechanisms involved in rotation tend to result in the most severe and obvious damage occurring in the laminar corium, with the other coria being less affected. It is easy to see how this has resulted in attention being focused on the laminae. Once the presence of more systemic inflammation in LGL suggests the possibility that this may be true in full-blown laminitis as well, the arguments for secondary damage to the solar and coronary coria are rather easily demolished. As we saw in Chapter 1, the equine foot has evolved a high degree of redundancy of structure. We know from the way horses recover from injuries resulting in the loss of a large part of the hoof wall (*see* Fig. 15 in Chapter 1) that this does not result in secondary damage to the solar coria. In such cases there is no wall on the ground, and hence the laminar corium cannot be weight bearing, and neither can the coronary corium. The entire weight is adequately supported by the solar corium without damage. If the solar corium were not inflamed as a primary component of full-blown laminitis, then the failure of the laminae would merely result in a shift of weight from the laminar corium to the solar corium, and the solar corium would be more than able to fulfil this role. If, however, the solar corium *is* affected by the underlying disease processes, then this more than adequately explains why catastrophic damage often occurs in this corium as well, particularly where failure of the laminae results in increased pressure on the solar corium. Once the involvement of the solar corium in the underlying disease processes of

laminitis is accepted, it is no great leap to recognize the possibility that the other coria might also be affected.

Whilst all the coria are affected in LGL (and, it would seem, in full-blown laminitis also), they are not necessarily all equally affected. Any number of factors may influence how inflamed each corium becomes in any individual case. These factors may include genetics (different breeds tend to have subtly different responses to the same underlying problem); conformation; the current presence of shoes (which of course modify the distribution of forces in the foot); the history of shoeing or other forms of hoofcare, and the effect this has had on the shape, quality and balance of the foot; and the history of any past injuries or previous laminitis attacks that may have weakened the internal structures of the foot. As a result of this, the presenting signs of LGL (and full-blown laminitis, for that matter) vary significantly from horse to horse. For example, an unshod horse where the sole is badly affected but other coria less so, may show only minor changes in the shape of the hoof capsule but is likely to be sore on hard or uneven ground. In contrast, an unshod horse where the sole is largely unaffected but the laminar and coronary coria are badly affected will tend to have much more visible defects in the hoof, and yet may be more or less sound.

IMMEDIATE SIGNS OF LGL

The clinical signs of LGL are divided into two categories. The immediate signs are those that are present at the time the horse is actively laminitic. The delayed signs are those that show up as changes in the hoof capsule, which can be observed in the weeks and months after the actual LGL attack. The following sections describe the immediate signs.

Raised Pulses

During an LGL attack, the foot is inflamed. It follows that you would expect raised digital pulses, and this is indeed the case. However, the strength of pulse in an LGL case is rarely as high as that in a full-blown laminitis case. The degree of lasting damage inside the foot is often low enough that the raised pulses disappear again within minutes or hours of the underlying cause of the laminitis going away. Given that laminitis often comes and goes hour by hour (for example, in response to changes in grass sugar levels, which in turn are a response to the time of day, the amount of sunshine and so on), it is not uncommon for an LGL case to have raised pulses that only show intermittently. One of the author's horses only had raised pulses twice in a one-month period of taking them twice daily, and yet that was enough for him to show varying degrees of tender footedness.

Heat

As with full-blown laminitis, LGL causes the foot to be hotter than normal – just not as dramatically so. As a result, the increased heat is usually only detectable in more severe LGL cases.

Tender Footedness

An inflamed foot is likely to be a tender foot. However, it is important to recognize that other factors can affect how tender a foot is. Clearly a horse with a thin sole is going to feel stones more than one with a thick sole. So when looking for signs of LGL, what you are looking for is a horse that is more tender footed than would be expected, given the thickness of the sole. This might be a horse with a 10mm thick sole that is feeling large stones, or a horse with a 5mm thick sole that is sore even on smooth tarmac. That said, a horse with a less-than-ideal sole thickness will (at least in the author's experience) often prove fairly confident over stony ground if any underlying LGL is entirely eradicated, so long as the sole isn't really thin.

Swollen Coronary Band

It is not uncommon to find the coronary band swollen and puffy during an active LGL attack. This is particularly true in the worst acute attacks. This is consistent with the suggestion that all the coria are affected by the underlying inflammatory event in LGL.

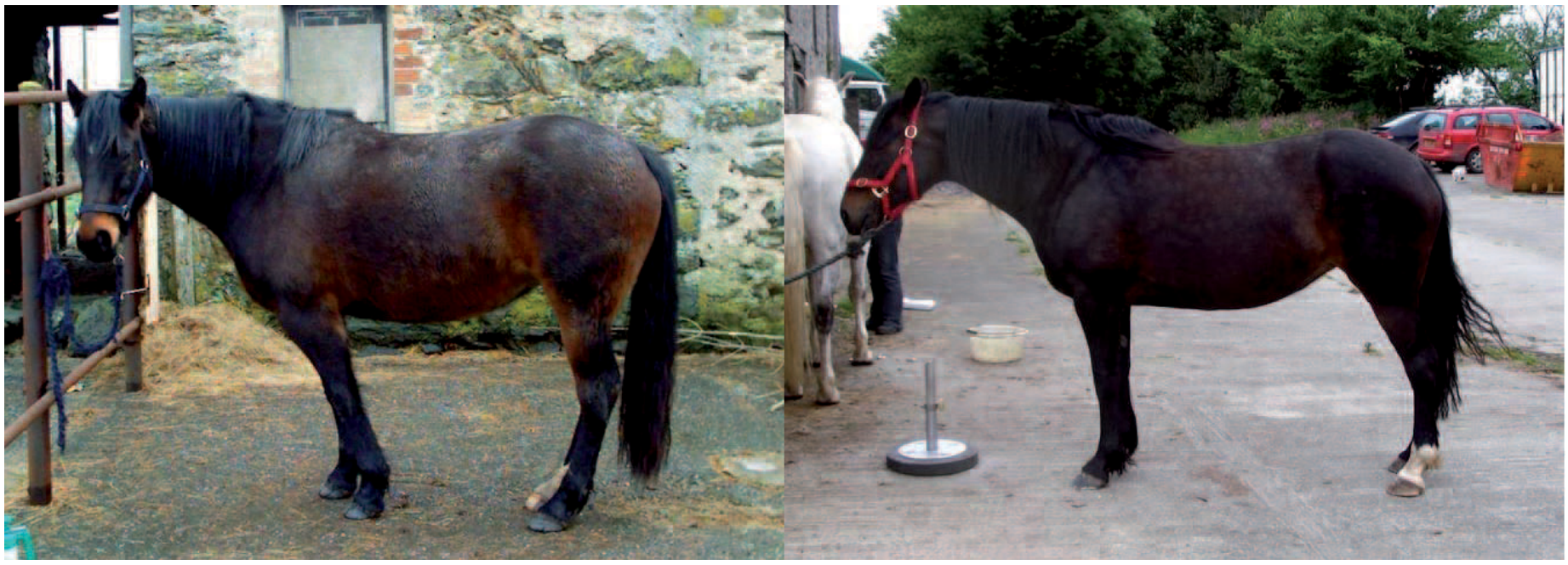


Fig. 56 LEFT: *A horse showing an abnormal stance as a result of LGL.* RIGHT: *The same horse at a later date after a successful diet change resulted in a dramatic reduction in the severity of the LGL.*

Altered Stance

A horse with sore feet is going to try to find a way to stand that minimizes that pain. However, unlike full-blown laminitis cases, LGL cases typically do not show the classic rocked-back stance. A healthy horse will, on average over time, stand with all four cannon bones vertical, as this requires the least muscular effort. In contrast, the typical LGL case will (assuming that the focus of discomfort is in the front feet) shift weight from the front feet towards the hind feet. The horse can achieve this by placing the hind feet slightly further forward than normal, bringing the hind cannon bones away from the vertical. This has the effect of shifting the centre of gravity closer to the hind feet.

In contrast to the full-blown laminitis case which tends to place the front feet forward of their normal position, an LGL case often does the exact opposite and places the front feet further backwards. Whilst there is as yet no proven reason for this, the suggestion is that it may reduce circulation in the toe by weighting the toe more than the heel. This is similar to a human who has just hammered a thumb, squeezing it to reduce throbbing.

The combination of the front feet placed further backwards and the hinds further forwards creates a stance that is variously described as the ‘elephant on a drum’ or ‘goat on a rock’ stance. It is important to recognize that horses who are mooching around may end up standing in all sorts of weird poses – what you are looking for here is a clear bias towards standing with the fronts and hinds closer together than normal. Horses that consistently show this stance almost always turn out to have sore front feet, and correcting the underlying cause of the soreness usually results in a fairly rapid return to a more normal stance.

It should be said that the exact stance adopted by an LGL case depends on the relative severity of the problem between the front and hind feet. As a result, the exact abnormality of stance can be quite variable. As with full-blown laminitis, it is most common to see the front feet significantly more affected than the hinds, but the opposite case does sometimes present.

As a result of habitually standing in an abnormal position, many LGL cases develop a range of associated soft tissue problems, most commonly in the form of muscle tension. The key areas where muscle tension is likely to be found are either side of the lumbar spine, the buttocks and the inside of the thighs. In long-standing cases these muscles are often over-developed. This is easiest to see on the buttocks (the semitendinosus muscles tend to develop a pointy appearance rather than a round

one), but it can sometimes also be seen as raised lumps of muscle on either side of the lumbar spine. The affected muscles often feel very hard to the touch (though be careful, as the horse may not appreciate these muscles being touched, especially on the inner thigh). A good body worker can often make such a horse far more comfortable, but these muscle problems will recur if the sore feet are not addressed.

Another area that is commonly affected is the stifle joint. This joint is designed to lock (by the patella – the same bone as our kneecap – moving across the end of the femur to block movement of the stifle joint) to allow the horse to sleep standing up. The correct locking and unlocking of the stifle is at least in part dependent on the balance between the muscles behind and in front of the hind leg. Where the muscles behind the leg become overdeveloped in comparison with those in front, there is an increased tendency for the stifle joint to lock accidentally, resulting in a clicking or even locking stifle. Stifle problems in LGL cases often improve once the underlying LGL is brought under control, although other factors such as conformation clearly play their part as well.

Altered Gait

A horse with sore feet will change the way it moves in an attempt to reduce discomfort. As with full-blown laminitis, the pain is usually focused in the toe area so the horse is going to show the same modified gait as in a full-blown case, but at a lower level. LGL cases typically won't show the very obvious stilted gait of a full-blown case, but will normally land excessively heel first on the affected feet, at least to some extent. As with full-blown laminitis, the excessive heel-first landing will show up most on hard ground because of the increased vibration. The alterations in gait seen in LGL are very commonly missed because they aren't sufficiently dramatic. The result is a horse that is equally (if subtly) lame on both front feet (or all four feet), but does not appear unlevel.

With mild LGL cases, the excessive heel-first landing may be the only abnormality in the gait. With more severe cases you may also see a shortened stride, especially in trot. A lack of elevation in the trot is another common response – the higher the horse picks up its feet, the harder they hit the ground, and hence the greater the discomfort. And of course a horse with sore feet out on a hack will tend to head for the verge so as to avoid the impact of hard ground. Unsurprisingly, such horses are also more likely to nap when asked to move on hard ground.

More severe LGL cases often alter their gait to compensate for the muscle tensions that result from incorrect stance and gait. To the trained observer, stiffness in the back – especially the lumbar spine area – can be a useful indicator. Pain in the lumbar region of the spine (which is directly under the cantle of the saddle) often results in a horse that will tend to buck. This particularly shows up during upward transitions into canter. During such a transition, the horse needs to strike through with the outside leg, and this results in a larger than normal flexion of the lumbar spine during the transition. This brings any sore muscles into contact with the saddle and causes pain – with a predictable response. Sadly, this response is all too often misinterpreted as 'bad behaviour'. More severe cases may struggle once in canter as well, with the canter becoming 'bunched up' and lacking in forward motion.

Heel Pain

Bizarrely, a very small minority of LGL cases appear to show more pain in the heels than in the toe area – particularly in the early stages of an acute attack. This can result in the horse landing toe first rather than heel first. It is tempting to suggest that this is not LGL but some other condition, but it responds to the same dietary interventions as LGL, suggesting that it is the same underlying condition. The reason for the heel pain is not currently known, but may be related to a situation where there is already damage to the back part of the foot (for example from early navicular disease), which results in the LGL affecting this part of the foot more severely.

DELAYED SIGNS OF LGL

The following sections describe what happens to the hoof capsule over time as a result of LGL. The typical case will only show some of these. Where only one sign is present, it is important to question whether there might be another cause for that sign. However, where a significant number of the following signs are present, LGL is almost certainly the explanation.

Differential Growth Rate

As with full-blown laminitis cases, an excessive heel-first landing creates an increased growth rate in the heels. However, there is little evidence of a corresponding drop in growth of the wall at the toe. This is to be expected because catastrophic damage to circulation in the coronary band is not present in LGL.

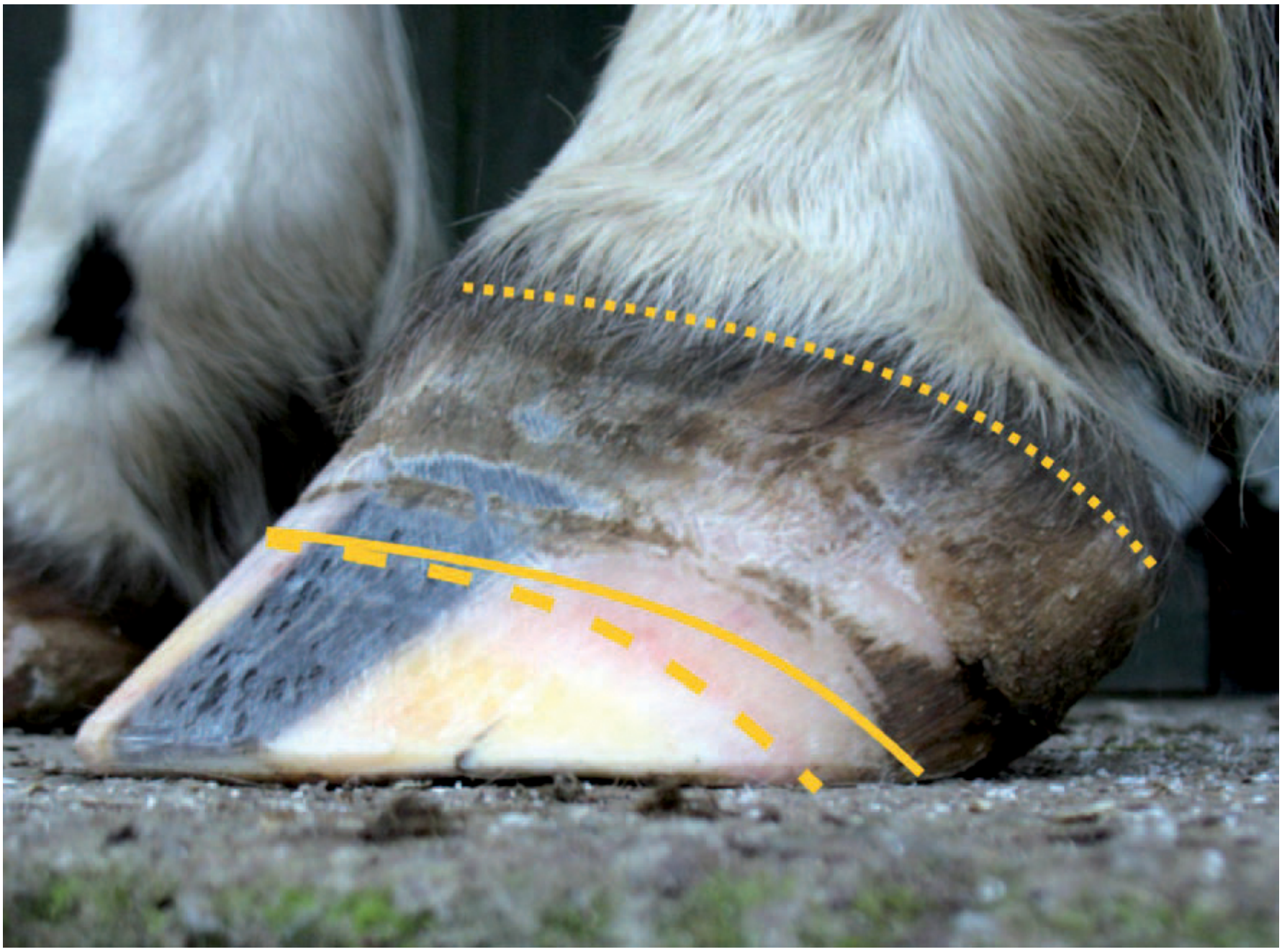


Fig. 57 Divergent growth rings in an LGL case. The solid yellow line is parallel to the coronary band (dotted line). A growth ring is easily visible because of bruising (dashed line) but is not parallel to the coronary band.

Because the adaptations in gait are less severe than in full-blown laminitis, the degree of excessive heel growth tends to be less obvious; however, growth rings that diverge towards the heel can still usually be seen.

Excessive Flaring

As with full-blown laminitis, we may see flaring at the toe in LGL cases. In a full-blown laminitis case, the flaring is associated with the wall being pulled away from the laminar corium, resulting in laminar wedge forming behind the flared wall. The definition of low-grade laminitis specifically excludes the

situation where laminar wedge is formed – which leaves an interesting conundrum: if there is no laminar wedge, how can the wall be further away from the pedal bone at the bottom of the foot than it is at the top?

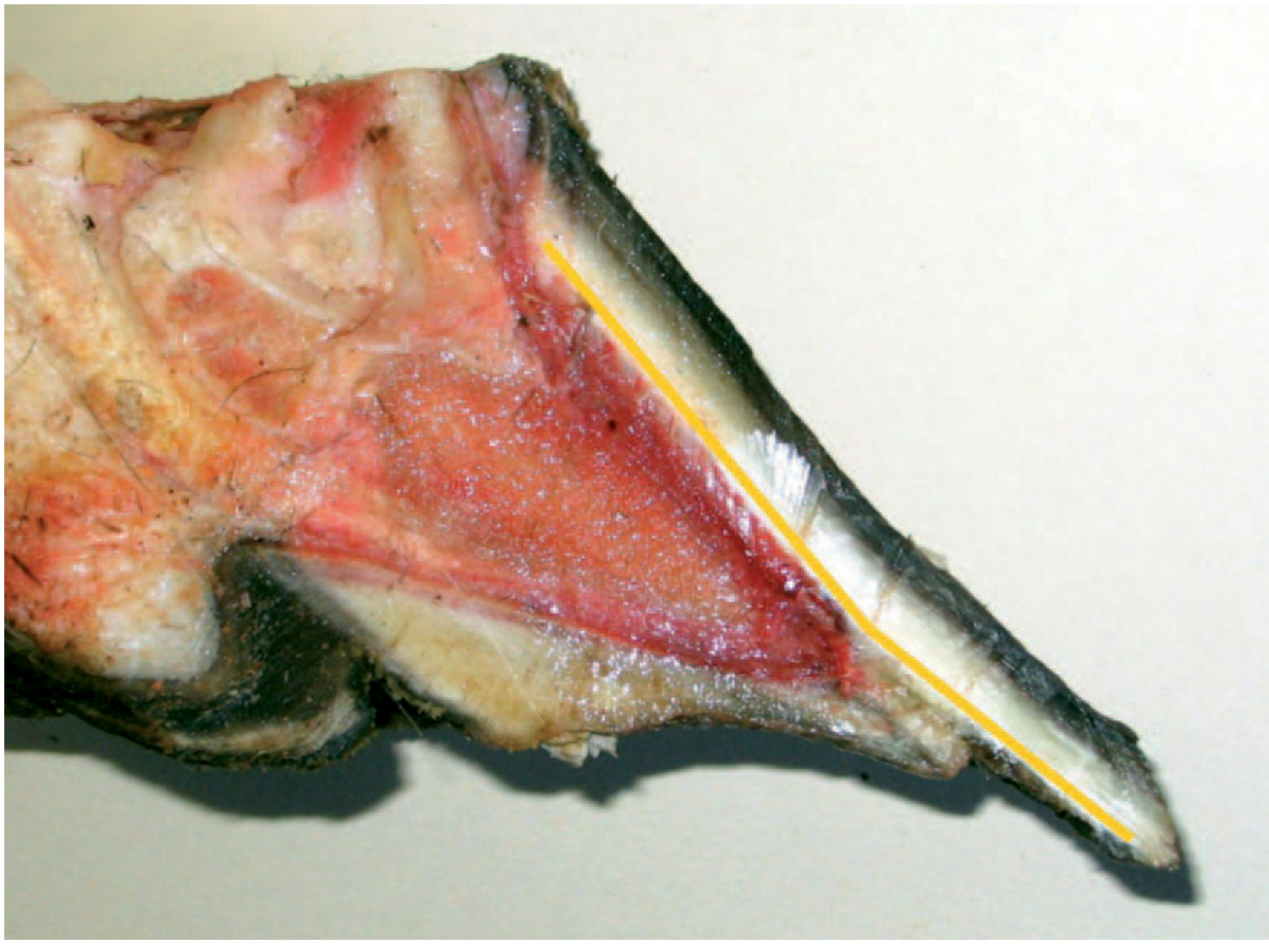


Fig. 58 Flaring at the toe of an LGL case caused by deviation of the white line from its normal orientation. The yellow line indicates the position of the inner wall and hence the location of the flaring.

It seems that there are three possible mechanisms. The first is that the flaring is entirely confined to the region below the bottom of the pedal bone. In this situation, the wall is straight until near the ground, and then flares. The flare results in the white line being pulled forwards in a curve such that it starts at the bottom of the laminae in the normal way, but does not then continue in line with the laminae. The white line in this situation is typically fairly normal, but the edge of the sole gets pulled forwards. Where an excessively long hoof wall and the associated leverage is not the cause, the driver for this form of flaring is excessive heel growth. The hoof is trying to form a slipper shape, just as in chronic full-blown laminitis, but the laminae are still strong enough to resist being stretched or torn, and instead the hoof horn deforms to accommodate the excess heel growth without the wall being pushed forwards at the toe. However, below the bottom of the laminae, the horn can flare mechanically.

The second mechanism for toe flaring is where the primary laminae do stretch, but do so subtly and evenly along the length of each lamina. This situation is on the cusp of meeting the criteria for full-blown laminitis, but the degree of laminar stretch is very slight and the laminae, although longer, tend to remain largely undamaged. This results in a slightly wider white line than normal, but not usually more than 50 per cent wider. Even such small increases in the length of the primary laminae can produce visually obvious flaring, but the deviations involved are often only a millimetre or two.

In the final flaring mechanism, the base of each primary dermal lamina stretches. In full-blown laminitis, the speed of the movement of the hoof wall away from the front surface of the bone provides enough force to tear the laminae apart. But where the movement is more gradual and steady, the base of each primary dermal lamina can stretch slowly, such that the section covered in secondary laminae

moves further away from the pedal bone. In this scenario, the primary and secondary epidermal laminae remain largely unchanged, and in particular, whilst the primary dermal laminae become longer, the primary epidermal laminae do not. This means that as the epidermal laminae grow down and reach the bottom of the dermal laminae, they merge with the rim of the sole to form a more or less normal white line, albeit a white line that is further forward than normal. For this to happen, the solar corium also has to be stretched forwards, such that it extends further than normal in front of the tip of the pedal bone. This final mechanism allows for there to be simultaneously significant toe flaring and yet a normal external appearance of the white line. The shape of the foot viewed from underneath will typically be elongated.

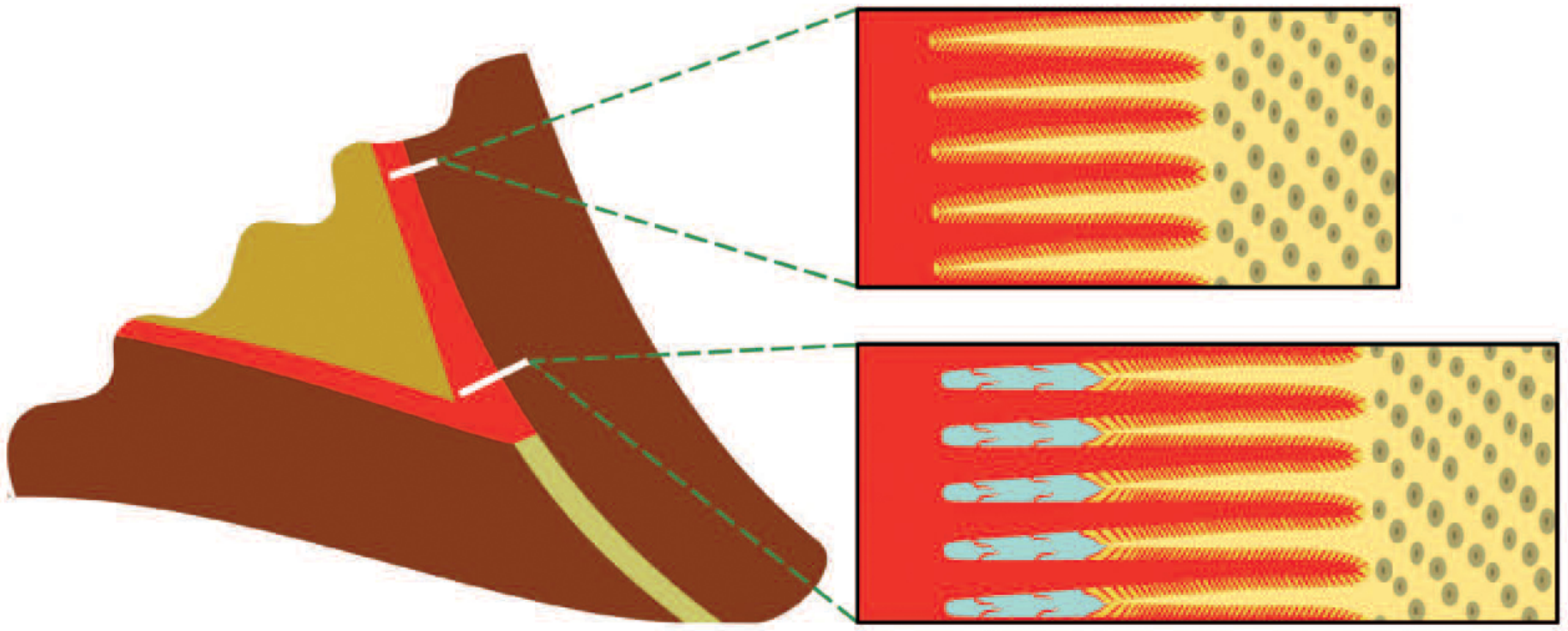


Fig. 59 A diagrammatic representation of flare caused by stretching of the base of each primary dermal lamina. Two sections through the laminae are shown (the locations are shown by white bars). Above the region of flaring, the laminae are normal. In the flared region, the bases of the primary dermal lamina are stretched. There is some damage to a few secondary dermal laminae where the stretching has occurred, and the spaces between these are filled with fluid. The primary epidermal laminae are normal length.

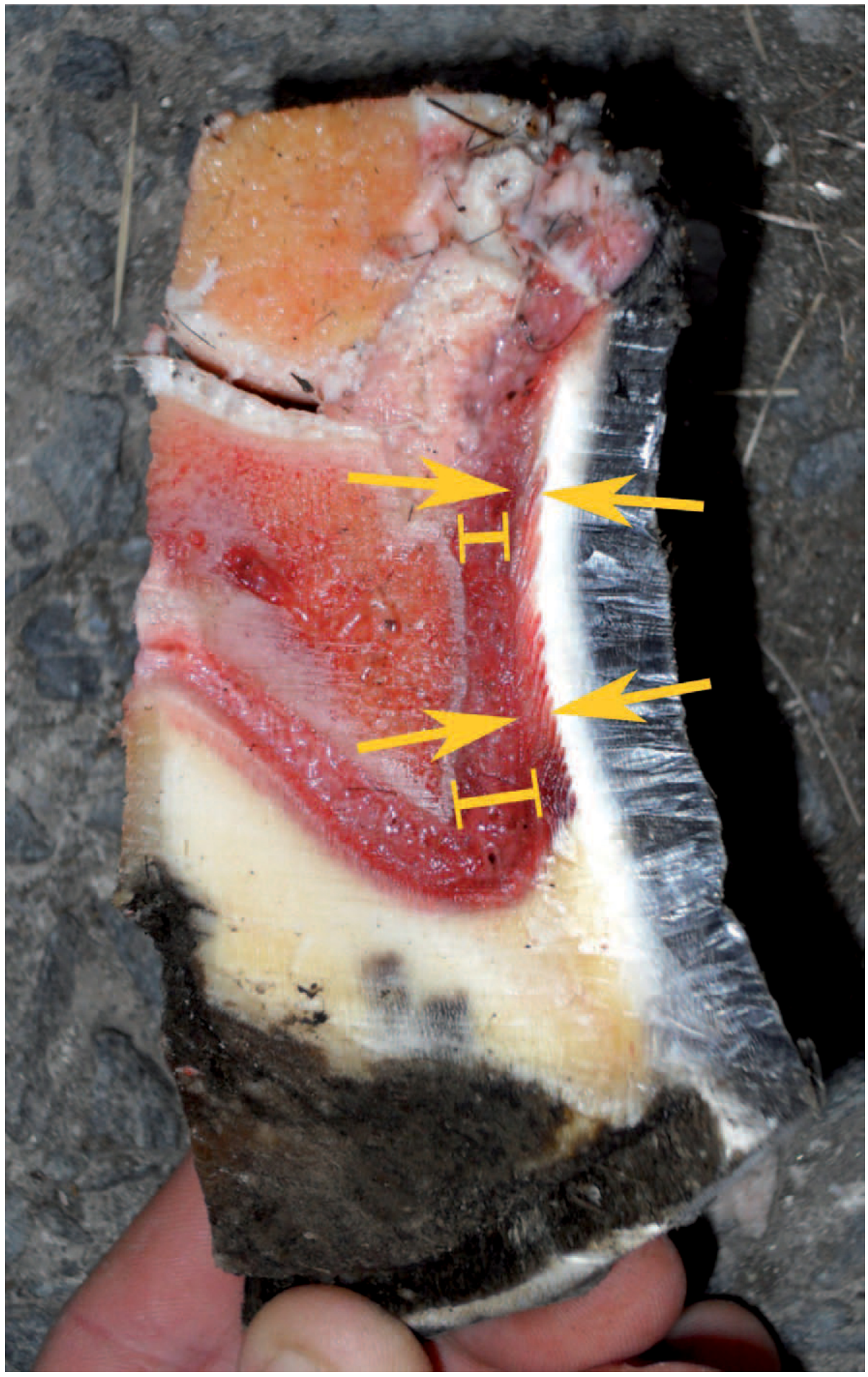


Fig. 60 A section through a hoof showing quarter flaring. The laminae appear to follow the flaring, but otherwise look normal. The apparent width of the laminae is marked (arrow heads). The apparent width of the underlying connective tissue is also marked (bars). More work is needed to confirm that this flaring is caused by stretching of the connective tissue between the laminae and the wall, rather than stretching of the laminae themselves.

It is theoretically possible to get a similar effect caused by stretching of the connective tissue between the dermal laminae and the pedal bone. Although some dissection samples appear to show this (particularly with quarter flaring), more work is needed to confirm whether it is indeed the connective tissue that stretches, or whether these cases are also examples of stretching of the bases of the primary dermal laminae.

Where the wall is pulled away from the pedal bone for a long period of time, the tension this places in the connective tissue between the laminar corium and the bone results in the front surface of the pedal bone being constantly pulled forwards. Where a tissue is subject to a constant unexpected force, it tends to remodel in a way that reduces that force. In this case, the pedal bone changes shape so as to become more elongated – effectively filling the space behind the flared wall at the toe. This process

is imperceptibly slow and may take some years to occur to any noticeable extent, but it does tend to be largely permanent, meaning that long-term laminitic feet sometimes end up with permanently elongated pedal bones, which in turn impacts on the external shape of the hoof capsule. It is, of course, very important to recognize where this has occurred, and not to attempt to 'make' the foot a normal shape.

As with full-blown laminitis, acute attacks produce sudden changes of angle in the wall, whereas chronic attacks produce a more curved form of flaring.

It is important to be aware that flaring can happen for reasons other than LGL. For example, lack of trimming can result in a wall that is significantly proud of the sole, and in this situation, the lower portion of the wall will often flare (sometimes dramatically) by the white line deviating from its normal location, as described above.

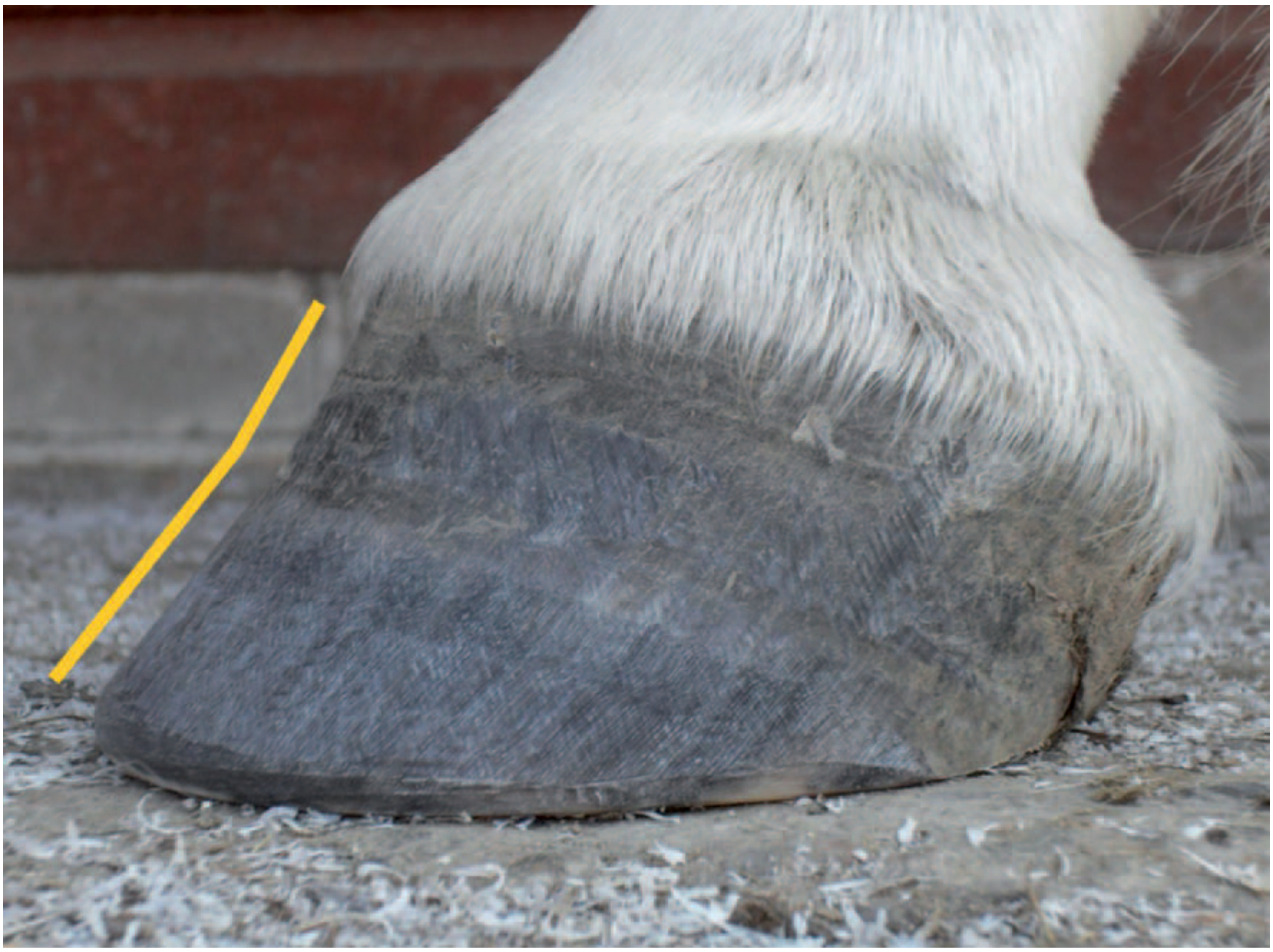


Fig. 61 A sudden change in wall angle at the toe (indicated by the yellow line) as a result of an acute LGL attack around five months previously.

Similarly, conformation defects or injuries that cause a less than perfect landing (that is, landing to one heel of the foot before the other) tend to result in increased growth on one side of the foot and flaring on the opposite side. The hoof will curl away from the side with the greatest stimulus in a similar way to the hoof capsule in chronic laminitis curling away from the heel. However, where no laminitis is present, the degree of flaring in such cases is usually minimal. This is because the laminae are strong enough to resist the curling action for the most part. Where LGL is not present in a foot with an uneven landing, the increased growth on one side of the foot results in that side being more upright (the wall in that quarter will be closer to vertical) and the other side being less upright; however, the wall will be nearly straight from top to bottom. The pedal bone will also tend to remodel in a similar fashion. Where LGL is present, however, the laminae will weaken to the point where the flaring that occurs as a result of the uneven landing will be more dramatic. For that reason, the key to recognizing LGL is not simply

the presence of flaring, but the presence of flaring that is greater than would be expected given any other factors involved.

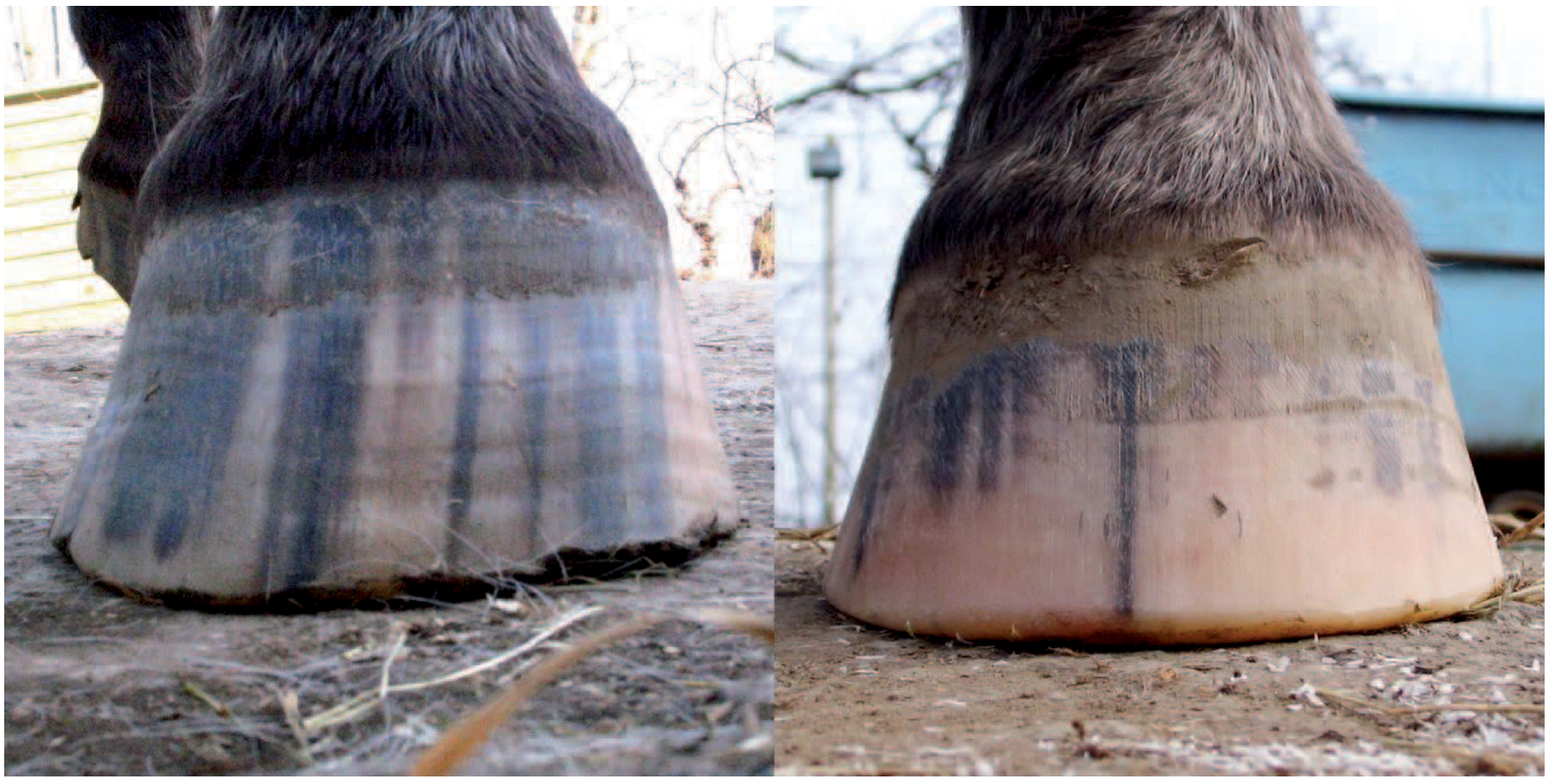


Fig. 62 LEFT: *A left fore foot showing flaring to the inside quarter as a result of poor conformation and LGL. The increased growth rate of the outside quarter can be seen in the growth rings.* RIGHT: *The same foot after LGL has been eliminated by diet changes. The difference in the wall marking pattern is the result of minor flare removal, with around a millimetre of outer wall removed. The differential growth rate is largely unchanged, but the flaring is significantly reduced. Note that the hoof-wall markings at the centre of the toe are now straight and more vertical.*

White Line Damage

Where toe (or quarter) flaring occurs as a result of LGL that involves the laminae stretching, this may result over the following weeks in the formation of a sub-standard white line. In some cases, blood products will be clearly visible in the white line; in others the white line may be slightly stretched. Severe stretch to the white line is outside the definition of LGL. Occasionally the white line will appear stretched and yet otherwise healthy, but more often there will be some degree of damage to the white line.

Wall Bruising

The coronary band is often inflamed in LGL, and an inflamed coronary band will bruise more easily than normal. Bruising occurs as microscopic damage to blood vessels, allowing them to leak. If damage to the blood vessels is limited, then tissue fluid (serum) leaks from the corium and gets incorporated into the hoof wall as it is produced. This tends to stain the horn a dark yellow or orange colour, although this is only easily visible in white hoof horn. Where there is greater damage, red blood cells may leak out and stain the horn bright red. Again, this is only easily visible in white horn, although really severe bruising can sometimes be seen as a red tinge in black hooves. Such bruising marks are easiest to see when the hoof wall has just been trimmed.

Unfortunately, any new wall bruising is initially hidden behind the periople. It typically takes around four weeks for newly produced wall to progress past the periople and become visible. So bruising in the wall can only be used as an indicator of LGL damage that is at least four weeks old. On the plus side, the history of past events (from four weeks to around twelve months) can be read off the hoof wall by looking at the pattern of bruising. This can sometimes be helpful in unpicking causative factors. For

example, bruising that consistently corresponds with the horse spending time in a specific field might suggest that something about that field is exacerbating the LGL.

The severity of bruising depends on two factors: the degree of underlying inflammation, and the mechanical stress the hoof is placed under. It takes a lot to bruise a healthy coronary corium. A mildly inflamed corium will only bruise as a result of concussion, and so may not show bruising if the horse is resting at pasture. With more severe inflammation bruising may happen even without any concussion.

Because the entire coronary band is inflamed, the bruising typically appears as rings that go right around the hoof, roughly parallel to the coronary band. These rings are rarely even all around the hoof, but usually have regions where the bruising is worst. For example, a horse that lands more to the outside heel as a result of poor conformation will often have rings that have the worst bruising in the outside quarter.

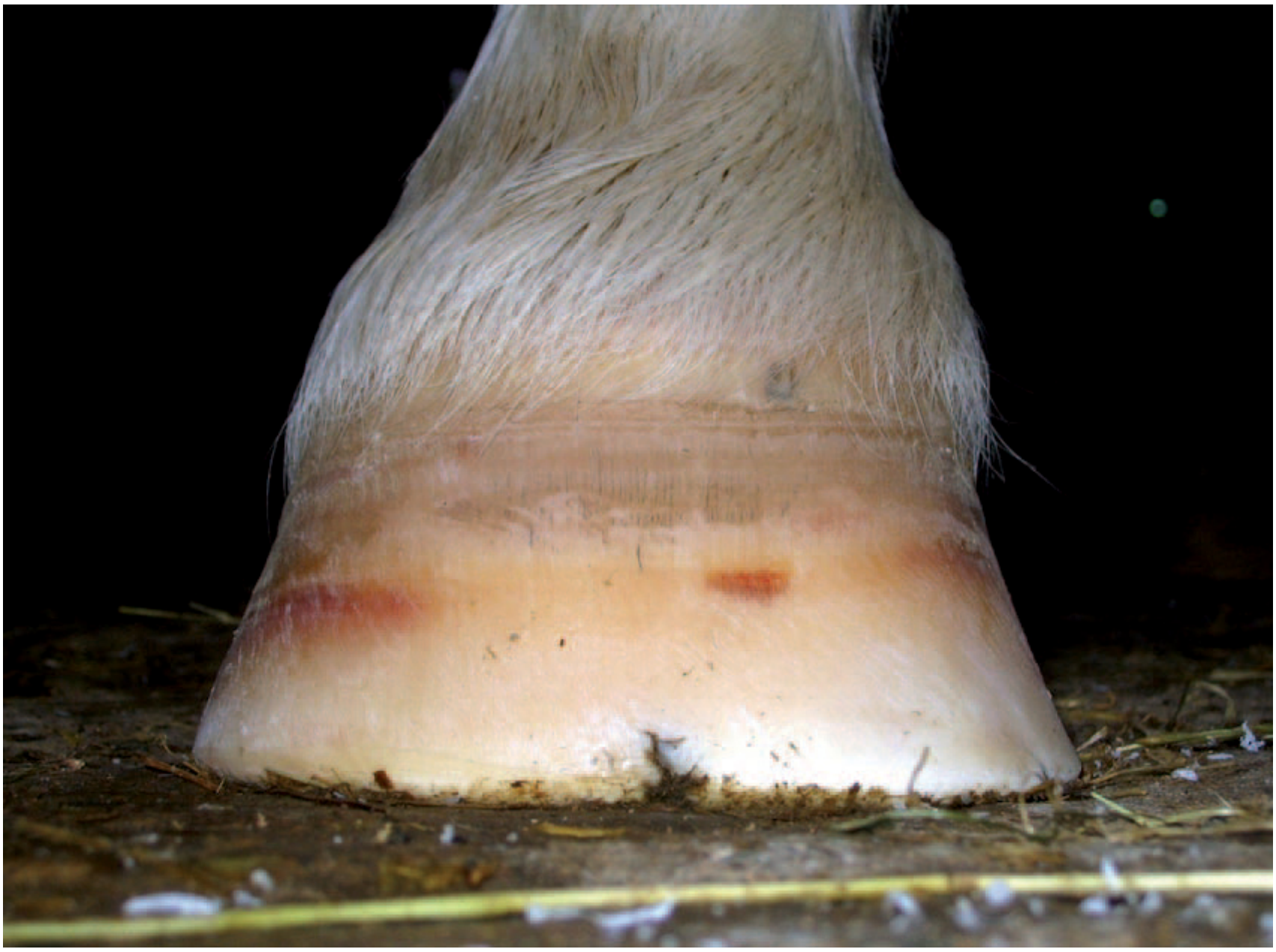


Fig. 63 A pony foot showing wall bruising typical of LGL. The bruising ring is roughly five months old. The orange ring is slightly further from the hairline on the left, showing that this side of the foot grew faster (a minor conformation defect resulted in heavier landing to this side of the foot). The area subject to impact is strongly red. The area where the most flaring resulted is also somewhat red. There is a small localized bruise in the toe area from blunt trauma to an already inflamed coronary corium.

It is important to differentiate ring bruising caused by LGL from localized bruising caused by traumatic injury to the coronary band. There are several things that help differentiate between these two scenarios. Where the horse mechanically bruises the coronary band – for example, by kicking a stable door – the bruise will be fairly localized and not part of a ring that goes from heel to heel. Such a bruise will also have a sudden start and then fade away as the coronary band heals. As the hoof wall is produced, this results in a wall bruise with a sharp lower margin that then fades away upwards. In contrast, most LGL-related bruises do not start suddenly and hence tend to fade in and fade out again as you read the history upwards on the hoof wall from oldest to newest horn. Of course it is possible for a horse with LGL to end up with a blunt trauma injury to the coronary band, and the localized bruising in this case will tend to be stronger than it would have been if the coronary band hadn't already been

inflamed. This scenario can often be recognized because a localized bruise with a sharp start and slow fade sits within a ring of more diffuse bruising.

Dissection of LGL cases helps to confirm that the bruising seen in the wall does indeed originate at the coronary corium. In milder cases, the bruising is typically confined to the outer wall (which is harder than the inner wall and hence transmits vibration to the coronary corium to a greater extent). This proves that the bruising does not originate at the laminar corium. In more severe cases, the bruising extends through the full thickness of the wall. The shape of the bruise when the wall is viewed in cross-section is always an exact match for the shape of the coronary corium, demonstrating that the bruising has been produced there before being pushed downwards by further wall production.

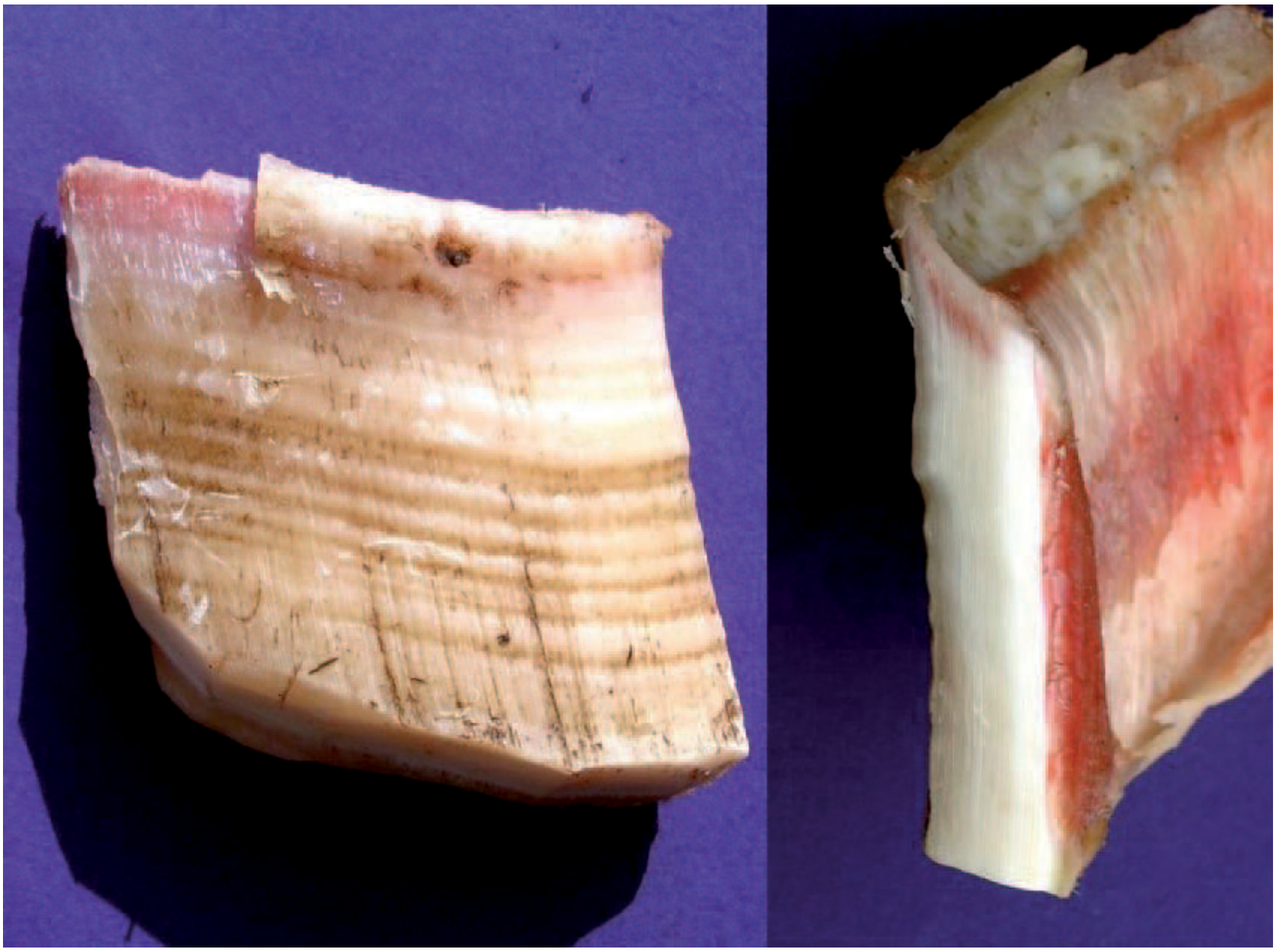


Fig. 64 Two views of the same section of hoof wall showing a recent (two to three weeks old) acute LGL attack. LEFT: ***Externally, the bruising ring is only visible where the periople has been removed.*** RIGHT: ***Sectioning through the wall reveals that the bruising ring matches the shape of the coronary corium.***

White Line Disease

The presence of blood products in the hoof wall and white line provides a substrate for bacteria and fungi to colonize the horn. The result is known as white line disease – a rather unhelpful term because it affects the wall as well as the white line. White line disease can happen for other reasons – for example, mechanical damage to the hoof wall as a result of leverage caused by lack of trimming – but in the author's experience, persistent white line disease despite good trimming is almost always associated with LGL. Even where mechanical factors such as lack of trimming play a part, LGL, if present, will make the severity of the resulting white line disease worse.

In a healthy hoof, the horn cells are so tightly packed together that moisture and infectious agents cannot easily penetrate the horn. The outer surface of the horn forms a breathable membrane that allows moisture produced within the foot (as part of the mechanisms that keep the hoof horn at the

correct level of hydration) to evaporate off the surface, whilst preventing moisture (and infection) from getting in. This works in a similar fashion to ‘breathable’ waterproof jackets.

Where LGL is present, some cells may be damaged and blood products may become incorporated between the horn cells, so making the horn permeable to water, bacteria and fungi. Once inside the horn, the bacteria and fungi feed on damaged cells and blood products and rot the horn. The surrounding horn may become damaged as a result, allowing infection to spread, although this effect is fairly minimal – the infection largely confines itself to horn that is already damaged.

The bacteria and fungi involved in white line disease are anaerobic – they prefer warm, moist conditions, and in particular do not cope well with being exposed to air. As a result, exposure to air kills horn infections, and the resulting waste products typically are black. The effect of this is that any blood products in the hoof wall and white line that are close to the surface of the hoof, particularly at the bottom of the wall where there is more contact with moisture, tend to rot and go black. For this reason, blood products in the white line especially are often missed because no red discoloration is seen, only black rot. Even in the outer hoof wall where any bruising can be seen growing down the wall, the role of blood products in providing a substrate for white line disease is recognized far too infrequently.

Fig. 65 Three examples of white line damage in LGL cases. LEFT: ***A mild LGL case where the white line is not stretched and is mostly a healthy colour but has some blood products in it. The blood products are clearly visible to the left of the toe, and the more common appearance, where the blood products have rotted and turned black, is visible to the right of the toe.*** CENTRE: ***A moderate LGL case with a normal thickness white line showing numerous rotting epidermal laminae.*** RIGHT: ***A severe LGL case where the white line is starting to stretch and exhibits the typical orange discoloration of more severe cases, as well as a lot of rotting laminae. This example is on the cusp of meeting the definition for full blown laminitis.***

It is common in LGL to see a fairly healthy white line (sometimes very slightly stretched) but with a few of the epidermal laminae broken or damaged (with blood products filling some of the space that would have been occupied by each damaged lamina). If the white line can be trimmed into sufficiently, this can be seen as occasional fine red lines radiating out from the sole, representing the damaged laminae. In real life cases, however, it is not usually desirable to trim that far into the white line, and so the external appearance is similar, but with the damaged laminae appearing black rather than red as a result of the blood products rotting. The less damaged areas of white line are also frequently more yellow than normal (normal white line is closer to a grey colour), which may indicate the presence of blood serum within the white line. In more severe cases, the white line may become orange in colour.

The other form of white line disease (which perhaps would be better termed ‘wall disease’) seen in LGL is where damage and blood products in the hoof wall itself rot, leaving the wall weakened. This typically allows the hoof wall to delaminate, the outer layers of hoof wall often breaking away as a result. Such damage is usually confined to the bottom of the hoof wall. There are several reasons for this. The first is that the horn in this area is the furthest from any corium in the entire hoof and hence

receives the least nutrition from the various coria. The second is that, of the hoof wall, the bottom surface is the one most exposed to moisture. And finally, the mechanical stresses placed on the hoof wall during breakover tend to pull the hoof wall forwards at the toe – any slight damage in this area allows the hoof wall to delaminate, allowing moisture into the anaerobic space created as the wall separates. Once small amounts of rot get into the bottom surface of the wall, delamination allows the damage to spread, feeding on blood products further up the wall as it goes.

Fig. 66 Wall bruising seen on the bottom surface of the outer wall. This was revealed when an excessively long wall was trimmed back. Where such bruising appears at the bottom of the hoof wall, it will normally allow bacteria and fungi to enter, resulting in wall delamination.

Fig. 67 A dissection showing wall delamination as a result of rot in a foot with LGL. Bruising is present at the top of the wall consistent with a recent acute LGL attack (also accompanied by a matching layer of bruising in the sole). More diffuse bruising consistent with a period of chronic LGL is apparent half way down the wall. Infection has rotted the blood products that would have been present in a further region at the bottom of the wall, with delamination (mostly in the outer wall in this case) playing a part in the spread of the infection. The ragged appearance of the bottom surface of the wall is typical of wall infections.

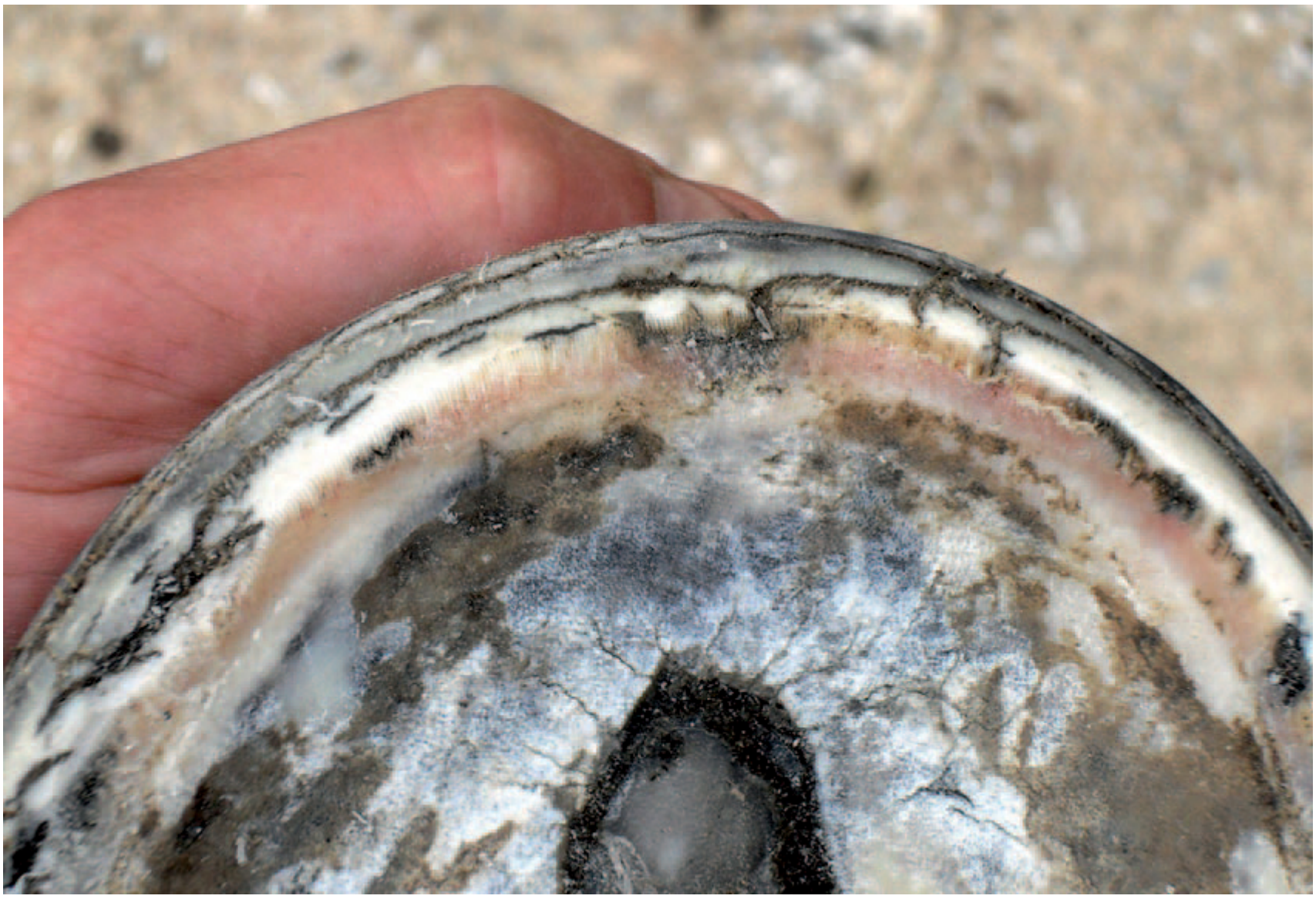


Fig. 68 An example of a foot with LGL showing bruising to the white line and extensive wall infections.

Where a horse with wall bruising is shod, the nail holes provide a useful route for moisture and infection to penetrate into areas of damaged horn that would otherwise not be as accessible. The result is that rot spreads around the nail holes, weakening the horn in that area. This is easily visible in a white hoof but much less obvious in black horn. Eventually the nails pull through and the shoe falls off. Where a horse repeatedly loses shoes due to poor quality horn around the nail holes, LGL should be considered as a possible contributory factor.

Fig. 69 White line disease developing around nail holes in an LGL case. Some wall bruising can be seen, which explains how the infection is managing to spread so rapidly from the nail holes. This horse had already lost a shoe soon after shoeing as a result of the weakened wall, as can be seen from the presence of two sets of nail holes at the same level.

Sole Bruising

As with the coronary band, an inflamed solar corium results in blood products being incorporated into the sole. Unfortunately this bruising is only visible as it reaches the surface. Acute LGL events result in fairly thin layers of bruising in the sole that appear and disappear at the surface in a matter of days. In contrast, chronic LGL tends to result in a sole that has multiple, thicker but less strong layers of bruising within it. It should also be noted that the sole typically takes around three months to grow out in a domesticated horse, and hence any bruising seen at the surface of the sole represents LGL that occurred around three months earlier.

The sole of an unshod horse is particularly vulnerable when inflamed because it is the main weight-bearing structure. Whereas a horse with healthy, thick soles can walk across stones without issue, the same horse with LGL is much more likely to bruise the sole as a result. In milder cases, this might be isolated bruises associated with standing on individual stones. In more severe cases, even the concussion from hard ground is enough to cause bruising across the entire surface of the sole.

Fig. 70 A section through the quarters of an LGL case showing very recent sole bruising (marked). Recent mild bruising from the same attack can also be seen at the top of the hoof wall (marked).

More severe bruising tends to lead to a reduction in circulation to the sole, and this in turn results in a slowing of the growth rate. A slower growing sole will tend to be thinner – exfoliation occurs at the same length of time after production, but less new horn has been produced in the meantime. This thinner sole then provides less mechanical protection to the solar corium, and hence the risk of bruising increases. As a result, a relatively low degree of LGL can sometimes lock the horse in a cycle of poor sole production and continued bruising. Where the sole has become very thin, even correcting the underlying causes of the LGL may not break the cycle unless steps are taken to mechanically protect the sole and allow it time to heal.

The problem of sole bruising and tenderness of the sole is often the biggest issue when it comes to more severe LGL cases, and such horses are often shod as a way to ‘fix’ the problem. Whilst shoeing the horse may result in an increase in comfort because the sole is no longer in contact with the ground, it does not address the underlying inflammation, and the vibration from the shoe may actually worsen the underlying problem.

False Sole

Just as in full-blown laminitis, the sole of an LGL case will sometimes produce more horn in response to mild inflammation. As would be expected, the result is usually less dramatic than in the full-blown case. It is also usually localized to the area immediately surrounding the frog. In milder cases, false sole tends to develop just in front of the bar. The result is a bar that appears to extend further forward than normal and so is sometimes referred to as ‘false bar’. However, it is important to recognize that this is not bar tissue as it is made of sole, not of wall and white line. As the severity progresses, the false sole may extend along the side of the frog and even around the apex of the frog, resulting in a raised ridge that completely encloses the front half of the frog.

This effect will be exacerbated if anything causes more mechanical stress on the underlying solar corium. One thing that will cause this is poor structure in the back half of the foot. This allows the two heels to move independently of each other to an excessive degree, and the sole then has to flex to accommodate this. This flexion force is concentrated in the region of the sole immediately adjacent to the frog on either side. Hence a horse with a poor frog and digital cushion is more likely to develop false sole around the frog, especially if it also has LGL.

Fig.71 Examples of false sole as a result of LGL. LEFT: *Mild false bar with the approximate end of the true bar marked.* RIGHT: *False sole completely surrounding the frog in a recovering LGL case. The false sole can be seen to be coming away from the underlying sole, and is ready to be removed as part of trimming.*

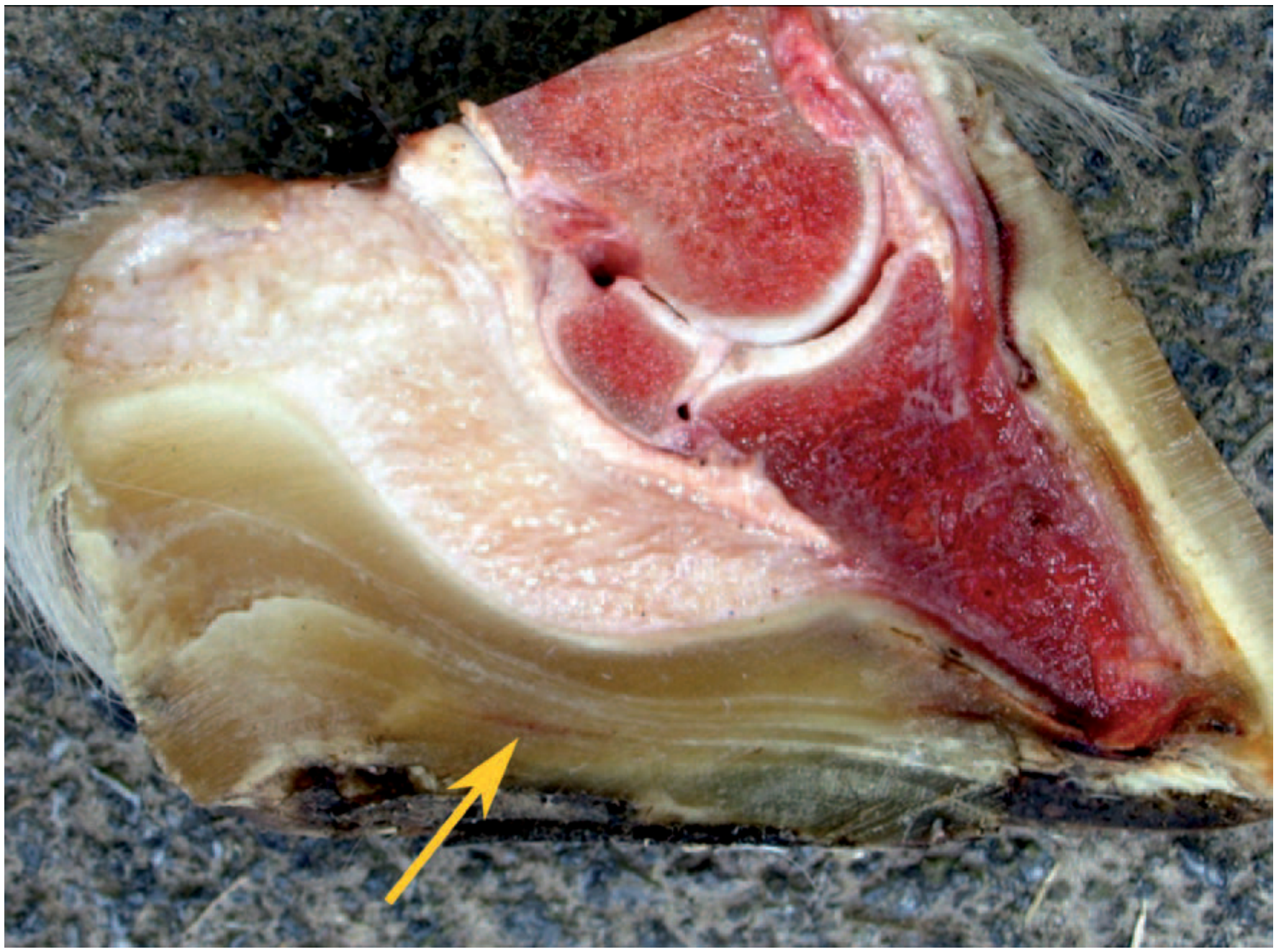


Fig. 72 A dissection showing layers of subtle damage in a frog. In one area (marked), blood products are visible within a layer of damage.

Frog Bruising

Given that the sole becomes bruised in LGL, it should not be surprising that exactly the same effect can be seen in the frog. Just as with the sole, layers of blood products can be incorporated into the frog horn as a result of bruising to the frog corium. As with the sole, these tend not to be visible until they reach the surface. A thin layer of bruising – say, from an acute attack that only lasted twenty-four hours – may appear at the surface and then exfoliate off again in a day or so. As with sole bruising, any bruising seen at the surface of the frog tends to relate to LGL that occurred around three months earlier.

Fig. 73 A recovering severe LGL case during frog trimming. A very thin layer of frog bruising has been exposed in places (in other places it has been trimmed through, as it was extremely thin). The sole also exhibits severe pitting as a result of sole damage that occurred at the same time.

Thrush

Thrush is the term used where the horn of the frog or sole becomes infected.

An unshod horse that is working regularly on abrasive surfaces will wear away the surface exfoliating layers of frog and sole as fast as they are produced. This results in a clean, hard surface to the frog and sole. Where there is insufficient wear to the surface of the frog or sole (as is common in domesticated horses, particularly when they are shod), some of the exfoliating material is retained on the surface of the hoof. Because this material no longer has the tightly knit structure of healthy horn, it is vulnerable to rot from similar infectious agents to those that cause white line disease. However, unlike white line disease, this is actually helpful to the horse. Without the infection, the exfoliating material (which is still surprisingly hard if not wet and rotting) builds up on the bottom surface of the hoof, and ultimately can get in the way. For example, where exfoliating material builds up just in front of the bars, this can cause bruising and corns.

A build-up of exfoliating material can sometimes be seen in horses on box rest (particularly when they are kept on dry bedding such as hemp or shavings), and in such cases will often have to be trimmed away regularly to prevent problems. In contrast, the horse out in a field can benefit from the softening effect of infection to allow the excess material to wear away easily, even under conditions where there is little abrasion.

Fig. 74 A typical example of thrush tracking forwards in layers through the frog of an LGL case.

The frog, being softer than the other horny structures, tends to be subject to nicks and scratches as a result of work on any abrasive surfaces such as tarmac. Normally this causes no problems to the horse, as the frog is far thicker than the depth of any damage, and under any surface exfoliating layer, the frog is healthy and will not rot. However, if there are layers of bruising in the frog as a result of previous LGL attacks, this normal minor surface damage can provide a route for moisture and infectious agents to track into a layer of bruising. The end result is that the outer layer of frog becomes detached from the rest by the layer of rotten horn. Because most nicks in the frog happen close to the heel bulbs (because the foot lands heel first), these layers of rot typically track forwards from the heel bulbs, often leaving a layer of shedding frog hanging on by the front portion. In addition, the apex of the frog is not in contact with the ground in most horses, and so experiences less mechanical bruising. As a result, the layers of damage that allow thrush to track through the frog are often not as severe towards the apex of the frog, resulting in less thrush.

In more severe LGL cases, infection can track through the frog a layer at a time, using lower levels of damage between the main layers; this results in fairly thick layers of frog shedding away.

Fig. 75 LEFT: *Damage to the periople and outer surface of the wall as a result of, in this case, full-blown laminitis. LGL cases show a milder form of this effect.* RIGHT: *The same foot seven months later after successful diet changes, showing an improvement in the periople and the very top of the wall.*

It is important to note that the infection in both white line disease and thrush normally remains in the outer surface of the hoof horn and will not make it anywhere near blood vessels. As such there is a very low risk of live tissue becoming affected or of an abscess resulting. Horn infections are merely the rotting of damaged horn on the outer surface of the hoof.

Perioplic Damage

Although not often considered, the perioplic corium may also become damaged in both laminitis and LGL. This results in a poor quality periople being produced. In some horses, this may be the first sign of a problem – others can have significant degrees of even full-blown laminitis without showing much damage to the periople. The periople is responsible for protecting the outermost surface of the outer wall after it is produced to allow it to harden, so any damage to the periople also tends to result in damage to the surface of the outer wall. This outer wall damage, which leaves the surface of the hoof wall looking scaly, is often more noticeable than the damage to the periople itself. Because this damage only affects the very surface of the hoof, any rasping of the hoof wall lower down during normal trimming of the hoof will go through the damage. As such, the damage is usually confined to the top of the hoof. Where this effect is present, an improvement in the quality of the wall at the hairline is often one of the first signs that an intervention aimed at reducing the severity of the underlying laminitis has been successful.

Low-Grade Sinker

The potential for the pedal bone to drop to a lower position within the hoof capsule in full-blown laminitis is well recognized, as discussed in Chapter 2. Less well recognized is the possibility of the pedal bone sinking in an LGL case, a situation described as low-grade sinker.

It is generally assumed that the founder distance in a healthy horse is somewhere in the range of 1–7mm. When ‘healthy’ horses are studied, the typical founder distance varies with breed, with thoroughbreds having the highest values and native ponies the lowest. These studies are typically performed on shod horses. However, when barefoot horses and those known to have little or no LGL are x-rayed or dissected, the founder distance is typically near to zero. What this seems to suggest is that any founder distance significantly above zero is the result of LGL in a shod horse.

It is not too hard to see how this could happen. The laminae in an LGL case are inflamed, but not to the point of dramatic tearing. However, as seen in Chapter 3, applying a shoe to the foot creates a force that acts to shunt the hoof wall upwards along the laminae. Healthy laminae can resist this shear force; severely inflamed laminae tear as a result. But slightly inflamed laminae stretch a little instead, allowing

the pedal bone to sit lower in the hoof capsule (or taking a horse-centred viewpoint, the hoof capsule gets shunted slightly up the bone column). The result is a hoof wall that is a few millimetres longer than normal. This can give the impression that more material needs to be removed from the bottom of the hoof, although in reality, the pedal bone is no further from the ground than normal.

THE ROLE OF LGL IN OTHER PATHOLOGIES

So far this chapter has covered the clinical signs directly attributed to a current LGL attack, and the changes in the hoof that occur in the weeks and months after an LGL attack. However, LGL can also contribute to a range of other problems, the more common of which are described below.

High Heels

Because LGL typically results in toe pain, horses tend to shift weight to the heels at gait, just as they do in full-blown laminitis. The result, as has already been described, is excess heel growth. Where the horse has access to hard ground, this excess growth may be at least partly matched by increased wear as a result of an excessive heel-first landing. However, in all other cases (and especially in a shod horse), the excess heel growth is not matched by increased wear and hence the heels will become high in relation to the toe over time. If the excess heel height is not regularly removed by trimming, then this can cause the horse tendon problems in exactly the same way as described in Chapter 3 in relation to full-blown laminitis.

There are, however, some additional problems associated with long-term high heels that tend to be particular to LGL cases, if only because they are more likely to be working despite long-term laminitis.

Contracted Heels

If the hoof wall is removed from the foot during a dissection it tends to want to curl up. The wall has a natural springiness that pushes the heels together. In a healthy horse, this force is resisted by the frog and the digital cushion, which sit between the heels holding them apart. For the frog and digital cushion to be full, firm and capable of holding the heels apart, these structures need to be stimulated via pressure on the frog from the ground. Where the heels are left high for long periods as a result of poorly managed LGL, the frog is lifted off the ground somewhat and loses stimulus. This effect is exacerbated in a shod horse where the frog may already be lifted slightly off the ground by the thickness of the shoe. With insufficient stimulus, the frog and digital cushion begin to shrink, allowing the heels to become closer together – a condition described as contracted heels.

Fig. 76 LEFT: *An example of contracted heels. (Photo: Jayne Hunt)* RIGHT: *A healthy foot for comparison.*

In mild cases, heel contraction does not appear to cause the horse any significant discomfort, but as the problem becomes more severe, heel pain often results, either directly or as a result of knock-on problems.

Deep Central Sulcus Infections

A healthy frog corium, when seen in cross section from the back of the foot, has a gentle M shape (with the sole pointing upwards as it does when the foot is being picked out). The centre of this M is a depression in the corium which corresponds to a gentle depression in the centre of the back half of the frog, called the central sulcus. This depression should be very shallow but is often far deeper in domesticated horses than should be considered healthy.

Where the heels contract as a result of being left overly long, the M shape of the frog corium also becomes compressed. The normal gentle curve of the central portion typically becomes a V-shaped crease. At this point, frog horn produced from one side of the V crushes against the horn produced from the other side, rather than these two areas contributing to a region of horn that flows smoothly away from the corium. The result is a narrow region of damage that grows out in the very centre of the central sulcus. As this reaches the surface, it allows thrush to track back towards the frog corium separating the two halves of the frog.

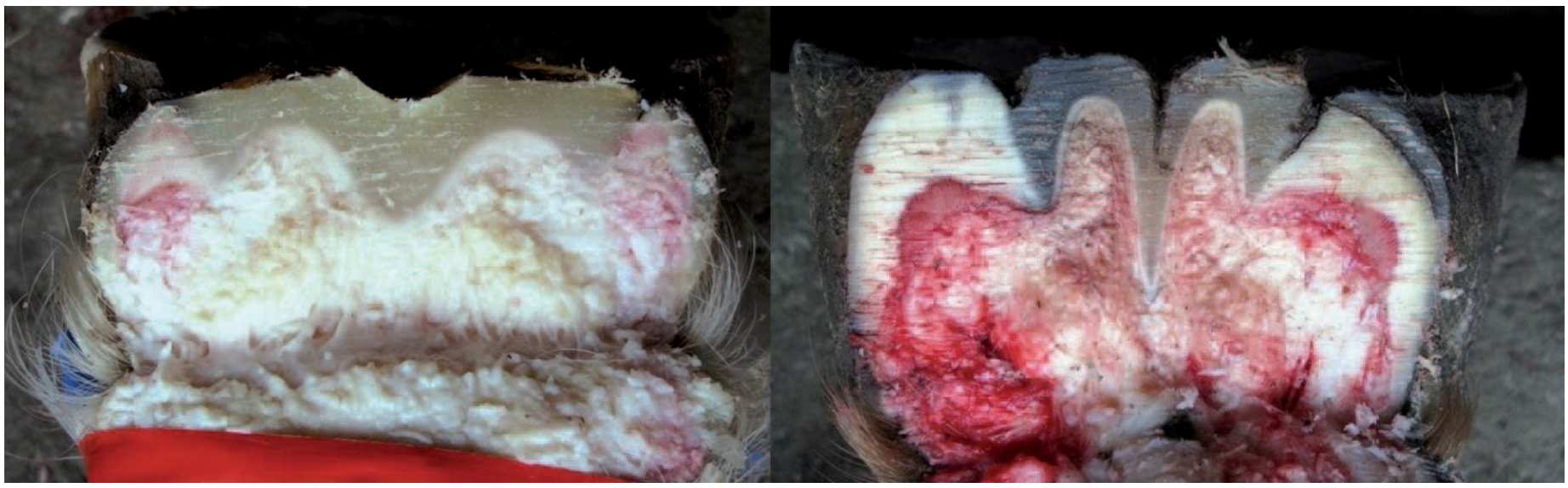


Fig. 77 Sections through the back of the heel bulbs of two feet (looking from behind the foot with the sole uppermost). LEFT: *A normal healthy frog with an M-shaped corium.* RIGHT: *A horse with a long-standing history of deep central sulcus infections. The corium shows the typical crease in the centre, and it has also retreated deeper within the foot. The contraction of the heels can be clearly seen.*

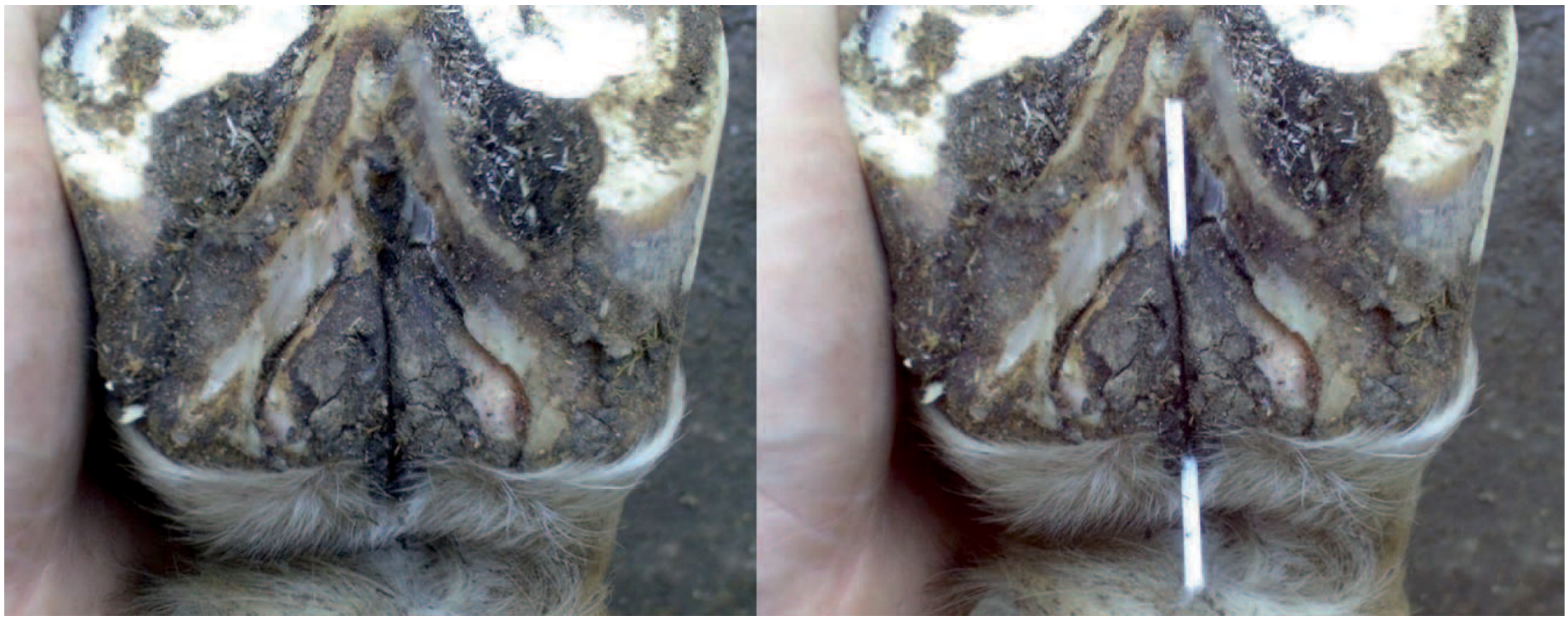


Fig. 78 The external appearance of a deep central sulcus infection in a live horse. The insertion of a probe (right) gives an indication of the extent of the defect. Thrush from the central sulcus has also tracked into a layer of LGL-related damage deep within the frog, causing a section of the back part of the frog to shed.

The resulting deep crack between the two sides of the frog cannot extend further forward than the normal extent of the central sulcus, as forward of that point there isn't a depression in the corium to become V shaped. It can, however, track right through the frog until it reaches live flesh and causes bleeding. This crack is called a deep central sulcus infection and it is extremely common in the UK. It should not be surprising that, where the infection reaches corium, the result is pain. Horses often switch from an excessive heel-first landing to a toe-first landing as a result – the pain from the deep central sulcus infection being worse than from the underlying LGL that originally contributed to it. Once a horse starts landing toe first, there is an increased risk of tripping, particularly when moving at speed.

Deep central sulcus infections will not always go away if the underlying LGL is removed. One reason for this is that the area is often filled with dirt and grit, which abrades the sides of the crack as the horse moves, causing more damage and allowing more thrush to take hold. Sometimes the deep central sulcus will allow easy access to layers of damage within the frog, allowing thrush to cause much thicker than normal layers of frog to shed away. This reduces the size of the frog and hence makes it even less likely that it will get enough of a stimulus to grow to a healthy size again.

Long-standing deep central sulcus infections are sadly very common. For some reason this is not something that many vets and farriers routinely look out for, even where the horse is exhibiting lameness associated with the heels. Where the problem is long standing, there may be permanent scar tissue in the frog corium, and the frog corium may also retreat further away from the ground in the

central portion. These factors make the likelihood of repeated central sulcus problems much higher even where LGL is no longer an issue, and a minority of horses will battle these problems for the rest of their lives.

Navicular Disease

Navicular disease is defined as changes to the quality of the navicular bone that result in heel pain. There is also navicular syndrome, where there is pain centred in the area of the navicular bone, but no changes are detectable on an x-ray – this is often seen as the precursor to navicular disease. Whilst a detailed description of navicular problems is beyond the scope of this book, a quick foray into this subject is relevant.

Both navicular disease and navicular syndrome have been poorly understood for many years. However, work by Dr Robert Bowker at Michigan State University has started to shed some light on the disease itself, and the processes that lead to it. It would appear that the early focus of damage in such cases is the impar ligament. This ligament attaches the navicular bone to the pedal bone and causes the two to move together more or less as a unit. A major part of the blood supply to the navicular bone is provided by blood vessels that pass through the impar ligament. If this ligament gets damaged, the blood supply to the navicular bone is compromised, and this in turn leads to erosion of the surface of the navicular bone. In particular, the part of the navicular bone over which the DDFT runs tends to become rough. This inevitably results in heel pain, which typically encourages the horse to start landing toe first.

The most interesting part of the proposed new model is the suggestion that the toe-first landing in itself contributes to damage to the impar ligament, creating a vicious circle. This may be because landing toe first bypasses the shock absorbers in the back of the foot and allows the vibration from the foot hitting the ground to be transmitted directly up the bone column. The impar ligament then becomes one of the first structures in line to have to cope with a massive increase in vibration, and potentially will suffer damage as a result. This model opens up the interesting possibility that navicular disease might actually start with a toe-first landing (caused by something other than navicular disease).

If so, then one possible route to navicular disease is via a deep central sulcus infection causing heel pain, and central sulcus problems are most common in LGL cases. If this model is correct, then it may be that navicular syndrome and navicular disease are actually, at least in a good proportion of cases, the result of LGL. The practical experience of EPA members in the UK is that a very significant proportion of navicular cases also show clear signs of LGL and long-standing central sulcus damage. Correcting both the LGL and the central sulcus infections typically leads to the progression of navicular disease halting, and even reversing. Early cases can often be brought back to full soundness. This experience lends weight to the suggestion that LGL may play a part in the development of a good proportion of navicular disease cases.

Under-Run Heels

More rubbish is written on the subject of under-run heels than almost any other subject in equine foot care! The most common misconception is the idea that a long toe somehow pulls the heels forwards. This is a classic example of assuming that because long toes and under-run heels tend to occur together, then one must cause the other. In reality both problems are actually caused by the same underlying condition – LGL.

In a healthy hoof, the angle at which the tubules of the wall sit at the heel is roughly the same as that at the toe. This is also the same angle as the front surface of the pedal bone, and the angle that all three phalanges sit at. This is no coincidence. The foot is designed to hit the ground in a very specific direction (remember the foot is travelling forwards at the same time as it approaches the ground), such that the force of impact travels up the leg entirely aligned with not only the angle of the phalanges, but

also the angle of the tubules in the hoof wall. In this orientation, these structures are immensely strong and can withstand the phenomenal forces involved in a heavy horse hitting the ground at speed.
The problem comes when a horse starts to land excessively heel first. This means that the foot at the point of impact is tipped up such that the heel tubules are more horizontal to the ground than they would normally be. The force of impact no longer travels directly along the length of the heel tubules, but rather acts partly across them. There is a component of the impact force that then acts to push the heel tubules forwards.

The hoof capsule itself is fairly plastic, as are the lateral cartilages, which have a role in holding the heels in the correct shape. These structures will bend to an extent, but repeated excessive bending results in them remodelling to a new shape. In this case, the heels will tend to move forward of their normal position. If the heels are artificially long (as is often the case in LGL), then the same force acts on the lateral cartilages via a longer lever, and hence has a bigger effect.

Once this process has started, it can easily become a runaway train. The wall at the heels is now at a shallower angle, which brings the heel bulbs closer to the ground. A farrier or trimmer will then be tempted to leave the heels longer in an attempt to prevent the heel bulbs becoming too close to the ground. This in turn makes for a longer lever, and hence the problem accelerates. You can end up with heel tubules that are growing almost horizontally forwards and are massively long. At this point it becomes impossible to leave the heels long enough to actually provide any degree of height, and the hairline at the heels ends up very close to the ground. The lateral cartilages are then no longer oriented in a way that allows them to hold the back of the pedal bone up, and the internal arch of the foot collapses. This brings the pedal bone flat to the ground (or worse, to a negative angle) which in turn tends to result in heel pain (because the back part of the pedal bone did not evolve to be that close to the ground). In the worst case scenario, the horse will then start to land toe first. This may temporarily improve the under-run heels, but risks pushing the horse in the direction of navicular disease.

Under-run heels are easy to recognize. Firstly, the furthest back part of the heel in contact with the ground ends up significantly forward of the widest part of the frog. Secondly, the angle of the tubules of the wall at the heel ends up shallower than at the toe. And finally, because the heel tubules always remain roughly at right angles to the coronary band that produces them, the coronary band at the heel gets pulled downwards. A healthy coronary band is roughly straight when viewed from the side of the foot. In a case of under-run heels, the coronary band curls downwards towards the heels. In addition to this, the failure of the internal arch results in the front wall of the hoof becoming less steep (giving an illusion of the toe being longer, even if there is no toe flare involved), although this takes some experience to recognize.

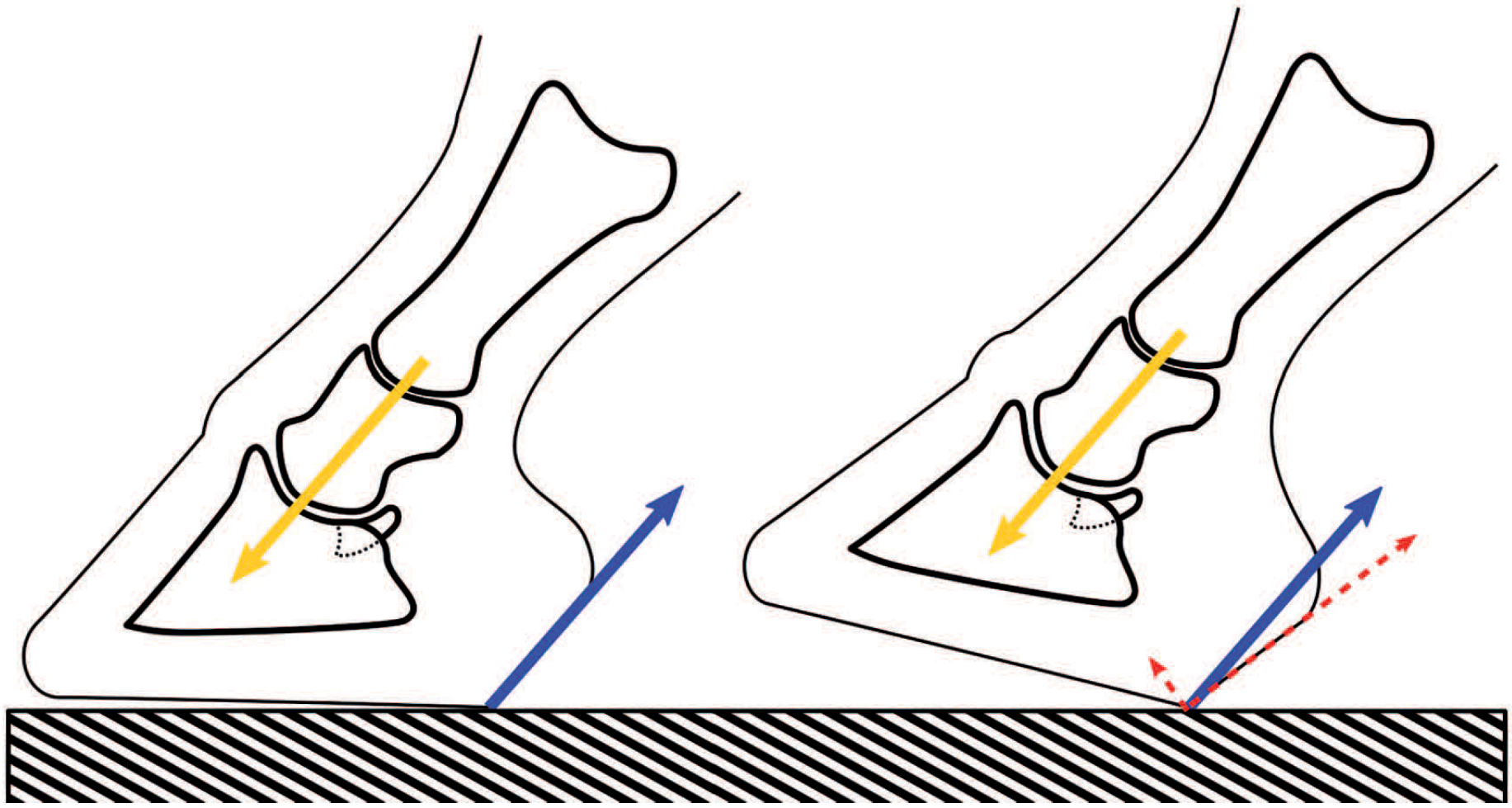


Fig. 79 LEFT: ***A foot landing correctly.*** RIGHT: ***A foot landing excessively heel first. The direction of impact is marked in yellow, and the force of impact will be parallel to this. At the point of impact there will be an equal and opposite reaction force from the ground on to the foot acting at the heel (blue arrows). With a normal landing, this reaction force acts directly along the tubules of the heel. With an excessive heel-first landing, the reaction force is not quite parallel to the tubules of the heel. This force is equivalent to two component forces at right angles (dotted red arrows). The main component still acts along the heel tubules, but there is now a smaller component, which acts to push the heels forwards.***

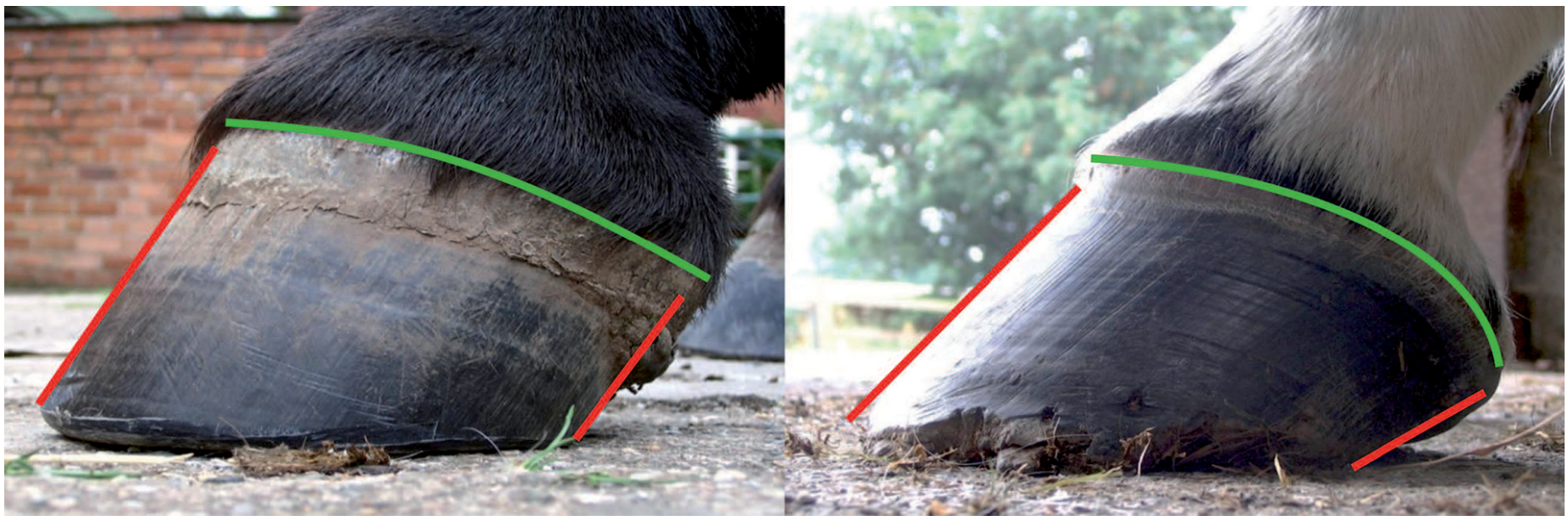


Fig. 80 LEFT: ***A healthy foot.*** RIGHT: ***A foot with under-run heels. The red lines show the angle of wall tubules. In the healthy foot these are parallel between the heel and toe; in under-run heels, the heel tubules are at a much shallower angle. The slightly shallower angle of the front wall of the hoof in the under-run heels case also indicates that the internal arch has collapsed, leaving the pedal bone parallel to the ground, or even tipped backwards slightly. The green line shows how the lateral cartilages (as indicated by the hairline) become distorted as the heels under-run, with the heel tubules always approximately at right angles to the hairline.***

Flat Feet

With the exception of feet that have suffered catastrophic bone damage as a result of severe full-blown laminitis, the bottom surface of the pedal bone is always concave. In a healthy horse, the sole should be largely of uniform thickness throughout so it is reasonable to state that a healthy horse should

always have a concave sole. Any attempts to blame flat-footedness on conformation are flawed. It is also a mistake to claim that certain breeds of horse are inherently flat-footed. This is an accusation often levelled at thoroughbreds in the UK, and yet the same breed (and even the same blood lines) in more arid countries shows no sign of flat-footedness. It is also possible to show that a thoroughbred brought up in a well-designed environment even in the UK can have upright and concave feet more reminiscent of an Arab's. And of course flat-footedness is never observed in young foals. So it would appear that flat feet are an acquired disorder associated with the environment in which the horse is kept, rather than something that can be blamed on breeding.

Fig. 81 An example of a flat foot. The normal concavity of the sole is filled with false sole. The front portion of the frog is also artificially thick, and the collateral grooves either side of the frog have filled with additional horn, as well leaving an almost totally flat surface. Early signs of the false sole exfoliating can just be seen at the rim of the sole.

There are three component effects that contribute to flat feet, all of which have already been described. The first is flaring. If the white line is pulled to a shallower angle to the ground, this in turn does the same with the rim of the sole. The solar tubules remain the same length but are at a shallower angle, resulting in the pedal bone being marginally closer to the ground and the sole being marginally less concave.

The second effect is the collapse of the internal arch associated with under-run heels. This brings the back part of the pedal bone closer to the ground, and with it, the apex of the frog (which sits just under the back of the pedal bone in the centre of the foot). Once the front part of the frog is in contact with the ground, this results in additional stimulus to the frog. This, combined with an already inflamed sole from the underlying LGL, results in the production of false sole around the frog, which is the final thing needed to create a flat foot. There may already be a degree of false sole produced as a direct result of the underlying LGL, and any lack of structure in the heel area will also contribute to the formation of false sole.

If the severity of the underlying problems is sufficient, then the entire central portion of the sole becomes inflamed and hence produces false sole. Because the stimulus comes from the centre of the foot (the apex of the frog) it tends to be strongest in the centre and tapers off towards the rim of the sole. The result is a plug of false sole that is thickest in the middle and thinnest at the edge, and hence neatly fills in any concavity in the sole. This situation is often easy to recognize because the groove between the sole and the apex of the frog appears significantly deeper than normal. However, in some cases, even this groove fills in with a combination of excess frog and sole, and the entire foot can look fairly normal apart from being table-top flat.

Of course, if the underlying LGL is removed and the mechanical issues are correctly addressed, the entire process can be reversed, although this is not necessarily a trivial undertaking. If the correct changes to the horse's environment are made, the false sole usually exfoliates away gradually as the foot recovers. Rarely, the false sole will jettison in a single piece allowing the mechanism of recovery to be clearly seen.

Wall Cracks

Wall cracks can happen for a number of reasons, but LGL is often a contributing factor.

Toe cracks usually form for one of two reasons. The first is where the hoof wall is allowed to become excessively long. The horse may be able to wear or break away some of the excess wall at the toe during breakover, but will find this more difficult in the quarters. The result is a hoof with flared wings on either side of the toe. These tend to act as levers, pulling the wall at the toe in two directions. A split in the toe is a common outcome.

The other common cause of a toe crack is a defect in the laminae called 'seedy toe'. This defect is not well understood, but seems to be very common in LGL cases. The defect is thought to start when a single horny lamina becomes damaged all the way from the white line to the coronary band (possibly as a result of the stretching effect previously discussed). This almost always happens close to the centre of the toe, and it creates a defect that allows rot to travel up the back of the hoof wall close to the laminae. Surprisingly, this does not usually result in an abscess, which suggests that the sensitive laminae may respond by producing a thin layer of additional horn to seal themselves away from the seedy toe. However, such a defect behind the wall weakens it, and small toe cracks near the ground are a common result. Such cracks can then get out of hand if the shape of the hoof capsule is not good or if the hoof is trimmed too infrequently.

There is also a suggestion that in some cases a seedy toe may be the result of a small benign tumour in the laminae called a keratoma. Most of these tumours do not grow aggressively and there is some suggestion that they can spontaneously disappear. They certainly appear in a surprising number of dissections. Rarely such tumours can grow rapidly and cause major problems – in such cases surgery is needed to remove them. There is a suggestion that keratomas may be more common in horses suffering from LGL, but there is just not enough data as yet to confirm this.

Quarter cracks form when the hoof wall is distorted beyond its tolerance. The hoof wall is designed to be a continuous curve from heel to heel, and always convex. Where heels become under-run, the wall just in front of the heel has to go somewhere to get out of the way of the heel. Typically this results in the quarter bulging outwards. This in turn then creates a concave crease in the wall just in front of the quarter (roughly where the lateral cartilage attaches to the pedal bone – the cartilage can distort, whereas the pedal bone cannot). The hoof wall does not cope well with being bent the wrong way, and will usually split to some degree at this point.

Cracks often allow moisture and infection to penetrate deep into the wall and into areas where blood products from LGL may be present. It is not uncommon to see a minor crack develop into a major problem as a result of infection.

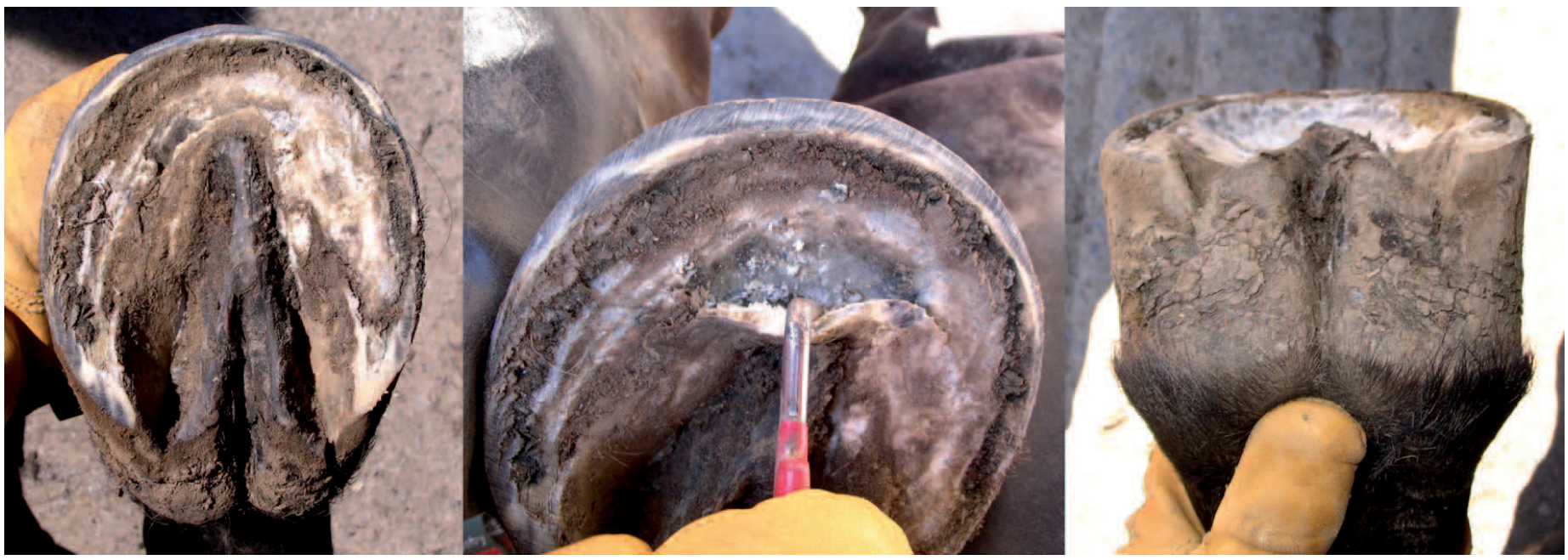


Fig. 82 A flat foot at the point of recovery. LEFT: *The foot at the start of a trim.* CENTRE: *The false sole has exfoliated as a single piece and is ready to remove.* RIGHT: *With all exfoliating material removed (no further sole or frog horn was removed), the foot now has a good level of concavity, although the overall health of the foot is still a work in progress.*

Abscesses

As with full-blown laminitis, LGL creates an increased risk of foot abscesses. Even fairly minor damage to the white line can be sufficient for bacteria to penetrate through to the solar and laminar coria. Bruising, particularly of the sole, then combines with the infection to create the right conditions for an abscess to form.

It might be expected that the risks of an abscess in LGL would be significantly lower than in full-blown laminitis. In reality, however, LGL is often missed, resulting in the horse being worked despite inflamed soles. This results in significant bruising to the sole and hence a high risk of abscessing, whereas a horse with full-blown laminitis is often put on box rest.

It is particularly worth noting that pulling shoes from a significant LGL case and then working it on roads without any form of protection to the feet creates a high risk of abscessing. It is suggested by some inexperienced proponents of keeping horses unshod that a newly barefoot horse should be worked on the roads to toughen up the feet. Sadly, not every horse with sore feet will give clear indications to the rider that all is not well, and even where the horse does attempt to communicate, not every rider will correctly interpret the signs and realize that the horse is footsore. It is not unheard of for a newly barefoot horse with LGL to abscess on all four feet within a few days or weeks of the shoes being removed. This is a key reason why it is important when removing shoes from a horse to have expert input from someone with sufficient training to be able to recognize any LGL that may be present, and who can give appropriate advice on the transition process.

Part 2
What Causes Laminitis?

5 The Established View on the Causes of Laminitis

Every few years, a new theoretical model is announced purporting to explain all or part of the underlying causes of laminitis. Sadly a good number of these models turn out to be incomplete, inaccurate, incorrect or less useful than originally claimed. The result is that, despite decades of research, we are still a long way from understanding the disease mechanisms that lead to laminitis. This chapter will attempt to outline some of the best known proposed models for the underlying causes of laminitis, and will discuss their strengths and weaknesses as well as examining some of the predisposing factors and triggers for an attack of laminitis. Few of the models attempt to describe the entire mechanism behind laminitis – most of them describe just one aspect.

CARBOHYDRATE OVERLOAD

The carbohydrate overload model is one of the best known models of how laminitis can occur, and seeks to explain how a horse develops laminitis after consuming excessive quantities of starch-rich grains. This can occur either through inappropriate feeding (for example feeding an over-sized bucket feed with a high grain content), or as the result of an accident such as the horse breaking into the feed store.

The horse evolved to eat a diet that is mostly fibre. Starches would have been present in the diet, but not in large quantities. The horse has also evolved as a trickle feeder, eating small amounts throughout the day, rather than discrete meals. Domestication introduced patterns of work that often prevented grazing/browsing for significant periods, and the feeding of cereals was introduced to compensate the horse for the calories lost from periods of being unable to eat. Any compensatory feed needs to have a higher calorie density than normal forage in order to supply sufficient calories in a short space of time, and cereals such as barley and oats fit this requirement well. However, this means that the horse is fed significant quantities of starch.

Starch is a polysaccharide molecule made up of many glucose molecules joined together to make very long chains. Like humans, horses cannot use starches directly for energy, but need first to break them down into individual molecules of glucose, a simple sugar. The main stage of this process is achieved by the use of the enzyme amylase, which is present in small quantities in saliva and in much larger quantities in the digestive juices secreted into the small intestine by the pancreas. Amylase reduces the starches to maltose (a disaccharide made up of two glucose molecules joined together) and maltotriose (three glucose molecules joined together), which can then be further broken down into single glucose molecules by the enzymes produced in the lining of the small intestine. The resulting individual glucose molecules are then absorbed into the bloodstream through the lining of the small intestine, to be used for immediate energy or stored for later use.

For this process to work correctly, the horse must be able to produce sufficient enzymes to fully break down any starch. If too much starch is fed in a short space of time, there is a risk that the

production of enzymes will not keep up. Undigested and part-digested starch can then make its way from the small intestine into the hindgut – a situation that evolution has not prepared the horse for well.

To add to the problem, the horse's stomach is relatively small, with a usable volume of around five litres. Where a horse is given a bucket containing more food than this, there is a risk that the ingestion of excess food results in the stomach contents being pushed through into the small intestine too early. This in turn pushes whatever food is currently being digested in the small intestine through into the hindgut too early. Furthermore, where the excessive meal contains significant starch, this dramatically increases the risk that undigested or part-digested starch ends up in the hindgut.

Where sugars and starches make it as far as the hindgut, they create havoc in the fermentation process that normally breaks down fibre. Large quantities of lactic acid are produced in response by the gut bacteria, and this upsets the pH balance of the hindgut, making it more acidic. Depending on the bacteria present, much larger than normal quantities of gases such as methane and hydrogen may be produced, which again may cause problems with digestion by their chemical action on the lining of the gut, as well as propelling food rapidly through the gut and increasing the risk of colic.

The resulting abnormal conditions tend to upset the balance of bacteria in the gut, favouring gram-positive bacteria such as *Streptococcus bovis* and *Streptococcus equinus*, which are particularly responsible for producing lactic acid and hence will acidify the gut further. The hindgut is normally only very slightly acidic, with a pH of around 6.8 (7 being neutral, and lower values representing increasing acidity). Where excess starches and sugars are dumped into the hindgut, the pH can reduce to as little as 4.0. This condition is referred to as 'hindgut acidosis'. It can be detected by testing the pH of droppings, or by testing blood for D-lactate, which is only produced in the body by bacterial fermentation of sugars and starches in the hindgut, and hence gives a good indication of the levels of acid being produced as part of acidosis.

Hindgut acidosis tends to significantly damage the lining of the hindgut. Within as little as twenty-four hours, the cells of the lining of the colon start to become damaged, and severe damage can have occurred within two to three days. The healthy gut lining is coated in a mucosal layer that protects the cells of the gut lining. The gut lining itself acts like a sieve, allowing small molecules that typically represent useful nutrients to pass through into the bloodstream, whilst larger molecules that are more likely to be toxic to the horse are kept inside the gut and ultimately excreted in faeces. As acidosis damages the lining of the gut, causing varying degrees of colonic ulcers, the mucosal lining is damaged and the gut wall develops microscopic holes. These holes allow larger molecules, including potential toxins, to pass through into the bloodstream – a condition known as 'leaky gut syndrome'. The degree of leaky gut can be measured in a research setting by introducing easily identifiable safe or inert molecules of various sizes into the diet, and then measuring the largest size of these molecules that reaches the bloodstream.

As well as increasing the risk of colonic ulcers, hindgut acidosis tends to cause the death and breakdown of some of the gut bacteria, forming toxic substances in the process. These toxic substances are largely made up of two components: endotoxins, which are originally contained in the cell membranes of bacteria, and exotoxins, which are chemicals normally stored within cells and secreted at low rates, but which may be released in large quantities as cells die and break down. If leaky gut syndrome is also present, then these toxins can leak through into the bloodstream with the potential to poison the horse.

Early advocates of the carbohydrate overload model proposed that endotoxins and exotoxins could be directly responsible for causing laminitis in the foot by some as yet unknown mechanism. Whilst this model is attractively simple, it sadly doesn't appear to be correct. Injecting these toxins directly into the blood does not result in laminitis, and the use of drugs known to be able to neutralize the effects of these toxins does not prevent the development of laminitis. Whilst disappointing, these findings don't necessarily entirely invalidate the carbohydrate overload model. It is possible that endotoxins and exotoxins may indeed cause laminitis, but only in the presence of some other pre-disposing condition. This pre-disposing condition could be entirely unrelated, or it could be something that also happens as

a result of hindgut acidosis. It is also possible that hindgut acidosis can cause laminitis by some other mechanism yet to be discovered.

Interestingly, researchers have found that if the horse is given the antibiotic virginiamycin (marketed for horses under the trade name Founderguard) for a few days before administering a carbohydrate overload, this seems to prevent both hindgut acidosis and laminitis from developing. The suggestion is that it prevents the explosion of the *Streptococci* bacteria that are thought to be the major cause of the acidosis. If this antibiotic treatment prevented all laminitis cases, then the problem of laminitis could be solved, but unfortunately this is not the case. Whilst laminitis associated with grain overload can be prevented by treating the horse continuously with virginiamycin, laminitis that occurs at pasture (the overwhelming majority of cases) does not seem to respond to this approach. Grain-overload cases are easily avoided anyway by using sensible feeding regimes and keeping the feed-store door locked, so this discovery isn't really as helpful as it would first appear.

FRUCTANS

There has been much hyperbole in recent years around the suggestion that a type of carbohydrate called 'fructan' that is present in grass might be the trigger for laminitis. If the articles in the popular horse press are to be believed, the cause of laminitis has been definitively identified, and simple changes to grazing management will prevent laminitis from occurring. Sadly, horse owners who follow such advice often find that the laminitis becomes worse rather than better. It is important, then, to take a good look at the fructans model to try to understand whether it has any merit or not.

Fructans are a family of molecules each made up of a number of fructose molecules joined together to form a chain, usually with a sucrose molecule at one end. There is a range of different types of fructans (each with varying chain length) including inulin, levan and oligofructose (fructooligosaccharide). Many plants use fructans as storage carbohydrates. Where the plant is producing more sugars than it can use (for example where there is strong sunlight but the air temperature is too cold for significant plant growth), it can convert sugars produced by photosynthesis into fructans that can be stored for later use.

It has long been suggested that high levels of water-soluble carbohydrates (WSCs) in the diet can be a trigger for laminitis in susceptible horses. WSCs include the simple sugars such as glucose and fructose as well as fructans (but not starch). The total level of WSCs in pasture can vary significantly both from day to day and seasonally, but it has been argued that fructans are responsible for far more of this variation than simple sugars. This might mean that sudden rises in fructans levels in grass have the potential to trigger a laminitis attack, and that any management approach that minimizes fructans levels would help to prevent laminitis.

The exact mechanism by which fructans might induce laminitis has not been pinned down. It is known that longer chain fructans can be broken down to shorter chain ones in the stomach and small intestine, but it is believed that fructans cannot be fully digested and absorbed until they reach the hindgut. Here they are fermented by bacteria to form volatile fatty acids (which the horse can use for energy) and lactic acid (which, as in the case of starch overload can, in large quantities, cause leaky gut syndrome). It has been suggested that the increase in acidity of the hindgut caused by the ingestion of large quantities of fructans might be part at least of the mechanism by which they might cause laminitis.

There are a number of aspects to this model that deserve detailed examination. The first key question is the level of fructans required to create a laminitis attack. Researchers have managed to trigger laminitis experimentally in horses with doses of around 2.5kg of oligofructose administered in a single dose to a 500kg horse. This is actually a fairly high dose of carbohydrates, given that the total daily intake of dry matter (that is, ignoring water content) for a typical 500kg horse is only 10kg. It should also be borne in mind that there are a large number of different fructans molecules, and the type of fructans present in UK grasses tends to be different from those used in research into laminitis.

In real life, the levels of fructans in UK grasses don't often get that high. There is only limited research into this area, but even the worst case studies (done on highly fertile dairy pasture under ideal conditions) only manage to reach a level of around 25 per cent fructans (by dry matter) during peak sunshine hours. For a 500kg horse to ingest 2.5kg of fructans it would have to eat grass totalling 10kg of dry matter (a whole day's intake) with this level of fructans in it. Of course, the 25 per cent figure only relates to the middle of the day, so for a good proportion of the day (including the night), the level of fructans will be significantly lower, making it difficult for the horse to ingest a daily total of 2.5kg even in these worst case conditions.

It is also important to recognize that the studies where laminitis is induced using fructans use a nasogastric tube to force the fructans into the stomach in one go. In real life, the fructans would be consumed slowly over a twenty-four-hour period – a very different situation, and likely to have a far less dramatic impact on the digestive system.

Another serious problem with the fructans model is that the research cases where fructans are used to induce laminitis show a range of clinical signs and symptoms that just don't show up in horses that develop laminitis at pasture. These include raised temperature, diarrhoea and changes to white blood-cell counts, as well as other easily detectable changes in blood results. This strongly suggests that the mechanism by which laminitis is caused in the lab by fructans differs from the mechanism by which horses acquire laminitis at pasture.

Moreover the seasonal variation in fructans levels doesn't match the typical pattern of incidence of laminitis in the UK. The seasonal peaks in fructans levels tend to happen in early spring and late autumn when there is strong sunlight for photosynthesis but the ground isn't warm enough for leaf growth. This doesn't really line up with the peak laminitis seasons, which tend to be *late* spring and *early* autumn. There is also a tendency for pasture-induced laminitis cases to happen on a warm day when it has just rained and the sun has come out – exactly the situation when the grass is best able to produce new leaf growth and hence least likely to convert excess sugars into fructans.

It would also be expected from the fructans model that all horses in a field with high fructans levels would get laminitis. In reality a few horses may have severe attacks whereas other horses are totally unaffected. This suggests that, even if fructans are the trigger for laminitis, there must be some pre-disposing condition that makes a horse sensitive to the fructans.

It is interesting to also examine the advice that is frequently associated with the fructans model. The general principle behind this advice is that grass creates large levels of fructans when it is 'stressed' – meaning that it is unable to grow despite being able to manufacture sugars in the leaves through photosynthesis. The problem comes when advice is given on what might constitute 'stress'.

There is plenty of evidence in published plant physiology research that lack of water and frost can cause grass to accumulate significant quantities of fructans. However, the idea that drought might cause laminitis goes completely against the observed evidence, and in the real world, laminitics tend to do well in drought conditions. Frost makes more sense, as discussed below. For all other forms of 'stress' there is absolutely no evidence at all that fructans levels rise as a result.

Heavy grazing or cutting is often cited as a form of stress that will increase fructans levels. Owners are therefore advised to avoid grazing their laminitic horses on pasture that has been grazed down significantly, and to constantly move horses on to new areas of pasture that haven't been heavily grazed. Sadly there is no evidence to back up these assertions. When cut or grazed, grasses respond with a rapid growth of new leaves. When cut or eaten, the total leaf area is dramatically reduced, so there is less photosynthesis to produce new sugars – and yet the plants need increased levels of sugars to fuel regrowth. The result is that fructans present in the stem are used, and overall fructans levels in the plants drop.

A big problem with many laminitics is preventing them from over-eating pasture (they often have increased appetite, and are also very sensitive to any excessive consumption of grass). The grazing schemes advocated by the supporters of the fructans model typically make it extremely difficult to limit total intake, and so horses over-eat and develop laminitis as a result. Recent research also shows that,

contrary to the fructans model, shorter grass not only contains lower levels of non-structural carbohydrates than longer grass, but also places less stress on the insulin system (*see below*).

Another common myth is that failure to fertilize a pasture results in ‘stress’ and raises fructans levels. This misconception results from a lack of understanding of the complexities of pasture ecology. A pasture is an ecosystem in which a more or less diverse collection of plant species compete for resources. Over time, the rule of the survival of the fittest results in a stable mix of species that are best suited to the conditions present at the time (factors such as grazing patterns, soil acidity, soil fertility, aspect, altitude, rainfall, temperature and so on). This is the main reason why attempting to create a ‘wild flower’ meadow by ploughing a dairy pasture and reseeding it is doomed to failure. The ‘wild flower’ species are poorly adapted to the high nutrient levels resulting from the use of agricultural fertilizers, and are rapidly out-competed by species such as rye grass that are far better adapted to such conditions.

If no chemical fertilizer is applied to a previously fertilized pasture over a period of years, the levels of nitrate, potassium and phosphate gradually drop. Nitrate levels drop fastest, taking two to four years to drop to more natural levels in a typical pasture. Potassium and phosphate levels drop far more slowly, taking as much as twenty years to reach more natural levels. Over time, the environment changes imperceptibly slowly, and the ecology of the pasture alters accordingly. Individual plants are out-competed, become stressed, die, and are replaced by other plants – but at an incredibly slow rate. The overall level of ‘stress’ in the pasture in this scenario is absolutely tiny. The idea that this situation creates high fructans levels is laughable. Indeed as the fertility gradually drops, the percentage of rye grass plants (which appear to be the best at producing high levels of fructans) gradually drops, and these are replaced by plants that are likely to produce far lower levels of fructans.

In contrast, the application of chemical fertilizer consisting of high levels of nitrate, potassium and phosphate (NPK) changes the soil conditions suddenly and dramatically. The rate at which the existing plants die off and are replaced by rye grass plants is likely to be far more rapid than in the previous scenario. So if ‘stress’ and hence high fructans levels result from plants being out-competed, then applying NPK fertilizer is going to create far higher levels of fructans than leaving the pasture to revert to a more semi-natural habitat. And that assumes that die-off through competition really does increase fructans levels – something for which there is no evidence whatsoever.

It is also claimed that frost increases the fructans levels in grass. This assertion is far better supported by the available research evidence. It makes sense that a plant that is frozen cannot grow, but potentially may be able to photosynthesize and produce sugars on a sunny but frosty day. There is also evidence that some plants use fructans as a form of anti-freeze in frosty conditions. This might explain the strong anecdotal evidence that some horses develop laminitis on frosty but sunny days. Of course, it is only a minority of horses that appear to be sensitive to this scenario, which again suggests that whilst fructans might be a trigger for laminitis, the horse has to have some pre-disposing condition first so as to be susceptible to frosted grass. Where a horse has a history of laminitis during frost, it makes good sense to keep the horse stabled during frosty periods, particularly if the sun is shining.

In summary, it looks extremely unlikely that fructans alone can cause laminitis, and despite the popularity of this model, many experts have expressed serious concerns about its validity. That doesn’t mean that fructans aren’t at least in part responsible for triggering laminitis – probably as part of the mix with other water-soluble carbohydrates – just that the emphasis on them has been over-stressed. It is also very important to recognize that much of the popular advice that has been attached to the fructans model is both wrong and potentially dangerous.

ARTERIO-VENOUS ANASTOMOSES (AVAS)

Arterio-venous anastomoses (AVAs or AV shunts) are short cuts in the circulatory system that can divert blood from an artery directly to a vein, so bypassing the capillary bed that supplies blood to extremities of the body. With AVAs closed, blood has to flow through a network of fine capillary blood

vessels, and the resistance that these capillaries presents limits the overall volume of blood that can flow down the artery and back up the vein.

In human limbs, some AVAs are used for cooling. In hot conditions, these AVAs open, allowing extra blood to flow down the artery and back up the vein in addition to the normal flow of blood through the capillary bed. The veins in question lie close to the surface of the skin and hence are well placed to act as radiators, dumping heat into the surrounding air and hence cooling the body. In colder conditions these AVAs close, so reducing the amount of blood flow to the limb to the minimum needed to keep the capillary bed and associated tissues healthy. With less blood flowing through surface veins, less heat is lost. In even colder conditions, the capillary blood vessels will also contract, reducing the flow of blood through the limb even further – ultimately this is at the risk of compromising the blood supply to (and hence damaging) peripheral tissues, though it will preserve life by maintaining the core body temperature.

In contrast, the AVAs in a duck's foot are designed to open at low temperatures so as to increase blood flow to the foot and hence prevent the foot from becoming cold enough to suffer damage. It would seem that AVAs in different parts of the circulatory system can perform diametrically opposing functions.

In the early 1990s, researchers suggested that AVAs might open inappropriately in acute laminitis cases (perhaps as a result of toxins released from the gut), resulting in so much blood being diverted that none would make it to the capillary bed of the laminae. This would then lead to tissue death due to lack of oxygen, starting the whole process that we call laminitis. This proposed model has caused a degree of argument between researchers, with some claiming that the relevant AVAs can't open sufficiently to fully divert the arterial supply to the laminae, and others claiming that there aren't sufficient AVAs in the toe area of the foot to explain the pattern of damage seen in laminitis. Today, this model is generally seen as discredited as a mainstream cause of laminitis. It is now widely accepted that any opening of AVAs during acute laminitis is the result of damage that has already occurred to the capillary bed, rather than a causative factor. There is, however, one situation in which AVAs may still be relevant.

If a horse's foot behaves like a duck's, then there may be AVAs that are responsible for increasing the overall throughput of blood, and hence the delivery of heat to the foot in cold conditions. There does seem to be some support for this suggestion, not least in the ability of horses to survive in extremely cold weather without getting frostbite in their feet. It is tempting to suggest that such AVAs could open too far in laminitis and divert blood from the laminae, but this does not fit with the evidence from real life. If this were the case, then cooling the foot down (and hence opening the AVAs further) would make things worse. In reality, the opposite is true, and placing the feet in ice in the early stages of an acute attack helps to reduce the severity of the attack.

An intriguing suggestion, though, is that chronic laminitis might, over time, damage these AVAs to the point where they are less able to open in cold temperatures. This would result in a failure of the body to keep the foot adequately warm in icy conditions, with the potential for sensitive tissues such as the laminae to become badly damaged as a result. This might be one explanation for why horses generally cope well with icy conditions, and yet in a minority of horses, cold weather can trigger laminitis attacks.

VASOCONSTRICTION

The walls of arteries contain significant amounts of smooth muscle that allow the artery to contract, reducing blood flow, under certain circumstances. This process is known as 'vasoconstriction' and may occur for a number of reasons. Vasoconstriction in peripheral tissues can reduce heat loss in cold weather or reduce bleeding from an injury site. Vasoconstriction is also involved in the maintenance of blood pressure by reducing the volume of the circulatory system and hence increasing blood pressure.

One suggested mechanism for the causation of laminitis is that vasoconstriction is triggered abnormally in the foot (perhaps as the result of circulating toxins produced in the gut), resulting in a

near total loss of the blood supply to the foot (known as ‘ischemia’). If this lack of perfusion occurs for any length of time, then the tissues of the various coria will be starved of oxygen and will start to die, losing their strength in the process. This then would allow the foot to fail mechanically under load, as described in previous chapters.

It might seem reasonable that the end of the abnormal vasoconstriction event would return blood and oxygen to the damaged tissues and prevent further injury. In reality the damaged cells are poorly prepared to deal with the influx of oxygen, and so further chemical damage occurs (known as a ‘reperfusion injury’). The combination of damage from both ischemia and reperfusion injury can be devastating, and is well understood in human medicine as a potential risk from a crush injury.

Researchers have been able to show that circulation to the toe area of the foot is indeed dramatically lost during an acute laminitis attack, which might suggest that the vasoconstriction model has merit. Unfortunately it has not been possible to show that the loss of circulation occurs at the start of the laminitis attack, and hence might be the cause rather than a result of the damage. Furthermore, there is strong evidence that cooling the foot down with ice (and hence increasing the degree of vasoconstriction in the various coria) during the early stages of a laminitis attack actually reduces the severity of the attack – a finding that is not at all consistent with the vasoconstriction model.

It now seems more likely that the loss of circulation is the result of damage that has already occurred during previous stages of the development of acute laminitis.

VASODILATION

Studies of hoof temperature during acute laminitis attacks have shown that the foot becomes significantly warmer in the early stages of an attack. This tends to suggest that vasodilation – the relaxation of blood-vessel walls so that they dilate, allowing increased blood flow – may be part of the process in acute laminitis. As ever, the difficulty is to establish cause and effect. One suggestion is that the increased blood flow interferes with normal tissue fluid balance, resulting in the collection of fluid in the foot, and that this fluid then causes damage to the various coria.

A more credible suggestion is that vasodilation increases blood flow through the foot, and hence increases the foot’s exposure to any toxins circulating in the blood. Hence whatever toxin triggers the laminitis will do more damage if vasodilation occurs at the time. But what triggers the vasodilation? If the underlying disease process triggers the vasodilation, then that would suggest that the same processes both trigger vasodilation and separately cause sufficient damage to the coria to result in laminitis. This would seem to be something of a coincidence. Perhaps a more credible suggestion is that vasodilation is a knock-on effect from the damage initially done by circulating toxins within the foot, which allows more toxins to arrive and hence accelerates the rate of damage. It all depends on the timing, and in the absence of a definitive answer on the mechanisms by which the coria become damaged, the relative timing of the various stages of damage is difficult to prove.

MATRIX METALLOPROTEINASES

Matrix metalloproteinases (MMPs) are zinc-containing enzymes capable of breaking down the proteins that bind cells together in connective tissue. There are a number of different MMPs, and they have important roles within the body relating to structural changes at a cellular level.

Two MMPs, MMP-2 and MMP-14, are known to be present in fairly large quantities in the basement membrane of the laminae, and the hemidesmosomes that attach the basal cells of the secondary laminae to it. These MMPs are normally in an inactive form, and in a healthy horse only a small proportion of them would be activated at any one time. It is suggested that this forms the mechanism by which the epidermal laminae can slide past the dermal laminae, allowing the hoof wall to travel downwards whilst maintaining a strong attachment to the bone via the laminae. If MMPs are activated selectively, the basal cells can detach from the basement membrane, move and reattach to the basement membrane in ripples. Despite this selective detachment and reattachment, the majority of

basal cells are firmly attached to the basement membrane at any one time so as to provide a strong junction.

The MMP model of laminitis suggests that some blood-borne trigger (maybe endotoxins produced in the hindgut) activates most, or all of the MMPs in the laminae simultaneously. The result would be that the normally strong junction between the basement membrane and the basal cells would detach, resulting in mechanical failure of the laminae consistent with the damage seen in the early stages of laminitis.

The MMP model has been studied extensively in horses that have had laminitis induced using fructans. In this scenario, it appears to be a credible model. The big question is whether the same mechanism occurs in pasture-induced laminitis – the variety that forms the overwhelming majority of cases in the UK.

INSULIN RESISTANCE

Insulin resistance increasingly appears to be a key part of the mechanisms underlying laminitis in the majority of real life cases. To understand insulin resistance, it is first necessary to understand how the body regulates blood glucose.

The horse has evolved to get most of its energy from volatile fatty acids (VFAs) produced by the bacterial fermentation of dietary fibre in the hindgut. After VFAs, the next most important energy source is glucose, some of which is present in food as glucose, but much of which is the result of enzymatic breakdown of disaccharides and starches.

As any diabetic human will know, it is vitally important to the body that the level of glucose in the blood is kept within certain bounds. If glucose levels drop too low, a situation known as hypoglaecemia, the horse will tend to show signs such as lethargy, incoordination, sweating and confusion (and eventually death). If glucose levels become too high, this is termed hyperglaecemia. With severe hyperglaecemia, the horse will tend to show signs such as excessive hunger, excessive thirst and urination, and sleepiness (and eventually death). Lower levels of hyperglaecemia do not produce the same dramatic effects, but over a long time period can have severe detrimental effects on, for example, the vascular system.

As glucose is absorbed into the bloodstream through the lining of the gut, it causes blood glucose levels to rise. This rise is detected by the pancreas, which responds by producing insulin. Insulin is a hormone that signals to cells (mostly fat and skeletal muscle cells, but also liver cells) to take glucose out of the bloodstream and store it for later use, so reducing blood glucose levels. As blood glucose levels drop back towards normal, the production of insulin by the pancreas tails off, allowing blood glucose levels to stabilize.

Where blood glucose levels fall (for example, as a result of exercise where muscles will use glucose and deplete blood levels, or as a result of a failure to eat sufficient glucose-containing food), this is detected by the pancreas, which releases a hormone called glucagon. Glucagon causes the liver to release stored glucose. A drop in blood glucose is also detected by the hypothalamus gland (at the base of the brain), which instructs the pituitary gland (next to the hypothalamus) to produce a hormone called ‘adrenocorticotropic hormone’ (ACTH). This in turn signals to the adrenal glands (one of which sits on top of each kidney) to produce the hormone cortisol. Cortisol opposes the effects of insulin (although it also has many other actions). It blocks the uptake of glucose in response to insulin by fat and muscle cells, and promotes the release of stored glucose by the liver. As a result cortisol, like glucagon, acts to raise blood glucose levels.

Although there are significant further subtleties and complexities to the system, the fundamentals of glucose regulation are that cortisol and glucagon both boost blood glucose, and insulin reduces it, under feedback mechanisms such that blood glucose is always maintained within healthy bounds.

Insulin resistance is defined as the situation where the response of cells to insulin is reduced. As a result, the pancreas has to produce more insulin than normal (raising blood insulin levels to a higher level) in order to trigger the same amount of glucose to be stored away. The pancreas has a

mechanism to detect whether its insulin production is having the required effect on blood glucose, and where insulin resistance occurs, the pancreas compensates for this by increasing the level of insulin produced for a given rise in blood glucose. This situation is then termed ‘compensated insulin resistance’. In compensated insulin resistance, the blood level of insulin is higher than normal in response to a given quantity of glucose being absorbed into the bloodstream from the gut, but the regulation of blood glucose is still effective, so glucose levels stay within the normal range.

If the underlying level of insulin resistance becomes too great, the pancreas eventually reaches a tipping point where it is no longer able to produce ever larger amounts of insulin to compensate for the resistance. The pancreas may now be able to fully compensate for small rises in blood glucose, but any large rise in blood glucose results in a failure to produce sufficient insulin to compensate for the resistance. The situation is now termed ‘uncompensated insulin resistance’ (more commonly known as Type 2 diabetes), and blood glucose will start to rise above acceptable levels, causing a huge range of short- and long-term health problems. Whilst it is not unheard of for a horse to reach the point of uncompensated insulin resistance, it is rare.

The levels of insulin produced in response to even modest ingestion of glucose-containing food can be massive in some insulin-resistant cases. In a healthy horse, the maximum serum level of insulin normally seen when the horse is eating a high sugar diet is around 30mIU/l (milli international units per litre). It is possible to see insulin-resistant horses with serum insulin levels in excess of 700mIU/l, despite attempts by the owner to keep sugar and starch intakes low.

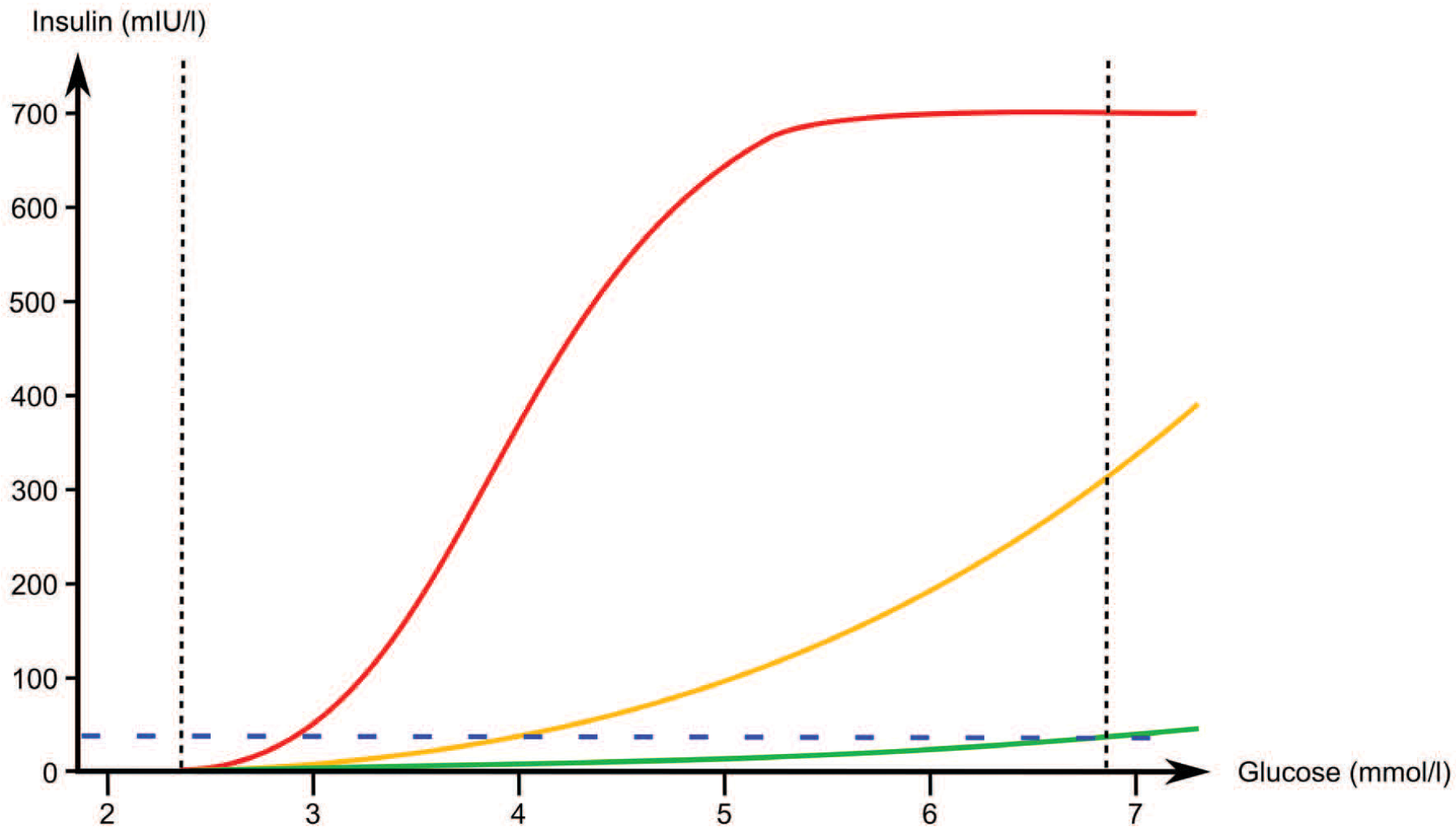


Fig.83 A stylized graph of the relationship between the blood insulin level and the steady state blood glucose level. In a healthy individual, glucose should stay between the two dotted black lines, and insulin should be below the dashed blue line. The green line shows the response of the pancreas to a specific glucose level in a healthy horse. The yellow line shows the response in a horse with compensated insulin resistance. The red line shows the response in uncompensated insulin resistance (Type 2 diabetes). In reality the response is more complex, because, for example, the pancreas produces an additional transient insulin spike in response to any sudden increase in blood glucose.

Recent research has strongly suggested that horses that develop laminitis at pasture tend to have at least some degree of insulin resistance, though the exact mechanism by which insulin resistance causes laminitis is not yet known. It is becoming increasingly clear, however, that insulin resistance is the pre-disposing factor, with the consumption of sugary or starchy food being the trigger. Given that

blood glucose levels typically remain normal in these horses, it is likely to be the sudden and dramatic increase in insulin which triggers damage in the feet. This might explain why so few horses develop Type 2 diabetes – they are usually euthanized as a result of laminitis before the insulin resistance becomes sufficiently severe to result in a loss of compensation.

In addition to a predisposition for laminitis, horses with insulin resistance (particularly more severe cases) often show a number of other clinically recognizable signs. The most obvious of these is a tendency for the horse to lay down fat abnormally, a condition known as ‘regional adiposity’. If an otherwise healthy horse puts on excessive weight, the additional fat tends to be distributed fairly evenly throughout the body. In contrast, an insulin-resistant horse will usually put fat down preferentially in the crest, shoulders and hips, as well as sometimes in the loins, sheath/udders and on either side of the tail head. In the most severe cases, randomly placed lumps of fat may appear almost anywhere on the torso.

Fig. 84 An example of an insulin-resistant horse showing regional adiposity in the crest of the neck. This horse was the correct weight overall.

When assessing the condition score of a horse, it is important to note that regional adiposity can skew the results. The condition score is normally assessed in three sections: neck, back/ribs and pelvis. A horse that scores far higher in the neck and pelvis than in the back/ribs is likely to be insulin resistant. It is common for horses to be incorrectly described as obese as a result of abnormal fat deposits in the neck and pelvis, despite having obviously visible ribs. An assessment of the degree of fat covering the ribs alone provides a far more reliable indication of overall body condition, with any higher scores in the neck and pelvis being indicative of additional fat laid down as a result of insulin resistance. Putting a horse with a poor covering of fat on the ribs but a large crest and rump on a diet to reduce weight is unlikely to be helpful in terms of laminitis (and may prove counterproductive).

It is suspected that, as in humans, regional adiposity in horses represents the deposition of abnormal fat that is in a pro-inflammatory state and produces adipokines (chemicals that affect the behaviour of fat cells). That in turn helps to perpetuate the pro-inflammatory state and encourages

further fat deposition. This type of fat also appears to have a multiplier effect on the severity of insulin resistance because the same adipokines appear to increase insulin resistance both locally and in other parts of the body.

As well as putting fat down unevenly, insulin-resistant horses tend to put on weight more easily than other horses, and often have an increased appetite. There is some debate about how much of this increased weight is due to increased appetite, and how much is due to the horse converting more energy to fat than normal. It is often argued that in order for a horse to put on weight, it has to overeat (leading to owners of insulin-resistant horses almost always being blamed for their excessive weight). Recent research in mice, however, has shown that changes in gut bacteria can significantly affect the efficiency with which the animal converts food into fat. The result is that two otherwise identical mice with different gut bacteria that are fed the exact same diet can have very different rates of weight gain. It would appear that the gut flora types that are associated with obesity are also associated with insulin resistance. This is not to say that overfeeding doesn't play a part in the creation of obesity in some insulin-resistant horses, just that it is dangerous to assume that the owner is always entirely to blame. With more severely insulin-resistant horses it can be extremely difficult to reduce their weight without starving the horse to the point of risking other adverse health issues, such as gastric ulcers.

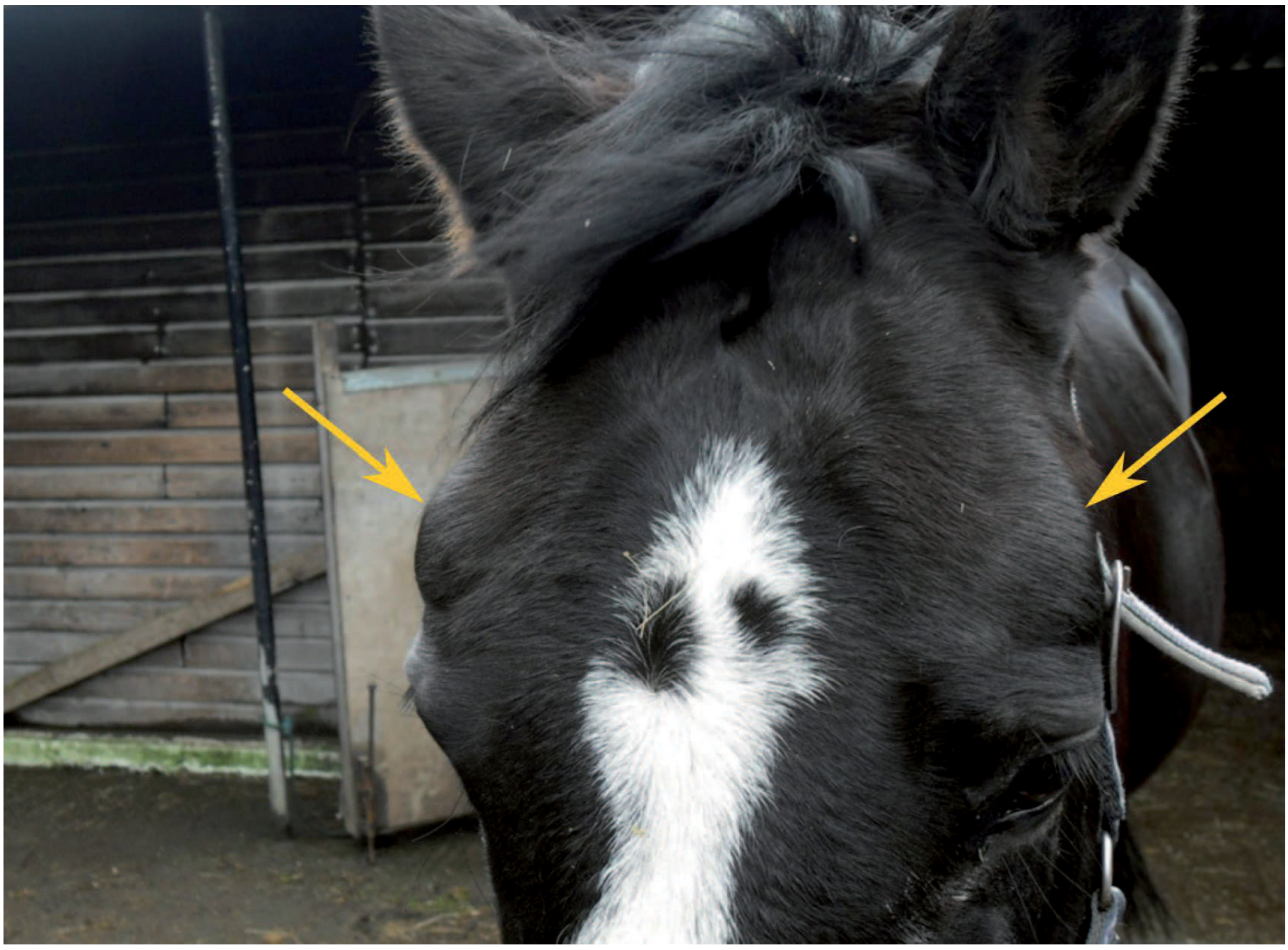


Fig. 85 An example of oedema in the supra-orbital fossae (arrowed).

To complicate the picture further, obesity itself appears to contribute to the causation of insulin resistance. A horse that is consistently overfed so as to become obese appears more likely to develop insulin resistance than an under-weight horse. So if a horse is allowed to become obese, it is more likely to develop insulin resistance, and the obesity will then make that insulin resistance worse. For that reason it is important not to allow horses to become overweight, and where insulin resistance has already occurred, it can often be reduced to a significant extent by slimming down an obese horse.

Another common misconception is that insulin resistance only affects overweight horses. If this were the case, then insulin resistance would be an avoidable condition. In reality, it is entirely possible for a horse to be severely insulin resistant and yet be the correct weight, or even underweight. This suggests that obesity isn't the only (or perhaps even the main?) cause of insulin resistance. There is even an argument that the assumption that obesity can cause insulin resistance may be incorrect. It is possible that horses develop insulin resistance for some other reason, and then develop a tendency to gain weight, which in turn increases the severity of the insulin resistance. This would create a correlation between obesity and insulin resistance without obesity causing insulin resistance.

An underweight or correct weight horse with insulin resistance is harder to spot because there is often far less, or even no obvious regional adiposity present. For this reason, such cases are often missed, and hence all horses with laminitis should be blood tested for insulin resistance regardless of their weight.

As well as the abnormal distribution of fat, horses with insulin resistance have a tendency to develop oedema – the collection of excess fluid in tissues. The oedema tends to be particularly focused on any areas where there is already abnormal fat deposition. Most notable is the crest. Where a horse has an abnormally fatty crest, that crest will tend to go hard at certain times. This hardening of the crest represents a build-up of significant amounts of fluid within the fat, and seems to be associated with periods where glucose intake has risen and hence insulin levels are dramatically high.

Another location where fluid collection is particularly noticeable can be the supra-orbital fossae (the depressions just above the eyes). In some (but not all) insulin-resistant horses, these depressions fill to a small extent with abnormal fat. Where this happens, the fat in the supra-orbital fossae seems particularly susceptible to oedema, and this can result in bulging pads appearing above the eyes at times of high glucose intake. These pads are sometimes incorrectly described as 'fat pads', but the majority of the volume is made up of water, as evidenced by their ability to appear and disappear within a matter of hours.

Fluid often also accumulates towards the bottom of the abdomen. Sheaths and udders can become swollen – sometimes dramatically so. The sheer quantity of fluid that can accumulate is surely more than could be generated by the small quantities of abnormal fat in this area. That might suggest that perhaps the fluid is being generated in abnormal fat around internal organs within the abdomen, and is then draining to the lowest point in the abdomen.

Whilst not all insulin-resistant horses have sufficient abnormal fat to result in visible oedema, in those that do it can be an extremely useful indicator. The presence and severity of oedema seems to accurately predict the likelihood and severity of a laminitis attack. Owners can monitor the levels of fluid above the eyes and/or in the crest, and use any significant increase as a warning sign of potential laminitis. The specific level of oedema that corresponds to a significant laminitis event is individual to each horse, but over time, owners can learn to spot the warning signs and take appropriate early preventative action, such as reducing the area of grazing available and increasing the intake of hay.

The combination of insulin resistance and the tendencies to obesity, abnormal fat deposition and fluid retention (along with other clinical signs such as raised blood pressure and abnormally high levels of triglycerides in blood) is collectively referred to as 'equine metabolic syndrome' (EMS), although the terms 'insulin resistance' and 'EMS' are often used interchangeably.

Accurate testing for EMS is difficult. Whilst signs such as elevated blood triglycerides are useful circumstantial evidence, a definitive diagnosis needs the identification of the severity of the insulin resistance. The gold standard test is the hyperinsulinemic- euglycaemic clamp technique. In this test, insulin is infused into a vein to maintain a constant artificially high level, whilst the blood glucose level is continuously monitored. On its own, this would cause blood glucose to drop below normal levels in a healthy individual. Glucose is then also infused into a vein at gradually increasing rates until the blood glucose comes back up to normal levels. The level of glucose infusion needed to keep blood glucose levels at a constant normal level is measured. This gives a measure of insulin resistance. In a healthy horse, the insulin has a large effect and a lot of glucose is needed to bring blood levels back to normal.

In an insulin-resistant horse, less glucose is needed because insulin has less of an effect. Unfortunately this test is complex and expensive and cannot realistically be used in daily clinical practice.

At present the most common means of testing in practice is to take a spot measurement of the blood insulin level. A higher than expected level suggests insulin resistance. Unfortunately this approach is poor at picking up mild cases, as is discussed in the next chapter.

GLUCOSE DEPRIVATION MODEL

The glucose deprivation model results from a set of experiments where samples of laminae were taken from recent cadavers and incubated in various solutions. Those kept in solutions containing glucose remained intact for up to eight days, but in those kept in solutions without glucose, the laminae separated within thirty-six hours. This separation occurred at the junction between the basement membrane and the basal cell layer, as is typical when acute laminitis is studied in the lab. This suggests that the laminae require adequate glucose to remain structurally strong, and laminitis will result if the laminae get too little glucose. This in turn suggests that the process of holding the laminae together is an active one that requires energy.

The issue with this model is the need to explain why the laminae would become starved of glucose. The study's authors suggested that the underlying conditions that cause laminitis might cause the peripheral tissues, such as the foot, to gradually shift away from using glucose as an energy source. They suggested that if this shift happened too fast, the foot wouldn't be able to adapt, and hence would be temporarily starved of glucose, resulting in laminitis. Whilst the finding that glucose deprivation can cause laminitis is clearly of interest, it is a huge leap to assume that the laminitis we see in real life is caused by this particular mechanism. The arguments for why glucose deprivation would occur seem somewhat tenuous and poorly justified.

PITUITARY PARS INTERMEDIA DYSFUNCTION (PPID, OR CUSHING'S SYNDROME)

Cushing's syndrome is a term used to describe the effects on the body of being exposed to higher than normal levels of the hormone cortisol. Cortisol is the main long-term stress hormone in the body, as compared to epinephrine (traditionally termed adrenalin) which is produced in response to short-term stress. Cortisol is produced where an animal is under psychological or physiological stress for a period of time, and is also produced in response to a drop in blood glucose.

Clinical signs of excess cortisol in a horse include: rapid weight gain (but weight loss in more severe cases) with increased appetite; fluid retention; hirsutism (the development of a longer coat, sometimes lighter in colour, that does not fully seasonally shed or sheds late compared to healthy horses); increased drinking/urination; abdominal bloating; muscle wastage (resulting in a sway back in more severe cases); lethargy; suppression of the immune system (often resulting in skin infections) and abnormal sweating patterns (too much, too little, or patchy sweating). In addition to this long list of signs, the high levels of cortisol act to oppose insulin and hence create insulin resistance, which in turn causes regional adiposity, oedema and laminitis. Traditionally, the first sign of Cushing's was thought to be a long curly coat. This results from the combination of hirsutism and excessive sweating. Recent research has shown that the long curly coat is often far from the first clinical sign to manifest, with laminitis, regional adiposity, weight gain and skin infections often being among the first presenting signs.

Laminitis in Cushing's syndrome cases is often worse in spring and autumn. At first sight this might suggest an association with sugar-rich grass from the spring and autumn growth flushes, but even cases that have no access to grass sometimes show this pattern. One suggestion is that the increased rate of laminitis is associated with hormonal changes that occur when the winter or summer coat is shed.

Cushing's syndrome can occur for a number of reasons, including as a result of the administration of cortisol (as hydrocortisone) or other steroids as a treatment. For that reason, vets have to be extremely cautious when prescribing steroids for horses, especially where there is a history of laminitis. Prednisolone is commonly used in veterinary applications as a drug to suppress the immune system (for example in the treatment of auto-immune conditions such as pemphigus). Prednisolone is a corticosteroid that has a similar effect to cortisol, and hence acts to mobilize glucose from cells into the bloodstream. This means that a higher than normal level of insulin is needed to reduce blood glucose by a given amount, and hence prednisolone also causes insulin resistance. Recent research has suggested that the use of prednisolone is reasonably safe in horses that don't have a prior history of laminitis. However, prednisolone can potentially trigger serious laminitis attacks in horses that already have EMS. For that reason, the use of prednisolone in horses with a prior history of laminitis should be avoided wherever possible.

Hydrocortisone injections into joints as a treatment for arthritis (which are metabolized to cortisol in the body) are normally less of a risk despite huge doses being used, because the hydrocortisone should remain sealed within the joint cavity. The main risk with joint injections is that the injection misses its intended target and is delivered directly or indirectly into the bloodstream. Such cases typically result in severe, acute laminitis.

In humans, the most common cause of Cushing's syndrome, after the side effects of medication, is a tumour called an adenoma in the part of the anterior pituitary gland responsible for producing ACTH. In this condition (known as Cushing's disease), the tumour cells produce too much ACTH, which in turn causes the adrenal glands to produce too much cortisol. Cushing's syndrome in horses was originally assumed to occur via the same mechanism, but this is now known not to be the case.

Neurons of the hypothalamus instruct cells called melanotropes in the pars intermedia region of the pituitary gland to release the chemical dopamine (which is used to send messages between nerve cells), which in turn triggers dopamine receptors on the melanotropes. Activation of these receptors inhibits the production of a chemical called pro-opiomelanocortin (POMC), which is used in the pituitary gland as a precursor for the manufacture of various hormones. In equine Cushing's syndrome, far less dopamine than normal is produced, resulting in runaway production of POMC. Where a gland is instructed to produce far too much of a chemical, the attempt to keep up with the increased demand results in the enlargement (hyperplasia) of the gland over time. In equine Cushing's syndrome, the pars intermedia enlarges, potentially causing pressure and damage to adjacent structures within the pituitary or even in adjacent tissues such as the hypothalamus.

POMC is cleaved to form two chemicals, one of which is ACTH. In a healthy horse, the amount of ACTH produced via this route is insignificant (around 2 per cent of the total). The majority of the ACTH is produced in the anterior region of the pituitary under feedback control via the hypothalamus. But in the presence of excess POMC, the levels of ACTH produced in the pars intermedia dwarf the levels produced under control of the hypothalamus, resulting in increased cortisol levels and hence Cushing's syndrome. This cascade of events is termed 'pituitary pars intermedia dysfunction' (PPID), and this is the correct term to describe the underlying pathology that causes Cushing's syndrome in horses.

The exact cause of the drop in dopamine production in the pars intermedia is the subject of continued research, but the current favoured proposal is that the nerves from the hypothalamus that normally control production become damaged as a result of oxidative stress. This might be due to a local or systemic deficiency in the anti-oxidants used to protect against normal chemical wear and tear from free radicals, or it might be the result of a significant increase in the levels of free radicals present in the pituitary gland. Either way, more research is needed to chase the chain of causative events back to their source. It is known that the damage appears to be largely irreversible, and in the absence of a known means of preventing further damage, the condition tends to be progressive. Interestingly, cases often tend not to show a linear rate of increase in the severity of PPID, but rather sudden and unpredictable step increases from time to time. This might suggest that there is a degree of external environmental influence over the underlying causes of PPID.

Although more severe PPID cases show a range of obvious and fairly distinctive clinical signs, diagnosis (especially in mild cases) can only be confirmed by the use of blood tests. The simplest test available is to measure the level of ACTH in blood. In all but the mildest cases of PPID, the level of ACTH will be well above the normal levels seen as a result of normal physiological regulation. The normal range of ACTH varies seasonally, with higher levels typically seen in spring and especially autumn. For some reason, this seasonal increase in ACTH is magnified in PPID cases, which makes it easier to detect borderline cases between August and October – hence this is the preferred time of year to perform blood tests for PPID.

The main issue with ACTH testing is that it is not sensitive enough to detect mild cases of PPID. ACTH is a stress hormone and hence can be raised in a healthy horse for good physiological reasons (such as hunger, fear, pain). As a result, the top end of the reference range for ACTH represents the elevated level seen in a healthy individual under a degree of mild stress (the presence of a vet armed with a needle, for example!). A horse with mild PPID may exhibit levels below this threshold and yet still have clinical signs such as mild laminitis. This is for two reasons: firstly, the normal peaks of ACTH due to stress are generally transient, whereas the levels in PPID are constant; and secondly there are other, as yet poorly understood, hormonal changes as a result of elevated levels of POMC that may have clinically significant effects even where the level of ACTH alone is not yet high enough to cause problems in itself. As well as a proportion of false negative results, false positive results are sometimes seen. This happens where the level of physiological stress (for example, pain from laminitis caused by something other than PPID) is sufficient to push ACTH above the levels seen in a normal healthy individual. For that reason, testing for ACTH during an active laminitis attack doesn't always give a reliable answer.

A potentially more accurate way to detect PPID is the dexamethasone suppression test. In a healthy individual, an increase in ACTH stimulates additional cortisol production, which in turn provides feedback to the pituitary gland to reduce ACTH again. Dexamethasone is a drug which the pituitary gland cannot distinguish from cortisol. The injection of a large quantity of dexamethasone should result in ACTH production and cortisol levels dropping to near zero as the pituitary recognizes what appears to be an over-production of cortisol. In PPID, ACTH production from the anterior pituitary is still suppressed, but the ACTH produced from POMC by the pars intermedia is not. Hence a failure of ACTH and cortisol to drop in response to the dexamethasone indicates the presence of PPID. Unfortunately, the natural rise in ACTH in autumn seems to result in false positive results from this approach, which tends to suggest that this seasonal rise is not under the control of the feedback mechanism. The test does, however, appear to be accurate if performed in the winter months. Of more concern is the potential for the dexamethasone itself to trigger laminitis, making this a test that vets are reluctant to use on horses with a history of laminitis.

The other approach commonly available is the TRH stimulation test. This test involves measuring ACTH before and after injection of thyrotropin-releasing hormone (TRH). The main impact of TRH in the body is to stimulate the pituitary to release thyroid-stimulating hormone, which in turn controls the levels of thyroid hormones in the body. However, TRH in addition stimulates an increase in the production of ACTH by the pituitary that is far more marked in PPID cases than normal cases. As such, TRH can be used to highlight borderline cases of PPID. This method is potentially safer than the dexamethasone suppression test; however, it also has some limitations in its usefulness.

The limitations of the various blood-testing approaches mean that mild cases of PPID are often missed. For this reason, where there are reasonable grounds to suspect PPID but testing does not give a positive result, retesting at yearly intervals (or sooner if the severity of laminitis worsens) should be considered.

One further thing needs to be considered. PPID always results in EMS as a result of elevated cortisol levels. However, not all EMS is caused by PPID. It is therefore possible for a horse to have PPID and EMS where some of the EMS is not caused by the PPID. If appropriate treatment of PPID does not fully reverse the EMS, coincident PPID and EMS should be considered as a possibility. This

scenario is, in the author's experience, far more common than one would expect. One possible reason for this might be if EMS increases the risk that a horse goes on to develop PPID.

KNOWN TRIGGERS

Even though the mechanisms that underpin laminitis are not well understood, there are a number of factors that are known or suspected to trigger specific attacks.

Dietary Change

Dietary change is perhaps the best known trigger factor for laminitis. This can take many forms, but the most common trigger factor appears to be the variations in the nutritional value of grass that occur at certain times of year (mostly in spring or autumn, but also at any time when there is a flush of grass growth as a result of favourable weather patterns). Frosty conditions also appear to trigger laminitis in some sensitive horses, although it is not known whether this is related to eating frosted grass, or the drop in the surface temperature of the feet.

The other commonly recognized dietary trigger is the classic feeding accident where a horse breaks into the feed store and eats an entire bag of grain or other similar high-energy feed. However, recent research is starting to hint that this kind of incident triggers a significantly different mechanism to that triggered by changes in pasture.

Shock

Any form of clinical shock has the potential to trigger a laminitis attack. This can happen as a result of extreme pain (for example, from colic or colitis), infection (such as pneumonia or infection of the womb), or from other stresses to the body such as dehydration (often as a result of some other medical crisis) or severe blood loss. Septicaemia resulting from an infected wound or abscess that isn't appropriately treated is another common cause of laminitis, as is endotoxaemia – the production of toxins by bacteria within the body (typically within the gut as a result of major problems with digestion). These trigger factors are something any vet should be aware of, and the risk of laminitis as a secondary condition resulting from other medical emergencies should always be considered and planned for.

Mechanical Trauma

Given that laminitis is just inflammation of the laminae, it is entirely possible to create such inflammation mechanically rather than biochemically. Repeated mechanical bruising to the laminae will result in inflammation that can be classed as laminitis. It is often stated that excessive concussion from road work or similar can cause laminitis – a situation sometimes termed 'road founder'. In practice this is not a common occurrence compared to other forms of laminitis, but well documented cases do exist.

If concussion alone could cause laminitis, then any horse trotted for long periods on the road would tend to develop laminitis. And yet plenty of horses, both shod and unshod, routinely work on roads at speed without showing any signs of laminitis. Even attempts to argue that certain horses are genetically more susceptible to mechanical laminitis don't stand up to close examination: sometimes horses that are described as having developed 'road founder' have previously managed to do far more work on hard ground without any problems.

It would seem that, for concussion to lead to laminitis, there has to be some predisposing event or condition that makes the foot more sensitive to concussion. A healthy foot, landing correctly (that is, just heel first) is extremely good at absorbing the vibration associated with impact. If, however, that horse experiences heel pain, it may switch to landing toe first, effectively bypassing most of the shock-absorbing mechanisms in the foot. Even this is not enough on its own to trigger laminitis. Horses with

navicular disease that land toe first do not typically go on to develop full-blown laminitis. That said, navicular disease often presents with accompanying LGL – but it can be argued that the LGL typically precedes, and most likely contributes to the navicular disease, rather than the other way around.

To understand the true underlying causes of concussion laminitis, you need to bear in mind that inflamed tissue bruises far more easily than healthy tissue. For otherwise healthy laminae to bruise purely as a result of concussion is highly unlikely. But if a horse already has a degree of LGL, the already inflamed laminae will bruise at far lower levels of vibration. The inflammation from the bruising will in turn make the laminae more sensitive still to vibration. Hence a horse that is asked to trot on roads when it already has a significant degree of LGL can potentially go on to develop full-blown laminitis as a result. Of course, we also know that LGL affects all the coria in the foot, not just the laminae, and the same arguments apply to the other coria equally well. Hence the presence of LGL combined with significant concussion has the potential to damage all parts of the foot, resulting in either worsening of the LGL or, in the worst case scenario, rotation or sinker. As such, when faced with an apparent case of concussion laminitis, it is important to look for evidence of prior LGL in the hoof capsule that pre-dates the concussion event.

The other association of mechanical trauma with laminitis is contralateral limb laminitis (laminitis caused by the horse weighting one foot excessively when another is lame). This is described in the next chapter.

Plant Toxins

The list of plant toxins that can trigger laminitis is huge. Some of these are well known and well understood and some are not.

One of the most studied toxic triggers for laminitis is black walnut. Black walnut trees (as well as some other related species) produce a toxin called juglone, which they secrete into the surrounding soil to prevent other plants from establishing and competing for resources. Unfortunately, this toxin (which is present throughout the tree and its fruit) is highly toxic to horses, and ingestion of even fairly small amounts triggers severe laminitis within a day or two. This is a particular problem in areas such as parts of the USA and Canada where these trees are native, and horses may inadvertently ingest fruit hulls or bark. It has even been suggested that black walnut pollen may trigger laminitis. The mechanism of toxicity of juglone is not yet known.

Black walnut produces high quality timber that is used in cabinet making, and as a result, black walnut can find its way into wood shavings used for animal bedding. The presence of significant quantities of black walnut shavings in bedding is sufficient to trigger laminitis (possibly as a result of the horse eating the bedding) so it is important when sourcing shavings as bedding to ensure that there is no contamination with black walnut.

There are a number of common UK plants that are toxic to horses, although most are not well known as triggers of laminitis in the way that black walnut is. Perhaps the most commonly known laminitis trigger in the UK is the oak tree. Horses that ingest either acorns or oak bark in any quantity tend to get colic and laminitis as a result. Few of the other UK toxic plants are well known as triggers of laminitis, but this may be because the levels needed to create full-blown laminitis are fairly high. These plants probably cause serious illness or death from other mechanisms, such as liver or nerve damage, before sufficient is ingested for the feet to rotate. However, lower levels of consumption may in some cases cause LGL to occur, or pre-existing full-blown laminitis to worsen, whilst not being sufficient to cause other obvious clinical signs.

A good example of this is bracken, which contains an enzyme called *thiaminase*, which destroys thiamine (vitamin B1). It is thought that the bracken uses this as a protection against insects. Horse tails, a common plant in UK ditches, also contain thiaminase. Vitamin B1 is essential for many bodily functions, and deficiency in vitamin B1 causes a condition known as ‘beriberi’. Beriberi can present in a number of ways, but in horses it typically results in damage to peripheral nerves and hence a loss of sensation and motor control. For this reason the condition in horses is known as ‘bracken staggers’. As

well as the loss of coordination, affected horses will tend to lose their appetite and lose weight. In more serious cases, death results. It is easily treated if caught early enough by removing the bracken source, and feeding or injecting additional vitamin B1 to bring blood levels back to normal.

Less commonly recognized is the importance of vitamin B1 in the body's processing of blood glucose. Deficiency in vitamin B1 results in poor glucose metabolism and hence insulin resistance, which, as already mentioned, is a known predisposing factor in laminitis. So horses with mild bracken poisoning may show mild laminitis (typically LGL, rather than full- blown laminitis) at levels of ingestion that are not sufficient to cause the more classic signs of bracken staggers. In the author's experience, removing the horse from the bracken source, and feeding supplementary vitamin B1 for a few weeks, is sufficient to completely stop the LGL.

There is no definitive list of common UK plants that may cause laminitis, but the following list (which is unlikely to be complete) represents those that, in the author's experience, should be considered as possible suspects:

- oak
- box
- bracken
- hemlock water dropwort
- horsetails
- bullrush
- yellow flag iris (and maybe other iris species)
- laburnum
- rhododendron
- cherry laurel
- linseed (if not cooked)
- potato (including peelings)
- privet
- ragwort
- yew

Where toxic plants are implicated in laminitis in the UK, this is most commonly as a result of non-native garden plants either migrating out of gardens or being browsed by horses over the fence from adjacent pastures.

Most of these toxic plants are not readily palatable to horses. For example, most horses will not touch acorns as they are too bitter. The risk with toxic plants arises in cases where horses are short of food and may start to nibble at them despite the bitterness. Just as we can acclimatize ourselves to bitter tastes such as dark chocolate and coffee, once horses start nibbling at bitter plants they can sometimes develop a taste for them. Ironically, it is often the horses with a history of laminitis that are placed in 'starvation paddocks' to reduce grass intake and hence are sufficiently hungry to develop a taste for anything toxic that might be available.

One final plant that deserves a special mention is garlic. Veterinary text books from a century ago list wild garlic as toxic to horses. And yet today its cultivated form is added to horse feeds and even sold as a separate supplement for horses. It is hard to understand how this situation has come about, but it may be the result of confusion, given that garlic is considered beneficial in humans.

Garlic (and indeed every member of the onion family) contains a compound called N-propyl disulfide, which has the potential to cause damage to haemoglobin, the chemical that carries oxygen in red blood cells. This results in deformed red blood cells containing 'bubbles' of damaged haemoglobin called 'Heinz bodies'. The spleen then clears these damaged cells from the blood. If the garlic damages red blood cells faster than they can be replaced with new healthy ones, Heinz body anaemia results.

There is some argument about the exact amount of garlic a horse needs to consume in order to cause Heinz body anaemia, with studies variously reporting anywhere from 16g to 100g per day for a typical 500kg horse being the critical level. This compares with the typical recommended feeding rate

from feed manufacturers of up to 36g per day, so even with the more conservative studies, there is little headroom between the recommended dose and the toxic dose.

Another problem with garlic is that it has fairly potent anti-bacterial properties. Some researchers argue that garlic can damage healthy gut bacteria, with diarrhoea as a result. As already mentioned, abnormalities in gut bacteria are strongly implicated in the underlying mechanisms behind laminitis. In the author's experience, removing garlic from the diet often clears up persistent diarrhoea in horses as well as reducing levels of laminitis.

Mycotoxins

Mycotoxins are toxic chemicals produced by fungi (moulds) that live on plants. There are hundreds of different mycotoxins with varying degrees of toxicity, and they are common contaminants of nearly all horse feeds ingredients. With mycotoxins being so naturally widespread, it is not surprising that horses appear to have evolved to be able to tolerate them at low levels. Higher levels of mycotoxins in the diet can, however, lead to a range of health issues, including laminitis according to some sources. The problem is not confined to preserved forages and hard feeds – even fresh grass can be contaminated with mycotoxins.

Very little formal research has been carried out on the potential link between mycotoxins in grass and laminitis, but there is some anecdotal evidence from owners that products aimed at reducing the effects of mycotoxins have helped with laminitis.

One problem with this model is that grass rusts and similar fungi seem to be most active in late summer, and hence the likely peak in mycotoxin levels does not align well with the peak laminitis season. As such, mycotoxins from pasture are unlikely to be a major cause of laminitis, but may be a contributory factor. Preserved forages such as hay or haylage are more likely to contain high levels of mycotoxins, particularly in forage that has been stored for a significant length of time, but this would lead to an expected peak in laminitis towards the end of winter – which again does not match reality. However, one possibility is that mycotoxin ingestion over winter creates insulin resistance, which in turn makes the horse more sensitive to spring grass. More research is needed to explore the possible role of mycotoxins in laminitis.

Other Toxins

The list of other toxins that horses can potentially be exposed to is endless. It can sometimes take significant detective work to identify toxins in the horse's environment that have the potential to be the cause of a laminitis attack. A few examples, described below, should help to suggest the kinds of things to look out for.

Lead poisoning: Old paint was often made with lead pigments mixed with linseed oil. As the paint degrades and flakes it can be eaten by horses, some of whom find it palatable (presumably because of the linseed content). Old cast-iron fencing, painted woodwork and drainpipes are all the sort of places where old paint can lurk. Water sources are also a potential risk. Whilst few houses these days are supplied with drinking water via lead pipes, many old horse troughs still are. In hard water areas the inside of lead pipes fur up, protecting the water from contamination, but in soft water areas, the more acidic water will actively strip lead from the insides of the pipes. Water collected from roofs in areas subject to acid rain might also be an issue if lead work is present in the roof valleys.

Toxic metals: Some pastures can be contaminated with toxic metals including lead, aluminium and even arsenic. This can be the result of contamination from chemicals spread on agricultural land in the past. For example, lead hydrogen arsenate was frequently used by farmers as an insecticide until the 1940s and was only banned in 1988. This particular chemical was mostly used in orchards, but was also used on some potato crops, and contamination is still present in some fields in the UK.

Contamination can also occur from adjacent human operations – for example, water flowing out of old mine workings may be contaminated with a range of toxic metals. It is also possible for land to be naturally high in toxic metals. In any area where there is a history of mining for toxic metals, the possibility that the local rock and hence local soils may be naturally high in toxic metals should be considered. Where toxic metal contamination is suspected, the best practice is to test grass or hay samples, as some toxins may be locked in the soil in chemical forms that plants cannot easily access. Soil testing may show up toxins that are inaccessible to plants and hence not a problem, whereas forage testing gives an accurate indication of the levels of toxins the horse is actually exposed to.

Jeyes Fluid: This product contains a number of unpleasant chemicals, including phenols – and yet it is routinely used around horses. The safety data sheet for the product states that it is harmful if swallowed and should only be used in a well-ventilated area. And yet horse owners are actively encouraged to wash down stable walls with it, and even treat straw bedding with it daily in an attempt to prevent the horse from eating its bedding. Whilst there is no formal evidence of a link between Jeyes Fluid and laminitis, there is a significant body of anecdotal evidence. The author has personal experience of a case where a horse that was recovering extremely well from laminitis had a severe recurrence within hours of eating straw bedding that had been treated with Jeyes Fluid.

Chemical fertilizers: There is a very large body of anecdotal evidence linking chemical fertilizers (those containing a mix of nitrate, potassium and phosphate, known as NPK fertilizers) to laminitis. This seems to be a particular problem where horses are reintroduced to the field too soon after fertilizer application. Although it is not known which of the three ingredients is responsible for the laminitis, nitrate is often cited as the most likely suspect. There is some evidence in other animals that chronic exposure to nitrates can increase insulin resistance. Hence it is advisable to keep horses away from fertilized fields until rain has adequately washed the fertilizer into the soil. Better still would be not to fertilize the land at all, as discussed in the next chapter.

Overspray of pesticides or herbicides: Overspray of these substances from adjacent fields appears to be a significant problem for some horses, as can the use of herbicides to remove weeds from pastures. Even horses kept away from treated land for the manufacturers' recommended exclusion period can sometimes develop laminitis within hours of being returned to the field. A good precautionary approach is to double any exclusion periods stated by manufacturers. As with fertilizers, the best approach is to avoid the use of such chemicals altogether if at all possible. For example, docks can be effectively controlled by regular topping of the field, and the old-fashioned approach of scything docks is surprisingly quick and easy and extremely effective if repeated several times during the growing season.

Unintended Consequences of Drugs

Drug interventions for a range of unrelated medical conditions can have the unintended consequence of causing or exacerbating laminitis. The most clear cut of these is the use of steroids, as already discussed, but there are a few other drugs that are well known to carry a laminitis risk. A good example is the use of antibiotics, which upsets the balance of gut bacteria and causes diarrhoea, with laminitis being a potential side effect of such treatment.

More controversial is the assertion by some observers (and hotly contested by others) that more routine treatments may either cause or exacerbate laminitis. Of particular interest are vaccinations and worm treatments, neither of which appears to cause laminitis in otherwise healthy horses. This tends to suggest that, where laminitis is observed immediately after these treatments, it is because the horse was already unwell in some way that makes it more sensitive.

Vaccinations can cause transient fever in some humans, so it is possible that a horse that is already on the edge of laminitis might be pushed over that edge by a vaccination. For that reason, it is perhaps

wise to avoid unnecessarily vaccinating any horse known to be susceptible to laminitis, and to carry out any necessary vaccinations at low risk times of year (that is, avoiding spring and autumn).

Wormers work by selectively poisoning parasites within the gut. Some wormers work on the principle that the wormer molecule is too big to pass through the lining of the gut into the bloodstream, and hence the horse cannot be poisoned by the wormer. Of course, if the horse develops a leaky gut as previously described, it is possible that the wormer *will* reach the bloodstream, and this might explain the apparent link between worming and laminitis in susceptible horses.

The other potential risk stems from worming a horse with a high worm burden. Modern wormers are extremely powerful and will kill every worm within a matter of hours. This results in a lot of dead worms starting to decompose within the gut and releasing toxins, which might also be potential triggers of laminitis.

If wormers are of concern, then it makes sense to do regular worm egg counts to determine if worming is actually necessary. Where egg counts are very high, it may make sense to use a slower acting wormer (such as a five-day programme) to reduce the potential ‘shock to the system’ of killing all the worms at once.

Whilst there is insufficient research evidence to back up the suggestion that vaccinations and wormers can trigger laminitis, there is a significant body of anecdotal evidence. It therefore makes sense to take the precautionary principle and avoid unnecessary treatments, or to treat at safer times of the year.

Unknown Triggers

Unfortunately in a significant minority of cases none of the known triggers seem to be responsible for the laminitis. Sometimes this is due to obvious triggers being missed – one of the author’s clients eventually discovered that a regular passer-by was feeding the horse chocolate bars! But in some cases, there really is no obvious trigger. What that tells us is that we still have a lot to learn about laminitis.

6 Further Thoughts on the Causes of Laminitis

The models described in the previous chapter give infuriating glimpses of mechanisms that might explain parts of the process by which laminitis occurs, but while there are significant areas where the models complement each other, there are also significant areas where they appear to be in contradiction. One reason for the confusion over these mechanisms of causation is the growing realization that laminitis is not one single condition, but rather a group of medical conditions that happen to have the same or similar final outcomes.

Within the equine podiatry profession there has been a strong feeling for a number of years that there are likely to be multiple disease processes that ultimately lead to laminitis, and maybe even different mechanisms by which the damage actually occurs within the foot. The suggestion has been that laminitis is a clinical sign of disease, rather than a disease in its own right. This suspicion is based on years of experience from a good number of people looking at real life laminitis cases in real life situations. There seems to be no clear pattern with risk factors, no clear pattern with trigger factors, and no clear pattern of the progress of the laminitis once it happens. When clear patterns are so stubbornly difficult to detect, it tends to suggest that you are not studying a homogeneous group. The laminitis research community are finally catching up with this way of thinking, and a recent research paper has suggested that there are three distinct mechanisms by which the laminae can fail within the foot: inflammatory laminitis, endocrinopathic laminitis and contralateral limb (sometimes called supporting limb) laminitis.

Inflammatory laminitis: This form of laminitis seems to be mainly associated with overload of the gut with various carbohydrates from starches to fructans, and is characterized by evidence of a strong inflammatory state within the horse (raised temperature, increased white blood cell count). Black walnut poisoning also appears to fit into the same category, for reasons that are not clear. These cases, perhaps as a result of the release of toxins from the hindgut, seem to show MMP activation followed by fairly rapid mechanical failure of the laminae. As such, inflammatory laminitis tends to have an acute onset rather than a chronic one, with changes to the structure of the foot happening within twenty-four to forty- eight hours of the trigger event. Interestingly, the author's dissection studies have suggested that these very rapid onset cases may show pulping of the solar corium in addition to the laminar corium in the early stages of an attack. Of course, because the received wisdom is that the laminae alone are the initial site of failure in laminitis, there have been no studies into MMP activation in the solar corium.

Endocrinopathic laminitis: This is laminitis that has its origins in the endocrine (hormone) system. This group is largely made up of horses that exhibit insulin resistance, whether from PPID or for other reasons. This group tends to show more chronic onset of clinical signs, and the damage to the laminae is characterized more by slow stretching of the laminae prior to failure. This then fits well with the models of chronic laminitis described in Chapters 3 and 4. A key feature of this form of laminitis, which distinguishes it from inflammatory laminitis, is that there is no clear inflammatory state within the horse as a whole. The overwhelming majority of grass-induced cases seem to fit into this category.

Contralateral limb laminitis: A healthy horse tends to shift weight between the feet, albeit often imperceptibly, even when stood still for long periods. This helps to stimulate circulation to the feet, keeping them supplied with the nutrients and oxygen needed for them to remain healthy. Contralateral limb laminitis can occur when a horse develops a severe and prolonged lameness in a single foot that

results in the foot being unable to bear weight. Typical examples might be the recovery period from a bone fracture or a prolonged foot abscess. As a result, the horse loads the opposite (or contralateral) foot continuously, without the ability to rest it. This increases the loading on the contralateral foot significantly, and also reduces circulation. There is some evidence to support the idea that this loss of circulation reduces the delivery of glucose to the laminae, weakening them to the point where they can fail.

Contralateral limb laminitis is a well documented phenomenon, and this explanation for it makes a lot of sense. And yet far from every horse with prolonged single foot lameness goes on to develop contralateral limb laminitis. So it is possible that any horse that already has a degree of LGL is more susceptible to supporting limb laminitis. Perhaps the additional loading and reduced circulation in the contralateral limb is the last contributory factor that tips the horse from LGL into full-blown laminitis? In the author's experience, horses that develop contralateral limb laminitis frequently show clear signs of LGL in both front feet, or all the feet, which pre-date the contralateral limb rotating or sinking.

The idea that there are three separate mechanisms that can lead to laminitis makes a huge amount of sense, but unfortunately it also means that, as most of the research to date on laminitis has been carried out on inflammatory laminitis cases, we know very little about the endocrinopathic mechanism that is now thought to underpin the overwhelming majority of laminitis cases in the UK! Given that inflammatory and supporting limb laminitis cases appear to be rare in the UK, the rest of this chapter will focus on the endocrinopathic model.

Current research seems to be converging on the idea that endocrinopathic laminitis always has insulin resistance at its core. There are two key questions to be answered in such cases: why does the insulin resistance happen in the first place, and how does insulin resistance cause laminitis?

THE CAUSES OF INSULIN RESISTANCE

In some cases (such as PPID and reactions to corticosteroid drugs), the causes of insulin resistance are easy to identify, but in the majority of cases, they are not. This is a problem that is baffling human doctors as well, with insulin resistance and Type 2 diabetes being linked to obesity as well as a range of circulatory diseases such as stroke and heart disease. This means that there is a wealth of human research that can be drawn upon when trying to understand insulin resistance in horses. Sadly this body of human research really doesn't yet provide the answers that horse owners are looking for.

If the research aimed directly or indirectly at pharmaceutical interventions (a large proportion) is weeded out, an interesting pattern of research emerges, suggesting that insulin resistance has a variety of environmental causes. One especially interesting angle is the potential for nutrient deficiencies to cause or contribute to insulin resistance.

Nutrient Deficiencies

For insulin to have its correct effect, molecules of insulin have to bind to receptors at the surface of target cells. This then triggers an active process where glucose from outside the cell is drawn into the cell and stored or used for energy. The cell's response to the insulin, transporting glucose into the cell across the cell membrane, uses a number of nutrients. If any of these nutrients are deficient at a cellular level, then active transportation of glucose is impaired, leading to insulin resistance. The precise list of nutrients required for this process is difficult to pin down (more research is needed), but magnesium, zinc and chromium are all strongly implicated, and there appears to be some evidence for B vitamins, particularly B7 (biotin) and maybe B6. Nutrient deficiency is unlikely to be the only mechanism for insulin resistance to occur, but it is a mechanism that has the potential to be easy to correct.

So why would a horse be deficient in some or all of these nutrients? The most obvious answer would be that perhaps the horse's diet is deficient in some nutrients. For example, government research shows that a significant proportion of people in the UK eat a diet deficient in magnesium. The

blame for this appears to be laid largely at the door of modern agricultural practices, and it would seem that the same problems affect the diet of horses (as discussed later in this chapter).

Even if the levels of minerals in the diet are appropriate, there may be reasons why the horse is deficient at a cellular level. For example, the role of toxins such as heavy metals in laminitis was mentioned in the previous chapter. One possible mechanism for this is that such toxins may adversely impact the function of the kidneys. A key job of the kidneys is to maintain correct levels of mineral nutrients such as magnesium, calcium, zinc and so on in the blood (and hence in the rest of the body). If the correct functioning of the kidneys is being blocked by heavy metals, for example, this can lead to excessive excretion of minerals such as magnesium in the urine, deficiencies within the body, and potentially, insulin resistance.

The other key area of interest is digestive health. Even if the diet contains sufficient levels of minerals, they may not be absorbed in sufficient quantities if the gut isn't fully healthy. The lining of gut is covered in microscopic spikes called villi. This pattern of spikes increases the surface area of the gut dramatically, providing more opportunity for nutrients to be absorbed. The villi also have very thin walls (just one cell thick), which makes it easy for nutrients to pass through from the gut into the bloodstream – but this also makes them fragile. Anything that damages the villi will reduce the uptake of nutrients significantly. It is interesting that the minerals whose deficiency is most implicated in insulin resistance (magnesium, chromium and zinc) are all absorbed in the small intestine. Might this suggest that damage to the villi lining the small intestine could be another potential underlying cause of insulin resistance? As ever, more research is needed.

Direct Toxicity

Little is known about the role of environmental pollutants in insulin resistance, but there are tantalizing glimpses within the published research of a possible model whereby heavy metals and other toxins might cause or contribute to insulin resistance. One good example of this is cadmium, a toxic metal that is a common contaminant in soil and hence in plant crops. Cadmium has a number of mechanisms of toxicity, but one suggestion is that it may reduce the effectiveness of GLUT-4 transporters, which are responsible for moving glucose into cells under the control of insulin. Hence the presence of cadmium in the diet has the potential to cause insulin resistance.

Indirect Toxicity

Toxins can create insulin resistance without necessarily having to act directly on the glucose metabolism of the horse. Anything that impairs digestion, particularly in respect to absorption of nutrients in the intestines, has the potential to cause or exacerbate insulin resistance by reducing the availability of key micronutrients needed for the correct handling of glucose.

One compound which is increasingly being raised as a potential concern is glyphosate, a broad spectrum herbicide which has been around since the mid-1970s. Glyphosate works by interfering with an enzyme called EPSPS, causing a chemical called shikimate to build up and block the normal processing of energy within the plant. The herbicide industry has claimed that glyphosate is safe because the EPSPS enzyme is not present in mammals, and hence there is no mechanism by which glyphosate can act on mammalian tissue. Safety studies have largely appeared to back up this assertion, although it can be argued that the majority of these studies focus on the effects of high-level acute exposure, whereas horses are likely to suffer more from low-level chronic exposure as a result of feed contamination. Longer-term studies are starting to emerge that show evidence of carcinogenic properties, on the basis of which some countries, at the time of writing, are attempting to block the re-licensing of glyphosate within the EU.

One significant concern with glyphosate is that, whilst mammalian cells do not contain the EPSPS enzyme, the bacteria in mammalian guts do. This opens up the possibility of a mechanism of action of glyphosate on gut bacteria, with the potential for this to adversely affect digestion and metabolism of

nutrients. There is growing evidence of glyphosate causing a shift in gut bacteria towards the more harmful bacteria, and away from the beneficial ones.

A recent review paper identified glyphosate's blocking of an important group of enzymes called the cytochromes P450 as a potential cause for concern. These enzymes have many roles, including a role in the body's mechanisms for excreting toxins that don't originate within the individual. As a result, the authors claim that glyphosate increases the damage done by low levels of toxins present in food, air and water, with the damage being slow and insidious (this is an example of the cocktail effect – *see below*). The authors go on to describe mechanisms by which glyphosate might contribute to a range of Western diseases in humans, including (amongst others) gastrointestinal disorders, obesity and diabetes. If these researchers are right, then this opens up the question of whether glyphosate in the horse's diet might contribute to gut dysfunction, obesity and insulin resistance – the very issues that are now thought to underpin laminitis.

Recent changes in farming practices have dramatically increased the levels of glyphosate present in horse feeds. In the last few decades, farmers have taken to applying herbicides to their cereal crops a few days before harvest. This was originally done as a way of killing any weeds present amongst the crop so that the field could be reseeded earlier. However, more recently, herbicide manufacturers have pushed this approach as a way of getting crops to ripen faster and more evenly (reducing the need for a long window of dry weather for harvesting) – a process known as crop desiccation. So long as the crop is already nearly ripe, the amount of herbicide that makes it into the grain is minimal, and the grain from such crops generally passes the restrictions placed on herbicide residues in food intended for human consumption. There are concerns, however, that the remaining parts of the plant (particularly oat straw and oat chaff) are being used as ingredients in animal feeds. Whilst desiccation results in fairly low levels of contamination of the grain, the contamination of the chaff and straw is significantly higher. The restrictions placed on such contamination in animal feed are also much more lax than in human food. The suggestion is that the levels of glyphosate in horse feeds have rocketed in the last two decades as a result.

The Cocktail Effect

There are many hundreds of toxic chemicals that can potentially be present in a horse's environment. Some of these are naturally occurring (for example, lead and aluminium are present in some rocks and hence can turn up in some soils), while some have been accidentally released into the environment as a result of human activities (for example, overspray of herbicides from adjacent fields). Most of these toxins, considered individually, are probably present in a horse's environment at sufficiently low levels as to be considered fairly safe. However, the combination of many such toxins may be more of a problem to the horse. Even more worryingly, there is growing evidence that different toxins may combine so as to become more toxic than the effects of their individual toxicities – dubbed the 'cocktail effect'. It is complacent to assume that any individual horse is not exposed to a significant number of toxins. Many of these potentially toxic substances are not immediately obvious to the lay person. For example, a typical horse might be exposed to:

- plasticizers from plastic feed bags
- preservatives in bagged feeds
- traces of heavy metals from feed crops grown on contaminated soils
- herbicide and/or pesticide residues from feed production practices
- overspray of herbicides/pesticides from adjacent farmland
- cleaning agents and surfactants present in shampoos
- air pollution from adjacent road traffic
- possibly carcinogenic compounds from creosote applied to stable doors
- phenols given off by disinfectants such as Jeyes Fluid used to wash down stables
- airborne and waterborne pollutants as a result of domestic and industrial chemical discharges

Each one of these in isolation may be insufficient to trigger a medical problem, but once the cocktail effect is taken into consideration, is there a possibility that the combined effects could be one cause of insulin resistance in horses?

The Role of Fibre

Fibre is the part of the horse's carbohydrate intake that cannot be directly absorbed or broken down to form useful nutrients by the enzymes produced in the gut by the horse. It consists of long chain carbohydrate molecules such as cellulose and pectin (but not starch, as this can be broken down). The horse can only get nutrients from fibre by using bacteria in the hindgut to ferment it. The fermentation process produces volatile fatty acids which the horse can absorb and use for energy. As a useful by-product, significant amounts of heat are also produced, which helps the horse to keep warm in cold conditions. The horse has evolved to get almost all its energy from the fermentation of fibre to volatile fatty acids. These volatile fatty acids do not immediately increase blood glucose and so do not form a trigger for endocrinopathic laminitis. For that reason, even a horse with severe insulin resistance would not get laminitis if fed a diet that was 100 per cent fibre. Unfortunately, the most readily available source of fibre in the UK, grass, is not pure fibre and contains significant quantities of sugars and starches.

UK pasture (especially since the advent of chemical fertilizers) tends to be very lush, and hence a significant proportion of the energy content is not in the form of fibre. This is not what the horse evolved to eat. Indeed the whole idea that a horse is a grazing animal is somewhat flawed. Horses have evolved to eat a diet that is a mixture of pasture plants and woodier plants such as trees and shrubs. A horse can happily eat tree branches up to the diameter of a human finger. Given free choice, horses spend around 20 per cent of their foraging time eating woody plants. As such, a horse is actually halfway between a grazing animal and a browsing animal (think of the cross between a sheep and a goat). And yet we often confine horses to rich pasture and actively discourage them from eating hedge plants with fencing. They then start to eat wood (fences, stable doors and so on), and we see this as a vice rather than as a natural expression of a horse's need to eat an appropriately varied diet.

The Role of Lignin

There is a significant difference between the type of fibre present in a pasture plant and that present in a woody plant – the woody plant has much more of a type of fibre called 'lignin'. For a tree to stand up, the wood has to have a degree of rigidity, and it is the lignin in the plant that gives it that rigidity. The more structural strength a plant needs, the more lignin it has in its structure. A grass leaf can blow around in the wind because it has very low levels of lignin; however, a grass-seed stem needs to stand up to encourage wind-blown distribution of seed, or for the seeds to catch in animal fur. So the seed stem of a grass has a higher proportion of lignin. Cereal-seed stems (often fed to horses in the form of straw chaff) are even higher in lignin. And a tree needs to have a very high level of lignin in its woody parts so as to maintain its shape.

Traditionally it has been claimed that horses cannot digest lignin, and that lignin's only role in the diet is as a bulking agent. It turns out that this is not the case. Horses can utilize lignin as a source of energy via bacterial fermentation in the hindgut. This works well as long as there are sufficient bacteria present to do the job. Of course if the horse has been kept on a diet low in lignin, the quantity of lignin-digesting bacteria in the hindgut will be low. If the horse then eats a significant amount of lignin (as happens, for example, when a horse who is used to a shavings bed is moved to a straw bed and decides to eat it), the lignin will not be properly digested and the resulting plant material may block the gut, causing colic. This may be where the myth about lignin being indigestible comes from. If a high-lignin diet is to be introduced, it must be introduced slowly to allow the relevant bacteria in the hindgut to proliferate.

Given that the horse has evolved to eat a diet that is fairly high in lignin, could some of the problems we are seeing in UK horses be linked to there being insufficient lignin in the diet? There is some

research in other farm animals suggesting that a higher lignin diet acts to improve digestion (including improving the quality of the villi on the lining of the gut). So it is entirely possible that lignin is actually something the horse needs in its diet in order to be fully healthy. That might explain why, as horses were historically moved from countries with a more arid climate to more temperate areas, problems such as laminitis started to occur. More research is needed in this area, but some circumstantial evidence backs this suggestion. We know that laminitic horses do better on late cut hay than early cut hay – and late cut hay contains a far higher proportion of seed stems and hence has a higher lignin content. There has also been research looking at laminitis in donkeys (who are marginally more sensitive to problems from a high sugar, low fibre diet than horses), which suggests that very high lignin diets (for example, a mixture of oat straw and ash branches) provide adequate energy intake and do not trigger any laminitis, even in susceptible individuals.

Based on this thinking, it makes sense with overweight laminitics to substitute a proportion of the hay for straw (a good source of lignin). This needs to be introduced slowly so that the gut flora has time to adapt, otherwise there is a risk of colic, but it is not uncommon for 'good doers' to do well on a 50:50 mixture of hay and straw. In Portugal, some horses are fed 100 per cent straw and still maintain weight well. They eat a larger weight of straw than a horse would eat of hay, but that may well be a good thing, mimicking the high volume, low nutrient diet the horse has evolved to eat. It is important to ensure that the straw used is either barley straw or, preferably, oat straw; wheat straw is not suitable. It is also extremely difficult to obtain straw that isn't either mouldy or contaminated with significant quantities of herbicides, and the difficulties in obtaining suitable straw (for example clean, organic oat straw) are often the biggest problem with this approach.

Gastric Ulcers

There has been a rush of interest in gastric (stomach) ulcers in horses in recent years. This used to be thought of as a rare problem, but research now suggests that more than 90 per cent of racehorses, around 50 per cent of competition horses and at least 10 per cent of pleasure horses have some degree of ulceration in their stomachs. This tends to suggest that we are doing something very wrong in the way we look after domestic horses!

The established view of how gastric ulcers occur is that they are the result of the build- up of excessive levels of acid in the stomach. Humans are designed to eat discrete meals with long gaps in between, and so we have evolved a mechanism that turns off stomach acid production between meals. Horses have evolved to eat constantly throughout the day with only short gaps for sleeping, and hence do not have a mechanism to turn off acid production. Once a horse has stopped eating and the contents of the stomach have been processed and passed into the small intestine, the level of acid in the stomach starts to gradually increase. If this is allowed to continue for long enough (more than a few hours), the level of acidity can reach the point where the protective mucosal lining of the stomach is no longer able to protect the tissue beneath it. The result is raw flesh (an ulcer) which is extremely painful for the horse. It is hard for the ulcer to heal because the damaged stomach lining is less able to produce a new protective coating, and the ulcer is constantly bathed in strong acid.

It is easy to see how racehorses are particularly prone to ulcers, given that they are often fed a diet that is high in concentrates and low in forage. It is common for racehorses when stabled to be given insufficient hay to last until the next haynet, and hence long periods are spent not eating. Psychological stress is also known to increase stomach acid production, which may explain why horses that are routinely subjected to travel and competition stress are more likely to suffer from ulcers. But why do other horses get ulcers – especially those that are kept at grass and so are eating constantly? One possible answer to this is that any horse that has previously had gastric ulcers may still have them. In the author's experience, many ex-racehorses turn out to have gastric ulcers years after they have left the racing industry, despite being given constant access to forage since leaving. Those ulcers may have improved significantly because the constant access to forage means that acid levels in the stomach are not as severe, but it seems possible for ulcers to persist at a low level for years even in

these conditions. There are still, however, some horses with ulcers that have never had limited access to forage, so there must be some other route to this problem.

In humans, it has been known for several decades that gastric ulcers are not caused by acidity alone (although the medical profession as a whole has had to be dragged kicking and screaming to that realization). The traditional management approach of taking antacids neutralizes a proportion of the acid and hence reduces the symptoms (less acid means less pain) but does not reliably promote healing of the ulcers. More modern drugs, such as proton pump inhibitors, are also designed to reduce the production of stomach acid. These, too, whilst allowing some ulcers to heal (the level of acid suppression can be significantly higher), are not a reliable treatment (despite still being heavily prescribed). It turns out that human gastric ulcers involve a bacterium called *Helicobacter pylori* (first discovered in the pyloric region of the stomach, hence the name). Unless this bacterium is effectively eradicated with antibiotics, the ulcers tend to keep coming back.

It is not too great a leap of imagination to question whether equine gastric ulcers might also have a bacterial involvement, and indeed early research is providing tantalizing glimpses of this possibility. A new species of bacterium, related to *Helicobacter pylori*, has been identified in the caecum, colon and rectum of some horses (and is particularly common in young foals). This has been named *Helicobacter equorum*. So far, though, conclusive evidence that this bacterium is involved in equine gastric ulcers is proving stubbornly difficult to obtain, although some studies are finding limited evidence of *Helicobacter* species in the stomachs of ulcer cases. If bacteria are involved in equine gastric ulcers, then this has implications for treatment. Suppressing stomach acid with either ant-acids or proton pump inhibitors may actually make things worse by removing the very acid that is supposed to make conditions too harsh for bacteria to survive in the stomach.

All of this is of interest in the context of laminitis because of a growing body of anecdotal evidence suggesting that horses with gastric ulcers are more prone to endocrinopathic laminitis. There is no proven link as yet, and no proven mechanism of causation, but it is possible to develop credible theories as to those mechanisms. The gut consists of a series of compartments, each with a particular job to do. The output from each compartment is supposed to be food processed so as to optimize the function of later compartments. If there is a fault in the processing of food in any compartment, the output of that compartment is sub-optimal, and the functioning of the remaining compartments of the gut can potentially be adversely affected in a form of chain reaction.

There are various ways in which gastric ulcers might do this. For example, the process of ulceration produces waste products (serum, dead skin cells and so on), which can pass into the rest of the gut and potentially form a substrate for undesirable bacteria to colonize later parts of the gut. If the bacterial model is accepted, there is also the question of how the bacteria colonize the stomach in the first place. Does this suggest that the horse is producing too little stomach acid in the first place? If so, does that result in problems further down the line – poorer protein digestion, as well as a failure to fully sterilize food, resulting in low levels of pathogens passing through the stomach, for example?

Small Intestine Bacterial Overgrowth (SIBO)

Equine podiatrists have one advantage over vets in that they see horses on a regular cycle and so can observe patterns of overall clinical signs that appear to be associated with adverse events in the foot. This kind of observation has led to the suggestion that there may be sub-groups within the endocrinopathic group of cases.

Of particular interest is a subgroup of horses that show significant levels of abdominal distension, the severity of which seems to track the level of laminitis present. Within this group, a further subgroup tends to suffer from severe flatulence. Abdominal distension is most likely to happen as a result of either a build-up of fluid in the abdominal cavity, or the presence of significant quantities of gas within the gut. It is fairly easy to argue that the flatulence cases are likely to be distended because of gas, but at first sight it is harder to make this assumption with the remainder of cases. However, it turns out that

the majority of gas produced within the gut – particularly in the early stages of digestion – is reabsorbed through the lining of the gut into the bloodstream, and then exhaled via the lungs.

This effect is routinely used in humans to diagnose problems with abnormal bacterial activity in the gut. The only process within the mammalian body that can produce significant quantities of hydrogen or methane is bacterial fermentation in the gut. As such, any hydrogen or methane detected in breath must be the result of bacterial fermentation. Feeding a small amount of carbohydrates to a previously starved patient results in a pattern of hydrogen/methane production over time, which indicates the amount of bacteria in the various parts of the gut as the carbs pass through. A very early peak suggests bacteria in the stomach (and is used to diagnose gastric ulcers in humans). A fairly early peak indicates too much bacteria in the small intestine (which should have only small quantities of bacteria in it).

The use of different carbohydrate loads can also provide valuable information. For example, the simple sugar fructose should be absorbed in the first part of the small intestine, well before any significant numbers of bacteria are present. Hydrogen or methane production a few hours after a fructose load suggests that the fructose hasn't been fully absorbed and has made it through to the large intestine – a condition known as fructose malabsorption. The use of breath testing in horses is theoretically possible but would be complicated by the need to starve the animal for a significant period before testing so that it had an empty gut.

Given that gas can be reabsorbed into the bloodstream, it would make sense that horses producing too much gas early in the gut (in the small intestine, for example) would not exhibit flatulence, whereas those producing too much gas in the later stages of the hindgut would. Whilst this doesn't rule out the possibility that some of the bloating cases might have fluid retention, it does leave open the possibility that most, or all of them are suffering from excessive gas production.

The question then would be why so much more gas than normal is being produced in these horses. One possible answer comes from research into irritable bowel syndrome (IBS) in humans. Researchers in the USA have argued that a high proportion of such patients have far more bacteria in the small intestine than normal. In a healthy human gut (and also in a horse's), there is a gradual increase in levels of bacteria found as you progress through the small intestine. The stomach should be sterile, and the duodenum (the first section of the small intestine) should be very nearly so. By the ileum (the last part of the small intestine), levels of bacteria are noticeably higher, but still way lower than in the caecum. Between the ileum and the caecum sits the ileo-caecal valve, whose job is to allow food through from the ileum to the caecum whilst preventing the majority of bacteria from the caecum from flowing back into the ileum. Where excessive numbers of bacteria colonize the small intestine, this is termed 'small intestine bacterial overgrowth' (SIBO).

SIBO is known to be a complication of some other medical issues (for example, bowel surgery) but the reason it appears to happen in IBS cases is not yet understood. However, once SIBO is present a number of things go wrong. The small intestine is where monosaccharides, disaccharides and starches are absorbed. Normally these carbohydrates are broken down into simple sugars and absorbed before progressing far enough to encounter significant numbers of bacteria. But if bacteria are present too early in the digestion process, then these can ferment the sugars and starches, producing large quantities of gas. The small intestine is a fairly narrow pipe, and is not well adapted to dissipating large quantities of gas.

The result is that food is jet propelled in either direction from the location of fermentation. Food that is propelled onwards passes through the small intestine too fast, so reducing the absorption of key trace elements such as magnesium, zinc, copper and chromium. Food that is propelled back up towards the stomach takes bacteria and bile with it. The bacteria potentially colonize further back in the gut, and the bile potentially burns areas of the duodenum that aren't designed to cope with it. Some bile may also be propelled back into the stomach, creating damage to the stomach lining known as 'chemical gastritis'. It is not difficult, then, to see how further digestive problems and nutritional imbalances might result from SIBO, which could potentially lead to laminitis. Research is needed to see if SIBO does indeed occur in horses, but if SIBO is an underlying factor in some laminitis cases, there is the potential to develop antibiotic-based treatments.

HOW DOES INSULIN RESISTANCE CAUSE LAMINITIS?

Whilst there is a wealth of evidence that insulin resistance is central to pasture-induced laminitis, and that laminitis attacks appear to be triggered by rising levels of blood glucose and the resulting hikes in insulin levels, the exact mechanism by which the various coria in the foot become damaged is still unknown. One very promising line of enquiry is the suggestion that oedema (the build-up of fluid that has leaked from capillary blood vessels into the spaces between cells) is seen very early in the processes that lead to laminitis within the foot (work undertaken by Dr Susan Kempson at the Royal (Dick) School of Veterinary Studies, Edinburgh). We know that EMS cases have a tendency to develop oedema in areas of regional adiposity such as the crest just prior to and during laminitis attacks. The idea that oedema might occur in the foot is therefore not entirely random.

In a healthy individual, tissue fluid is delivered by the arterial side of capillary blood vessels to a tissue so as to bathe the cells in a range of nutrients. At the same time, fluid is returned to the venous side of the capillaries so as to maintain the correct level of fluid within the tissue. If capillary blood vessels are damaged at a microscopic level, they can leak excessive quantities of tissue fluid and so upset the normal balance of fluid in the tissue. The result is oedema. Some of this oedema can be returned to the circulatory system by the lymphatic system – a separate system of ducts that drains fluid from peripheral tissues back towards the heart. However, unlike the main circulatory system, the lymphatic system has no dedicated pump, and relies instead on lymph ducts passing through muscles, such that activity in the muscles helps to pump fluid back towards the heart. Therefore a horse that is standing still has little or no lymphatic drainage.

If there is a problem with fluid balance and capillaries are leaking, this will tend not to result in significant oedema if the horse is moving around a lot. However, if the horse is stabled, there will be little or no lymphatic drainage, and fluid will pool in the legs causing oedema. In the leg, this is known as stocking up. This explains why filled legs improve once the horse is turned out.

It is of note that many laminitis cases, including LGL cases, tend to stock up when stabled. This tends to suggest that there is a degree of oedema happening in the leg as a whole in these cases, which again supports the suggestion that maybe oedema is present in the foot during laminitis.

It is fairly easy to see how oedema might lead to laminitis. In the limb as a whole, oedema results in swelling and the leg enlarges. However, in the foot, the laminar and solar coria are sandwiched in a tight space between the pedal bone and the hoof capsule: there is no room for these coria to expand, and hence any oedema will instead result in a build-up of pressure. The pressure will, in the first instance, result in pain. This pain is likely to be worst in the toe area, as this is where the coria are most confined by hoof and bone. In contrast, the coria in the heel area have soft tissues beneath them and there is room for the foot to expand upwards between the heel bulbs to a small degree. This might explain why laminitis typically affects the toe most strongly.

The pain from oedema is sufficient in itself to cause alterations in gait and stance, and hence alterations in growth patterns as discussed in Chapters 3 and 4. But the build-up of pressure will also start to damage the various coria, weakening them and hence allowing them to distort more in response to the mechanical imbalances caused by the altered gait and stance. This would explain the stretching and/ or tearing of the laminar corium. If the pressure in the coria increases to the point of equalling arterial blood pressure, then there is no way the arterial pulse of blood can make it into those tissues, and the result is a total loss of circulation (ischemia). This might explain what happens when a pasture-induced LGL case suddenly slips into full-blown laminitis.

One further piece of evidence that might help to confirm the oedema model comes from the study of blood flow in the arteries and veins of the foot during laminitis. X-ray pictures of the blood vessels can be obtained by injecting a dye into them that shows up on an x-ray. Where the dye is injected into the main artery of the lower leg, an arteriogram is obtained which shows all the main arteries in the foot. Where dye is injected into the main vein, a venogram is obtained which shows all the main veins in the foot. It has been noted that arteriograms of severe pasture-induced laminitis cases typically show fairly normal quantities of blood in the arteries, whereas venograms show very little blood in the veins (*see* Fig. 19 in Chapter 2). Could this be because the build-up of pressure is sufficient to collapse the veins

but not the arteries, given that arteries operate at significantly higher pressures than veins? This might suggest that a build-up of pressure within the foot is a key part of the mechanism of laminitis in these cases, supporting the oedema model.

TESTING FOR INSULIN RESISTANCE

The standard approach to testing for EMS is to attempt to measure the degree of insulin resistance. The approaches taken in equines are closely based on those developed for humans. One approach that is routinely used is to fast the horse for six hours. The idea is to remove the effect of any glucose being absorbed from the gut on the level of insulin (all glucose from food should have been absorbed within six hours); the blood glucose level in a fasted horse should be towards the bottom of the reference range. With blood glucose low, the insulin level should also be fairly low, so any high level of insulin in this case indicates a degree of insulin resistance.

Unfortunately, fasting insulin levels are not that reliable an indicator of insulin resistance, particularly in mild cases. Furthermore, as well as false negatives, there are also some false positives, where stress from fasting itself raises the insulin level.

In an attempt to get around this, some researchers have suggested using various formulae to try to get more accurate results. One of the two most common ones is ‘RISQI’ (the reciprocal inverse square of insulin). This still relies on a single insulin sample and so adds nothing to the actual sensitivity of the test. The other is ‘MIRG’ (modified insulin ratio to glucose), which attempts to combine the insulin and glucose levels from a single fasted blood sample so as to make more accurate predictions of the level of insulin resistance. Sadly, MIRG has also been shown to be fairly inaccurate and is increasingly falling out of favour.

One possible workaround to this that appears not to have been explored as yet is to measure the degree to which the pancreas compensates for insulin resistance by increasing the level of insulin production for a given level of glucose. In humans, many of the subjects being tested have already reached the point of Type 2 diabetes and hence the pancreas is already failing to fully compensate for insulin resistance. Where Type 2 diabetes is present, the degree of pancreatic compensation will underestimate the degree of insulin resistance. In horses, however, the overwhelming majority of cases do not show Type 2 diabetes, and hence this approach has more value. Even if Type 2 diabetes is present, there will still be a high degree of compensation and hence a clear positive test for insulin resistance.

In order to measure pancreatic compensation, it is important to keep blood glucose levels constant (as sudden increases in blood glucose create additional spikes of insulin production). This again is difficult in humans who typically eat discrete meals, but is fairly easy in a horse. Any horse eating only hay or haylage at a fairly constant rate is likely to have a fairly stable blood glucose level. The measurement of pancreatic compensation (and hence of insulin resistance) then becomes a case of measuring glucose and insulin from a single blood sample, and comparing the insulin level to that which would be expected for the level of blood glucose. Of course, this would require testing a range of known healthy animals to establish what the normal pancreatic response is.

The level of blood glucose alone in this approach also provides useful information about whether the forage being used is suitable for a laminitic or not. Appropriate forage will produce a blood glucose level towards the bottom of the reference range – if glucose is towards the top of the range, this tends to suggest that the forage is too rich for a laminitic.

A useful recent addition to the armoury of a vet attempting to identify the presence or absence of EMS is the adiponectin blood test. Adiponectin is a beneficial adipokine (signalling protein) produced by fat cells. It has anti-inflammatory effects, and also increases insulin sensitivity (hence reducing the degree of insulin resistance). Overweight horses tend to show lower adiponectin levels than normal, but horses with EMS tend to show significantly reduced adiponectin levels. Hence if a blood test shows normal levels of adiponectin, this suggests that EMS is an unlikely diagnosis, whereas low levels of adiponectin indicate an increased possibility that EMS is present. Unfortunately the test is not fully

diagnostic, as some horses without EMS show lower than normal adiponectin results. However, a normal level of adiponectin is fairly useful for excluding the possibility of EMS. Adiponectin testing is probably not sufficiently powerful to be used in isolation, but it is a very useful add-on to testing a spot sample of glucose and insulin.

PASTURE MANAGEMENT AND FERTILITY

Changes in modern farming practices since World War II have radically altered the nature of pasture in the UK. Most notable amongst these is the move from fertilizing with animal manure to the use of chemical 'NPK' fertilizers, which supply high levels of nitrate, phosphate and potassium.

Both pasture plants and the animals that feed on them rely on a large range of essential minerals (minerals that must be present in the diet) for survival and health. These minerals are often divided into two groups: macrominerals, which are those needed in large quantities (calcium, magnesium, potassium, phosphate, sodium and sulphur); and microminerals, which are the remainder of the essential minerals, needed in far smaller quantities.

Historically, all these minerals would have been constantly recycled, with a range of waste products from all the way up the food chain being spread back on to the land (ranging from animal urine and droppings, through blood and bone meal, to composted waste food and even human excrement). Today, far less of this waste makes it back on to the land, resulting in a gradual stripping of minerals from the land. In the meantime, the spreading of NPK fertilizer and lime (as a source of calcium) supplies in abundance those nutrients that would otherwise limit plant growth, and hence dramatically increases yields both of the forage and the animals that feed on it. The result is a shift in the mineral balance of the forage, with the trace minerals that are not routinely fertilized being depleted. In addition, those macrominerals that are not routinely added to the land also get depleted, especially magnesium, which tends not to be that abundant in the UK in the first place. In cattle and sheep, magnesium deficiency (which is often worst in spring, when faster grass growth reduces the rate of uptake of magnesium from the soil) can cause 'grass staggers' – a serious neurological condition that starts with irritability and leads to incoordination and ultimately death.

Because it is generally considered not to be cost effective to spread magnesium back on the land, farmers typically supplement their livestock by providing molassed licks high in magnesium. The volume of these licks sold by the typical agricultural merchant, and the claims made in large letters across them to be 'a good source of magnesium', is all the evidence needed to show that farmers are acutely aware of this problem.

Given how well understood all of this is, it seems incomprehensible that, until very recently, horse owners were blissfully oblivious to the scale of the problem. It is only in the last ten years that it has become fashionable for equine feed companies to add significant quantities of magnesium to their products, and even now, many horse owners are just not aware of the problem. Sadly, very little formal research has been done into the role of magnesium deficiency in laminitis, but the experience of alternative hoofcare professionals both in the UK and internationally has provided an overwhelming amount of anecdotal evidence that magnesium supplementation helps a good proportion of (though not all) laminitis cases.

The problems caused by NPK fertilizers are not confined to mineral deficiencies, however. A pasture does not behave like a well-tended flower bed: rather it is an ecosystem that runs according to the rule of survival of the fittest – because no one ever routinely hand-weeds a pasture so as to remove unwanted plants (with the exception of ragwort)! Because traditional meadows are not ploughed and reseeded, the soil has had hundreds or even thousands of years to achieve a structure and diversity of plant and animal species that is truly astounding. Even the number of different species of higher plant (those that you tend to notice, as opposed to mosses and suchlike) can be well in excess of a hundred. Every meadow has a different species profile – one that represents the community of species best adapted to the soil conditions, rainfall, altitude, aspect, latitude.

When NPK fertilizer is applied to such a pasture, the delicate balance of the ecosystem is broken. Even without reseeding, there will be a rapid shift away from the traditional meadow species (which can't compete in higher fertility conditions) towards species such as rye grass (which dominates in high fertility conditions). The diversity of higher plant species can drop from over one hundred to as little as four. Typically, rye grass ends up as the overwhelmingly dominant species. And if other species that can cope with high fertility (such as docks, thistles, nettles) start to take over, the field is typically ploughed and reseeded with a mix that is usually based on rye grass.

Once NPK is added to the soil, it is very difficult to wind the clock back. It is utterly pointless to plough the pasture and reseed it with a 'traditional meadow' mix. Quite apart from the fact that many of these mixes are far from authentic (often containing varieties of seed imported from abroad that are less well adapted to UK conditions), the high soil fertility means that the seeded species will be out-competed by rye grass within a year or two. Over time, constant grazing and hay cutting will reduce the levels of N, P and K, but this process is painfully slow, often taking as much as twenty years to complete. Even then, the delicate balance of soil bacteria, fungi and invertebrates that supports a traditional meadow is unlikely to just reappear, and so the diversity of higher plant species rarely equates to that seen in an untouched meadow.

As a result of all this, the vast majority of pastures in the UK are dominated by rye grass, and traditional meadows are extremely rare. Those that are left are supposedly protected by law, but even with that protection in place there is a significant rate of attrition.

It is tempting to say that the increased productivity of a fertilized pasture outweighs the loss of species diversity. However, it is very important to understand what has actually been lost. Firstly, rye grass has very shallow roots. This means that, instead of pulling up trace minerals from as much as a metre deep, the pasture is sourcing its trace minerals from perhaps the top 50mm of soil. The result is a far more rapid depletion of trace minerals and magnesium.

In addition, rye grass has been selectively bred to increase milk and meat livestock yields. The researchers responsible for this promote the degree to which they have increased the sugar content of rye grass over the last few decades. This selective breeding has resulted in a grass that is very high in sugar and fairly low in fibre – almost exactly the opposite of the profile a horse ideally needs to minimize the risks of laminitis. Ironically, the gradual increase in sugar in rye grass has now reached a point where even dairy cattle are getting laminitis, and this in turn is actually reducing milk yields again!

Part 3
Management of the Laminitic Horse

7 Established Approaches to the Management of the Foot

It is helpful when looking at how to manage the foot itself in laminitis to start with a discussion of well-established approaches. Full-blown laminitis should always be considered an emergency, and calling a veterinary surgeon to attend the animal should be the very first step in managing any new full-blown laminitis case. Any LGL case that suddenly becomes lame or footsore should also be treated as an emergency, but clearly where LGL does not cause overt lameness, the urgency of the situation is less clear cut. The rest of this chapter will focus on the management of full-blown laminitis as the established approaches do not as yet include the possibility that a horse might have LGL.

FIRST AID

While waiting for the vet to arrive, a number of things can be done that may help. If at all possible, the horse should be brought in off pasture and stabled. Ideally a deep bed of shavings, at least 30cm (12in) deep, and ideally 45cm (18in) deep, should be provided across the entire stable floor. This serves two purposes: firstly it provides a soft, conforming surface that will mould to the shape of the bottom of the foot and provide support to the pedal bone via the sole, even to some extent if the horse is shod; and secondly, it allows the horse to shuffle its feet until it finds a position that is comfortable. A horse may want to dig its toes into the bed slightly to reduce any tension in the DDFT that might be contributing to rotation, but equally it might want to weight the heels so as to avoid pressure in the toe area. Either way, a soft bed allows the horse to find its own optimum posture so as to minimize pain (and hence any mechanical damage that might be occurring).

Under no circumstances should the horse be starved. Continuous access to forage is important, as starvation increases the risk of gastric ulcers and may also cause distress and discomfort to the horse, which in turn may increase the severity of the laminitis through increased stress hormone levels. The ideal is to feed hay that has been soaked for thirty to sixty minutes to reduce the sugar content. Such hay should, at least in the first instance, be given ad lib so as to minimize stress.

As a general rule, pain relief should not be administered without advice from the vet. If the vet is unable to attend in person within a short space of time, and appropriate pain relief drugs are available on site, then the vet should be able to advise by phone on whether and how to use them.

EARLY VETERINARY INTERVENTION

The actions of the vet on arrival may vary somewhat depending on the exact nature of the case, but the following are common early interventions: pain relief, the use of sedatives and aspirin, cold hosing and/or cryotherapy, frog supports, and removing the horse’s shoes.

Pain Relief

Initial pain relief in serious cases is usually administered by injection (which is faster acting than oral pain relief). Vets will usually leave the owner with further oral pain relief to be administered over the following days or weeks. The use of non-steroidal anti-inflammatory drugs (NSAIDs) such as phenylbutazone (bute) not only reduces the suffering of the horse, but also acts to some extent as an anti-inflammatory, reducing inflammation and hence damage in the foot.

The Use of Sedatives

Some horses will become agitated when put on box rest for laminitis. This might be a horse that is not used to being stabled, or it might be more directly related to the discomfort from the feet. Such agitation can further contribute to the laminitis in two ways. Firstly, excessive movement puts more strain on already compromised structures within the foot. The administration of significant amounts of pain relief may make this situation worse by removing or reducing the pain stimulus that would normally encourage a horse with laminitis to remain fairly still. Secondly, agitation is associated with raised levels of stress hormones, which are likely to then contribute further to the underlying laminitis. Where the horse is not calm and still within the stable, the vet may choose to use sedatives. This will tend to encourage the horse to stand still or even to lie down for some of the time, and will also tend to reduce the levels of stress hormones.

Sedatives such as acetylpromazine (ACP) have some degree of pain-relieving effect, and hence reduce the need for the use of NSAIDs. This may help to reduce some of the harmful side effects of the use of NSAIDs, such as an increased risk of gastric ulcers (*see* the section on drug interventions in Chapter 8). In addition, there is some evidence that ACP helps to slow clotting by preventing the aggregation of platelets, and this may reduce the severity of any micro-thrombosis that would otherwise act to clog blood vessels in the foot, causing more damage.

The Use of Aspirin

Aspirin is known to reduce the effects of clotting by blocking platelet aggregation, in a way similar to ACP (which is why it is used for human patients at risk of heart disease and stroke). It has been suggested that micro-thrombosis forms part of the mechanism of damage in laminitis, and as such, aspirin would appear to be a sensible treatment. Unfortunately there is little research evidence to date to support or refute the use of aspirin in laminitis, so its efficacy is not yet known. That said, the use of aspirin carries few risks, so there may be arguments for using it in the hope that it might do some good.

Cold Hosing/Cryotherapy

There is a suggestion that cooling the feet down in the early stages of a laminitis attack may reduce the severity of the attack and improve the long-term prognosis. The idea behind this is that the underlying laminitis is caused by toxins entering the feet via the bloodstream and poisoning the laminae. Cooling the feet down reduces the circulation and hence reduces the quantities of these toxins reaching the foot. It should also be noted that cooling the feet down will reduce pain and also reduce swelling (which may cause secondary damage to the foot).

There are a variety of suggested approaches for cooling the foot, ranging from standing the horse in boots filled with a mixture of crushed ice and water, to hosing with very cold water. Where a static mixture of water and ice is used, it needs to be replaced regularly so as to keep the temperature of the foot low (under 5°C). Whichever method is used, it needs to be used continuously for a few days rather than hours until the underlying laminitis event has subsided, and this makes the practicalities of implementing cryotherapy difficult. However, research comparing two front feet in the same horse (one

untreated and one treated with cryotherapy) shows that cryotherapy can significantly reduce the amount of damage done to the foot by acute laminitis.

There is a long-standing tradition in equestrian circles that standing a horse in a stream for a day will reduce the severity of an acute laminitis attack by cooling the feet. This may be an example of tradition representing the accumulated wisdom of many generations.

Frog Supports

Some vets like to apply frog support pads (sometimes called lily pads) as a first aid measure. These are small plastic or foam pads that are taped on to the frog. The idea is that they encourage the horse to weight the frog in preference to other parts of the foot, so supporting the weight of the horse via the back of the pedal bone and avoiding weighting the laminae. There is, however, some argument that, when combined with a deep bed, these achieve absolutely nothing because the bedding moulds to the shape of the foot and creates an even pressure regardless of any devices added to the foot.

Fig. 86 A frog support pad being fitted to a shod foot. This would then be held in place with a cohesive bandage or similar.

Where the bedding isn't sufficiently conforming, there is an argument that a frog pad converts a standard shoe into a heart-bar shoe, and hence supports some of the weight of the horse via the frog,

reducing the load on more damaged structures within the foot. This makes good sense, but the same effect can also be achieved by removing the shoe and applying an appropriate trim. As such, frog pads are normally only useful for short-term first-aid use where neither the deep bedding nor the skills to remove the shoes are available.

Removal of Shoes

Some vets like to remove the shoes at the first sign of laminitis, although this is an area where the approach is highly variable from one vet to another. Robert Eustace of the Laminitis Clinic recommends that shoes should not be removed if the sole is flat or convex.

The detailed advantages and disadvantages of removing shoes will be discussed further in the next chapter.

ONGOING DIAGNOSTIC AND TREATMENT OPTIONS

Once the initial first-aid measures have been put in place, the traditional view is that there is little that can be done other than to wait. The hope is that the severity of the initial underlying laminitis will reduce, and that the rate of rotation or sinker will gradually decrease. Once the worst of the initial crisis is past, the next phase of treatment moves to the management of what are now badly damaged feet. Chronic laminitis cases that don't have an acute onset are also typically managed in the same way. The main approaches to ongoing diagnosis and treatment are outlined below.

X-Rays

If the vet suspects that there is any significant degree of rotation or sinker (usually in the early stages because there is a depression in the coronary band at the toe), then x-rays are almost essential to assess the severity of the damage and also to guide the subsequent trimming of the hoof. Normally a latero-medial (looking from the side) shot is taken of each foot as this gives the most useful information in a laminitis case. Occasionally, in very serious cases, other shots may be taken to assess damage to the pedal bone (or if there is a suspicion that the pedal bone may have rotated sideways to some degree).

A number of key measurements can be taken from an x-ray, which help both in assessing prognosis and also in trimming the hoof. These are usually dependent on suitable markers being placed on the outside of the hoof so that key external landmarks can be seen on the x-ray. The most helpful markers are as follows:

The front surface of the hoof wall: The degree of rotation can then be determined by measuring the angle between the external hoof wall and the front surface of the pedal bone. This gives an indication of the degree of damage present in the laminae. Furthermore, the quality of the laminar wedge when viewed on x-ray can tell us whether the onset was sudden (a haematoma, visible on x-ray as a gas pocket, is typically present just behind the wall in this case) or gradual.

The position of the hairline at the front of the foot: This allows the founder distance to be measured, which indicates the degree to which the front portion of the pedal bone has descended within the foot, as well as the likely degree of damage to the coronary band.

The position of the apex of the frog: This helps a farrier or trimmer to transfer information from the x-ray on to the external surface of the hoof prior to trimming.

The position of the external surface of the sole directly under the tip of the pedal bone: This allows the thickness of the sole under the tip of the pedal bone to be measured. In the first few days

after an acute attack, the thickness of the sole is unlikely to be affected and we cannot use this to gauge the degree of damage to the solar corium, but within a few weeks, any significant damage to the solar corium will result in a thinning of the sole, particularly just under the tip of the pedal bone. In addition, even if the underlying corium is starting to heal, a thin sole is far less good at spreading weight away from damaged areas, and hence will provide less mechanical support to healing tissues.

The position of the ground under the foot: This is required to measure the degree to which the pedal bone is incorrectly oriented with the ground. This is usually achieved by placing the foot on a wooden block that contains markers such as screws or nails.

Sadly, not every vet places all these markers on the foot, which makes the job of the farrier/ trimmer somewhat harder. It is common to see a scalpel blade or hoof nail taped to the front surface of the hoof wall. If carefully placed, this can be used to indicate the angle of the hoof wall and the position of the hairline, but it does not work as well when the front surface of the hoof wall is significantly curved.

It is also common to see a drawing pin placed in the apex of the frog. There are two disadvantages to this approach. Firstly, the pin cannot be placed at the exact apex of the frog as there is no material there to push it into. Instead the pin is placed slightly back from the apex, and a pen mark placed on the frog to indicate where it was placed. Sadly, the pen mark is often neglected by the vet, making this marker less useful. Secondly, the frog may be sufficiently damaged that there is a risk that a drawing pin placed into it will actually penetrate close to, or even into the frog corium!

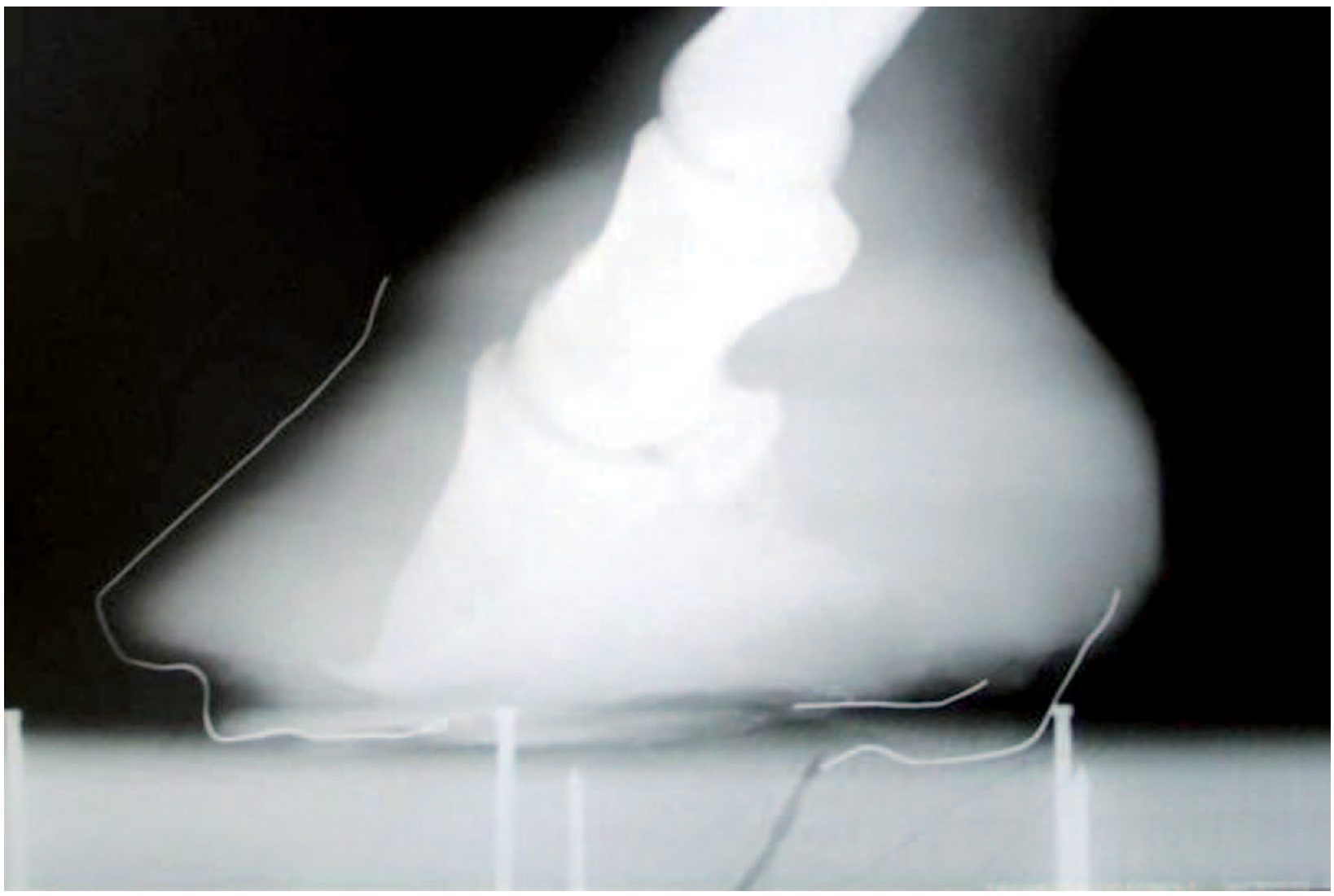


Fig. 87 An example of an x-ray marked with solder wire placed from the hairline to the apex of the frog. In this example, wire has also been used to indicate the position of the heels (which would otherwise be difficult to see). The ground surface is also indicated by a number of nails placed in the block on which the foot is stood. This x-ray suffers from parallax error – the marker nails within the block do not line up vertically. The heel markers also do not line up, again as a result of parallax error. Measurements taken directly from this x-ray may be inaccurate as a result.

One quick and easy way to create all the markers (apart from the ground level) in one go is to use a suitable piece of flexible wire (solder wire works well as it is flexible, and its high lead content makes it fairly opaque to x-rays). One end of the wire should be taped to the centre of the front wall of the hoof such that it ends at the hairline. The wire is then taped down the wall, around the toe and down the

centre line of the sole. The second end of the wire is either cut or folded so that it stops at the apex of the frog.

Another common problem with x-rays is parallax error. This occurs where the x-ray source is placed too close to the foot. The x-ray beam then diverges at a significant angle by the time it passes through the foot, resulting in parts of the foot that are nearer the x-ray source appearing bigger than those further away. This can cause a range of unhelpful distortions, which make measurements of distances and angles taken from the x-ray less accurate.

Heart-Bar Shoes

The mainstay of the current established approach to the management of laminitis is the heart-bar shoe. There is significant debate amongst vets as to which cases need heart-bar shoes and which do not, but in more severe cases, heart-bars are almost always recommended.

Fig. 88 A heart-bar shoe fitted to a laminitic foot. (Photo: Vikki Fear)

The heart-bar shoe adds a frog plate to the standard shoe. The frog plate sits directly under the frog and allows a proportion of the horse's weight to be supported via the back of the pedal bone, the digital cushion and the frog. A heart-bar shoe must be correctly fitted to the foot or serious harm can be done. For that reason, heart-bar shoes should only be fitted in reference to recently taken x-rays, and by a farrier experienced in fitting them from x-rays. It is also important that the pressure applied by the shoe to the apex of the frog is correct: too little pressure gives no benefit; too much pressure risks causing bruising and abscessing to the frog corium.

Plastic and Glue-On Shoes

One problem with the use of standard heart-bar shoes is the necessity of nailing them on to the foot. Given that a horse with an inflamed foot will be extremely sensitive to vibration, it can be argued that nailing a shoe on to the foot is unethical. There is also an argument that the mechanical vibration of nailing may cause further damage to the internal structures of the foot. As the horse recovers and is brought back into work, the vibration of a metal shoe impacting hard ground can also slow recovery. For these reasons the use of glue-on shoes, and in particular plastic glue-on shoes such as the Imprint shoe, has become common in recent years.

Fig.89 An Imprint shoe fitted to a foot.

Sole Pads and Fillers

It is widely accepted that many recovering laminitics have tender soles that are sensitive to pressure and/or vibration. In an attempt to work around this problem as the horse recovers, a pad is often fitted between the hoof and the shoe. A pad will, to an extent, protect the sole from stone bruising. However, the main benefit from pads is in reducing the amount of vibration from the shoe impacting on hard ground that reaches the foot. Because such pads are flexible, they do not significantly contribute to the support of the horse's weight unless a rigid plate is placed between the shoe and the pad (which is rarely done). Sometimes flexible fillers are used to fill the space inside the shoe between the sole and the ground. These can be a little more effective at supporting the sole than the use of pads so long as the filler reaches the ground.

Heel Wedges

Heel wedges are sometimes used in an attempt to reduce rotation by relaxing the deep digital flexor tendon. As previously described, this approach is flawed in all but a tiny minority of cases.

Dorsal Wall Resections

Since the late 1950s, it has been common practice to remove the front portion of the hoof wall in more serious laminitis cases – a procedure known as a dorsal wall resection ('dorsal' meaning front). This is a surgical procedure and should only be carried out with a supervising vet present. According to the established model of laminitis, the laminar wedge consists of dead horn, with the dermal laminae remaining at the inner surface of the laminar wedge. Hence trimming through into the laminar wedge is

only the removal of tissue that is no longer living or healthy anyway. As such, although technically a surgical procedure, it is often carried out without anaesthetic. The material is typically removed with a combination of a hoof knife and hand-held power tools.

The thinking behind dorsal wall resections is threefold. Firstly, there is a view that the formation of a laminar wedge somehow prevents the hoof wall from becoming reattached to the pedal bone. This view seems to have originated from the mistaken perception that the pedal bone could somehow be pushed back into place if only the laminar wedge weren't in the way! There is even research published in respected veterinary journals that claims that the presence of the laminar wedge slows down or even prevents the recovery of the foot. This research turns out to be deeply flawed (confusing the effects of the laminar wedge with the effects of ongoing uncontrolled laminitis), and should not be seen as an argument for resecting the wall.

In reality, the laminar wedge is attached more or less firmly to the inside of the hoof wall. Even where there is a haematoma in between the laminar wedge and the hoof wall at the toe, this typically doesn't extend far into the quarters, and hence the two structures will still be attached to each other further back in the hoof. As the hoof wall grows down in a recovering laminitic, it takes any laminar wedge with it, and any new wall growth attaches more or less normally to the healing laminae (a bit like a zip fastener being zipped up). Where laminar wedge continues to be present at the top of the hoof wall, this is because the underlying causes of the laminitis haven't been fully addressed and hence new rotation is occurring.

Secondly, there is a suggestion that the build-up of fluid behind the hoof wall as part of the inflammatory process either prevents healing or contributes to a worsening of the mechanical damage. There are claims that the pressure of fluid may actually push the pedal bone away from the hoof wall. Draining any fluid present by removing the hoof wall allows any internal pressure to dissipate.

The final suggestion is that much of the pain of laminitis is associated with the build-up of pressure in the toe area, either from the inflammation caused by the underlying laminitis or as a result of abscessing. By draining the pressure and any pus that may be present, the pain is relieved. This clearly has an ethical advantage to it, but may also help to reduce pain-related alterations in stance and gait, and hence further reduce the rate of rotation.

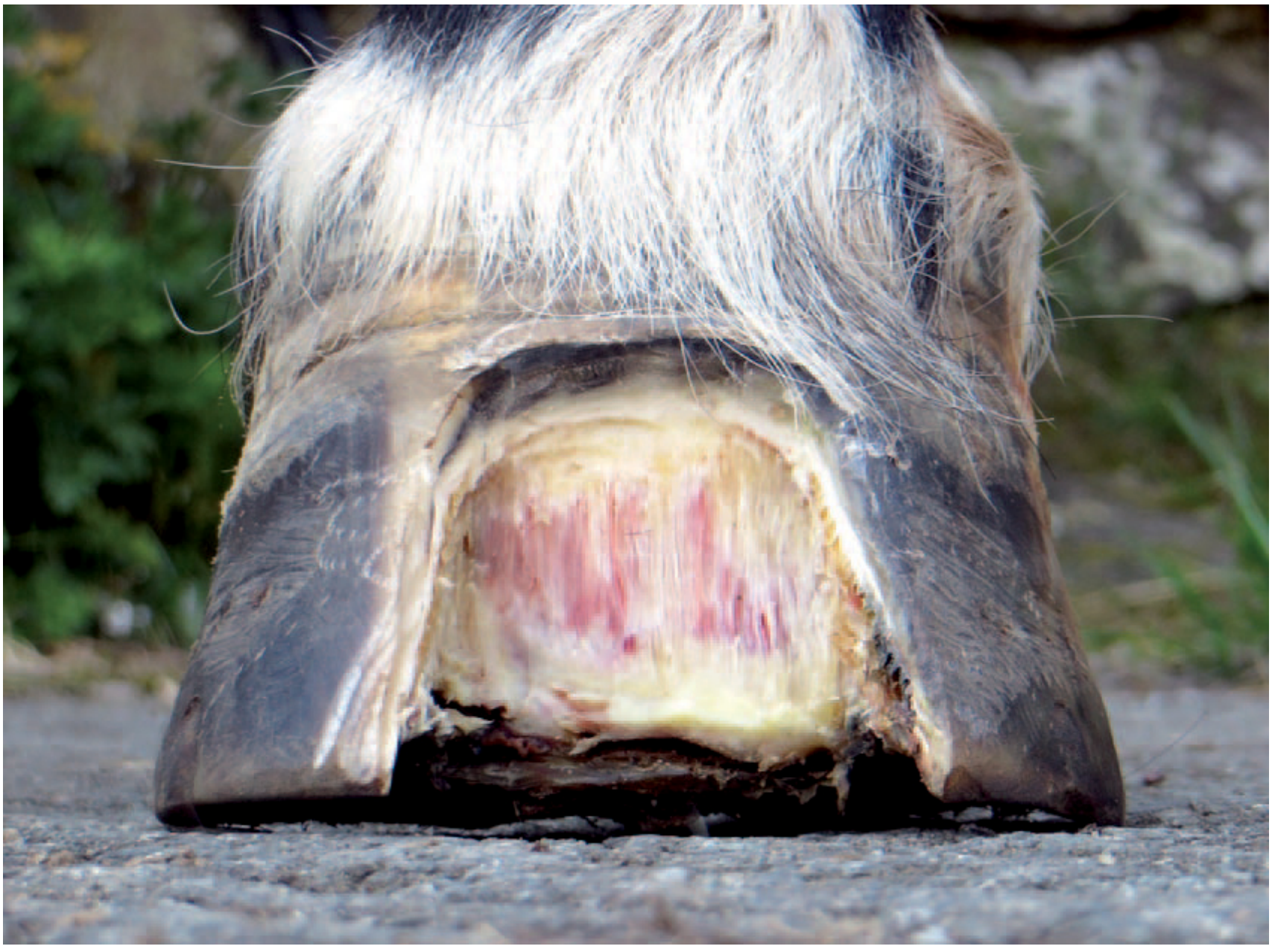


Fig. 90 A dorsal wall resection performed by the author on a cadaver leg as a demonstration. Most of the blood visible is not fresh and has been incorporated into the laminar wedge during the process of rotation. However, this trim did hit one viable dermal lamina, and in a live horse this would have significantly increased the risk of abscessing. There are plenty of similar photographs on the internet of live horses associated with claims that this approach represents best practice.

So of the three arguments for dorsal wall resections, one has merit, one may have merit and one does not. Interestingly, some vets do not recommend performing a dorsal wall resection until at least six weeks after the start of the acute rotation/sinker. If the underlying laminitis has not been brought largely under control by this point, then the prognosis is likely to be very poor anyway, regardless of whether or not a resection is performed. If the underlying laminitis is under control, then sufficient healing should have occurred by this point that the arguments for performing a resection lose much of their strength. There is also a risk with dorsal wall resections that live laminar corium is accidentally hit, and this can dramatically increase the risk of abscessing.

Deep Digital Flexor Tenotomy

Where rotation cannot be controlled, vets will sometimes cut the deep digital flexor tendon as a measure of last resort – a procedure called a deep digital flexor tenotomy. This is based on the thinking that tension in the DDFT will pull the pedal bone around, causing more rotation. The flaws in this thinking have already been discussed in Chapter 3. There is, however, one situation where a deep digital flexor tenotomy might be appropriate, and that is where there is permanent damage to the tendon, which prevents the horse from fully extending the coffin joint such that it cannot put weight into the heels. Where the underlying laminitis cannot be fully controlled and the short DDFT is contributing to continued rotation, severing the DDFT (usually around half way up the cannon bone) can provide a useful mechanism for reducing mechanical rotation.

Once severed, the cut ends of the DDFT will often heal on to the surrounding tendon sheath. Once any tension is placed on the lower half of the DDFT, these adhesions will strengthen, resulting in a

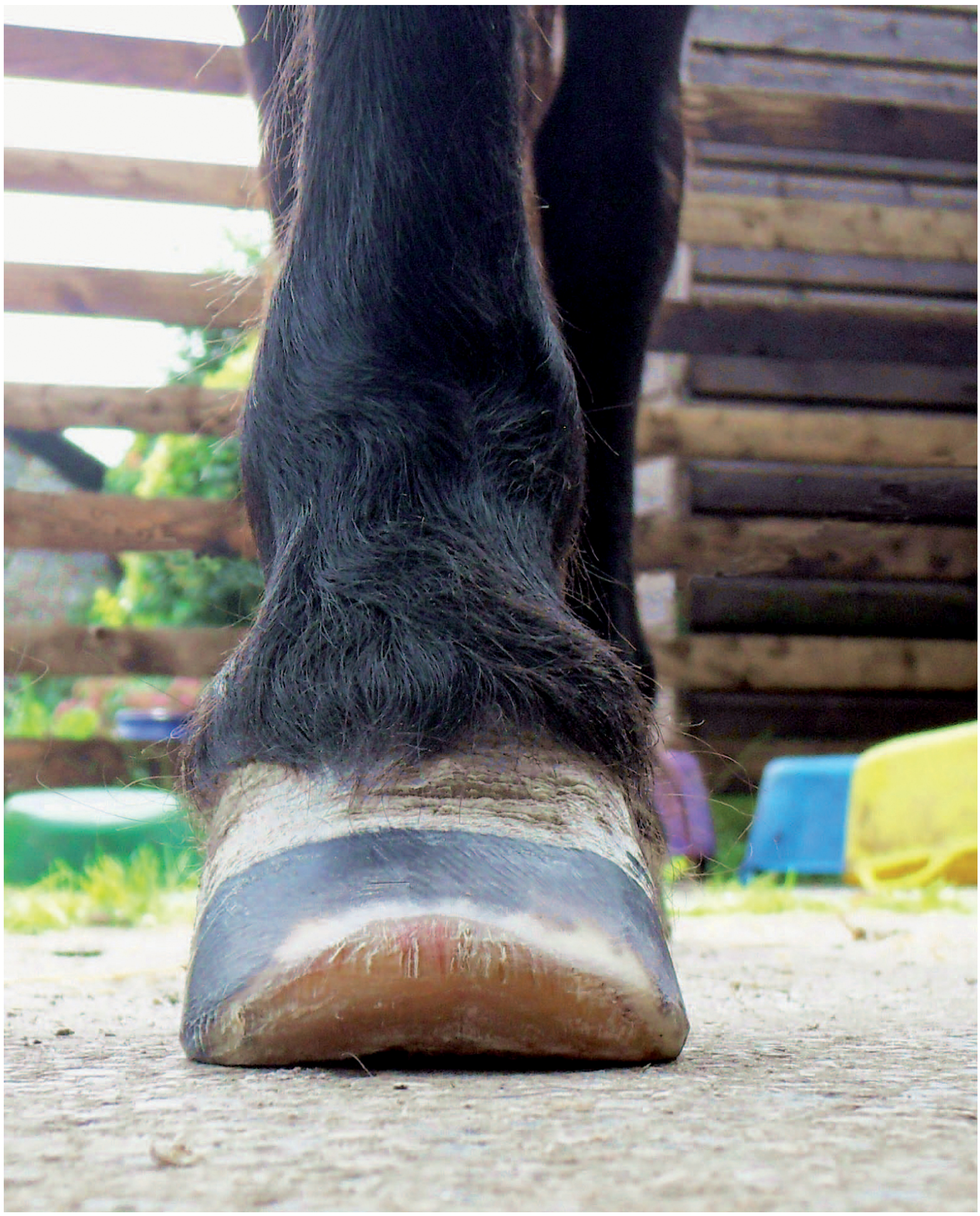
degree of support to the back of the foot from the remaining DDFT. This means that the DDFT supports the role of the suspensory ligament as it does in a non-severed case, but without the ability of the deep digital flexor muscle to control movement of the coffin joint.

Surprisingly, a horse with a severed DDFT can still move fairly well, although there should be no expectation of a return to athletic performance. The horse can still avoid tripping by lifting the toe just before impact using the extensor tendon. It can also lift the foot backwards and out of the way reasonably well during flight using the SDFT. What it can't do is use the DDFT to propel itself forward at the point of breakover (although it can use the SDFT to a lesser extent for propulsion). As such, acceleration is impaired, but once speed is achieved, it can be maintained. Given that propulsion comes more from the hind end than the front, a horse with both front DDFTs severed will still be capable of a surprising turn of speed once it has adapted to its disability.

The risk with a deep digital flexor tenotomy is that adhesions form between the cut end of the tendon and the surrounding sheath, but with the foot in an insufficiently extended position, resulting in a foot that will still not extend. For this reason, management of the case in the immediate aftermath of the surgery must be carefully controlled.

8 Horse-Centred Management of the Foot

The previous chapter outlined the established approach to managing a laminitic foot (the management of the underlying processes that cause the laminitis will be covered in the next two chapters). Just as the understanding of the processes of laminitis can be reframed as discussed in Chapter 3, the approaches that are taken to manage the laminitic foot can also benefit from the improved understanding that comes from reframing the problem.



A laminitic pony foot, trimmed with a toe dub, exposing laminar wedge.

PRINCIPLES OF HORSE-CENTRED FOOT MANAGEMENT

A number of principles need to be followed for the successful management and recovery of the foot in laminitis, whether full-blown or low grade; these are discussed below.

Supporting Weight via the Least Damaged Structures

If we want the foot to heal from the damage that laminitis causes, it is essential to manage the biomechanics of the foot, and in particular, the way the hoof is used to support the weight of the horse. The hoof effectively acts as an interface between the horse and the ground – the weight of the horse has to be transferred through the hoof in order to be supported. If we are to manage a damaged foot successfully in order to promote healing and recovery, then we need to aim to use those structures that are least damaged to support the horse's weight, and minimize the use of (or even totally avoid) those structures that are most badly damaged. Badly damaged tissue has far less structural integrity and so is far less able to support weight without sustaining further damage.

Of course this means that, before deciding on a plan for how best to support the horse's weight, we first have to understand which structures are the most damaged, and which the least, in the foot. The established model of laminitis would have us believe that the laminae are always the most badly damaged structure, and that other structures only become damaged as a result of mechanical stresses resulting from the inability of the laminae to support weight. But as we saw in Chapter 4, this is far too simplistic. It appears that all the structures in the foot become damaged to some degree, with the focus of damage being variable and dependent on such things as breed, the history of foot care and the current shoeing status. As such, it is critically important actually to assess each individual foot to determine where the areas of maximum damage lie. A strategy can then be developed to minimize the transfer of weight through these structures whilst maximizing the use of those structures that are more or less intact.

The use of x-rays is very important in determining what damage has been done, and where – particularly in acute cases where a lot of change may have happened internally in a very short time. So long as appropriate markers are placed on the outside of the hoof to allow us to see the outline of the hoof capsule, a latero-medial x-ray of the foot gives a wealth of information, as described in the previous chapter.

Whilst x-rays are a very important means of assessing the damage within the foot, an x-ray cannot actually tell you which tissues are currently inflamed, and what hurts. For that reason, it is important to use every technique available to build up a picture of where the damage is focused within the foot. Watching how the horse moves can give a lot of useful information (although the most serious cases may be too sore for it to be ethical to move the horse specifically for the purpose of gait analysis). For example, a horse that appears fairly comfortable on smooth tarmac but looks uncomfortable on sand is likely to have inflamed soles (the sand presses on the sole, whereas the tarmac mostly loads the wall).

Careful examination of the feet can also help. For example, a sole that moves significantly with thumb pressure applied over the tip of the pedal bone is likely to be fairly thin. If thumb pressure in that area also elicits a flinch response, this tends to suggest that the solar corium is inflamed. Heat can also be a useful indicator. If the front feet are significantly hotter than the hinds, then that suggests that the front feet are more affected by the laminitis than the hinds. Similarly, if the sole is significantly hotter than the coronary band, then this might indicate significant inflammation of the solar corium. It is of course important to rule out other causes of temperature differences, such as the horse standing with its front feet in the sun and its hinds in the shade! Also, different parts of a healthy foot also tend to exhibit different surface temperatures, so it is important to understand the temperature profile of a healthy foot before attempting to locate specific inflamed structures in a laminitic foot. Absolute heat is not a useful indicator because the surface temperature of the hoof varies widely, depending on the conditions. It is only differences in heat that are useful.

All of this unfortunately takes experience, which is one of many reasons why a competent professional should always be involved in the management of any laminitis case. Even professionals need to build a library of experience, and that cannot be done without carefully examining many cases, both healthy and laminitic, so as to gain knowledge of the subtle differences that can be used as clues.

Providing Support at both the Front and Back of the Foot

It is easy to forget when discussing how to support the horse's weight that it is important to provide support to both the front and back portions of the foot. With the flexor and extensor tendons relaxed, the foot can effectively flop either way, and will try to find a level. If only the front of the foot is supported, it will topple backwards, and if only the back is supported it will topple forwards. The horse can compensate for a lack of support to the back of the foot by tensing the deep flexor muscle so as to pull on the deep flexor tendon, but as we have already seen, this will tend to increase the rate of damage to the laminae. The horse can compensate for a lack of support at the front of the foot by tensing the extensor muscle so as to pull on the extensor tendon, but the relevant muscle is fairly small and will tire or cramp fairly rapidly if the horse relies on it. If a vertical line is drawn downwards from the centre of articulation of the coffin joint, there needs to be some degree of weight support both in front of and behind this line.

This helps to explain why the use of frog pads, as described in Chapter 7, is not that effective. In the absence of a shoe, a frog pad provides very little support forward of the centre of articulation of the coffin joint, and hence the foot tends to topple forwards on to the toe where the greatest damage typically lies. The horse can avoid this by tensing the extensor tendon, but this is not something it can do for extended periods, as the extensor muscle will become fatigued. To some extent the horse can reduce the effort needed in the extensor muscle by placing the feet further forwards, as in the classic laminitis stance. This works by using the natural tendency of the ligaments around the coffin joint to return the joint to a neutral position – but it is not a large effect.

The need for support to the front and back portions of the foot also explains why the heart-bar shoe does not provide as much benefit as is often claimed. The back half of the foot is typically already well supported in laminitis cases. Except perhaps in the most serious sinker cases, the wall at the heels will be well attached to the laminae and hence to the internal structures of the foot. What is needed is additional support for the front of the pedal bone. A heart-bar shoe attempts to provide this by providing support via the frog, and yet only the very tip of the frog is forward of the centre of articulation of the coffin joint. As such, any additional support must be provided via a fairly small area of frog. This area of frog is also close to the centre of articulation of the pedal bone, and hence provides little leverage on the pedal bone – a lot of force is required here to prevent the bone rotating, compared to the amount of force that would be needed if it were applied further forwards. The amount of force that can be applied in this location by a heart-bar shoe is limited – too much force results in severe bruising to the frog corium, which in turn removes this area as a viable means of support. The end result is that the benefit gained by the fitting of a heart-bar shoe (including plastic variants) is fairly minimal. *Hickman's Farriery* (second edition), a classic UK farriery text book, recognizes this failing of heart-bar shoes:

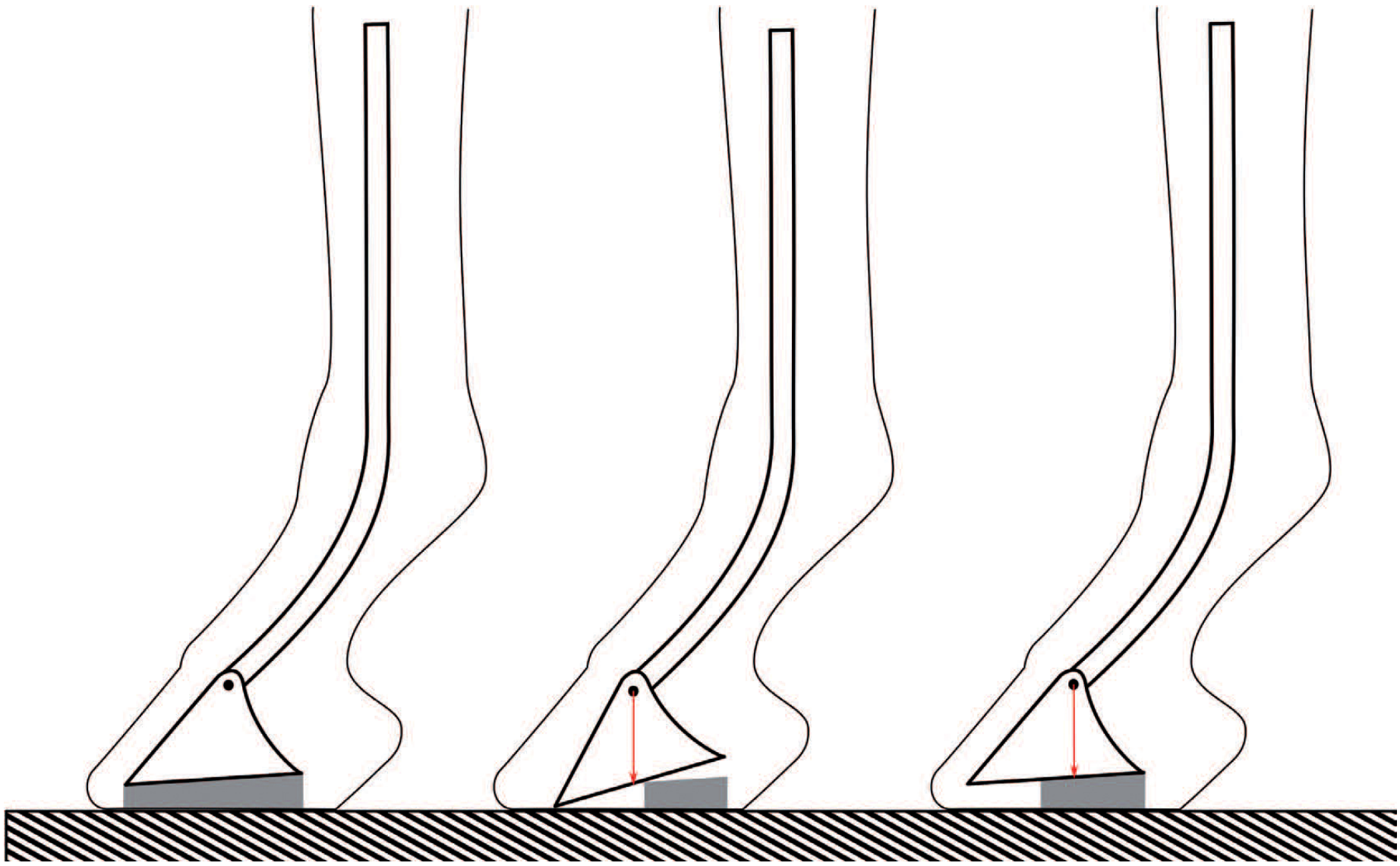


Fig. 91 A stylized representation of the lower leg, with the pastern, cannon bone and suspensory ligament represented as a leaf spring and the coffin joint as a pin joint. LEFT: *Without tension in the tendons, the pedal bone will find its own level based on the available support.* CENTRE: *If support is only provided to the back of the pedal bone it will topple forwards.* RIGHT: *As long as there is some degree of support forward of the centre of articulation of the coffin joint (red arrow), the pedal bone will not topple.*

A heart-bar shoe has a bar fashioned to press just behind the point of the frog with the aim of mechanically opposing rotation of the distal phalanx, but is incapable of doing so effectively.

The Preservation of Solar Material

Given the tendency of the sole to thin under the tip of the pedal bone as a result of compromise to the solar circumflex artery, anything that makes the sole artificially thinner in this area risks making the situation worse. A thinner sole means that: the weight of the horse is less well supported by the sole as the sole will tend to flex more; there is an increased risk of bruising to the solar corium; and ultimately the risk of solar penetration is significantly increased. For these reasons it is imperative that no solar horn is removed from under the front portion of the pedal bone when managing a laminitis case, even if the solar horn appears to be artificially thick. The one exception to this rule is where a recovering case has reached the point where the false sole is starting to exfoliate anyway, in which case it may, very occasionally, be appropriate to assist the horse in removing material that is no longer needed. It should also be said that donkeys sometimes build up extreme amounts of false sole (far more than would usually be seen in a horse) and cautiously removing some of this can be useful in certain situations so as to obtain correct balance.

The Avoidance of Vibration

We have already seen that inflamed coria are particularly sensitive to vibration. Vibration in a laminitis case results in further damage to inflamed coria, and hence needs to be minimized as far as possible. The use of metal shoes significantly increases the amount of vibration the foot is subjected to, and the typical frequency of vibration created by a shoe hitting hard ground is right in the range most likely to

cause damage to already inflamed tissue. In the initial stages of recovery from full-blown laminitis, the horse is likely to be on box rest and hence vibration is not a real issue. But in the later stages of recovery, when the horse is more likely to be crossing a yard to get to a field or may be gradually reintroduced to work, the vibration created by a shoe can be seriously counterproductive.

Because LGL cases are routinely missed (particularly where shoes are masking lameness to an extent), they are often in some degree of work despite the inflammation to the feet. In that situation, the potential for vibration to contribute to damage is significant.

The vibration associated with nailing a shoe on to the foot is also a major issue, particularly when the inflammation is at its worst. For that reason, any shoe that is applied to an actively laminitic foot should ideally be applied with glue.

Encouraging Movement

A key part of the inflammatory processes that occur in laminitis is the pooling of fluid in damaged tissues. If oedema is allowed to collect in the foot, the pressure from it can both mechanically damage tissue and also reduce circulation in the foot, causing further damage, as described in Chapter 6. The body's natural mechanism to prevent the build-up of excess fluid in tissues is the lymphatic system. This system consists of channels and nodes that move excess tissue fluid from the extremities back towards the heart. It works as a back-up system to clear any fluid that isn't sufficiently cleared by the venous system. But unlike the venous system, the lymphatic system has no heart to pump fluid. Instead, it relies on lymph ducts passing through muscles, such that any muscle movement massages the ducts and draws fluid away from the extremities. This system can therefore only function when the horse is moving: without movement, any excess fluid in the feet will pool and potentially cause further damage.

This leaves a tricky conundrum when it comes to managing the early stages of a laminitis attack. If the horse is allowed to move too much, too early, further mechanical damage may be done. On the other hand, if movement is limited too much or for too long, oedema will tend to pool in the foot, causing more damage. This leads to the fifth and final principle of horse-centred management of laminitis – the horse needs to be allowed to move as early in the recovery process as it is safe for him to do so.

The challenge is to know how much the horse can safely move at each stage in recovery, and the principle that pain equates to damage can be very useful here. So long as the horse is moving without pain, then it is unlikely that further mechanical damage is being done (assuming that no pain-relieving drugs/supplements are in use). So the goal in managing any case should be to get the horse off pain medications and comfortable in whatever mechanical support approach is appropriate as soon as possible. This places the emphasis on finding ways to support the foot mechanically so as to minimize pain as early in the process as possible (as well as getting the underlying causes of the laminitis under control). The horse can then be allowed limited movement as soon as it is comfortable without pain relief, so reducing the risk that oedema will further slow recovery.

It is very important that any movement is voluntary on the part of the horse. Forcing the horse to move (either physically or psychologically) may result in the horse ignoring pain cues and doing more damage. The ideal is to provide a small, safe space in which the horse can move around freely.

For more serious cases, there may be a significant period between the onset of lameness and safely achieving the goal of getting the horse moving again. During this period, the technique of manual lymphatic drainage (using massage to stimulate flow in the lymph ducts) can be very beneficial (as shown by at least one research study) – although sadly, practitioners experienced in this approach in horses are few and far between. Nevertheless the beneficial effects of manual lymphatic drainage provide a useful piece of evidence supporting the oedema model of laminitis.

MANAGEMENT OF THE FOOT IN FULL-BLOWN LAMINITIS

The following sections outline how the horse-centred approach to managing the foot in a full-blown laminitis case varies from the established approach.

Drug Interventions

While few would argue that pain relief is a bad thing, it is crucial to understand that pain has evolved for good reasons, and that if we are to circumvent the pain mechanism with drugs, we really need to understand the consequences of that. Pain is there to warn the brain that damage is being done. The brain can then attempt to take avoiding action so as to minimize the pain, so reducing the likelihood and/or severity of damage. For example, if a human accidentally touches a hot pan, the brain responds with a reflex withdrawal of the hand, so reducing the severity of the burn. If the pain sensation were absent, there would be a far higher risk that a more serious burn would result.

In a similar fashion, the toe pain a horse experiences during a full-blown laminitis attack encourages its brain to try to find ways to avoid that pain. This will consist of altering posture so as to reduce pressure on the laminae, reducing concussion on the feet by not moving so fast or so energetically, or standing very still or lying down so as to avoid any potential for movement to cause further damage. Therefore care must be taken with a horse on pain relief medication to ensure that it doesn't feel comfortable enough to charge around, and so do more damage to the feet. For that reason, any laminitis case should ideally be kept on box rest until the painkillers are no longer needed, or at least until the dose has been reduced to a minimal level.

As discussed in the previous chapter, the use of ACP can be helpful in encouraging a laminitic horse to move around less while on box rest. Where a horse copes particularly badly with box rest, it may be appropriate to find an alternative way to restrict movement. For example a horse can sometimes be kept in a very small area in the corner of a field using temporary electric fencing. This has the advantage that the horse can still take part in social behaviour with other horses in the field, which in turn reduces stress levels. Ideally the area should be small enough so that the horse does not have room to trot. A further advantage is that the resulting low-level movement helps with lymphatic drainage.

Another important thing to recognize is that the postures and habits that a horse with severe laminitis adopts do not represent suffering in themselves – they are rather based on an attempt by the horse to minimize discomfort and hence minimize damage. So for example, standing rocked back reduces pain in the front toes but also reduces damage to the laminae. If the horse can adequately adapt posture and gait so as to reduce pain to a minimum without the use of painkillers or with the use of fewer painkillers, then this is advantageous, as the adaptations not only reduce pain, but reduce damage. Removing or reducing the pain means that the horse has less motivation to stand in a posture that reduces mechanical damage to the feet.

Over-use of painkillers may therefore be disadvantageous in terms of long-term outcome, whilst not actually gaining the horse any significant additional level of comfort. Masking pain to anything more than a minor extent also removes the possibility of safely allowing the horse limited movement to help with lymphatic drainage. Of course the pain-avoidance postures may in themselves cause temporary musculo-skeletal problems over time, and for that reason, it can be helpful to have an appropriately trained body worker involved in the case to manage this aspect.

In addition, the side effects of the pain medications have to be considered. Most oral painkillers for horses carry a risk of causing or exacerbating gastric ulcers, especially when used at a high dose or for long periods of time. Given the suggested link between digestive disorders and laminitis, the last thing a laminitic horse needs is to develop gastric ulcers. Even if the gastric ulcers themselves do not directly contribute to the underlying laminitic processes, the pain associated with them is likely to increase stress levels and hence increase insulin resistance as a result. This is another reason why the dose of painkillers should be kept to the minimum needed to cover the ethical needs of the case – especially if the horse has a history of gastric ulcers.

As the horse cannot be asked for its opinion, this area is clearly extremely difficult to assess, and getting the degree of pain relief right can be tricky. For obvious ethical reasons, enough pain relief needs to be given to ensure that the horse is reasonably comfortable, but overdoing it carries risks too. Because owners usually know their horses well, the owner should ideally be involved by the vet in the process of deciding on appropriate levels of pain relief, and vets and owners should be prepared for the likelihood that doses will need to be adjusted upwards or downwards depending on response and the timing of any recovery. The pain relief approach also needs to be considered as part of a whole horse approach, taking into account the other issues already discussed.

Other drugs may be appropriate in the management of the underlying laminitis, but these will be discussed in a later chapter.

Removal of Shoes

The removal of shoes in full-blown laminitis is commonly (although not universally) suggested as part of the current mainstream approach. However, it is important to understand why this would be beneficial, and also under what circumstances removal of shoes alone may not be the right solution for the foot.

The argument for the removal of standard shoes is simple and compelling. The fitting of a standard shoe creates a scenario where the vast majority of the horse's weight is supported via the laminae (a small proportion is supported via the white line and sole or via the coronary band). In contrast, an unshod horse walks not only on the inner wall and white line, but also the frog and the rim of the sole. As such, more structures support weight in an unshod horse than in a shod one. In a healthy foot there is more than enough redundancy of structure to allow the horse to cope with supporting its weight via a more limited selection of internal foot structures. But where full-blown laminitis is present, the very structure on which a shoe relies primarily for weight support is the structure that is most likely to fail catastrophically: the laminae. The reasons why heart-bar shoes are little better than standard shoes has already been covered.

Another issue with shoeing a laminitic foot is that the rate of wall growth, particularly at the heels, can be rapid when compared to a healthy foot. Give that the hoof wall is slightly conical, allowing the hoof to grow longer results in a larger footprint. When this larger footprint is fixed to a rigid shoe that can't expand with growth, there is a potential for the hoof wall to be crushed inwards, applying pressure to the solar circumflex artery. For this reason, any shoe applied to a laminitic foot needs to be reseated much more regularly than with a healthy shod foot.

The issue of vibration means that many laminitics these days are fitted with glue-on shoes; but gluing a shoe on to a laminitic foot risks creating different problems. Within a few weeks of the onset of laminitis, the bottom of the hoof, and in particular the white line, is likely to be full of blood products and rotting rapidly. Keeping a shoe glued to this failing substrate can be challenging. More importantly, the presence of a shoe plus associated glue over this area removes any possibility of using topical disinfectants to manage the rot. The shoe also prevents the evaporation of moisture from the surface of the hoof horn – moisture that is created within the foot as part of the natural mechanisms for keeping the hoof horn at the correct level of hydration. Warm, moist and anaerobic conditions therefore result at the junction between the shoe and the hoof, conditions that are perfect for promoting bacterial and fungal infections. Within a few shoeing cycles, the bottom of the wall often rots significantly and it can become increasingly difficult to find sufficient structure to which to reapply a shoe. And of course, glue-on plastic shoes are very expensive to buy and to fit.

Removing the shoes has significant potential advantages. However, it is very important to recognize that removing the shoes alone is often not a sufficient solution to the mechanical problems the horse is struggling with. Once the shoes are removed, a strategy is needed to manage the foot according to the principles outlined above. It is particularly important to recognize cases where the sole is badly damaged. Simply removing the shoe in such a case risks shifting additional weight on to an already badly damaged sole, and hence increasing the risk of further damage. If appropriate alternative means of supporting the foot are not available, then it may be better in some cases to leave the shoe in situ

than to remove it. It cannot be stressed enough that the decision of how best to manage the mechanical forces on a laminitic foot is complex, with no one-size-fits-all approach. As such, a professional with experience in this field should always be involved in the case.

Trimming the Bottom of the Hoof

The detailed processes of trimming the laminitic hoof are beyond the scope of this book and should be left to a suitably qualified professional. However, an understanding of the broad principles used in the trimming of the hooves can be useful to the owner. It should also be stressed that, particularly in the early stages of an acute laminitis case, it is not possible to trim the hoof accurately without reference to recent x-rays.

In full-blown laminitis, the first priority of trimming is to return the pedal bone to its normal 3-to 5-degree angle to the ground. By the time they are ready to be trimmed, such cases usually have a pedal bone angle that is far greater than this as a result of excessive heel growth – particularly in chronic cases or within a few weeks of an acute case – so it is usual to have to reduce the height of the heels.

It is vitally important before changing the angle of the foot to check for the presence of any damage to the deep digital flexor tendon. As discussed in Chapter 3, any shortening of this tendon, or any adhesions to the tendon sheath, will mean that reducing heel height suddenly – or even at all in some cases – is likely to do more harm than good. Where there is a problem with the tendon, this makes it harder for the foot to recover – the tendency for the pedal bone to press on the solar circumflex artery where excessive heel height cannot safely be removed makes compromise of the solar circulation more likely. If such a case already has a good sole with little sign of damage, and the underlying laminitis can be controlled, then the prognosis is fairly good. If, however, there is a combination of a short tendon and significant existing damage to the solar corium, or the underlying laminitis proves difficult to control fully, then the prognosis is likely to be poor. As discussed in Chapter 7, this may be the rare situation that justifies a deep digital flexor tenotomy.

When reducing the angle of the pedal bone to the ground, it is usual to err on the high side – aiming for 5 degrees rather than 3 degrees. There is no need to reduce the angle below 5 degrees as this is sufficient to minimize the risk of damage to the solar circumflex artery. Reducing the angle too far makes it harder for the horse to shift weight to the heels and avoid weighting the toe.

More of an issue is the exact amount of material to remove. The constraint of placing the bottom of the pedal bone at 5 degrees to the ground still leaves a degree of freedom in terms of the overall height of the foot. The correct approach to take depends on the quality of the sole. If the sole is thick and reasonably healthy with some degree of concavity, then the foot can be trimmed such that the front portion of the sole is just on the ground, as is normal for healthy unshod feet. Where the sole is very thin under the tip of the pedal bone, this approach risks thinning the sole in this critical area (Fig. 92). Even if the sole isn't thinned, bringing an already badly damaged sole into greater contact with the ground is likely to be counter-productive. In such situations, the better approach is to leave the entire foot a little longer whilst still trimming at the same angle. This results in an appearance often described as a 'heel rocker', where a ramp is created at the desired angle between the heel and the quarters, roughly level with the apex of the frog. The portion of the foot forward of the frog (and hence the badly damaged area under the tip of the pedal bone) is usually lifted very slightly off the ground.

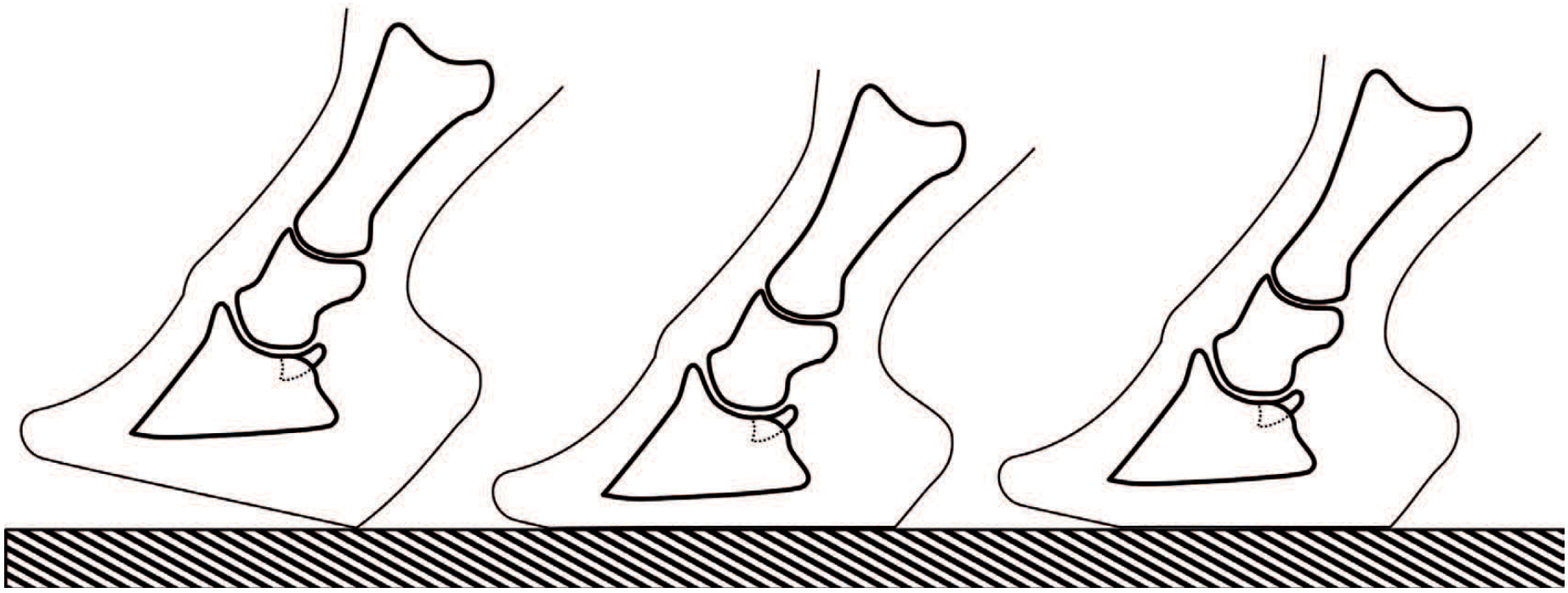


Fig. 92 LEFT: ***A significantly rotated hoof capsule typical of a chronic laminitis case before trimming, with the pedal bone correctly oriented to the ground.*** *CENTRE:* ***The same foot trimmed to bring the sole just into contact with the ground at the toe, as a healthy foot would be trimmed. However, the distortion in the hoof capsule means that the sole tubules are pulled forwards to a shallower angle at the toe, giving less vertical height, so the tip of the pedal bone is now too close to the ground.*** *RIGHT:* ***A heel rocker applied to the same foot. More vertical height has been retained whilst keeping the same balance. The tip of the pedal bone is now the correct distance from the ground, and the damaged solar corium directly under the tip of the pedal bone is no longer weight-bearing.***

The advantage of a heel rocker in a thin-soled case is that the most damaged area of sole – the area under the tip of the pedal bone – is preserved as thick as possible, but is also kept clear of the ground and hence is not weight-bearing. However, it is important not to overdo the heel rocker: there needs to be material on the ground a reasonable distance forward of the centre of articulation of the coffin joint, because if that rule isn't followed, then the foot may topple forwards off the heel rocker, resulting in weight being exclusively transferred to the most damaged structures in the foot!

Trimming the Toe and Dorsal Wall

Once the bottom of the hoof has been trimmed to an appropriate height and angle, the remainder of the trim tends to focus on sorting out the toe area, and in particular, the breakover position. This is the point at the toe of the hoof that initially remains on the ground as the heels lift during normal movement. In a healthy unshod foot, the breakover position sits just in front of the white line at the toe. This position is the optimal compromise between reducing leverage to the hoof wall as the foot breaks over, and providing some wall material in contact with the ground to resist wear. In full-blown laminitis, as the hoof capsule rotates away from the pedal bone, the breakover position can move significantly forwards of its normal position in relation to the pedal bone. The result is that, at the point of breakover, there is a greater leverage force that tends to pull the hoof wall away from the pedal bone. In a case where the laminae are already failing to keep the wall attached to the bone, this kind of leverage can cause further damage. For that reason it is important to trim the hoof such that the breakover position is back to its normal relationship with the pedal bone.

There is a range of options as to how to bring the breakover back in a laminitic foot. The most conservative approach, and the one typically favoured by the author, is to create a 'dub' to the toe. This involves bringing the breakover back to the correct point at ground level, and then removing the minimum of material above that. This is typically done by putting a 45-degree bevel on the bottom of the wall until the desired breakover is achieved, and then rounding off the toe to create a more curved outline. This approach achieves the desired goal of removing the excess leverage associated with the breakover being too far forward, but the combination of growth and wear will mean that the breakover will move forwards again as the trim grows out. The breakover often becomes a problem again within

two to four weeks, and hence the foot will need trimming again fairly soon. Of course, in a serious laminitis case, it is usually desirable to trim fairly frequently anyway to avoid the build-up of excessive heel height, so this in itself isn't a big disadvantage of the dubbing approach.

The more extreme option is to identify the position in which the outer surface of the hoof wall would be, relative to the pedal bone, if the foot were healthy, and then remove all material forward of that line. In any case where there is significant rotation, the hoof wall towards the bottom of the foot may be entirely outside the original outline of the hoof capsule. As such, this approach will tend to remove the entire thickness of the hoof wall at the bottom of the foot, and depending on the degree of rotation, often some way up the hoof. This then becomes a form of dorsal wall resection. The advantage of this approach is that, assuming that there is no further rotation, the trim will last for many weeks as there is no material to grow down that would bring the breakover further forward again. This can be done with a rasp in a similar (but more dramatic) way to the normal removal of flare.

A full resection as described in Chapter 7 also brings the breakover back, although often it brings it back too far. This is often compensated for by fitting a heart-bar shoe that has the breakover in the correct position.

There are two key disadvantages to dorsal wall resections in whatever form. The first is an increased risk of abscessing. Where the horse has fully recovered from the underlying laminitis, the dermal laminae will, as part of the early stages of healing, have covered themselves with a thin layer of 'repair horn'. This means that any infectious materials that get into the laminar wedge via the resection cannot penetrate through the repair horn to infect live tissue. The horse is therefore safe from the risk of abscessing. But if the laminitis is ongoing, or a further acute attack occurs, this thin layer of repair horn may be absent, or it may become damaged again. At this point, the presence of infectious material deep in the laminar wedge dramatically increases the risk of the dermal laminae becoming infected, resulting in an abscess. For this reason alone it is surely better to preserve as much of the external wall as possible so as to keep the laminar wedge covered, and to increase the distance that infectious material has to migrate through the laminar wedge in order to reach any damaged dermal laminae.

Fig. 93 A partial resection performed by removing with a rasp all the material outside the original outline of the hoof capsule relative to the pedal bone. (Photo: Gina Foulkes-Halbard)

The second disadvantage relates to the assumption that the laminar wedge is always composed solely of dead material and that there is no risk of penetrating live tissue. This tends to be true in acute laminitis attacks where the laminae tear catastrophically and then recover to a fairly normal length on the inside of the laminar wedge. However, in a chronic case, particularly a ‘slow and even burn’ chronic case, the laminae can stretch slowly without actually tearing. Normal healthy laminae are around 1½ to 2mm long, but in chronic laminitis they can stretch to as much as 10mm long and still remain viable, complete with a blood supply and nerves.

The dissection of hundreds of laminitis cases reveals a small but significant percentage that show stretched but otherwise largely intact laminae. Stretched laminae are indistinguishable from dead laminae on an x-ray (unless contrast material is injected into the vein so as to form a venogram, which is not routinely done in the UK). Only in the situation where there is a gas pocket immediately behind the wall (indicating the presence of a haematoma) can an x-ray suggest that the laminar wedge is likely to consist of dead material. Even then, there is no guarantee that there aren’t at least some stretched laminae immediately behind the haematoma. The quality of the white line gives some indication of what has happened, but this gives an out-of-date history of the damage to the laminae, and is not certain proof of what is going on at the present moment. As such, it can be argued that, under the principle of ‘first do no harm’, a conservative approach should always be taken, which assumes that there may be live tissue directly behind the hoof wall in any laminitis case. This is a classic example of the importance of understanding the full range of variation in the underlying pathological processes before attempting to trim laminitic feet.

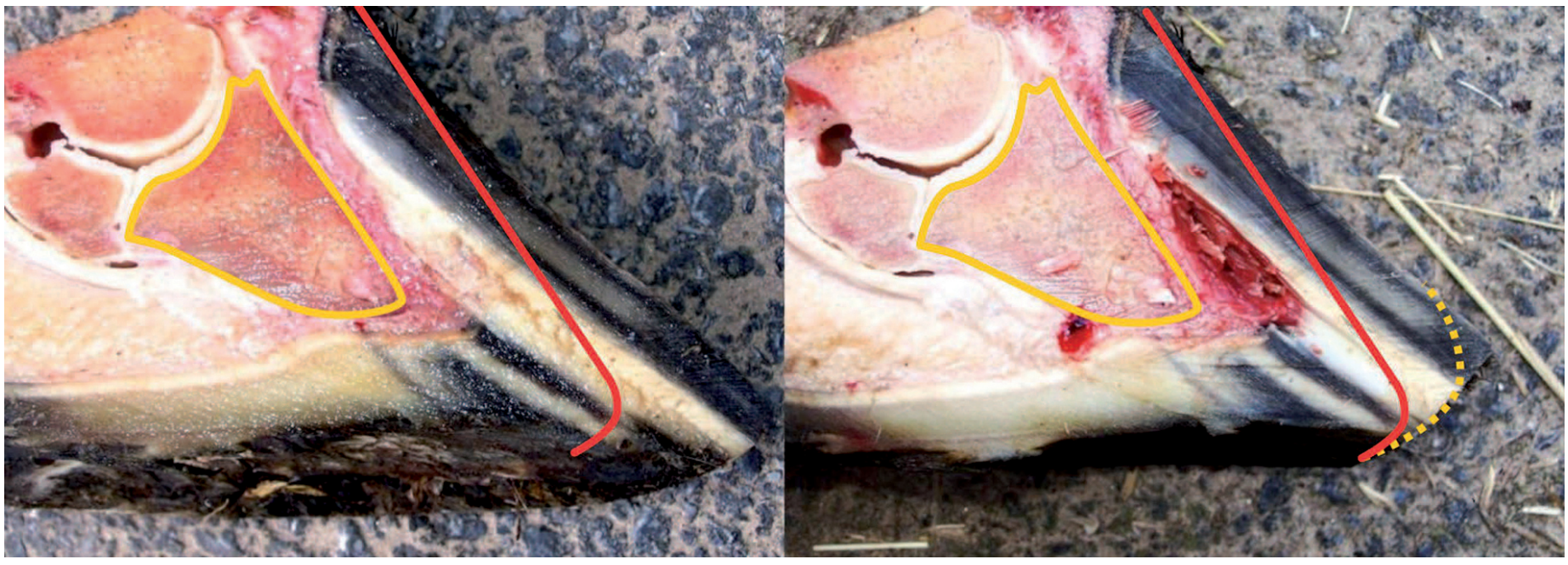


Fig. 94 LEFT: ***A dissection of a rotated foot showing the pedal bone (outlined yellow), and indicating the material that would be removed by a typical dorsal wall resection (red line). The laminar wedge appears to be dead horn, albeit of fairly good quality.*** RIGHT: ***Removal of successive stretched epidermal laminae on the same foot reveals that between them there is viable vascular tissue – dermal laminae that have stretched, rather than breaking. The resection line is dangerously close to live tissue. A more conservative dub that achieves the same breakover point over the short term is shown in dotted yellow.***

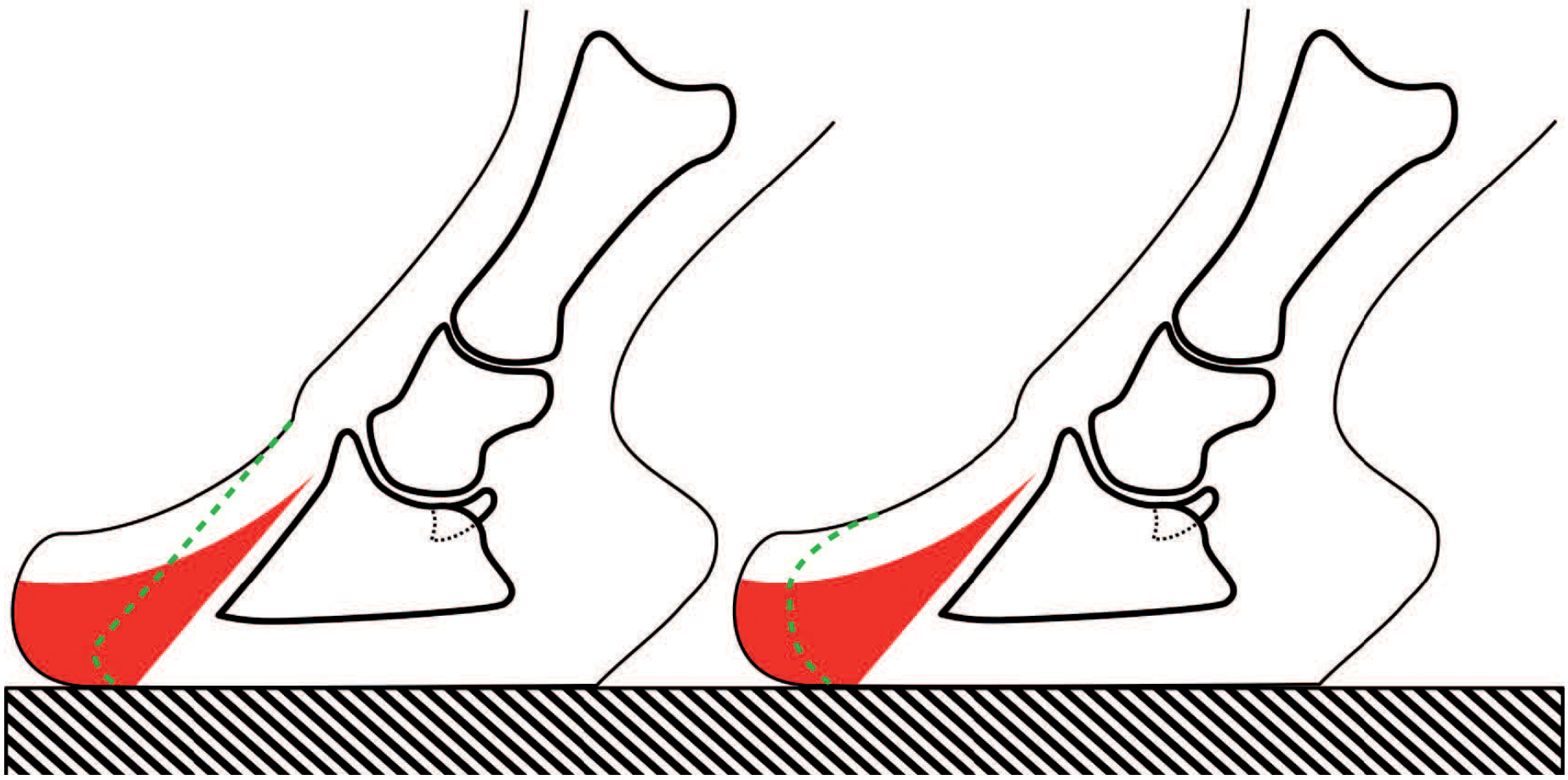


Fig. 95 LEFT: *A severely rotated foot with the laminar wedge shown in red. A typical dorsal wall resection is shown in dotted green. This exposes damaged laminar wedge close to the laminar corium. If the laminar wedge contains some stretched rather than broken laminae, this trim may cut into live tissue. Where power tools are used, the trim is often more severe than indicated here.* RIGHT: *The same foot with a conservative dub applied. The breakover is brought back to the same position, but minimal hoof wall is removed. The infection risk is reduced and the risk of trimming into live tissue is minimal. This trim will need to be reapplied far more frequently.*

It is possible to take a hybrid approach between a dub and a resection. This can be seen either as a minimal resection or a more aggressive dub. The increased risks of such an approach are, in the author's experience, minimal so long as the exposure of laminar wedge is kept below the level of the bottom of the pedal bone. This can be done easily in milder cases of rotation, but may not be possible in some extreme cases.

Despite the discussion above, the arguments for performing a dorsal wall resection if there is a build-up of pressure in the foot from oedema or abscessing still hold some sway. One possible compromise approach is to carefully drill a small hole in the front surface of the hoof wall just above the level of the bottom of the pedal bone (a technique called dorsal wall drilling). If fluid is present under pressure then this should spurt out of the hole as the wall is breached, providing immediate pain relief. In the case of abscessing, this will also allow the pus to drain, facilitating healing. So long as a sterile drill bit is used and the resulting hole is then plugged with sterile material once any pus has drained, the risks of further abscessing should be minimal. As with the dorsal wall resection, this is something that should only be carried out by a vet or under veterinary supervision.

The Management of Sinker

The most difficult cases to manage from a foot perspective are those where there is severe sinker. Given the catastrophic nature of the damage to the coronary band, it can be easily understood why sinker is seen as such a serious outcome of laminitis. However, by returning to the reframed view of the problem, a potential means of recovery becomes apparent. The distortion to the coronary band comes about because the horse's weight is being supported more by the hoof wall than by the sole. The traditional approach to sinker is to try to support the pedal bone by fitting a heart-bar shoe. Whilst such a shoe provides support to the back of the pedal bone via the frog, the front of the pedal bone still remains supported solely by the hoof wall via the failed laminae, so perpetuating the very forces that are creating the sinker in the first place.

If, on the other hand, the opposite is attempted – that is, the hoof wall is trimmed off the ground and the front half of the pedal bone is supported only via the sole, the forces pushing the hoof wall upwards can be removed. Distorted tissues always attempt to return to their correct shape, and incredibly, the coronary band can recover a more normal shape within two to three weeks once the abnormal forces are removed. It is sometimes claimed that this can only be achieved by performing a full dorsal wall resection, but in practice anything that removes load from the dorsal wall will work well. As the coronary band recovers, further hoof wall production reorientates back to a more normal direction, and the thickness of the wall and hence the spacing of the tubules also return to more normal parameters.

The pattern of wall growth in a recovering sinker case (looking at the toe area from the side of the foot) can be very instructive. The history of the case can be read, starting with the oldest growth nearest the ground. The pre-sinker growth is typically rotated significantly away from the front surface of the pedal bone, as sinker rarely occurs without at least some degree of rotation. As the sinker episode starts, the coronary band is sheared and crushed. New wall growth is now oriented more horizontally, but the growth rate in the toe area also drops significantly as a result of compromised circulation. The toe pain results in the horse shifting weight to the heels, and hence the wall here is stimulated to grow faster than normal. The result is that the growth rings corresponding to the start and end of the sinker episode are almost touching at the toe and yet far apart at the heels. If the underlying laminitis can be brought rapidly under control and/or the mechanical forces shunting the wall past the coronary band can be dramatically reduced, the coronary band relaxes back into a more normal shape, and the post-sinker growth is now roughly parallel to the front surface of the pedal bone. The overall effect is of a marked step or shelf that appears in the wall. In severe sinker cases this step can be dramatic, while in mild cases it can be very subtle.

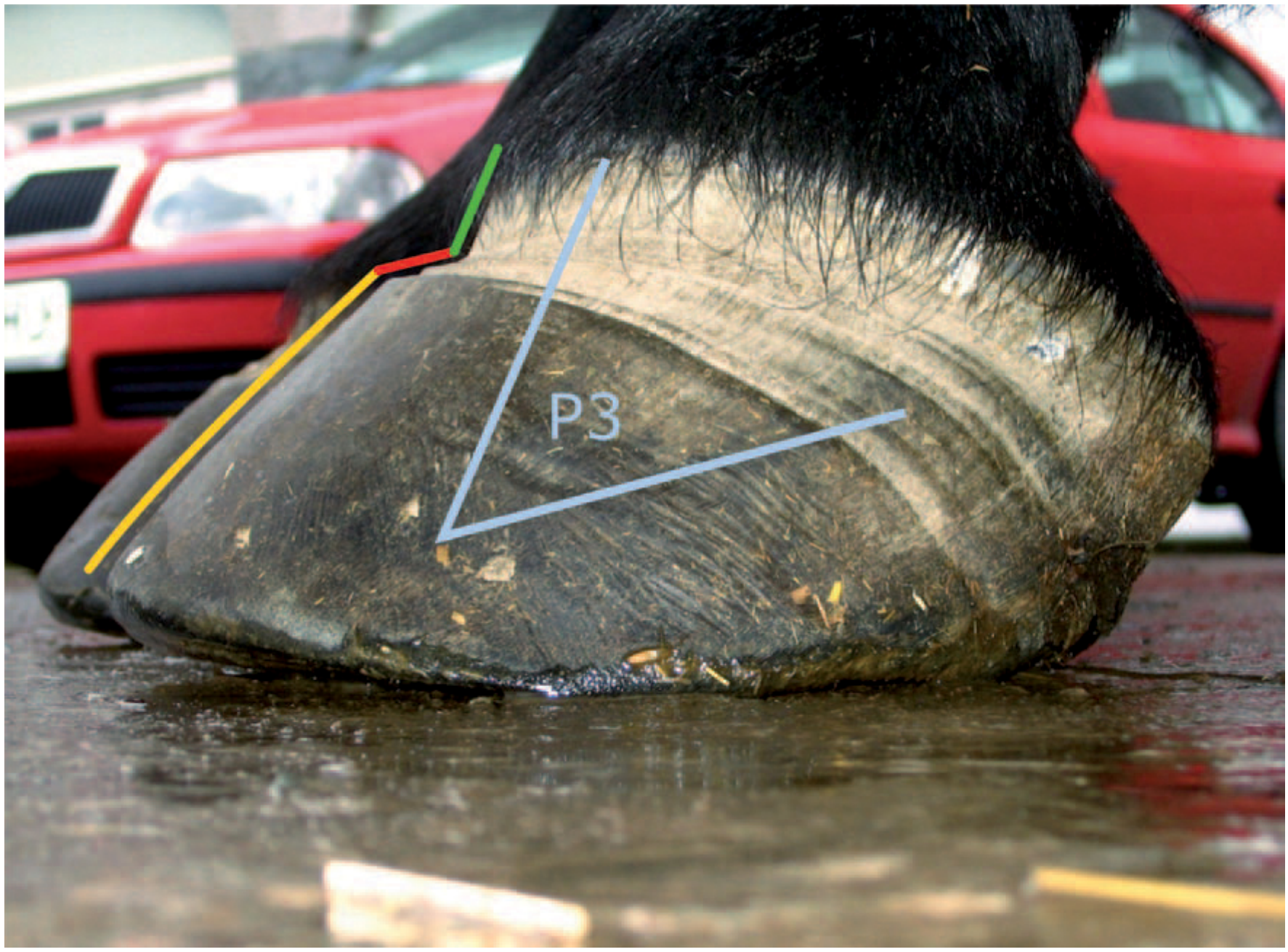


Fig. 96 Changes in hoof wall angle as a result of a severe sinker episode, with the approximate position of the pedal bone shown. The pre-sinker growth (yellow line) is rotated by over 20 degrees from the pedal bone. Wall growth during the sinker episode (red line) is almost horizontal. After recovery of the coronary band (green line), growth is near parallel to the pedal bone. A heel rocker has been used in this case to remove pressure from both the front portion of the sole (to reduce the risk of pedal bone penetration) and the front portion of the wall (to reduce pressure on the coronary band); the recovery visible at the top of the hoof wall is the direct result of that approach.

Of course shifting the weight from the wall to the sole relies on there being a healthy sole in the front half of the foot with which to support the horse's weight. In many severe laminitis cases this is not the case, and part of the art of helping the worst cases to recover is to find ways to reduce the amount of weight being carried by the wall and hence the coronary band, without transferring that weight on to the sole directly under the tip of the pedal bone, where it is typically most badly damaged. This can often be done by utilizing the areas of sole on either side of the toe area, which tend to be less badly damaged and hence capable of carrying at least some weight. Carefully designed padding arrangements are usually needed to ensure that only the appropriate parts of the foot are weight-bearing.

The Use of Pads

It is often difficult to manage the mechanical distribution of weight within the foot using trimming alone. For this reason, pads can be an extremely important tool in the management of severe laminitis.

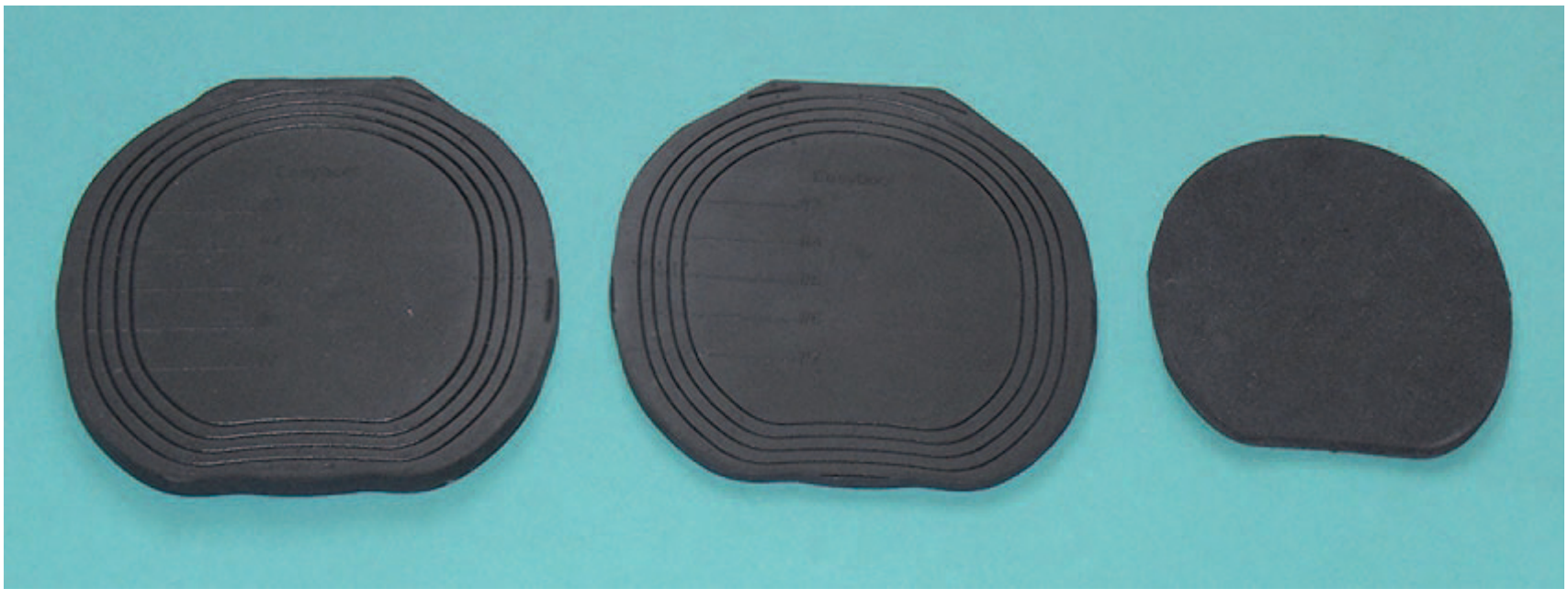


Fig. 97 A selection of elastic pads: two Easyboot pads of different thicknesses with pre-printed lines to assist with cutting to size, and a pad from an Easyboot RX poultice boot.

Pads divide into two broad types. The first type is springy and is typically made of rubber or similar materials. Such pads behave elastically according to Hook's law – the more you crush the pad down, the harder you have to press. This means that with a weighted foot placed on the pad, the areas of the foot that are most proud (typically the frog, the wall and the rim of the sole) crush the pad down the most and so are subject to the greatest pressure. Areas of concavity, such as the centre of the sole, are subject to the least pressure. This might seem like a useful property, but unfortunately, the pattern of reduced pressure doesn't map that well to the most typically damaged areas of sole, especially where there is significant false or dropped sole. The vast majority of commercially available pads are of this type. They are available in a variety of thicknesses and are often sold by boot manufacturers pre-marked for cutting to fit various boots.

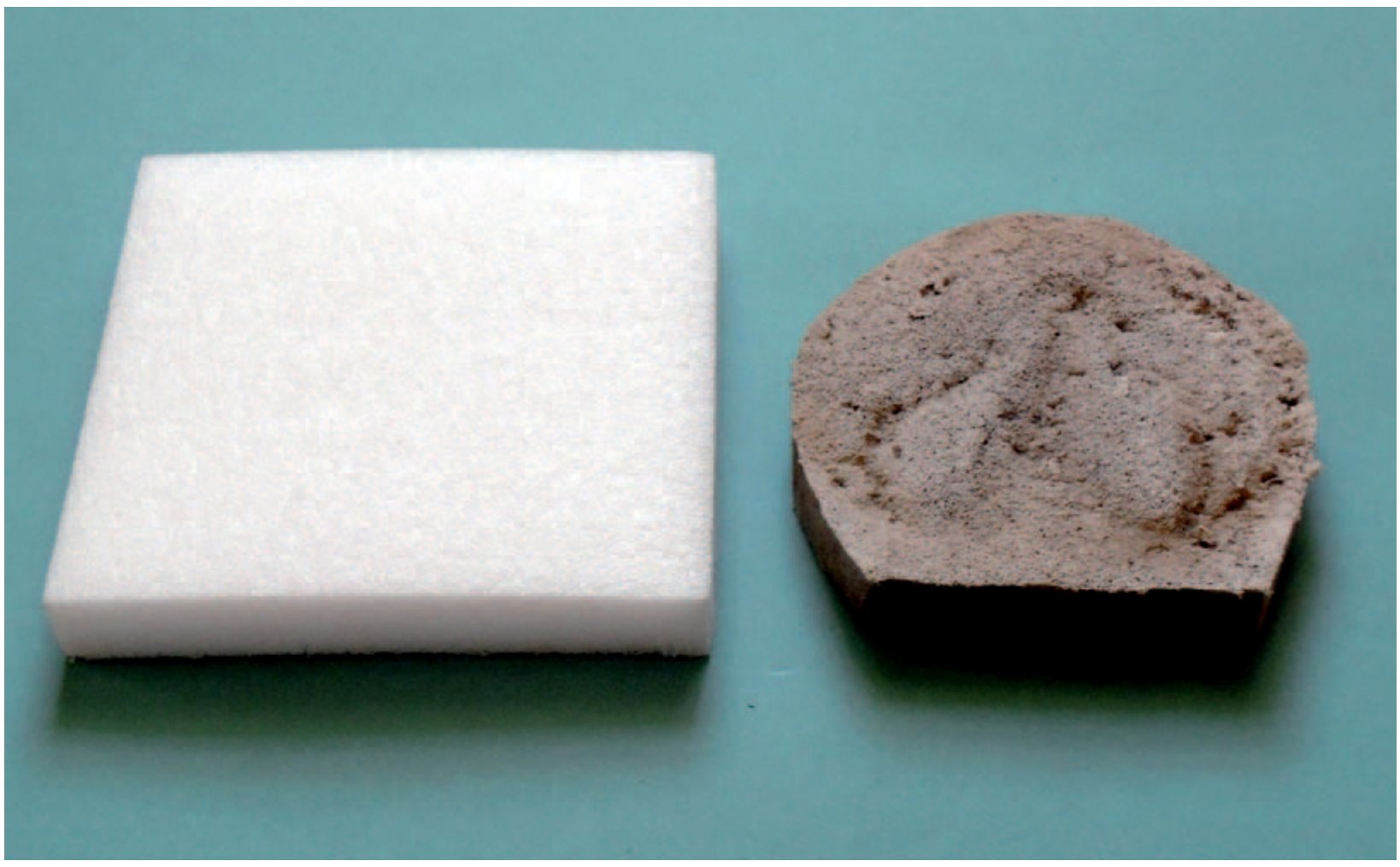


Fig. 98 An unused memory-foam pad, and one that has been used inside a removable hoof boot. The used pad is stained with dirt but shows the clear imprint of the hoof it was used to support, despite having had time to recover its shape somewhat after removal from the boot.

The second type of pad behaves more like memory foam. This is typically a closed cell foam pad that is initially quite thick (perhaps 25mm), but which crushes down in a semi-permanent fashion when loaded. The pad will take a little while to crush (perhaps an hour or more) and will behave elastically

during this time, but once fully crushed, the pad effectively makes a mould of the bottom of the foot and loses much of its elasticity. From this point on, the pad will provide even pressure across the entire bottom surface of the foot (with the exception of any deep channels such as the collateral grooves on each side of the frog). As the shape of the foot changes over time, the pad will to some extent change shape to accommodate this. Where a pad has become ‘tired’, it can be refreshed by placing it in a warm place (such as near a radiator) for a few hours. Because the foam cells are closed and contain gas, warming the pad causes the gas to expand, reinflating the pad to some extent. Different grades of pad should be used depending on the size of the horse, with ponies and smaller horses typically doing well in ‘4lb’ pads and heavier horses typically needing ‘7lb’ pads. This type of pad is the preferred choice of equine podiatrists working in the UK, but is difficult to obtain, with only a handful of specialist suppliers stocking them. They need to be cut to size, and this is most easily done with a serrated kitchen knife.

It is also possible to obtain liquid urethane packing material that is dispensed on to the bottom of the foot, and which sets to form a soft pad. The disadvantage of this approach is that the pad cannot then easily be removed daily to disinfect and inspect. It is also much harder to shape such a pad so as to remove pressure from selected areas.

Pads have three roles. The first is to provide even support to the bottom of the foot. A pad will mould to the shape of the foot and so distribute weight evenly across the bottom surface of the foot. This removes any pressure points associated with parts of the foot that are proud. For example, a horse with a dropped sole may be very uncomfortable standing on concrete without shoes, but the fitting of a foam pad to the bottom of the foot provides an instant and dramatic increase in comfort. For this role, a memory-foam pad often performs significantly better than an elastic pad.

The second role is in shock absorption. Inflamed coria are particularly susceptible to vibration, with any significant amount resulting in pain and potentially further damage. Where a horse needs to move on hard ground – for example, during rehabilitation once the initial healing from laminitis is complete – placing an appropriate pad under the foot can absorb a large proportion of the vibration of impact, so significantly reducing discomfort and minimizing the risk of further damage. Elastic pads typically provide a small degree of shock absorption and may be appropriate for mild LGL cases that are in work. Memory-foam pads typically provide a far greater degree of shock absorption and are the more appropriate choice for severe cases.

The final role is to allow more direct management of weight distribution. Pads can be easily cut and shaped so as to reduce or increase pressure on specific structures. Again, this is easier and tends to be more effective with memory-foam style pads. Where a structure is very badly damaged and needs to be removed entirely from weight-bearing, the relevant part of the pad can be completely cut away (similar to a corn plaster used on a human foot). Where more pressure needs to be applied to a specific area (most commonly this is the frog), a piece of elastic material such as an offcut from a Prolite® pad can be glued on top of a memory-foam pad so as to provide additional pressure in this area.

In severe laminitis cases where sole damage is an issue, the most common requirement is to remove pressure from the area under the tip of the pedal bone. The challenge in such cases is to find sufficient area of reasonably healthy structures forward of the midpoint of the foot to support the horse’s weight and prevent the foot from toppling forwards off the pad. The area of sole immediately under the tip of the pedal bone needs to be relieved of pressure, as does the front portion of the hoof wall. However, it is usually possible to load the sole and wall either side of the centre so as to get some support here. The result is a pad which is cut out from the ten o’clock to the two o’clock positions.

The most appropriate padding arrangement for a given laminitis case is often fairly obvious. For example, where the pedal bone has penetrated the sole or is close to doing so, it is reasonable to assume that this area of the bottom of the foot should not be used to support weight! But the relative ability of different parts of the foot to bear weight is not always easy to guess. For all but the most obvious cases, it is often best to use a degree of trial and error when it comes to padding. Indeed, such a process of trial and error can be a helpful aid in determining which structures are the least damaged and which the most. For example, a horse with at least some concavity to the sole, and which looks

fairly comfortable on smooth tarmac but goes significantly lamer when a plain pad is attached to the bottom of the foot, probably has laminitis where damage to the solar corium predominates.

Fig. 99 A memory-foam pad cut to prevent pressure to the solar corium under the tip of the pedal bone. This pad has been used for several weeks inside a removable hoof boot, and a clear impression of the bottom of the hoof is visible.

In contrast, a horse that becomes more comfortable when a plain pad is applied is likely to have lower levels of damage to the solar corium than to other structures in the foot, such as the coronary band or laminae. Because pain is an indication of likely damage, finding the padding arrangement that results in the least pain is a great way of minimizing damage to the feet. This trial and error process is easier to do before the horse is given any significant levels of pain relief, but ethical considerations must always take precedence over convenience.

The Use of Boots

Removable hoof boots are a very useful tool in the recovery of full-blown laminitis cases. They provide a degree of mechanical support and protection to the foot at least equivalent to that provided by traditional horseshoes, but they have a number of additional benefits:

- Typically they have soles made of rubber or similar materials. Therefore they actively reduce the vibration to which the foot is subjected. In comparison, shoes significantly increase the level of vibration.
- They can be removed daily, and the structures of the hoof checked. Any horn infections such as thrush or white line disease can be easily treated.
- They can be used on an ‘as needed’ basis. Whilst some severe laminitis cases may need to be booted twenty-four hours a day, milder cases during recovery may only need to be booted for turnout, or when being moved from stable to field over hard ground.

- Some styles of boot are very good at accommodating the changing shape of a laminitic foot between trims.
- A boot is an ideal way to keep a pad in place against the bottom of the foot. Attaching a pad to the bottom of a foot with duct tape or similar is possible, but keeping the pad in place by this means can be challenging. A pad can be placed between a traditional shoe and the foot, but the tendency is for the pad to bend and fall into the hole in the centre of the shoe, so reducing the mechanical support it should provide. In contrast, a hoof boot provides a firm, flat surface for the pad to sit on, and is a good means of holding the pad in place. The boot and pad can also be removed and checked daily, and disinfectants applied to both the foot and the pad if needed.
- If properly used, boots can last a very long time and hence can be a cost-effective approach when compared to plastic glue-on shoes, or even to traditional heart-bar shoes.

Of course, despite the benefits of hoof boots, there are some disadvantages, too:

- Hoof boots need to be removed and cleaned regularly (ideally daily where they are in use twenty-four hours per day), otherwise they tend to fill with mud if the horse is turned out, or bedding if the horse is stabled.
- Hoof boots are difficult to fit. Picking the right style of boot for a specific situation requires experience. Measuring charts are typically available for each make of boot, which supposedly allow the correct size to be chosen based on some simple measurements of the hoof. Unfortunately, these measurement charts are based on healthy feet and don't tend to work as well with laminitic feet. Even with a healthy foot, the variation in foot conformation can mean that a measuring chart may give a size too large or too small. For this reason, it is best if boot sizes are determined by an appropriate professional who is trained in boot fitting. This is especially true in laminitis cases.
- Hoof boots represent an expensive initial outlay. This is less of a problem where the boots are likely to be needed for some time, but some laminitis cases may only need them for a few weeks. Second-hand boots do, however, sell well on online auction sites.
- Although boots can be very flexible, full-blown laminitis cases often undergo dramatic changes in hoof shape and size during recovery, which may mean that more than one size of boot is needed during the recovery process.

Hoof boots fall into two broad categories. The first category is 'clamp'-style boots. These boots are designed to be a tight fit against the hoof and typically have a low profile, with the top of the boot sitting just below the hairline. Traditionally, these boots have some form of mechanism such as a clamp to tighten the boot against the hoof. More recent designs have no mechanism but are designed to be a tight push-on fit (often with a safety cuff that goes around the mid-pastern area so as to prevent the boot falling off the heels).

Clamp-style boots need to be a very good fit to the hoof. If they are too tight, they tend to crush the hoof and do damage. If they are too loose, they tend to fall off very easily. These boots are typically not a good choice for a laminitic horse. They rely on a good fit, and as such, need a hoof that is a fairly 'normal' shape. They are also very intolerant of any changes in shape or size of the hoof, and there is an argument that clamping a boot on to an already inflamed foot isn't necessarily a good idea! Clamp-style boots may, however, be appropriate for mild, long-term LGL cases where a little extra protection is needed for road work, and the hoof remains a fairly constant shape and size between trims.

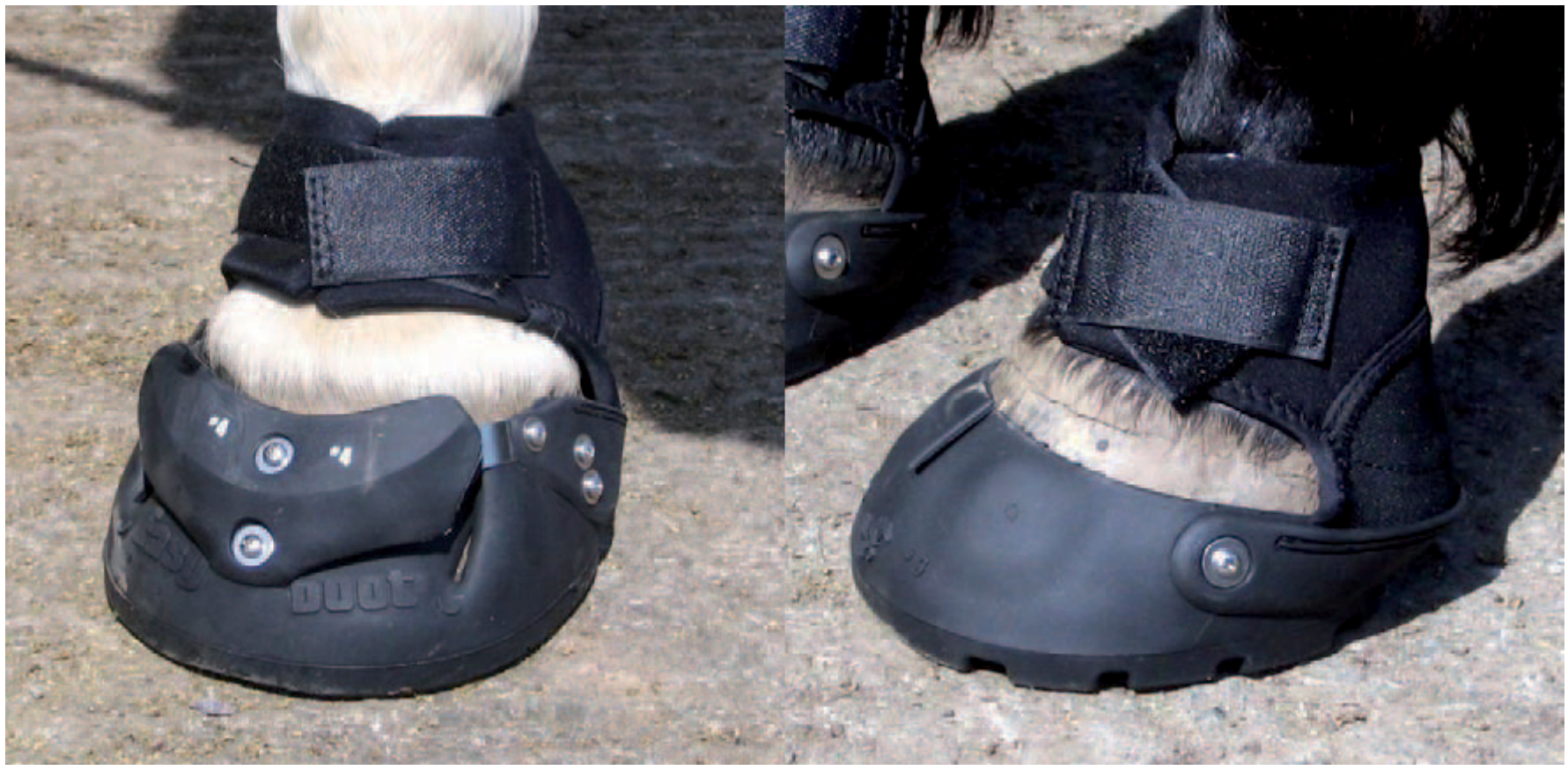


Fig. 100 Clamp-style boots: LEFT: *an Easyboot Bare;* RIGHT: *an Easyboot Glove.*

Fig. 101 Trainer-style boots: LEFT: *a Cavallo simple boot;* RIGHT: *an Easyboot Trail.*

The other category of boot works more along the lines of a human training shoe. These boots are a looser fit on the foot but extend above the coronary band, and tend to have a cuff round the bottom of the pastern. These boots are typically made of softer, more flexible materials, and are fastened by means of Velcro® straps, and in some cases, buckles. When correctly fitted, a 'trainer'-style boot should 'slop' slightly on the foot. It should be possible to twist the boot easily around the axis of the foot by about 5 degrees either way (making sure that the boot is actually slipping around the foot, rather than twisting the coffin joint). If the boot won't twist at all, it is likely to rub the heel bulbs; if it twists too much, there is a risk it will come off or spin round.

Trainer-style boots are generally better suited for use in full-blown laminitis cases. They are much more forgiving of any change in shape or size of the hoof during recovery, and they apply little pressure to the upper parts of the foot. Some designs also cope well with the insertion of a fairly thick pad in the bottom of the boot (something clamp-style boots will generally not cope with).

There is now a huge and ever-growing range of removable hoof boots on the market, so a detailed description of all the various boots available is well beyond the scope of this book. Ideally professional advice should be sought when considering which boot to use for a laminitic horse.

Twenty-Four-Hour Use of Boots

Few boots on the market are specifically designed to be used twenty-four hours per day. Those that are, typically take the form of poultice boots that are designed for very short-term use (a few days at most), and are typically less suitable for the full-blown laminitis case. Despite this, it is possible to use some brands of boot twenty-four hours per day. However, this needs to be done with care to ensure that harm isn't done.

The biggest risk in 24/7 booting is that the boot rubs the heel bulbs. If the heel bulbs blister as a result, the boots will need to be removed for long enough for the heels to recover. In a serious laminitis case, the loss of the boots as a tool in supporting the foot may prove an extremely serious setback. For that reason alone, it is vitally important to manage the situation to avoid rubbing. There are a number of ways whereby rubbing can be minimized:

- If possible use boots that are already 'broken in' (this may not be possible if boots have been purchased specifically for a new laminitis case).
- Ensure that the boot is the correct size for the foot, taking into account any pads or other therapeutic or anti-rubbing devices fitted within the boot.
- Use 'socks': just as socks help prevent human shoes from rubbing, a similar approach can be used with a horse. Some boots come with gaiters made of neoprene or similar materials. These go between the boot and the hoof, and aim to reduce friction between the two. Unfortunately, neoprene doesn't breathe well, and use of these gaiters for extended periods can cause moisture to build up between the gaiter and the hoof, which softens the heel bulbs and increases the risk of rubbing. A better material is wool or cotton (never nylon). Human hiking or sports socks can be stretched to fit over all but the very largest hooves, and these can work exceptionally well. One of the author's clients even used the sleeves cut off a worn-out cashmere jumper, as this was the only thing at hand in an emergency. Of course, when choosing the correct boot size, the thickness of any socks being used needs to be considered.
- Remove the boots daily and clean both the boots and the feet. This can be as simple as running the boots under a tap, or it may require mild disinfectants to manage any thrush present. It is very important that no gravel or grit is left on the hoof or inside the boot. Where socks are being used, these should be changed regularly and washed (so more than one pair will be required). Don't expect socks to last long – most horses will wear them into holes within two or three weeks.
- While cleaning the hooves and boots, check the heel bulbs very carefully for any sign of heat, puffiness or redness. If there are signs of rubbing, then check the boot fit very carefully indeed.

Whilst twenty-four-hour booting is not easy, many owners of laminitis cases manage this very well, and it can be the critical factor that helps a horse to make a good recovery.

Fig. 102 A horse in boots twenty-four hours per day. Old Mac boots are being used because of their flexibility, and the ease with which pads can be inserted into them. Wool hiking socks have been added to prevent rubbing.

MANAGEMENT OF THE FOOT IN LGL

Management of the foot in LGL cases tends to be more a case of managing a range of individual problems that can occur as a result of LGL. The key to improvement in the hoof capsule is always to get the underlying causes of the LGL under control. Because the level of pathologies present in LGL cases is typically far less severe than in full-blown laminitis, there are many cases where this alone is enough to resolve any hoof pathologies, assuming that the hoof is regularly maintained.

Removal of Shoes

As with full-blown laminitis, removing the shoes from an LGL case can be extremely helpful. Although the level of inflammation of the underlying tissues is less than that in full-blown laminitis, the vibration from a shoe can still contribute to both pain and increased damage. However, as with full-blown laminitis, it is important to have a plan in place for how to replace the protection provided by a shoe.

Almost all LGL cases are comfortable and cope well without shoes when in the field. Milder cases may be fully sound on hard ground as well, especially those cases where the solar corium isn't significantly involved in the inflammation. More severe cases, however, should be booted for any work on hard ground. Without boots, these cases tend to be footsore and have a significantly increased risk of sole bruising with the concomitant risks of abscessing, poor sole growth and so on.

In some cases, the use of pads fitted inside the boots will increase comfort levels (and hence decrease the potential for damage). As with full-blown cases, experimenting with different booting and padding arrangements until the optimum comfort is achieved is the right way to approach the problem. It is not usually necessary to cut relief holes in pads in LGL cases, as damage to the solar corium isn't usually severe enough that individual areas are unable to take any weight.

Trimming the Hooves

Because LGL cases show no rotation or sinker, there is normally no need to take x-rays (other than to confirm that there has been no rotation or sinker if that is in doubt). There is also no need to alter the basic trimming approach from that used on a healthy foot. It is very important not to remove any viable (that is, non-exfoliating) sole material from an LGL case, but that should not be done on a healthy foot either, except in very specialist circumstances.

There are, however, specific pathologies that can result from LGL, which may need specialist trimming or management, such as quarter cracks or deep central sulcus infections. Even with these secondary pathologies present, whilst trimming may play a part in recovery, getting the underlying LGL under control is far more important. It is possible to get away with a sub-optimal trim if the underlying LGL is fully controlled, whereas the best trim in the world will not promote significant recovery if the underlying LGL is not brought under control.

General Infection Control

LGL cases tend to be far more prone to thrush and white line disease than healthy horses. For that reason, infection control tends to become a significant issue – especially if the underlying causes of the LGL cannot be fully controlled.

Because horn infections live on damaged horn, any approach to disinfection that does further damage to healthy horn is likely to be counter-productive, particularly if used regularly. Most traditional approaches to the treatment of thrush and, sadly, many off-the-shelf products aimed at thrush do not meet this requirement. For example, substances such as Stockholm tar, iodine (even when diluted), hydrogen peroxide (again, even when diluted) and hoof oil all damage healthy horn. One-off use may avoid sufficient damage to cause a problem, but routine use of such substances tends to lead to a vicious cycle, where any attempt to cease their use results in a dramatic recurrence of the original problem, as newly damaged horn starts to rot.

Routine disinfection should only be performed with products that are both strong enough to kill bacteria and fungi, and yet mild enough not to damage healthy horn. This is actually a difficult balance to achieve. Given that hoof horn is (with minor differences) essentially the same tissue as human skin, a good rule of thumb is never to treat an equine foot routinely with something you wouldn't be happy to wash your hands in daily! One good standby for thrush and white line disease is Milton® Sterilising Fluid, which should be diluted at a rate of one capful (30ml) to 5ltr of cold water. The solution needs to be made up fresh each time, and can either be brushed on a clean hoof with a stiff brush, or the foot can be soaked for ten to fifteen minutes in a bucket or proprietary soaking boot. There are some good commercial products available that can be used, but a full description of these is beyond the scope of this book.

Central Sulcus Infections

To tackle a deep central sulcus infection, it is necessary to stretch the frog corium from side to side so as to pull out the V-shaped crease in the central sulcus. The moment this happens, the corium will start to produce more healthy frog material, which will push the crack outwards towards the surface of the frog. One way to get rid of the crease is to put a lot of pressure on the frog, but this is usually difficult because the frog is often badly damaged, and the horse is often so sore that additional pressure just results in the horse shifting weight more to the toe. Much more effective is to pack the central sulcus with a medicated packing material, such as Red Horse Hoof Stuff. The aims of this approach are as follows: to force the sides of the sulcus apart; to suppress any infection present; and to prevent dirt and stones from getting trapped in the sulcus and doing more damage. It only takes a thin layer of material in the bottom of the crack (perhaps 1–2mm wide) to push the corium apart sufficiently in that locale to

get rid of the crease. The use of this approach typically results in the central sulcus infection growing out in a matter of a few weeks.

Navicular Disease

Navicular disease is a complex subject, and a detailed discussion of it is beyond the scope of this book. Resolving navicular syndrome/ disease (assuming it can be caught before the damage becomes permanent) is largely a matter of getting the horse to land heel first so as to use its natural shock absorbers. This can sometimes be achieved just by fixing any central sulcus problem, but more often requires the provision of artificial shock absorbers in the form of closed-cell foam pads (usually placed in a removable hoof boot). Any underlying LGL (which is almost always present in navicular disease cases) clearly also needs to be addressed. The results in early navicular syndrome cases can be spectacular, and even in more advanced cases of navicular disease, a reasonable degree of improvement is usually possible.

Wall Cracks

Dealing with cracks is a matter of addressing all the underlying factors that contribute to them. Clearly any LGL needs to be addressed, but in addition the shape of the hoof needs to be carefully and sympathetically controlled, any infection needs to be treated with mild topical disinfectants (stronger ones damage the healthy horn as well, which just makes more bug food!), and any significant defects behind the wall from the likes of white line disease or seedy toe need to be packed with a medicated flexible putty to prevent stones getting in and making things worse. Nevertheless, with the right holistic approach even the worst of cracks will close.

Under-Run Heels

Because leverage is an important part of the process by which under-run heels occur, it is vitally important not to let the heels grow long. However, over-shortening the heels is not the solution either, as this can cause other problems. Rehabilitation of poor lateral cartilages and a poor digital cushion is needed to provide sufficient strength in the heel area to support a more upright heel, and hence a heel-first landing during work on hard ground is essential to recovery. Boots (sometimes with pads inside) can be very helpful in achieving this.

Flat Feet

As seen in Chapter 4, flat feet are typically the combined result of three separate pathological processes in the foot. All of these will reverse in time given good trimming so long as the underlying causes of the LGL are addressed and there isn't significant bone loss. As such, there are no specific techniques required to reverse flat feet other than to recognize that the soles may initially be thin and prone to bruising and hence in need of appropriate protection during the recovery process.

Sole Creep

Once any underlying LGL is dealt with, any wall flaring tends to gradually grow out, a process that can take many months. However, a new sole can grow out in as little as three months, and the tubules at the rim of this new sole are often aligned in a healthier, more upright orientation. The result is a stage during recovery in which the sole appears to creep back from the wall, leaving a shallow groove between the wall and the rim of the sole. This groove often appears just before a full new sole has

grown through because the last fragments of the original solar rim tubules will exfoliate as a result of being less well supported.

Sole creep is often mistakenly seen as a pathology, worrying owner and trimmer alike. But as long as the white line at the bottom of the groove is reasonably tight, sole creep is an indication of healing and should be welcomed. As the new, less flared wall grows down, the groove gradually decreases in size, and eventually vanishes. If the original sole and hoof wall diameters are measured prior to tackling the underlying causes of the LGL, subsequent measurements clearly show that the sole reduces a few millimetres in size after around three months, with the wall gradually following suit in the months after that. Not all cases show sole creep during recovery, but where it is present, it should not be a cause for concern.

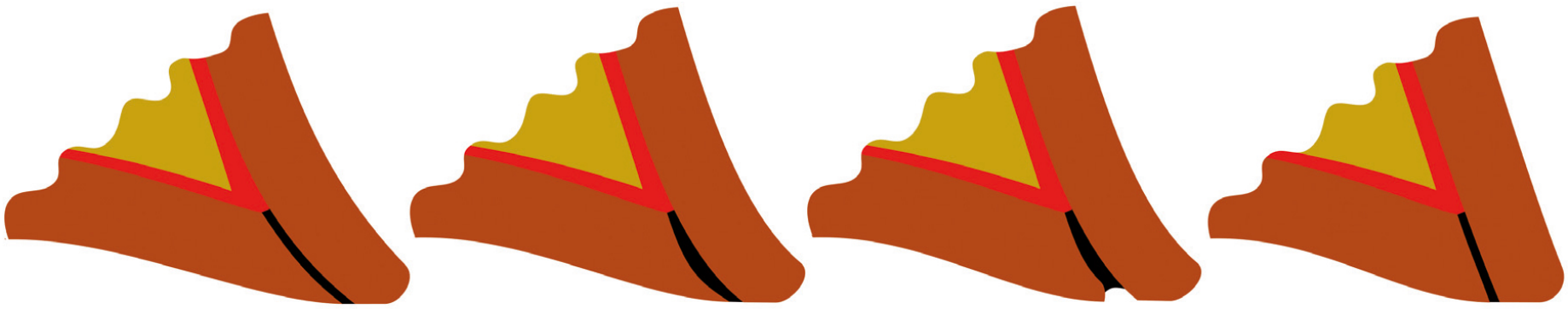


Fig. 103 A stylized representation of how sole creep occurs, with the white line shown in black for clarity. a) Flaring results in minor distortion to the laminar corium and a significant deviation in the white line. b) With the underlying cause of the flaring corrected, new wall growth is tighter against the pedal bone. New sole growth is also more upright, creating, at around two to three months, a lip at the rim of the sole. c) The poorly supported lip at the rim of the sole now exfoliates, along with a small part of the white line, leaving a shallow hollow gap between the rim of the sole and the hoof wall. d) Some months later, both wall and sole growth are correctly oriented and the white line is now tight again – there is no longer a gap between the sole and the wall.

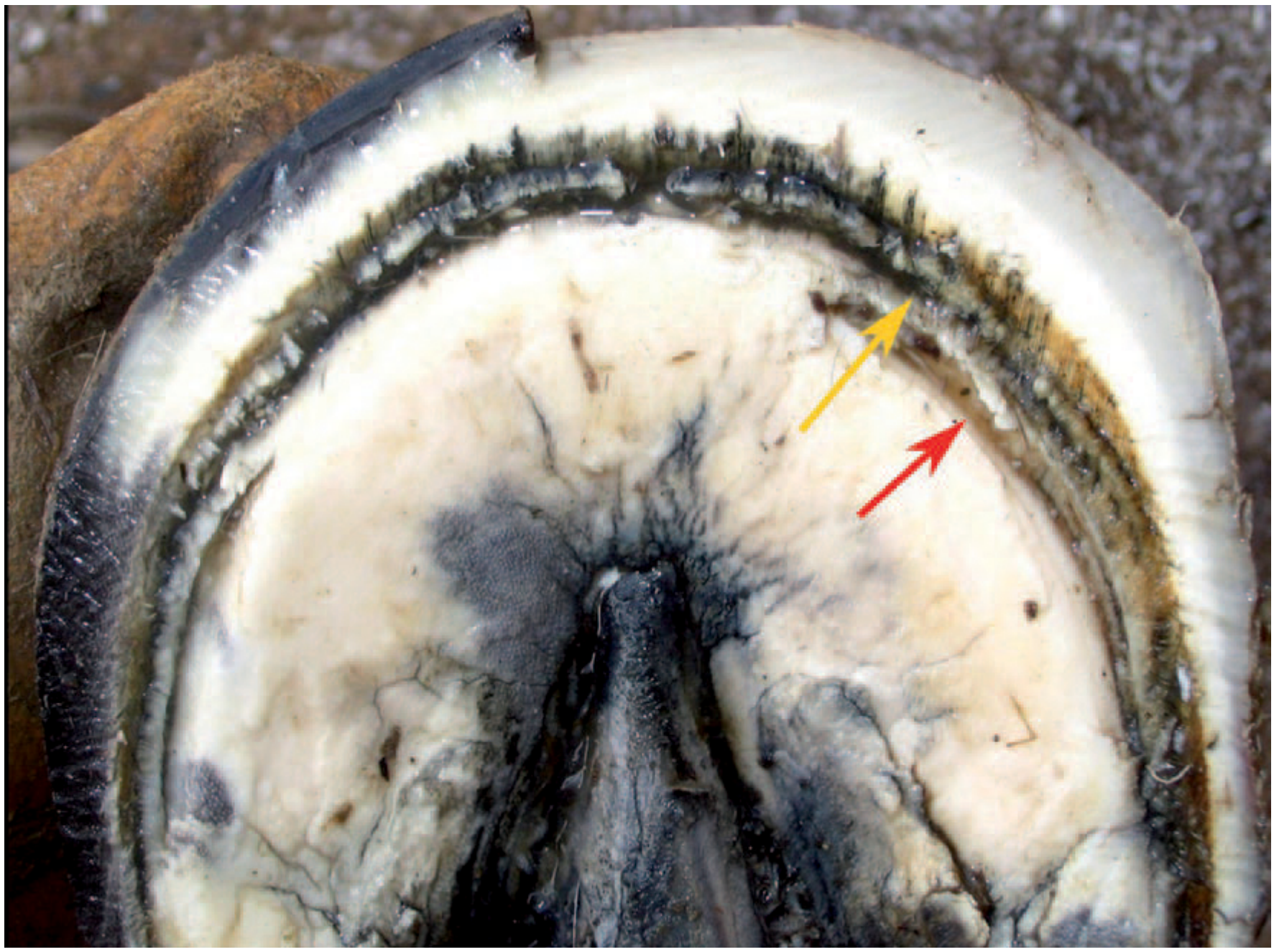


Fig. 104 An example of sole creep during recovery from flare. The yellow arrow indicates the outline of the original sole associated with the flare, and the red arrow indicates the more upright rim of the recovering sole. The last fragments of the old flared solar rim can be seen exfoliating in the quarters, but in the toe area it is taking a little longer to exfoliate. The bottom of the wall will move inwards in the following months, such that the groove between sole and wall closes again.

ABSCESSES AND HOW TO DEAL WITH THEM

As previously discussed, both low-grade and full-blown laminitis cases are particularly prone to foot abscesses. In the author's experience, common approaches to managing abscesses can, in some cases, cause more problems than they solve.

It is traditional with a hoof abscess to create a hole in the hoof capsule (usually in the white line) so as to drain the pus. This relieves the pressure within the foot and so provides instant pain relief. However, it can be very difficult to locate the abscess track accurately so as to make a drain hole in the correct place. Missing the abscess can have significant consequences, not least that an unnecessary hole has been made in the hoof capsule. If the hole is dug over healthy corium, bleeding results. This then creates a significant risk of infection getting into the bloodstream and creating a new abscess, adjacent to the existing one. This is particularly true given that the tool used to make a drain hole is often not disinfected first. More of a problem is a hole that hits both the abscess track and adjacent healthy tissue. An abscess is the horse's way of encapsulating infection and preventing it from spreading, and the pus from the abscess has now been introduced to healthy tissue, defeating this survival mechanism. This can result in a more severe abscess, and increases the risk of infection tracking up the leg and causing septicaemia.

Oral or injected antibiotics are also fairly pointless for foot abscesses. By the time a horse shows significant pain and the vet is called, the infection has been largely or completely encapsulated away from the bloodstream. In many cases, antibiotics fail to reach the abscess altogether and so have no effect. However, in some cases (particularly where antibiotics are administered early on), some

antibiotic effect will be seen and the horse will typically look a lot better (which may explain why some vets persevere with this approach). But the infection is unlikely to be fully suppressed, and in the author's experience, will often come back again with a vengeance a week or so after the course of antibiotics is finished. Of course, if there is *any* suggestion that the infection has broken free of the encapsulation and is tracking up the leg (a very rare occurrence), then the vet should be called immediately so that antibiotics can be administered without delay. Similarly, any abscess which fails to blow naturally after a few days should also be referred back to the vet for drainage and possibly antibiotics, as the risk of complications goes up significantly with slow-to-blow abscesses.

Box rest is also usually the wrong approach to take with an abscess. Not only does it prevent lymphatic drainage and hence increases the risk that oedema does more damage to the foot and leg, it also increases the length of time it takes nature to deal with the abscess naturally. For an abscess to blow naturally (and hence heal as nature intended), the bacteria need to build up enough pressure to force the abscess to track out of the foot. Heat from movement is the best way to encourage this process. Poulticing (particularly with anything that helps to insulate the foot and hence keep it warm) can also help. With box rest, there is no movement and hence the whole process takes longer. Although the length of time taken for an abscess to blow naturally can be highly variable and depends on the degree of other pathologies present, horses that are turned out generally blow abscesses far faster than those on box rest. It is common for a minor abscess to blow within twenty-four to forty-eight hours with twenty-four-hour turnout, whereas a stabled horse may take one or two weeks to blow the same severity of abscess. Painkillers such as phenylbutazone are generally a good idea, as they encourage the horse to weight bear and move around, speeding the natural resolution of the abscess. Care must be taken, however, with movement and pain relief in more severe laminitis cases, where movement may cause more mechanical damage to the foot.

As a general rule, in the author's experience, horses return to their pre-abscess level of soundness significantly faster if the abscess can be encouraged to blow naturally than if attempts are made to intervene. The ideal is for the abscess to blow quickly at the hairline so as to minimize the risk of reinfection and to maximize the ability of the hoof to repair itself. Turnout, a sensible degree of pain relief and poulticing, make complete sense in terms of achieving this goal. Clearly though, this has to be balanced against the ethics of leaving a horse in some degree of pain, and the risks associated with movement if there is any degree of full-blown laminitis.

It is also important to understand that the degree of lameness builds up to the point at which the abscess blows. There is no point putting the horse through the discomfort of waiting for the abscess to blow only to lose one's nerve when the lameness worsens (which is almost always an indication that the abscess is about to blow) and dig a hole.

It should also be noted that the ideas presented here on how to deal with abscesses are considered controversial, and some vets may not be happy with following this approach.

9 Established Approaches to Underlying Causes

Given that the mechanisms by which laminitis occurs are poorly understood, it should not be surprising that the established options for treating the underlying disease are somewhat limited. Where laminitis is triggered by another well-known condition (such as colic or metritis), the focus should be on appropriate veterinary interventions aimed at that underlying condition (for example, antibiotics where sepsis is implicated). However, the vast majority of cases in the UK are pasture-induced endocrinopathic cases. For such cases, the established approach generally falls under three headings: avoiding trigger factors, weight loss and drugs.

AVOIDING TRIGGER FACTORS

The biggest focus in dealing with the underlying disease processes in endocrinopathic laminitis is currently the attempt to avoid trigger factors. Although the exact triggers are still hotly debated, it seems that sugars and starches are the most likely culprits. As such, the aim is to keep the diet low in sugar and starch whilst providing the overwhelming majority of the required calories in the form of fibre.

Reducing sugar and starch intakes is extremely difficult when a horse is at pasture twenty-four hours per day. As discussed in Chapter 6, most modern UK pastures have been heavily fertilized and hence are dominated by rye grass and are highly productive. Traditional pastures, however, have lower soil fertility, and as a result, have higher species diversity and lower productivity. Where there is a choice of available pastures, choosing a more natural, lower productivity pasture can significantly reduce sugar and starch intakes. Whilst detailed identification of pasture types is a job for an experienced botanist, such pastures typically have a much higher proportion of broad-leaf species present, whereas those dominated by rye grass typically have few broad-leaf species present other than docks, thistles, nettles and white clover. Low productivity pastures also have little or no rye grass present, but this is only easily identifiable where some of the pasture has been allowed to run to flower/seed.

Fig. 105 LEFT: ***A typical dairy pasture dominated by perennial rye grass with some docks and thistles.*** RIGHT: ***A species-rich, lower productivity meadow illustrating the large variety of broad-leaved species present. (Photo: Stuart Smith)***

Where a low productivity pasture is not available (and even sometimes where it is), limiting time and/or area can be helpful.

Time-Restricted Grazing

Stabling the horse for part of the day can help to reduce trigger events. Grass generally has lower sugar levels overnight (because there is no sunlight to drive photosynthesis and so the grass uses up some of the sugars stored in the leaves). For that reason, turning the horse out overnight typically poses less risk than daytime turnout. It is tempting to reduce grazing hours further and further in an attempt to reduce overall sugar intake, but this is not always a productive approach. It is not the overall intake over a twenty-four-hour period that is most important, but rather the peak rate of sugar intake. Research has shown that horses quickly become wise to reduced turnout duration and just eat faster to compensate. This results in more grass being eaten per hour, a higher rate of absorption of sugars into the bloodstream, and hence higher peak blood-sugar levels – precisely the thing that should be avoided in a laminitic horse!

Area-Restricted Grazing

Because limiting time at grass typically just increases the peak rate of consumption, limiting the area of grass available to the horse often proves more helpful. It is traditional to put a laminitic horse on a ‘starvation paddock’. The terminology here is far from helpful as it encourages a mind-set of needing to starve the horse. Starvation increases stress (and hence boosts cortisol levels, adding to the severity of any insulin resistance) and also significantly increases the risk of gastric ulcers (which anecdotal evidence suggests also carries a risk of causing/exacerbating insulin resistance). In reality, a starvation paddock is simply a paddock that provides adequate calorie intake for maintenance of weight without providing ad-lib grass. Because most UK pastures provide high levels of energy, the actual volume of grass needed to maintain weight is not that large and hence starvation paddocks typically look fairly bare. It is not uncommon for well-meaning people to complain that there is no grass for the horse. However, if the horse is removed from the starvation paddock for a week, the amount of grass produced in that week can be seen and can be fairly large, especially in the summer months. Of course, you can’t then put the horse back in the field because it will binge on a week’s growth in one sitting! This is actually a problem when creating a starvation paddock in the first place, and it is usually necessary to graze the grass well down first either with horses that are not prone to laminitis or with other stock such as sheep or cows. Alternatively strip grazing can be used to gradually introduce the horse into the new field from an adjacent field or yard.

It can be very difficult to judge the required area exactly so that the new grass growth provides exactly the right amount of grass for the horse each day. This difficulty is exacerbated by changeable weather. An area that is insufficient to support a horse on a cold day when the grass is not growing might produce enough rich grass to cause a laminitis attack on a warm, sunny day in a particularly sensitive horse. One part of the solution to this problem is strip grazing. In its simplest form, a temporary fence is moved every day so as to slightly increase the area of pasture available to the horse. The horse then has a large area of already grazed pasture (the back grazing) which grows a little each day, and a small area of ungrazed tall grass which adds sufficient extra calorie intake to meet the daily requirements. Over the summer, the moving fence results in the back grazing becoming larger and hence providing more of the daily calorie requirements. As a result, the fence should be moved smaller and smaller distances to balance this. Eventually a point is reached where the back grazing alone is sufficient to provide the horse’s calorie needs and the fence should not be moved any more.

A variation on strip grazing involves two fences. The second fence follows the first across the field, removing some of the back grazing each day such that the area of back grazing remains constant. This allows the same amount of ungrazed grass to be given to the horse each day, but relies on there being enough area in the field not to run out of ungrazed grass over the summer.

The challenge with any starvation paddock approach is to get the area just right. Too much, and the horse may binge on grass, creating blood sugar spikes; too little, and the horse may become stressed and/or lose weight. One helpful measure is to place a small amount of hay in the corner of the field each day (enough to provide perhaps 10–20 per cent of the calorie requirements for the period of the day that the horse is at grass). The amount of hay eaten can then be monitored. If the horse eats all the hay, that suggests that there is not enough grass and the area can be increased (or the fence moved further per day in a strip-grazing scheme). If the horse does not eat any of the hay, then that suggests that there is too much grass and the area can be reduced (or the strip-grazing fence moved a smaller distance). The advantage of this system is that the owner can react on a day-to-day basis to changes in grass-growth rates resulting from changes in weather patterns. By reducing the boom-and-bust effect that is normally a feature of pasture grazing, the risks of stress or over-eating are minimized. This scheme also has the advantage that the horse never gets 100 per cent of its calorific requirements from grass (a proportion of the intake comes from the hay), so avoiding the scenario where the horse gets more calories than it needs from a sugar-rich source.

Height of Grass

There is some controversy around the way in which pasture should be managed for a laminitic horse. There is a school of thought that states that starvation paddocks are the wrong way to manage a laminitic, and that such a horse should only be grazed on a sward that is between 10 and 25cm in height. This approach seems to be based on the assumption that overgrazed grass will be stressed and hence higher in fructans, which, as we saw in Chapter 5, is not true. The problem with such schemes is that they encourage ad-lib feeding of grass, which typically results in the horse overeating (especially given that horses with EMS and PPID tend to have excessive appetites). This both increases the horse's weight (and hence the severity of any insulin resistance) and also increases the risk of the horse bingeing on sugar-rich grass and pushing its blood glucose levels too high. In the author's experience, such approaches result in a much worse outcome than the use of a correctly managed starvation paddock.

Factors Affecting Leaf Sugar Levels

It is useful to understand the factors that can affect the sugar levels in pasture. These sugars are initially produced by photosynthesis. For photosynthesis to occur, several things are needed: carbon dioxide, minerals, sunlight, warmth and water. Carbon dioxide is readily available from the air around the plant, but a lack of any of the others can be the limiting factor in sugar production. Sugars are then used for leaf growth. Both warmth and water are needed for leaf growth as well. Where either are lacking, any sugars that are produced are stored in the leaf in the short term and in the roots over the long term. Rye grass largely stops growing when the temperature drops below around 6°C, but equally, it doesn't generally produce much in the way of sugars at such low temperatures either. The sugar level in the leaf tends to peak when it is warm, when the roots have plenty of water and the sun is shining strongly. Hence, the highest risk period for a laminitic horse is in the middle of a warm sunny day when it has previously rained and the soil is moist. This information can be used with very sensitive horses to manage turnout so as to avoid the times of highest risk.

Preserved Forages

Preserved forages, such as hay or haylage, are typically less problematic to laminitic horses than fresh grass. There are three main reasons for this. Firstly, the cut grass continues to consume some of the stored sugars in the leaf after cutting, but photosynthesis is minimal – hence the sugar levels in the hay or haylage are lower than in the grass prior to cutting. Secondly, hay is typically made at times of the

year when sugar levels are lower. Thirdly, hay is typically made in hot weather when the soil moisture levels are lower and hence photosynthesis is limited. Late-cut hay (cut from August onwards) is also typically lower in sugars (because the grass has run to seed and growth has slowed) and higher in fibre (because the flower/seed stems need to be higher in fibre to hold up the seed head). All other things being equal, late-cut forage is always better for laminitics than early-cut forage.

It is also easier to identify the species content of late-cut hay because it contains seed heads (the identification of species content from leaves is far more difficult). Hay containing a high proportion of rye-grass seed heads is likely to be higher in sugar and lower in fibre than hay containing less rye grass or none. The presence of easily identifiable species such as sweet vernal grass, crested dog's tail, knapweed, yellow rattle, ox-eye daisy and devil's bit scabious are also useful indicators that hay has come from an unfertilized meadow and hence is likely to be lower in sugars.

On average, hay seems to be marginally better for laminitics than haylage. The process of making haylage involves bacterial fermentation of the forage, which starts the process of digestion before the horse even eats it. One suggestion is that this means that the haylage digests faster in the horse's gut and hence is more likely to create blood-sugar spikes. Other possibilities are that the presence of high levels of bacteria in haylage, or maybe the fact that it is more acidic, may be problems. Sileage should never be fed to horses as it can cause laminitis. Even haylage should be fairly dry. Any bale that leaks water when opened is likely to have been baled too wet to be safe for a laminitic horse

Fig. 106 Rye-grass seed heads in a highly fertilized pasture. This photograph was taken in late summer and hence the seed heads are typical of what would be seen in late-cut hay or haylage. The key identifying feature is that rye grass is the only UK grass to have flowers/seeds that alternate up the main stem. The flowers present earlier in the year look almost identical but are green.

Soaked Forages

Some severe EMS cases can react to the sugar levels present even in the best hay. For such cases, soaking the hay can prove to be beneficial. Haylage can also be soaked although there is a far greater risk of the haylage going off if it is soaked for a long period or not fed immediately to the horse after soaking. The correct length of time to soak hay for is hotly debated, with some experts arguing for thirty minutes and others for as much as twenty-four hours. Longer soaks increase the risk of undesirable fermentation occurring (particularly in hot weather) and so shorter periods are preferred where this works. In the author's experience, however, some horses do better when the hay is soaked for longer periods. Experimentation seems to be the order of the day, and the shortest soak should be chosen that achieves the desired results.

A critically important point to note is that water used to soak one batch of hay should never be reused to soak the next batch. Quite apart from the increased risk of undesirable fermentation, the water becomes saturated with sugars soaked from the hay to the point where further sugars will not be drawn out. Indeed some proponents of longer soaks suggest replacing the water half way through the process.

The exact mechanism by which soaking aids the laminitic horse is not entirely clear. Soluble sugars are indeed removed to an extent by soaking, but it may be that other effects come into play. For example, there is a suggestion that some of the potassium content of the forage can be removed by soaking, which may help to mitigate high potassium:sodium ratios. Further research work on the nutritional effects of soaking different types of hay would be most helpful.

Bucket Feed Contents

It is easy whilst worrying about forage to forget the importance of the contents of the feed bucket. There are many commercial feed components and 'complete' feeds available, and choosing the right one(s) is something of a nightmare for the typical owner. As a general rule, most pleasure horses do not need the extra calories represented by a bucket feed. Such horses are more than capable of maintaining weight on a forage-only diet, and the only reasons to feed a bucket feed are to make the horse easier to catch and as a vehicle for feeding any supplements that may be required. As such, it makes sense to use a bucket feed that is as close as possible to pure fibre.

There is an increasing range of high-fibre 'chop-based' products on the market. However, many of these are based on alfalfa and straw, and there are two issues with these. The first is that some laminitic horses appear to do badly on alfalfa-based products for reasons that are not well understood. The second is that straw products are often contaminated with herbicides as a result of modern cereal farming practices. It is well worth interrogating feed manufacturers as to their policies on testing for herbicide contamination before using their products.

A good alternative to chop-based products is unmollassed sugar beet. This has low sugar and starch levels and is a good source of easily digestible fibre. It is also useful for elderly horses with few remaining teeth.

Feeding a laminitic is even more difficult if the animal is already underweight. Clearly it is important to identify if there are medical reasons for weight loss (see the next chapter), but there is a temptation with any underweight horse to feed high energy feeds in an attempt to improve condition. This risks worsening any laminitis. High energy feeds based on sugars and starches (such as coarse mix and pony nuts) should be avoided where possible, especially where these feeds are processed (and hence release energy rapidly).

Ad-lib access to forage is critically important in underweight horses, as is giving small regular meals based on 'safe' ingredients such as unmollassed sugar beet. A degree of weight gain can be achieved safely using oil-based feeds such as linseed (micronized versions avoid the inconvenience of having to cook raw linseed), but excessive use of oil-based feeds can reduce the absorption of vitamin E and selenium. Micronized linseed can be safely fed up to around 150g per day for a 500kg horse (ideally spread over two feeds) without the need to supplement vitamin E and selenium.

Although there is some controversy about this, in the author's view, rolled oats can also be a useful way to increase weight so long as they are fed as part of small meals so as not to overload the stomach. A 500g feed of rolled oats twice per day can help with weight gain in an underweight horse, and help retain weight in a horse prone to weight loss. The idea of feeding oats to a laminitic horse often raises eyebrows, but despite the starch content, the lack of processing of the oats leads their energy content to be released slowly in the gut, hence avoiding sudden blood-glucose spikes. Oats also do not normally cause 'heating' if fed in small quantities as part of a small bucket feed. The 'heating' effect is more associated with feeding oats as part of a feed that is large enough for some to spill over part-digested into the small intestine. However, a small minority of horses do react badly to even small quantities of oats, so, as with any new feed, oats should be introduced slowly and the horse monitored carefully for any signs of laminitis. Given the controversy and the minority of horses that react badly, it probably makes sense not to try rolled oats unless other approaches to weight gain have failed.

It is important to be aware that most processed horse feeds are not suitable for a laminitic horse, including, sadly, many that are marketed as 'safe for laminitics'. The owner of many a laminitic horse has learned the hard way not to believe every marketing claim from the equine feed industry! Of particular note is the prevalence of micronized cereals in feeds aimed at laminitic horses. There has been a misconception in the feed industry (based on the false assumption that the carbohydrate overload model of laminitis represents the mechanism behind all laminitis cases) that starch is safe for a laminitic so long as it is fully broken down and absorbed as glucose before hitting the hindgut. Micronizing cereals makes them easier to digest, and hence reduces the risk that starch will reach the hindgut and infiltrate it undigested. This of course ignores the dominance of endocrinopathic cases, where a sudden rise in blood glucose as a result of easily digestible starch can actually be just as much of a problem as that starch reaching the hindgut undigested. For this reason, micronized cereals should be avoided for laminitic horses.

Glucose Production from Feeds

Given that endocrinopathic laminitis appears to be by far the most prevalent form in the UK, it makes sense to try to avoid any feed that might cause blood-glucose spikes. For a number of years there has been a tendency for people to try to keep the sugar content of both preserved forages and bucket-feed ingredients low. The level of water-soluble carbohydrates in the total feed has also been used in an attempt to predict suitability for laminitics.

What is needed is an accurate way to measure the effect each feed will have on blood glucose levels, reflecting both the total quantity of glucose released, and the speed with which it is released and hence the peak rise in blood glucose resulting from the feed. Recent guidelines from the Equine Cushing's and Insulin Resistance Group (ECIR) suggest combining the quantity of starch with the quantity of carbohydrate that can be extracted from the feed, using ethanol as a solvent. The recommendation is that the total quantity of ethanol soluble carbohydrate (ESC) plus starch in the horse's total feed intake should be less than 10 per cent. It makes sense to ensure also that each part of the horse's feed intake – grass, hay and bucket feed – individually meets the target of less than 10 per cent ESC + starch, as well as collectively. There is a growing body of anecdotal evidence that this target is a useful one when dealing with endocrinopathic laminitis.

WEIGHT LOSS

As obesity is known to exacerbate insulin resistance, it is clear that reducing the bodyweight of an overweight laminitic horse is important. This is often portrayed as an 'easy win', but many owners find that reducing the weight of horses with endocrinopathic laminitis is far from easy.

The horse has evolved to eat forage with a much lower energy density than that usually available in the UK. In order to keep the daily calorie intake of even a healthy horse correct, the total daily volume

of food has to be kept lower than the volume the horse has evolved to eat. This results in the horse feeling hungry, to some extent, most of the time. This is particularly true if the horse is not in work and hence has a lower calorie requirement. Furthermore, once a horse develops EMS or PPID, their appetite increases, and this exacerbates the problem. Add to this the growing evidence that horses with metabolic disease can lay down more fat for a given calorie intake than healthy horses, and reducing weight in such cases becomes a serious challenge.

In practice, reducing a horse's weight solely by reducing the calorie intake is likely to prove difficult. The key to weight loss is exercise (combined, of course, with strict control of diet). Clearly it is not possible to exercise a horse that has just had an acute laminitis attack, but the techniques described in earlier chapters can be used to get the horse to the point of being safely exercised as early as possible.

Of course, if the horse is not overweight in the first place (and, contrary to popular opinion, a good proportion of EMS cases are not), weight loss is less of a useful technique. In severe cases, deliberately keeping the horse slightly underweight can be a useful part of management (a vet should ideally be involved in such decisions), particularly in the short term, but the gains from this are not large.

Where weight is a concern (in either direction), the use of a weight tape is extremely helpful. Whilst weight tapes are not particularly accurate in absolute terms, they are very good at picking up changes in weight so long as they are always placed in the same position. It takes around 30–50kg weight gain/loss on a 500kg horse before this is easily visible, whereas a weight tape can pick up changes of around 5kg. It is good practice to use a weight tape fortnightly and to record the results. Where weight gain/loss is desirable, the maximum rate of change that is considered safe is around 1 per cent of bodyweight per week. More rapid change risks causing additional health problems. For example, where a 500kg horse is overweight, the target range of weight loss would be 2½–5kg per week.

DRUGS

Whilst researchers are actively looking for new drugs to tackle the underlying causes of laminitis, there are really only two drugs that are routinely used at present.

Metformin

Metformin is a drug developed for the treatment of Type 2 diabetes in humans. It works in two ways. Firstly, it increases insulin sensitivity in cells to some extent (hence reducing the degree of insulin resistance); and secondly, it reduces the release of stored glucose from the liver (reducing the amount of glucose the insulin system has to deal with).

Metformin is used in horses with EMS in the hope that it will reduce the severity of the EMS and hence reduce the incidence/severity of laminitis. Although there is some controversy about its effectiveness, metformin does appear to provide some benefit to many cases. Where the horse is initially overweight, metformin is often prescribed for a limited time with the aim of reducing the weight and hence reducing the severity of the underlying EMS to the point where the metformin is no longer needed. There is little evidence as yet that metformin assists directly in weight loss, but at the very least, reducing the severity of the laminitis often makes it safer and more ethical to exercise the horse, and it is this increased level of exercise that typically helps to reduce the weight. For a good proportion of horses, weight loss resulting from the use of metformin reduces the severity of the underlying EMS to the point where the horse can be weaned off the metformin. In the author's experience, though, a significant minority of horses still need metformin to prevent laminitis even once the ideal weight has been achieved.

The side effects of metformin in horses appear to be limited, although in the author's experience, a tiny minority of horses react badly to metformin, exhibiting a paradoxical increase in insulin resistance, which can lead to a significant worsening of laminitis. For that reason, the drug should initially be introduced at a low dose, with the dose only being increased if no adverse effects are observed.

Unfortunately, metformin is not currently marketed specifically for horses, and so human tablets are used. A common starting dose is twenty 850mg tablets, and hiding such a large amount of crushed tablets in a feed can be a problem with some horses.

Prascend

Prascend® (generic name, pergolide) is used primarily in the treatment of PPID. This drug was originally developed for use in Parkinson's disease in humans, but was phased out in favour of newer drugs as a result of its side effects. It works as a dopamine receptor agonist – triggering dopamine receptors in the pars intermedia region of the pituitary gland in the same way that dopamine does. As such, Prascend can compensate for the lower level of dopamine production due to failing nerve control that is seen in PPID. By stimulating the pars intermedia in the same way that dopamine does, it reduces ACTH and cortisol levels to more normal levels.

Prascend can be extremely effective in the early stages of PPID, but for some reason, there appears to be a limit to the severity of PPID that can be compensated for. This means that PPID cases typically have near normal lifestyles, usually laminitis free, for a number of years but eventually reach a point where the underlying disease process can no longer be effectively treated.

The appropriate dose of Prascend is hard to predict, and the required dose does not always follow the severity of the PPID or the size of the horse as much as would be expected. For this reason it is usual to start with a dose of 1mg per day for a horse (0.5mg per day for a pony). The horse is then retested after six to eight weeks, and the dose adjusted upwards or downwards based on the combination of the blood results and the degree of clinical improvement. This process is repeated until an appropriate control of both ACTH level and clinical symptoms is achieved. The horse should then be retested every six to twelve months to ensure that the underlying PPID has not worsened.

Unlike metformin, Prascend has a number of side effects, the most common being lethargy, depression and loss of appetite. These side effects seem to be most problematic when the horse is first given Prascend, or when the dose has been increased significantly (another reason to start with a low dose and increase it slowly). For most horses, any side effects tend to become less problematic after a few weeks. Where they persist, this can be an indication that the dose is too high (assuming that any laminitis is well controlled). The loss of appetite can be a persistent problem for a minority of horses, though, and this can make keeping weight on the horse difficult.

Although Prascend is licensed only for the treatment of PPID, some vets are controversially experimenting with it for the treatment of EMS – and with success in some cases. It is possible that those cases that benefit from Prascend are actually early PPID cases that have yet to become sufficiently severe to show up on the relevant blood test, but there is at least a suggestion that Prascend may have some alternative mechanism of action that allows it to help in pure EMS cases.

OTHER APPROACHES

Besides the techniques described above, there is little more that can be described as being backed by consensus. There are further drugs that are sometimes used – for example, aspirin is known to thin the blood, and it has been suggested that this may help in the recovery from laminitis by reducing clotting – but such approaches do not tackle the underlying causes of the disease.

There is also some interest in herbal approaches – for example, chasteberry (Latin name: *vitex agnus castus*) has been suggested as helpful in calming down the hormone system in PPID cases in particular, but whilst some researchers found a small beneficial effect, others could not recreate this. There seems to be agreement that chasteberry helps lift mood and improves coat shedding in PPID cases, but little consensus that it actually helps with the degree of laminitis. It might be tempting to try chasteberry alongside Prascend, but unfortunately there are some concerns that chasteberry may reduce the effectiveness of the Prascend. For that reason, whilst chasteberry might have marginal benefit in very early PPID cases, Prascend should be the preferred treatment.

Other dietary interventions such as mineral or vitamin supplementation are considered helpful by some, but there is no consistent description in mainstream literature of which supplements should be used in laminitis. The next chapter covers these in more detail.

10 Additional Approaches to Underlying Causes

The alternative viewpoint on tackling the causes of laminitis is little clearer than the established view. However, there are some angles that are worth considering.

TRACK SYSTEMS AND YARDING

Our horses' wild ancestors would have had to walk many kilometers per day to find sufficient food and water, and that level of exercise forms part of the environment required for a horse to be healthy. In contrast, domesticated horses in the UK typically have everything they need within perhaps a 50m radius, and walk fairly short distances per day. Lack of exercise is known to be a risk factor in the development of laminitis, and it is surprisingly difficult to introduce exercise regimes that provide sufficient exercise to fully counteract this.

There has been a lot of interest recently in track systems as a way of improving exercise levels. The problem with a typical rectangular field is that the horse can reach the boundary in any direction in just a few strides. If the same area of field can be made into a long narrow strip, then the distance from one end to the other is much further. If the horse can be encouraged to move between the two ends of the track – for example, by placing hay at one end and water at the other – then the number of steps it takes per day can be increased significantly.

Feral ponies are used to manage the sand dunes at Newborough Warren on the Isle of Anglesey. These ponies roam on hundreds of hectares of land and yet maintain the correct weight and have good feet, suggesting that over-availability of food alone does not cause obesity or laminitis.

In the USA, this concept has been combined with the idea that horses do better on hay than grass. A typical approach involves creating a track around the outside of a large field by installing a new fence inside the line of the existing boundary fence. The central portion of the field is kept horse-free and used to make hay. The horses meanwhile are kept on the outer track and fed hay throughout the year. Any grass on the outer track gradually gets eaten and uprooted, leaving bare soil, which turns to dust. The result is what is termed a ‘dirt lot’ in the USA. If sight lines can also be blocked (for example if the field spans the brow of a hill or hedges are planted in place of the inner fence), then the horse’s natural curiosity can be utilized to encourage even more movement.

Of course the problem with dirt lots in the UK is that they turn rapidly to deep mud lots, especially in midwinter. The horses sink into the mud and walking around is a struggle, so much so that they become depressed and barely move. US-style dirt track systems can be created in the UK, but only with the installation of drainage and surfacing (with the exception of a few rare areas where sandy soils have sufficient natural drainage), which is generally prohibitively expensive. Even in summer, the additional traffic created by a track system (particularly where the horses are channelled into a narrow track) can create poaching in all but the driest of fields.

Another problem in the UK is that land is at a premium. Many horse owners do not have sufficient land to make their own hay, and so buy in hay as needed. For such owners, creating a system where hay is fed all year round increases costs dramatically. Of course, even without moving to a hay-only system, there is significant advantage to be gained by increasing movement with horses that are at grass even if the increase isn't as dramatic as with a classic track system. The trick is to do so without creating pinch points that become poached. Similarly, a track system that can only be used for the summer months is still advantageous, given that this is precisely the time when the grass is richest and hence the risk of laminitis is highest.

Some proponents of track systems suggest placing various 'conditioning' surfaces at points on the track with the idea of stimulating improved hoof growth. This can work well in some situations, but it is important not to force horses to walk on surfaces that their feet are not strong enough to cope with. For example, if a track is created with one section made of loose cobbles, this may cause problems where one member of the herd has LGL. Other horses will cross the cobbles easily, forcing the horse with LGL to cross the cobbles in order to keep up with the herd, with a resulting increase in the risk of bruising or even abscessing. Similarly, placing aggressive surfaces around feeding or watering points is usually a bad idea. A better approach is to create optional routes (such as surfacing half of the track width only) so that horses with problem feet can avoid the more difficult surfaces.

Where increased movement is the goal, a useful alternative to stabling is yarding. By fencing off all exits from a yard and opening or removing stable doors, horses can be encouraged to come and go between stables and yard. Not only does this allow more natural social behaviour, but such behaviour typically leads to significant increases in movement. This can be particularly helpful for horses that stock up when stabled. Owners often worry about the risk of several horses getting stuck in a single stable, but in reality, most horses behave very sensibly in this situation. It is not uncommon to find three horses standing in the same stable, but it is surprising how little fuss there is when one horse decides it wants to leave!

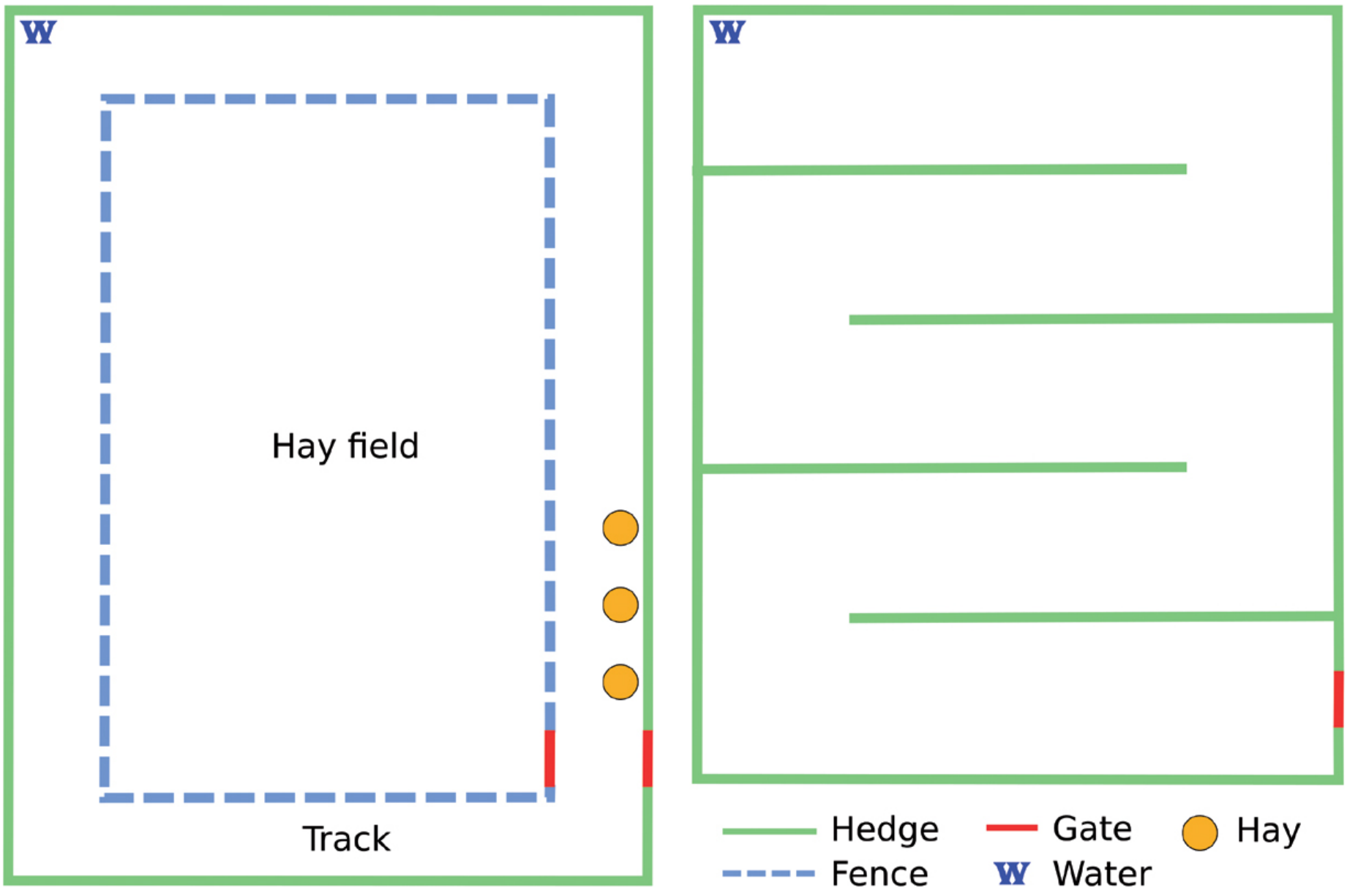


Fig. 107 Two examples of track systems. LEFT: ***A standard track system with the central portion of the field used for hay making.*** RIGHT: ***A baffle system with hedges planted to block straight line movement and remove sight lines so as to encourage increased movement without significantly reducing the area of grazing available.***

One issue with yarding is that concrete is a fairly hard and abrasive surface. As such, it is important to be aware of the quality of the hooves and the current degree of LGL present amongst herd members. Some horses will not cope with a standard yarding approach, either because the feet become bruised or because the hooves become over-worn. Booting the feet twenty-four hours per day is possible with care as a workaround to this, but is not ideal because of the risk of rubbing. Alternatively, part or all of the yard can be covered with rubber matting to allow those horses with less good feet somewhere safe to stand and eat.

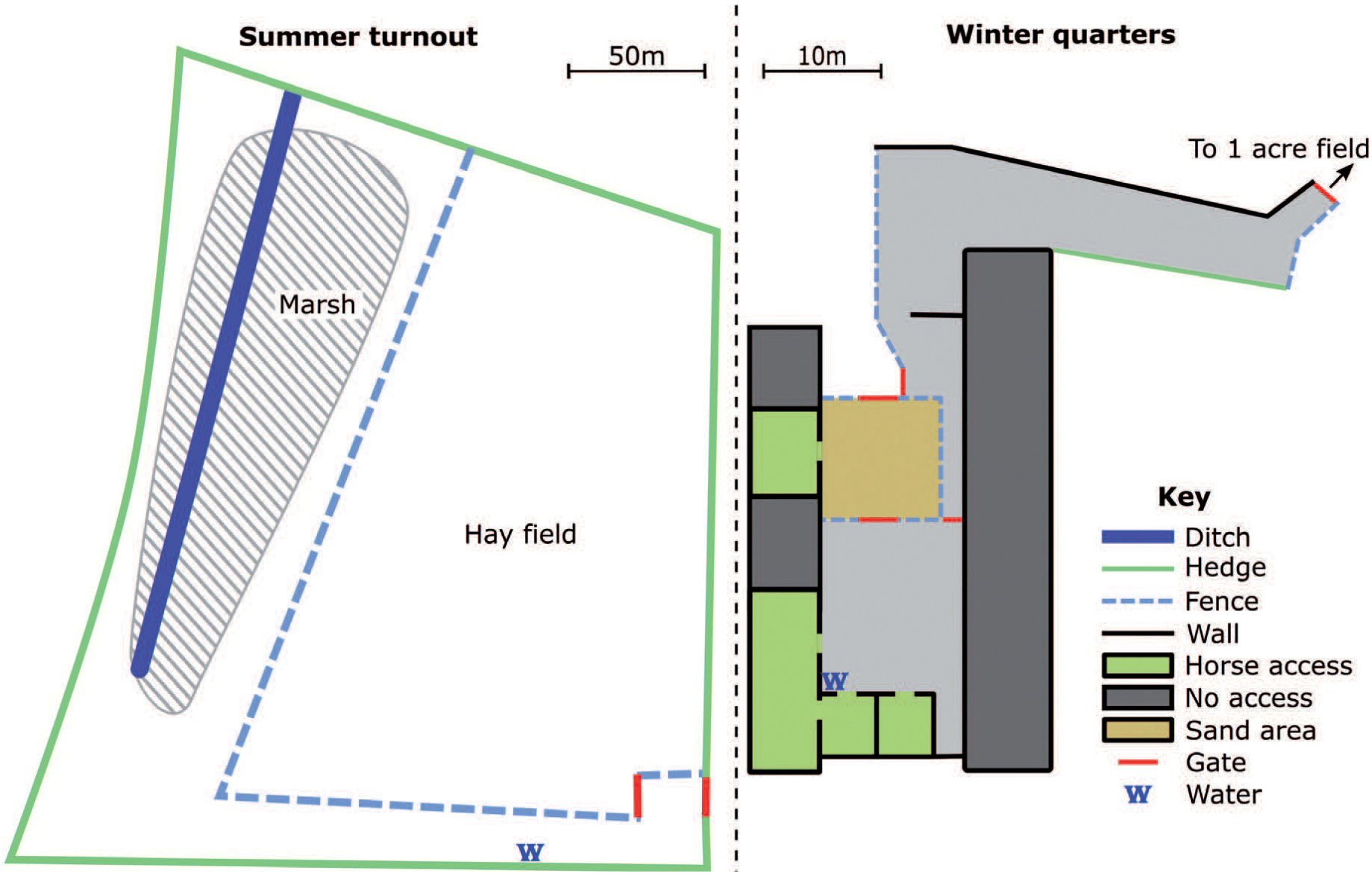


Fig. 108 A plan of two areas of the author's smallholding. LEFT: ***Summer grazing consists of an L-shaped fenced area around a hay field. Four horses are kept on this area and exist solely on the grass. It can only be used in summer because the marsh becomes severely waterlogged in winter. The field is on the brow of a hill and hence the horses cannot see one end of the 'L' from the other, which encourages curiosity-driven movement. The pre-existing drainage ditch is wide and deep enough to make crossing difficult, which encourages the horses to walk the long way round to get from one side of the marsh to the other. It is significantly wider than a normal track system, which keeps poaching to a minimum.*** RIGHT: ***In winter the horses are kept on a concrete yard and fed mostly on hay, although they have access to a one-acre field. They have free access to a barn and three stables, as well as a 10m square sand area which they use for sleeping and urinating. Piles of hay are placed around the yard (or in the barn during inclement weather). The yard is not visible from the field, and vice versa, which again encourages curiosity-driven movement. One horse (with a history of severe laminitis) needs boots on his front feet whilst on this yard, the others cope well barefoot. One stable has a cobbled floor and the horse with the strongest feet (who is also bottom in the herd hierarchy) uses this to escape from the other horses.***

It is important when considering tracks systems or yarding not to get stuck with fixed ideas about layout gained from the internet or books. The ideal is to work with what is already there, and apply the principles of track systems and yarding pragmatically. Even the most unpromising set-ups can often be improved by the judicious placement of a fence here and there. It is quite possible to spend large amounts of money creating 'ideal' systems, and yet good results can often be achieved with relatively small investments. Where there is uncertainty about the right approach, temporary electric fencing can be used to trial systems before more expensive fencing is used to make the preferred option permanent. Fig. 108 shows the author's track and yarding systems, created with fairly minimal amounts of fencing.

MINERAL BALANCING AND THE ROLE OF 'BALANCERS'

Not all nutrients present in the gut are just passively absorbed into the bloodstream. Some nutrients are absorbed passively, but some are actively transported through the lining of the gut. For example, the

simple sugar fructose can only be absorbed via the action of a protein called GLUT-5, which is present in the lining of the gut.

When it comes to minerals, the ratios of minerals to each other are often as important (or even more important?) than the absolute levels. It is fairly well known that a diet high in phosphate (for example, where large quantities of bran are fed) can result in an excessively low calcium:phosphate ratio. This blocks the uptake of calcium in the gut, and the horse will then have to 'steal' calcium from its bones in order to keep the myriad of bodily functions that depend on calcium functioning correctly.

What is less well known is that there are many other mineral ratios that are important. Some of these have been researched, but for some we have little data on what would be the ideal ratios. Amongst the most common to cause problems in the UK are the copper:iron and copper:manganese ratios. These can be a problem because many UK soils are fairly high in iron and manganese whilst being relatively low in copper. In contrast, it is extremely rare to see a UK soil that is deficient in either iron or manganese. There is some suggestion that copper (and possibly zinc) deficiency caused by high iron or manganese levels may contribute to insulin resistance. As such, the management of these ratios is something that needs to be carefully considered where laminitis is a problem.

When horse owners suspect problems with mineral levels, they typically reach for an off-the-shelf product that claims to 'balance' the ration. These products work by providing a good quantity of each essential nutrient on the understanding that this will then correct any deficiencies. Unfortunately, the idea that these products 'balance' the ration is deeply flawed. They will, of course, correct any minor deficiencies, but if anything is already too high in the diet, these products tend to make things worse. And if two minerals are seriously out of balance with each other, adding a little of each is not going to miraculously bring them back into balance. A more honest name for such a product would be a 'general purpose supplement'.

Balancing a Diet Using Forage Analysis

In order to actually *balance* a diet, you need to know the precise mineral content of the base forage and any bucket feed ingredients. The mineral breakdown of most commercial feed products is either published, or it can be obtained by asking the manufacturers directly. The forage (grass and hay) levels can only be obtained by sending samples off to a lab for analysis (at a cost typically approaching £100 per sample). It is important when sending samples for analysis to send them to a company that specializes in analysing samples for horses. There are many labs that will do such analysis for farmers, but the recommendations that come back are typically aimed at maximizing milk yield in dairy cattle rather than keeping a horse laminitis free!

Once you have the mineral breakdown for each component of the diet, these need to be entered into a spreadsheet and scaled according to the quantity of each component that the horse is eating. The quantity of hay or bucket feed ingredients being consumed can be weighed. However, analysis is typically quoted as proportions in relation to dry matter (that is, with all water removed). This has little impact on bagged feeds, which typically have fairly low moisture contents, but needs to be considered when estimating the intake of hay, and especially haylage. Estimating grass intake is far harder. One quick rule of thumb is that a typical 500kg horse needs 10kg of dry matter to maintain weight. So if a horse is surviving entirely on grass and is maintaining a steady weight, this figure can be used as a rough estimate. To some extent it can also be scaled for different weights of horse, and depending on how many hours per day the horse is turned out.

Even once you have the total intake of each mineral, you need to know what the recommended daily minimum and maximum intakes are for each mineral, as well as the acceptable ranges of a number of key mineral ratios. The most authoritative source of information for this is a book entitled *Nutrient Requirements of Horses*, which is published by the National Research Council of the National Academies in the USA. Sadly, even this book suffers from a lack of research into this field. There are few indications of the toxic levels of minerals, and very few indications in relation to important ratios.

Even the minimum daily requirements are not fully researched, with some recommendations based on research in other species (including hens).

Once any balancing problems have been identified, the solutions aren't necessarily trivial. Until recently, there were few commercial products available that had the appropriate mineral content to correctly balance typical UK pastures. In some cases, balancers had to be made from individual ingredients (for example magnesium oxide, copper aspartate, zinc gluconate). This again requires a degree of specialist knowledge, as getting the quantities wrong can cause serious side effects. Similarly, failing to mix the balancer correctly may result in individual ingredients forming clumps, rather than being evenly distributed through the batch. There is then the potential for the horse to get several weeks' worth of copper, for example, in a single meal, a quantity that could potentially be toxic.

A further problem is that it is fairly common to see forage in the UK with a mineral balance that is so poor that just correcting any outright deficiencies comes nowhere near to correcting the ratios of key minerals. A typical example of this is shown in Fig. 109. At first sight it would appear that this forage has plenty of iron and manganese and would just need moderate supplementation with copper and zinc.

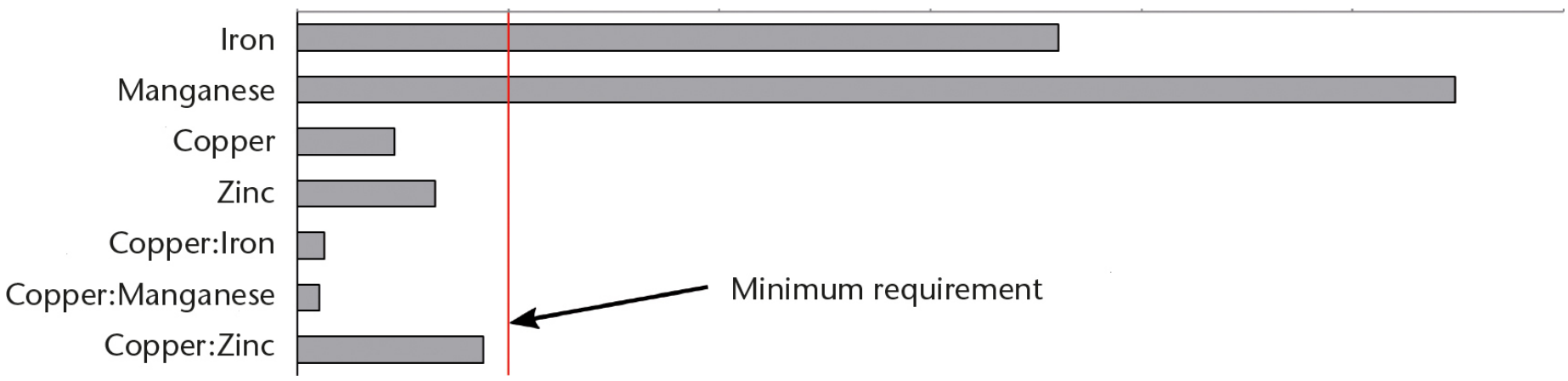


Fig. 109 The levels of iron, manganese, copper and zinc in a typical forage shown as a proportion of the recommended minimum daily requirement (red line). Also shown are key ratios of these minerals, again as a proportion of their minimum recommended values. The copper:zinc ratio is slightly low, but the copper:iron and copper:manganese ratios are extremely low.

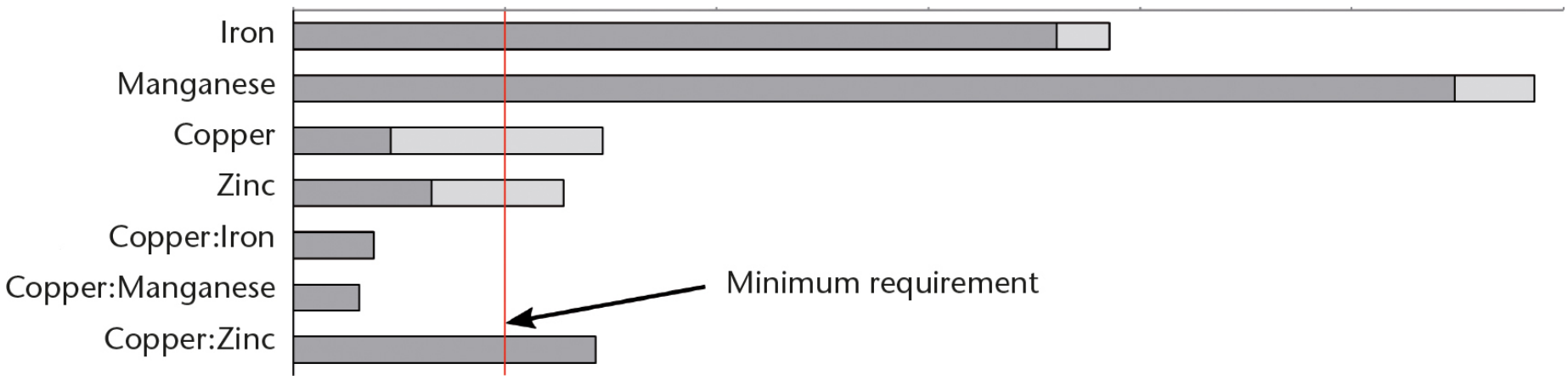


Fig. 110 The same forage as in Fig. 109, but with the addition of a commercial balancer (shown in lighter grey). All minerals shown are now above the minimum requirements, but the copper:iron and copper:manganese ratios are still fairly low.

Fig. 110 shows the same diet after the addition of a typical high specification commercial balancer product. The basic mineral levels are all now above the minimum daily recommendations, but the ratios are still poor.

The problem with this scenario is that the high levels of iron and manganese will reduce the uptake of copper in the gut (and similarly of zinc), effectively creating a copper deficiency even though there appears to be sufficient copper in the diet. Simply bringing the copper level above the recommended daily minimum is nowhere near enough to correct this 'functional' deficiency. Instead, the copper level needs to be boosted to well above normal recommended minimum levels to compensate for the reduction in uptake caused by the high levels of iron and manganese. And because the commercial balancer adds more iron and manganese, it actually makes the underlying problem slightly worse.

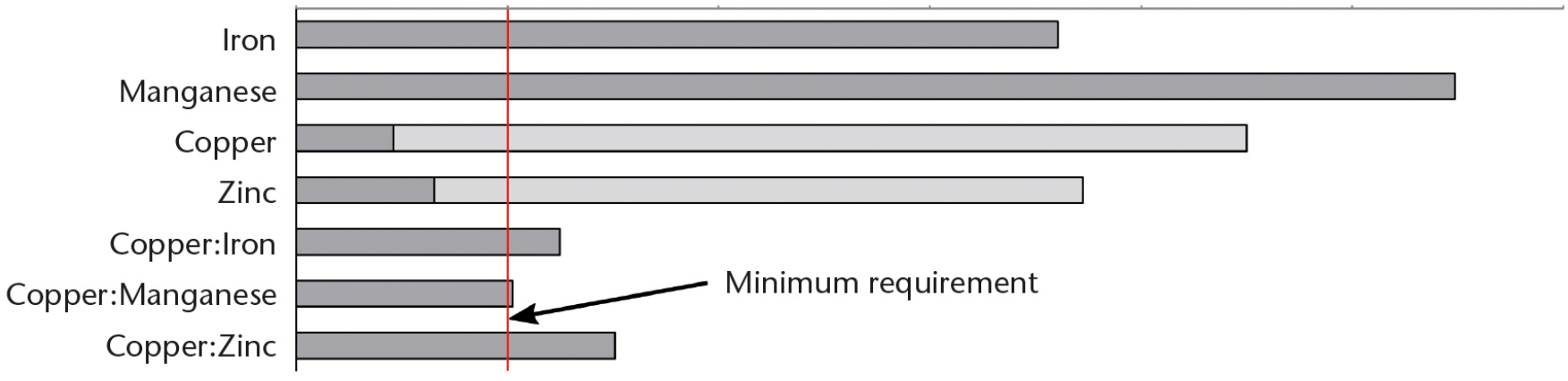


Fig. 111 The same forage as in Fig. 109, but with the addition of a bespoke supplement (shown in lighter grey). All minerals and ratios shown are now at or above minimum requirements.

In contrast, Fig. 111 shows the same forage with the addition of a bespoke supplement that contains no iron or manganese but contains higher than normal levels of copper and zinc. The levels of copper and zinc are nowhere near toxic levels, but are significantly higher than would normally be considered necessary in a supplement. The result of this is that not only are all minimum levels met, but that all the ratios are now within safe limits. This approach has corrected the outright deficiencies, but has also corrected the functional deficiencies created by poor mineral ratios. The result is that individual cells within the horse will now have enough copper and zinc to function correctly. A similar approach can be taken with other minerals.

Clearly, balancing the ration to the forage is a non-trivial exercise. There are few experts out there with experience of doing this (although all new members of the EPA are now trained in this approach). It would be tempting to ignore this approach in favour of easier wins if it weren't for the inconveniently large body of anecdotal evidence that it helps a lot of horses!

Intelligent Balancers

Thankfully, it is not usually necessary to go to the lengths described above. Over the last few years, various people involved in this approach have noted that UK forages tend to fit a pattern. It is extremely common to see high levels of iron and manganese, and fairly common to see borderline or low levels of copper, zinc, selenium and magnesium. In contrast, deficiencies in iron and manganese are extremely rare, as are significant excesses of copper, zinc and magnesium. Selenium is a little trickier, as some regions of the UK have moderately high levels, but luckily it is not usually necessary to supplement high levels of selenium to compensate for other issues.

Given this pattern, it is possible to design an 'intelligent' balancer which attempts to correct the typical mineral imbalances seen in UK pastures. The key features of such a product are that it shouldn't contain more than traces of iron or manganese, and it should contain sufficient copper and zinc to offset high levels of iron and manganese in the base forage. It is also a good plan to add a small amount of selenium (sufficient to correct deficiencies in most UK pastures without risking providing excess when combined with pastures already high in selenium) and some magnesium.

There are several companies offering ranges of intelligent balancers in the UK, including Progressive Earth, Forageplus and the Healthy Horse Company. Feeding appropriate products from these companies appears to provide at least some improvement for a fairly large proportion of laminitis cases. As with any such products, they should be added slowly, and it is very important not to feed these products alongside any other products that have a significant mineral content (for example hoof supplements or general purpose supplements).

The Role of Magnesium, Potassium and Sodium

The potential for magnesium deficiency to contribute to insulin resistance has already been discussed in Chapter 6, as have the reasons why most UK pastures (and hence also most preserved forages) are

deficient in magnesium. Mineral ratios, and in particular the potassium:sodium ratio, also have a part to play here.

The constant application of artificial fertilizer to fields over the last few decades has seriously increased the potassium content of grass. A typical dairy pasture may contain as much as 6 per cent potassium (by dry matter), which compares to less than 1 per cent in a traditional meadow. A 500kg horse requiring 10kg of dry matter forage per day might therefore ingest as much as 600g of potassium per day. Of that, even a hard-working horse will only need 40g. Most of the remaining 560g of potassium has to be excreted in the horse's urine by the kidneys (the rest remains in the droppings). Even when compared to the large volume of urine produced by a horse, this is a lot of stuff to dissolve in the urine, and the kidneys have to work hard to achieve that. The kidneys find it particularly hard to do so if the level of sodium in the blood is too low. Where this is the case, the kidneys will tend to leach magnesium as a side-effect, exacerbating any deficiency of magnesium in the base diet. This means that it is important to keep the potassium:sodium ratio in the diet reasonably low.

Those who have worked with horses for many decades and remember the time before bagged 'complete' feeds will remember the 'old-fashioned' advice to add a handful of salt per day to the bucket feed. This would have perhaps been a large handful for a horse in work and a small handful for one out of work. The introduction of 'complete' feeds led to the misconception that everything needed was in the bag. However, salt attracts water, so manufacturers aren't able to add significant levels of salt to a bagged feed without a risk that the feed will draw moisture from the air and go off. As a result, salt should always be added to the bucket even where 'complete' feeds are used.

The idea of adding salt directly to the bucket has gone out of fashion in recent years with people favouring the use of salt licks. Some of these licks have added minerals, which can be beneficial or problematic depending on the context. For example, using an iodized lick in an area where levels of iodine in the forage are already high can cause problems with the function of the thyroid gland. Conversely, failing to supplement iodine in an area such as Derbyshire, where soil iodine levels are notoriously low, can also result in poor thyroid function. Unfortunately, few horse owners have a good understanding of when to use each type of salt lick.

A bigger problem is that horses in the UK often struggle to regulate their own salt intake. The horse hasn't evolved to cope with a high potassium diet, and struggles to differentiate between high potassium and high sodium intake. As a result, many horses will instinctively under-use a salt lick because the high potassium intake is erroneously mistaken for sodium. An easy way to test this is to weigh the salt lick twice, two weeks apart, and from that work out the weight of salt consumed per day. Whilst some horses will consume adequate amounts, many will fail to do so. The presence of a salt lick reassures the owner that the horse is getting plenty of salt, when in reality that may not be the case. At the other end of the extreme, a tiny minority of horses will dramatically over-use a salt lick (possibly as a result of underlying health problems), potentially to the point of poisoning themselves. For that reason, any new introduction of a salt lick to a horse should be carefully supervised for the first day or two.

So where potassium intake is too high (and it almost always is in the UK), it is important to supplement both salt (to counteract the potassium) and magnesium (to correct outright deficiencies in the forage).

A safe and effective approach for a 500kg horse in light work is to add 25g of table salt per day to the diet. Cooking salt or table salt should be used, not Lo-salt (which provides potassium instead of sodium and hence makes things worse). That dose can be scaled up or down for heavier or lighter horses. A little more should be added for horses in heavy work, either directly into the feed or in the form of an electrolyte drink. This quantity may seem excessive to some. We are conditioned to see the addition of salt to food as a bad thing because human diets are typically already high in salt, and hence additional salt leads to health detriments. In contrast, however, horses' diets are typically low in salt and they need supplementation.

A good guideline for magnesium supplementation is 5g of extra magnesium per day for a 500kg horse. The easiest way to supplement this in isolation is to add magnesium oxide to the bucket. This

reacts with stomach acid to form magnesium chloride, which is well absorbed. Very roughly, 5g of magnesium is provided by around 10g of magnesium oxide. There are various grades of magnesium oxide available on the market, but for use in horses, higher quality grades such as pharmaceutical grade should be used. These typically come in one of two forms. Heavy magnesium oxide is a coarse powder (about the consistency of wheat flour), and a 25ml scoop of this provides the required 10g of magnesium oxide. Light magnesium oxide is a finer ground powder (more like talcum powder) and, having more air incorporated into it, a 50ml scoop is required to provide 10g. As with the salt, the dose can be scaled according to the horse's weight.

Any horse that shows any signs of laminitis, whether full blown or low grade, should be given supplementary magnesium and salt unless there is definitive evidence that the base forage already provides sufficient of each. Some supplements already provide the magnesium – such as the intelligent balancers described above – but the salt should always be added as well.

THE ROLE OF B VITAMINS

In a healthy horse, all the B vitamins (and vitamin K) required are produced by bacteria in the hindgut. As a result, horses don't need much in the way of B vitamins in their diet, and even where supplementation is needed, guideline amounts are fairly low. Of course, this assumes that the gut bacteria are healthy. Where the balance of gut bacteria is incorrect, it is possible for B vitamin production to tail off dramatically. The actual quantities of each B vitamin produced in the hindgut are not fully known, but it is likely that many of them are required in quantities of at least 1g, whereas supplementation guidelines are typically measured in tens of milligrams. So whilst a healthy horse should not need to be supplemented with B vitamins, an unhealthy one may gain benefit from fairly large levels of supplementation. For example, there is some evidence that allergies such as sweet itch can be improved by supplementation of vitamin B3 (ideally in the form of niacinamide) in quantities of at least 1g.

When it comes to laminitis, two particular B vitamins could theoretically be relevant. Both vitamins B6 and B7 are required for correct glucose metabolism, and deficiency at a cellular level would result in insulin resistance. There is little evidence of vitamin B6 supplementation helping with laminitis (although that may be just because there hasn't been enough research), but vitamin B7 is another matter. Vitamin B7 is also known as biotin, and biotin supplementation has been used for a number of years to improve hoof horn quality. The assumption has always been that biotin is somehow directly needed for the correct production of healthy horn at the corium. Whilst this might be true (at least in part), the horn quality issues that biotin appears to improve include cracks, flaring and horn infections, all of which turn out to be manifestations of LGL. So it seems likely that biotin supplementation may actually improve horn quality by reducing the level of LGL – presumably by correcting cellular level biotin deficiencies and hence reducing the degree of insulin resistance – and that this in turn has an indirect effect on horn quality.

One problem with biotin supplementation, however, is that biotin is an expensive ingredient. Probably for that reason, most supplements only provide around 15mg of biotin per day. This may help some horses, but in the author's experience, doses of around 60mg are more effective for many cases, and some horses seem to show further improvement at doses as high as 100mg per day. It is worth shopping around for supplements that provide sufficiently high levels of biotin. Of course if supplementing biotin shows no observable improvement in the hoof quality or soundness of the horse after a few months, then, as with any nutritional approach, it is not worth continuing with. Not every laminitis case will be deficient in biotin.

The ideal would be to try to improve digestion (as discussed below) such that the B vitamins are produced adequately by gut bacteria. In some cases, biotin supplementation can be phased out once gut health has improved, although sadly, some cases continue to benefit from biotin supplementation even when every avenue for improving gut health has been explored.

PROMOTING GUT HEALTH

Given that there are strong links between incorrect gut function and laminitis, it makes complete sense to try to improve the digestive health of any horse with laminitis.

Gastric Ulcers

Where gastric ulcers are suspected, the mainstream veterinary approach would be to pass an endoscope into the stomach to look for evidence of ulceration. Unfortunately, scoping for ulcers is expensive and not very pleasant for the horse, and also may not pick up very mild gastric cases (for example sub-mucosal ulcers) or ulcers further down the digestive tract (such as duodenal ulcers). Where ulcers are detected, the mainstream treatment is Gastroguard®, which works by reducing the production of stomach acid and hence reducing the acidity of the stomach to the point where damaged tissue has a better chance of healing. The cost of Gastroguard® is high enough to be a barrier to treatment in some uninsured cases. It is also questionable whether suppressing stomach acid production is always a good thing. Stomach acid is produced for important reasons, including assisting in the digestion of proteins, and sterilizing food to prevent pathogens making it into the rest of the digestive tract. There is therefore an argument that suppression of stomach acid as a treatment should not be undertaken without balancing the benefit against the potential risk of causing other problems.

Where ulcers are strongly suspected (for example a horse that does not want to be groomed in the girth area, and that is aggressive around food and/or shows stereotypical behaviours such as cribbing, weaving or windsucking), scoping should be performed and treatment with Gastroguard® initiated if necessary. But where there is only a slight suspicion, or a suspicion of mild ulcers, it may be appropriate (ideally with veterinary approval) to try herbal approaches to support gastric health. There are now a number of products on the market targeted at horses with poor gastric function, but the original (and in the author's opinion the best) is Hilton Herbs GastriX. This product contains mucilages such as slippery elm bark that help to coat inflamed tissue, protecting it from stomach acid, along with a range of other herbal ingredients that promote healing in the mucosal membranes of the foregut. Quite apart from the significant cost advantage over veterinary drugs, herbal approaches have the advantage that they can be used for longer periods and potentially will help with digestive problems further down the gut.

Whilst a good response to a product such as GastriX isn't fully diagnostic of gastric ulcers, the precise diagnosis is perhaps largely academic if the horse is much improved. A typical approach would be to start at the full dose for one month. If there is no difference in the horse's behaviour, or any reduction of the signs of metabolic disease within a month, then continuing with the product is probably pointless. Where there is a noticeable difference, a further two months at full dose followed by two months at half dose is a sensible regime. A minority of horses will show recurrences of problems a year or more later, and can be given a repeat course. A tiny minority benefit from being kept on a long term, low maintenance dose.

Probiotics

Much focus has been placed in recent years on hindgut health in laminitis cases, and it is tempting to try to influence the bacterial balance in the hindgut using probiotics. There are two main problems with this approach. Firstly, the hindgut is an ecosystem, with different bacteria species competing on the basis of survival of the fittest. The species populations present in any horse are the product of the environment in the gut of that horse. In many cases, beneficial bacteria may be present in small quantities, but are unable to compete with less beneficial bacteria because of the prevalent conditions. In such cases, 'seeding' the hindgut with beneficial bacteria in the form of a probiotic is unlikely to be effective. The quantities of probiotic needed to flood the gut with enough beneficial bacteria to make a serious difference are also unrealistic. However, probiotics can potentially be helpful where a course of

antibiotics has wiped out some species of gut flora, although this relies on the probiotic containing the species that have been knocked out.

Prebiotics

Prebiotics are perhaps a more sensible approach. These aim to provide nutrients that beneficial bacteria can use, but harmful bacteria cannot. The idea is to swing the environment within the gut in favour of the beneficial bacteria over the harmful ones. Of course, if you stop using the prebiotic, the benefit is lost, so this is not a one-off approach.

One of the most common prebiotics used is Saccharomyces cerevisiae (often described in the horse world as Yeasacc), which is a form of yeast. Whether fed in live or dead form, it cannot reach the intestines alive, and so any claims that it is a probiotic are false. But there is some evidence that Saccharomyces cerevisiae can act as a prebiotic, promoting a more healthy balance of gut bacteria.

Other commonly used prebiotics include mannan oligosaccharides (MOS) and beta-glucans, which are polysaccharides that are naturally found in the cell walls of yeasts, amongst other things. These substances can help with the excretion of harmful bacteria and promote beneficial bacteria. Short-chain fructo-oligosaccharides are also sometimes used as prebiotics for similar reasons, although these technically count as fructans… it is amusing to note that some of the same feed companies aggressively pushing the concept that fructans can cause laminitis are actually adding them to their feeds as prebiotics!

Whilst prebiotics make sense and may do some good, there is little evidence of them making significant differences in laminitis cases.

Whilst considering prebiotics it is perhaps worth mentioning that healthy gut flora depend on the horse being fed a rounded diet that contains all the nutrients needed by the gut flora. A restricted diet – such as is provided in a heavily fertilized field that is dominated by rye grass – is less likely to promote a diverse and healthy gut flora. Just as with humans, eating a varied diet promotes a healthier diversity in the gut flora. For that reason, allowing horses access to ‘rough’ grazing, hedgerows and suchlike, makes complete sense. And yet on so many yards, horses are fenced away from these more diverse food sources.

Other Approaches to Gut Health

Feeding the good bacteria is not the only way to create a beneficial change to the environment in the gut. There is a range of products on the market that approach this area from a variety of angles, some of which have more merits than others. However, a couple of specific approaches are worthy of a mention. First, various claims are made about feeding charcoal. ‘Activated’ forms of charcoal are thought to be good at absorbing toxins produced by non-beneficial bacteria and the sudden death of beneficial bacteria in the gut, and hence preventing them from being absorbed into the bloodstream. Unfortunately, activated charcoal may also absorb useful nutrients such as vitamins, making long-term use problematic. Less active forms of charcoal (such as Fine Fettle’s Happy Tummy) claim not to affect nutrient absorption, but are also unlikely to be as good at absorbing toxins. However, being somewhat alkaline, they may have a useful effect in de-acidifying the hindgut (and hence countering the effects of hindgut acidosis). There is certainly anecdotal evidence of Happy Tummy helping some laminitis cases.

Other products aim to improve the health of the lining of the gut. For example, a combination of Liquid Gold and Gut Restore (both by Thunderbrook Feeds) seems to help with improving the function of the small intestine and caecum. This is particularly useful in horses that show tenderness in the right flank, just in front of the hip (where the caecum sits). Such horses often hold themselves in a slight right flexion (and hence are stiff on the left rein), and tend to dislike bringing the right hind leg forward (which especially shows up when a hoof specialist is attempting to put the right hind on a hoof stand). Such products not only reduce the apparent right flank discomfort, but often seem to reduce the level of LGL. As with the GastriX, this approach would normally be used for a few months, and then be phased out.

SUPPORTING LIVER FUNCTION

There is some anecdotal evidence of a link between liver function and laminitis. Horses with poor liver function (as a result of liver disease, or poisoning, for example, by ragwort) seem to be more prone to laminitis. Therefore it makes some sense to try to detoxify the liver as a means of reducing laminitis. Detoxification products are typically based largely on a diuretic effect – they increase the volume of urine produced by the kidneys, and hence the amount of water the horse drinks. Such diuretics are claimed to have the effect of 'flushing' the liver and kidneys, helping the horse to clear toxins. It is possible that some herbal products also have other, less well understood beneficial effects on the liver. However, some care should be taken with liver 'detox' products. Where the liver or kidneys are severely diseased, strong diuretics can put more strain on already struggling organs and hence make things worse, rather than better. Where there is evidence or suspicion of significant liver disease, detoxing approaches should start with one of the milder products (such as NAF D-Tox). Should these be well tolerated and appear beneficial, stronger products (such as Hilton Herbs' Detox Gold) can be cautiously tried. If there is any evidence of increased bloating or fluid retention whilst the horse is on such products, they should be stopped immediately and veterinary advice sought.

Some specialist companies have also developed detox products aimed more specifically at laminitis cases. Anecdotal evidence suggests these can be helpful in at least some laminitis cases, especially as a 'first aid' approach for new cases. Good examples include Global Herbs' LamiPro Liquid, and Trinity Consultants' L94.

A TYPICAL 'FIRST AID' DIET

Whilst every case should be treated as an individual, it is perhaps helpful to outline a typical dietary approach to a new laminitis case. The following assumes a 500kg horse with moderate to severe new-onset acute laminitis:

- Remove the horse from grass entirely (using box rest or similar).
- Feed hay soaked for thirty minutes in clean water: 12kg per day where the horse is the correct weight, 9kg per day if the horse is overweight, and ad lib where the horse is underweight. Where access to hay is not ad lib, ensure that the supply of forage is reasonably constant by using haynets with smaller holes or double netting.
- Feed one bucket of feed per day containing the following:
  – Soaked, unmollassed sugar beet, 0.5kg dry weight
  – 10g of pharmaceutical grade magnesium oxide (for example, a 50ml scoop of light MagOx)
  – 25g of table salt (roughly a 20ml scoop)
  – Where the horse is underweight, consider adding micronized linseed (up to 150g)
  – Where there is any suspicion of gastric pain, or where significant doses of phenylbutazone or similar are being used, consider supporting digestion with a supplement such as Hilton Herbs GastriX
  – Dried mint can be safely used to improve palatability for fussy eaters.
- All other feed supplements should be stopped until the laminitis is fully controlled.
- Monitor weight carefully using a weight tape weekly to ensure that overweight horses are losing weight at an appropriate rate, and that horses with a correct weight, or that are underweight, are not losing weight (putting weight on an underweight horse in the immediate aftermath of acute laminitis is not usually advisable).

This diet can be safely fed for a few weeks whilst the laminitis is stabilized. With longer term use, consideration would need to be given as to whether soaking the hay might result in nutritional deficiencies, when additional supplementation would be needed.

Where this approach does not fully control laminitis, the diet may need to be further tweaked to try to address specific underlying health issues that may be contributing to the laminitis, as outlined earlier

in this chapter.

Once the underlying laminitis is well controlled, a more normal diet can be introduced in stages, but the magnesium and salt would normally be retained unless provided by some other feed ingredient.

11 Case Studies

The following case studies are based on real examples from the author's practice. Some of the horses' names have been changed to protect client confidentiality, and some minor details from older cases have been changed to reflect current best practice.

LADY

Lady was an elderly cob mare weighing around 400kg. She was originally bought by her current owner for use in carriage driving. At the point of purchase, she was shod all round and appeared sound, although she had a history of chronic laminitis. A year later the owner decided to remove the shoes and she was lame from that point on. Unfortunately, her feet were trimmed by an inexperienced hoof trimmer who apparently did not recognize the complexity of the case and lowered the heels too far without first checking the flexor tendons.

Lady came under the author's care as a veterinary referral some eighteen months after her shoes were removed, and by that point it was clear that the mare had fairly severe chronic laminitis affecting all four feet, with the fronts significantly worse than the hinds. The owner was dedicated to the horse and was doing everything possible to try to resolve the lameness. The owner's vet was involved throughout the case.

Lady was mostly kept in a rubber-matted yard and stable, but was allowed two hours turnout at grass per day with a grazing muzzle. She was being fed timothy haylage and a small amount of Spillers Happy Hoof®. The owner was concerned about her legs swelling up, and so was bandaging them at night to try to reduce the swelling.

All four feet showed growth rings that diverged at the heel, with evidence of significant recent rotation. There was a large amount of laminar wedge visible on the front feet and a very small amount on the hinds. The hoof walls of all feet were pink with bruising, some of which was in clear rings suggesting more acute laminitis attacks, and some was more diffuse suggesting lower levels of chronic laminitis.

At walk, Lady showed an excessive heel-first landing on all but the left fore, which showed a slight toe-first landing. She had mild raised pulses in all four feet, with the front pulses slightly stronger than the hinds.

The toe-first landing on the left fore raised concerns about tendon length, and a tendon length check confirmed that Lady was unable get the heels lower than the toe on that foot despite the heels already being too long – a condition known as club foot. The tendons on the other three legs appeared normal.

With the exception of the left fore, Lady was given a basic trim to put the pedal bones at the correct angle to the ground and also to bring the breakover back on each foot. On the left fore, no further heel material was removed. This initial trim was fairly conservative as there wasn't much spare hoof to work with.

Lady was fitted with a set of removable hoof boots, with 4lb grade, 1in thick, closed-cell foam pads fitted in the bottoms. These were to be used for exercise and turnout and also on the yard if she was not fully comfortable.

Some minor changes were made to the diet. The haylage was swapped to hay obtained from a species-rich meadow and soaked for one hour, 8g of magnesium oxide and 20g of table salt were added to the feed.

Lady responded to the dietary changes with a significant decrease in the rate of new rotation, but the underlying laminitis still appeared to be present at a low level, as evidenced by slightly raised pulses. It then transpired that the owner was feeding a significant quantity of fruit as treats, having read in a book that this might help laminitis. Once the fruit was removed from the diet, the laminitis almost completely disappeared, and Lady became sound apart from the effects of the club foot.

Fig. 113 Lady's front feet showing worsening club foot on the left fore.

Some months later Lady developed a foot abscess in the left fore, which resulted in a small amount of supporting limb laminitis on the right fore. In the aftermath of this abscess, Lady's club foot became significantly worse.

Within a few months, the decision was taken to sever the deep digital flexor tendon and remove a section – a surgical approach which can buy some improvement in such cases, though at the expense of an alteration in gait. The surgery initially worked well, but within a year the club foot had returned and Lady was starting to struggle. The owner then took the difficult decision to opt for euthanasia.

The owner very generously donated Lady's front legs for research, and an autopsy was carried out by the original referring vet and the author. This autopsy provided two surprising insights into the case.

The original cause of the club foot on the left fore turned out to be an adhesion between the deep digital flexor tendon and its surrounding sheath, which was preventing the tendon from moving through its full normal range. Whenever this adhesion was brought under tension (for example, by lowering the heels excessively) the adhesion would have become stronger and tighter. Severing the tendon in the mid-cannon region should have released the lower part of the tendon, allowing more normal movement of the coffin joint (albeit without the ability to move it under voluntary control). The surgery had ultimately failed because the two cut ends of the tendon had healed back together again. The new tendon tissue was of poor quality and had also adhered to the tendon sheath, so restricting movement of the lower part of the tendon again. This helps to explain why severing the tendon in club-foot cases often fails to provide a long-term solution.

Fig. 112 LEFT: ***Lady's right forefoot at the start of the case study.*** CENTRE: ***After four months.*** RIGHT: ***After fifteen months.***

The other surprising outcome was that there was no significant evidence of laminitis on either front foot. Indeed the quality of the dermal laminae and other coria was extremely good. Although this particular horse did not make a satisfactory recovery because of the complicating factor of the club foot, she did provide valuable evidence that appropriate dietary interventions can prevent further laminitis, and also allow the various coria to make a good recovery.

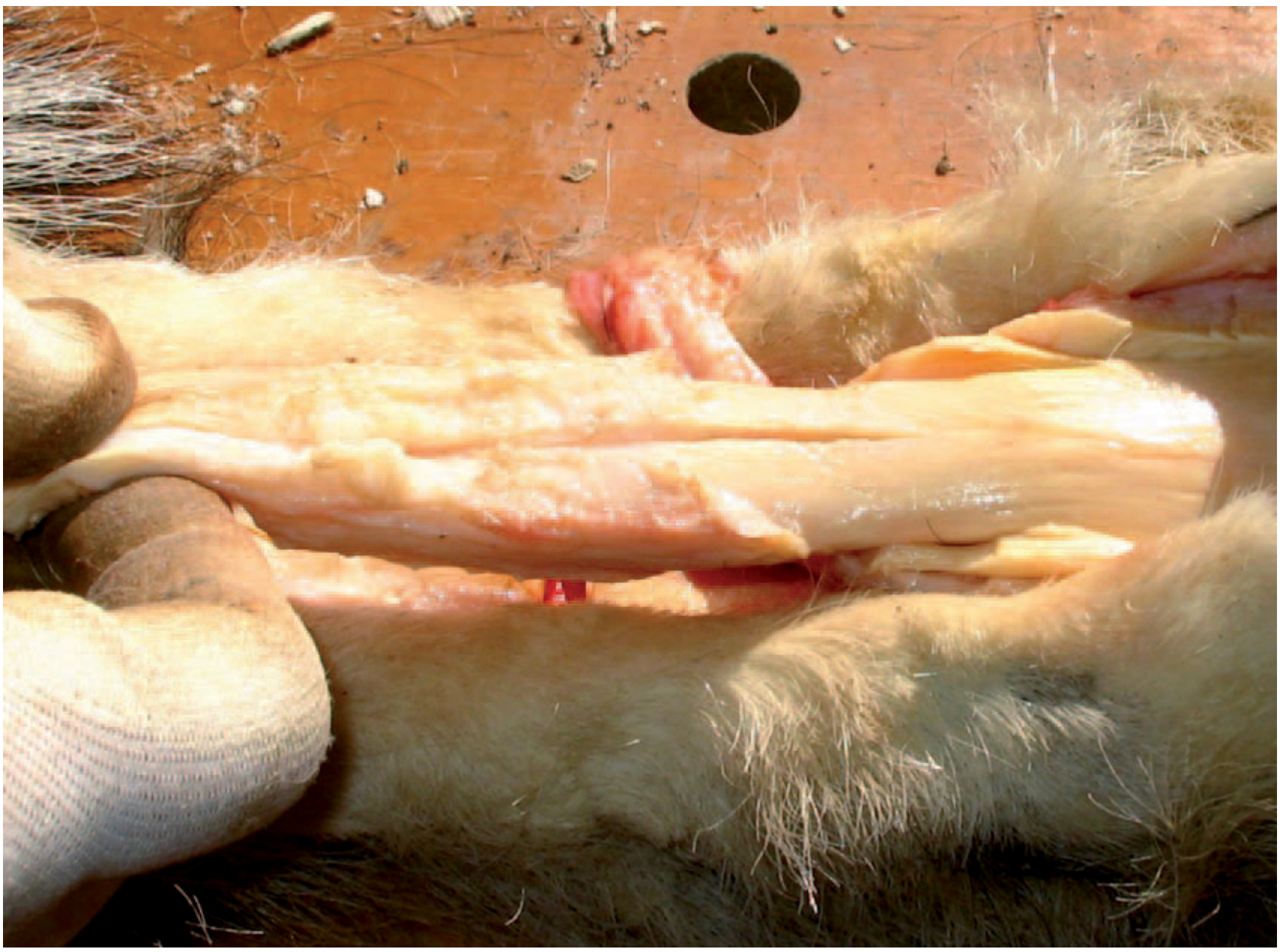


Fig. 114 A section of the deep digital flexor tendon from Lady's right foreleg showing new tendon material joining the severed ends. The longitudinal cut just visible along the tendon was made as part of the autopsy.

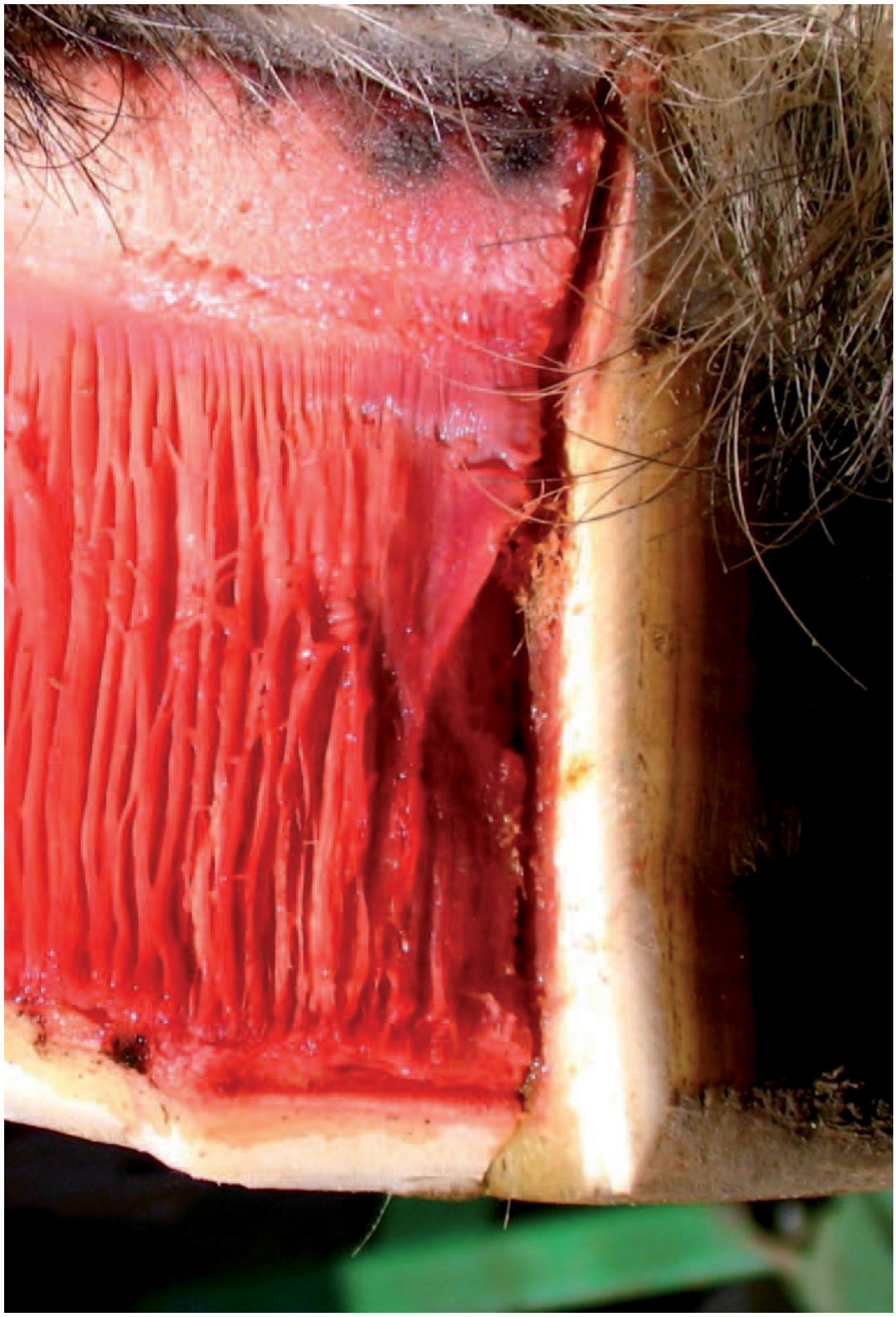


Fig. 115 A close-up of the dermal laminae on Lady's left fore. The hoof wall has been cut down the centre line of the toe to facilitate removal of part of the hoof wall, resulting in minor damage to the laminae immediately adjacent to the cut. Otherwise the laminae are in good condition with a good supply of blood and no evidence of the laminae stretching or tearing. The slightly thin sole is the result of excessive toe wear caused by the club foot.

PERCY

Percy is a Percheron/Lusitano cross who, at the start of the case study, was fourteen years old and had been with his present owner for one year. He had been shod since being backed and had a long history of severe quarter cracks to the outside quarter of each front foot. Various vets had been involved in the case and several farriers had attempted to resolve the problem cracks with a variety of remedial shoeing approaches, but none had managed to achieve a sustainable improvement. The owner had removed the shoes in desperation but had then realized that she needed professional help.

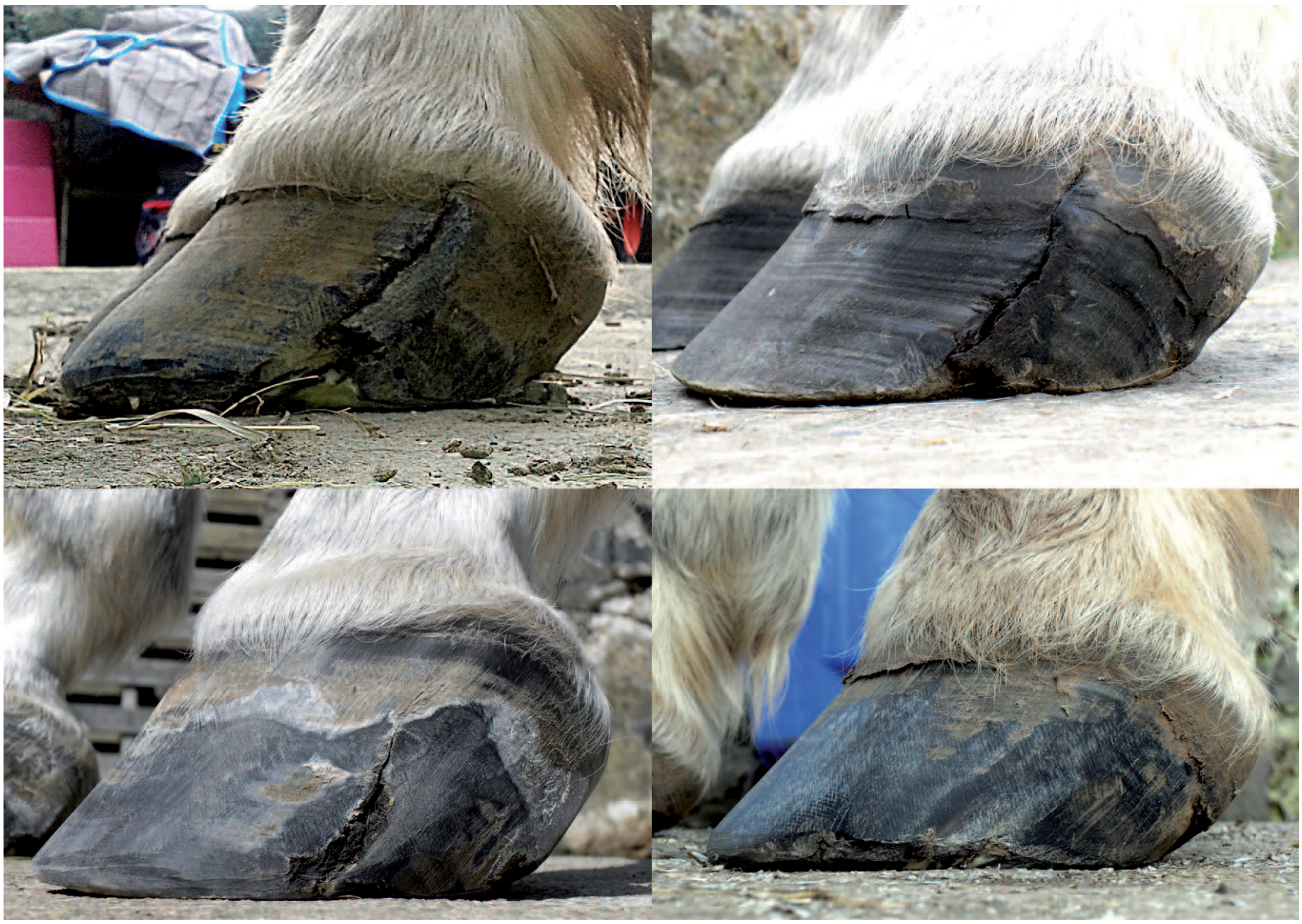


Fig. 116 TOP LEFT: ***A serious quarter crack resulting from a combination of LGL, a significant conformation defect, and rot.*** TOP RIGHT: ***The same foot after two months on a modified diet and with regular use of a topical disinfectant.*** BOTTOM LEFT: ***Five months later.*** BOTTOM RIGHT: ***Eleven months later. Interesting transient changes to the shape of the coronary band and the overall length of the hoof can be seen during the recovery period.***

Percy was living out on lush grass and being fed a bucket feed of Happy Hoof® and Super-Codlivine®. He was also slightly overweight with a large, firm crest. He had clear conformational issues with his front legs, which resulted in him landing strongly to the outside heel on both front feet. This was causing increased wall growth on the outside quarters, which was in turn putting mechanical strain on these areas. His feet were poorly balanced, although it wasn't clear how much of this was due to the previous shoeing, and how much was the result of uneven growth. The cracks were sufficiently severe to allow the outside heel of each foot to move slightly in relation to the rest of the foot. There was also a small quantity of dried blood at the top of one of the cracks, suggesting that this instability was causing damage to the coronary band at the top of the crack.

At first sight, Percy's problems appeared to be solely down to conformation and foot balance. However, the degree of flaring, both in the toe area and on the inside quarters, was more than would have been expected given his conformational and balance issues, suggesting that LGL might be playing at least a small part in the problem. There were also significant traces of blood in the white line on each forefoot. The working theory was that the presence of mild LGL combined with the uneven landing was causing the inside quarter to flare excessively, and that this was then putting more mechanical strain on the outside quarter, perpetuating the crack.

Percy's feet were trimmed regularly to achieve an appropriate balance (accepting that he would not be able to land evenly between the heels). His grazing was restricted using a strip-grazing approach, and his hay soaked in an attempt to reduce his weight slightly. The Super-Codlivine® was removed from his feed and replaced with appropriate quantities of magnesium oxide and salt. His owner was

advised to check his weight with a weight tape weekly, and to aim for a slow but steady rate of weight loss.

Percy's feet were soaked daily in a solution of Milton Fluid® (one capful of Milton Fluid to five litres of water) to try to slow the rotting of the damaged horn and to give any newly forming horn at the tops of the cracks a chance to grow down. Removable hoof boots were used for turnout so as to provide a degree of mechanical stability across the cracks whilst allowing some degree of normal distortion of the hoof capsule.

The initial improvement was not cosmetically obvious, but the cracks stabilized within a few weeks, removing the independent movement of the outside heels. At that point the cosmetic appearance of the cracks began to improve, with the cracks knitting together from the top of the wall. Over a period of eleven months, both cracks grew out entirely, leaving very little evidence of where they had been. The overall shape of the front hooves is still far from ideal, but they are significantly improved from their original state. Percy is now the correct weight and no longer has a crest. The boots and disinfectant have been dispensed with, and the cracks have not recurred.

This case shows that even pathologies that at first sight have nothing to do with laminitis can sometimes have LGL as a significant contributory factor. The LGL in this case resulted in excessive flaring which created mechanical strain on already damaged walls. The LGL also resulted in significant blood products being released into the hoof wall which allowed rot to spread away from the cracks, further destabilizing them. It is unlikely that such rapid improvements would have been gained without the dietary interventions.

DYLAN

Dylan is a 14.1hh Welsh Section D cob who was aged ten at the start of this study. His owner at that time had owned him for around one year and wasn't aware of a previous history of laminitis. He was being used for general hacking and fun rides, and had been shod in front intermittently. He had an acute laminitis attack a few weeks prior to the author's first visit. This had been dealt with by the vet and farrier, but he was still obviously sore on the front feet and slightly sore on the hinds. He was shod in front only, and these shoes were removed to aid in assessment.

With the shoes taken off, Dylan showed an obvious excessive heel-first landing on all four feet in walk. He was clearly sore in walk, so no attempt was made to trot him up. There was no obvious damage to the white line on any foot, but there was a marked sudden change of angle in the front wall of both front feet, and to a lesser extent on the hinds, suggestive of an acute laminitis attack within the previous three months. Dylan was kept at grass and not given any form of bucket feed.

Fig. 117 Dylan's right forefoot at the start of the case study. Note the change in angle near the top of the hoof wall at the toe in the side-on photograph. The bottom of the wall at the toe had been rasped back, making this change of angle somewhat less obvious

Fig.118 Dylan's right forefoot around five years later. The foot is not perfect but is highly functional.

Fig. 119 Dylan with his rider Benjamin showing how well a PPID case can perform when correctly managed.

After an initial set-up trim, Dylan was kept off hard ground, turned out 24/7 in a starvation paddock, and given a daily bucket feed of Hi-Fi and Speedibeet with 10g of magnesium oxide and 20g of salt. Within three months there was a marked improvement in his feet, and by the time five months had passed, he was being ridden five to ten miles per week on roads without problems.

Dylan was then loaned to a young boy and continued to do well apart from transient periods of being footsore, typically in spring and autumn. During these problem periods he was ridden in front removable hoof boots, which allowed him to work without pain. The pattern of seasonal soreness led to a suspicion around two years after the shoes were taken off that he might have either EMS or PPID. Blood tests showed that his ACTH was a little above the reference range, which resulted in a diagnosis of PPID. He was then put on 0.5mg daily of Prascend to treat the PPID. From that point onwards, he has been fully sound on all surfaces without boots or shoes. He is regularly hacked on roads, and has had a very successful career in show jumping.

LUNA

Luna is a thoroughbred ex-racehorse weighing around 500kg, being used for light hacking. At the start of the study, she was six years old. She lived out in the summer but was stabled overnight in winter. The pasture was ex-dairy pasture and hence was quite rich, but the area available to her was restricted to prevent weight gain. Aside from grass, she was given ad-lib hay when stabled and a daily bucket feed of molassed sugar beet and coarse mix. She was an ideal weight and showed no signs of abnormal fat distribution.

She had originally been shod, but her shoes had been taken off nine months earlier and her feet regularly trimmed by a farrier. At the start of this study, she hadn't been trimmed for some weeks. Her feet showed significant flaring with a sudden change of angle part way down the toe, but there was no laminar wedge, and the white line was tight and normal. She had mild thrush and moderate white line disease on all four feet.

In walk on a concrete surface, Luna walked with a very slightly excessive heel-first landing on the front feet, but landed correctly on the hinds. Her trot on concrete was cautious and lacked elevation, whereas on a soft surface, her trot was expressive.

The sudden change of angle in the front wall combined with the absence of laminar wedge was strongly suggestive of an acute low-grade laminitis attack perhaps five months earlier. The overall quality of the hoof (especially growth rings that diverged at the heel and significant horn infections) and the slightly abnormal landing and tender feet also suggested that there was a lower level of chronic low-grade laminitis present.

After an initial set-up trim, her level of discomfort was no worse. She appeared fully sound and confident in trot with Old Mac boots fitted to her front feet. Booting the hind feet appeared to make little or no difference. The owner was then advised to exercise her daily, but to use front boots for any work on hard ground.

The owner was also asked to replace Luna's current bucket feed with a daily feed consisting of small quantities of Speedibeet® and Coolstance Copra®, with 10g of magnesium oxide and 25g of table salt added.

Within three months, Luna was landing correctly on all four feet and was fully sound in trot on smooth tarmac or concrete. By seven months, all hooves had attained a much more correct shape, and the concavity of the front feet had improved significantly. The horn infections had also more or less disappeared. From that point, Luna was hacked without the boots and remained sound on the roads.

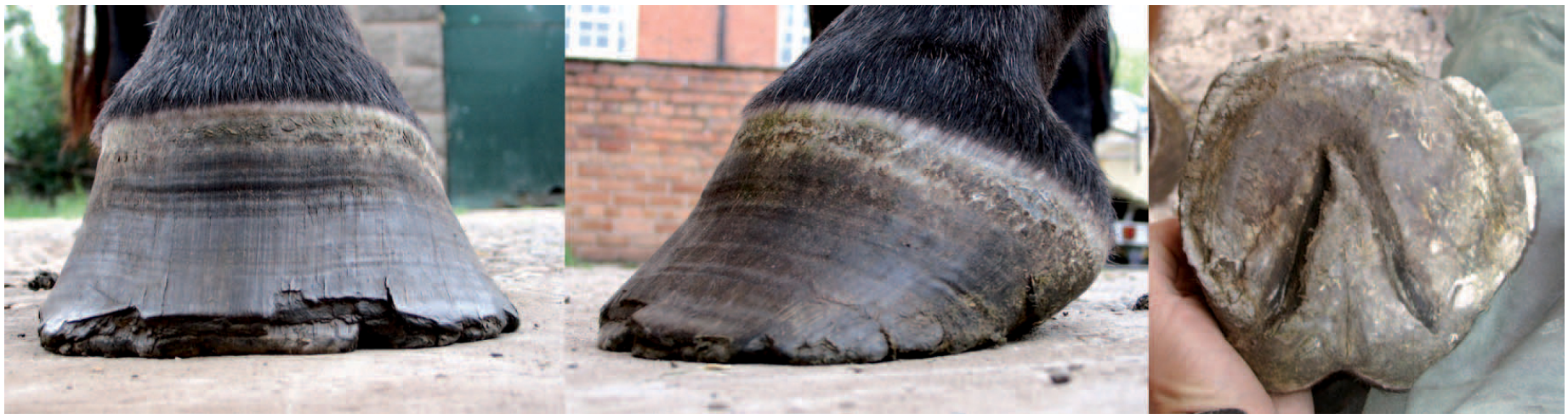


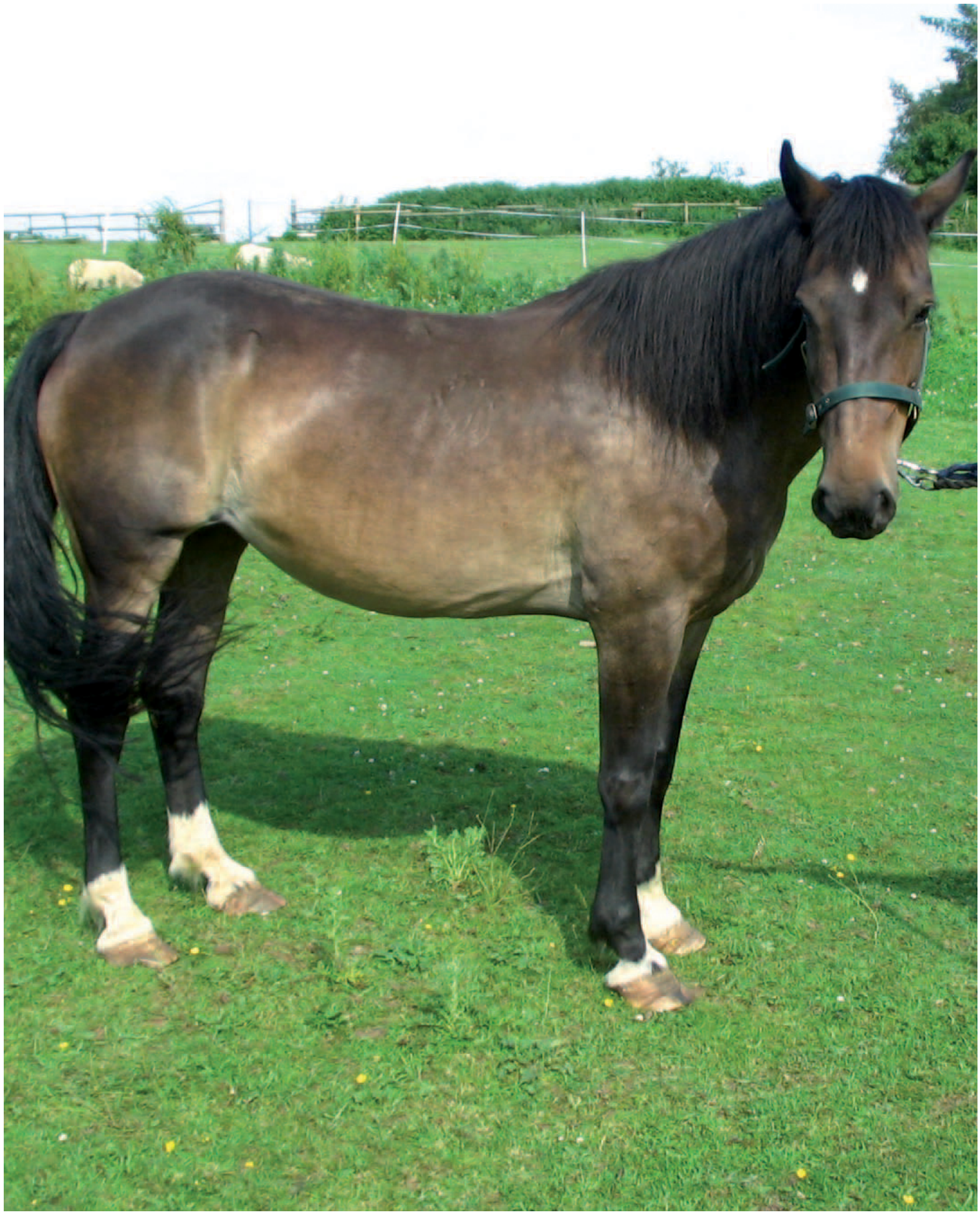
Fig. 120 Luna's left fore foot at the start of the case study.

Fig. 121 Luna's left fore foot seven months after diet changes.

Luna's case shows that sometimes fairly minor dietary fixes (in this case, reducing the sugar and starch content of the bucket feed and adding magnesium and salt) can result in significant improvements in mild LGL cases.

BRIDIE

Bridie was a Welsh Cob mare; at the start of this case study she was thirteen years old. She was kept as a pet, and wasn't ridden or routinely handled. She had never been shod, and had no history of foot problems, despite living feral without any routine hoofcare. She had had one foal at the age of four, and had been hobbled by a 'professional' horse trainer to enable the foal to suckle. This had resulted in her developing severe trust issues in relation to her hind feet.



Bridie, six months after her initial laminitis attack. By this point she stood square and was fully sound.

She developed laminitis suddenly, most likely as a result of accidentally being fed a large quantity of sheep feed whilst the owner was away. She was seen immediately by a vet, who provided pain relief and suggested box rest on a deep bed of shavings with frog support pads taped to her front feet. She was given Danilon® for pain relief. Unfortunately, because she had never been stabled before, she became severely distressed, and after two weeks damaged the stable so seriously that she had to be released back into the field in a limited area. The laminitis gradually became worse.

Bridie came under the author's care as a veterinary referral six weeks after the initial attack. At this point the pedal bones of both front feet appeared to have rotated and sunk to a significant degree. There were very strong depressions at the coronary bands, and the soles gave easily to thumb pressure (suggesting that they were fairly thin) on both front feet. She was clearly in a lot of pain in her front feet, and struggled to pick them up. It was agreed that x-rays would be useful to confirm the extent of the damage. Because of her history it proved impossible to pick up her hind feet to assess them, but

based on how she was moving, the assumption was made that if any laminitis was present in the hinds, it was less serious than in the fronts.

As an interim measure whilst awaiting x-rays she was given a cautious trim (mostly lowering the heels to bring the pedal bone closer to the correct orientation with the ground). She was then fitted with therapeutic pads cut out under the tips of the pedal bones and held on with ‘Old Mac’ hoof boots. This is the author’s standard approach where there is considered to be a risk of pedal bone penetration. In addition to the fairly dry haylage she was already being given, her diet was modified (she had been fed coarse mix) to a mixture of Speedibeet and Hifi Lite, with supplementary magnesium oxide and salt. The Danilon® was continued. The owner was advised to keep her fenced into the corner of the field, with her companion horses in the rest of the field to reduce stress. Bridie was immediately far more comfortable with the boots and pads fitted, and the size of her fenced off area had to be reduced to prevent her from trotting around.

For various reasons it took nearly two weeks for the x-rays to be taken, by which point Bridie had again deteriorated significantly. Whilst taking the x-rays, the vet became concerned that the soles were now paper thin. There also appeared to be evidence of abscessing on both front feet. Whilst examining the left fore, the abscess burst through the sole, revealing what the vet described as a prolapsed solar corium underneath. He sprayed the feet with Terramycin as an antiseptic and placed some wadding inside the boot to protect the damaged area. A joint visit with the author was then arranged for the following day.

By the next day, the sole on the right fore had also burst. The x-rays showed a high degree of sinker and rotation on both front feet. The tips of the pedal bones were very close to the external surfaces of the soles, and there were ‘gas pockets’ suggestive of large collections of pus under-running both soles and the frog on the left fore. The only saving grace in this extremely serious situation was that, as the sole of each foot had gradually thinned to nothing over the previous two weeks, it had bulged. This would have allowed the pressure from the abscess to drop, in turn allowing the solar corium to produce a small amount of poor quality horn again. This meant that, at the point where the abscess blew, there was a very thin skin of horn covering the solar corium. Hence, despite the original sole being holed, the corium was not directly exposed. In the author’s experience, this is usually the case where a pedal bone penetrates the sole. Where there is no covering of horn over the exposed corium, it suggests that circulation in the toe area has been lost more permanently and there is little hope of the horse recovering.

Fig. 122 Bridie's left fore sole eight weeks after the initial attack. The abscess has blown a large hole in the paper-thin sole. Through the hole can be seen the rim of the pedal bone, covered in corium, and a very thin layer of newly produced horn (yellow in colour). Behind this is a large quantity of congealed blood produced by the damaged corium before healing started. (The green discoloration is antiseptic spray applied by the vet on the previous day.)

Fig. 123 Bridie's left fore sole during recovery. TOP LEFT: ***Twelve weeks after the initial attack (and four weeks after the original sole was holed).*** TOP RIGHT: ***Twenty-five weeks post attack.*** BOTTOM LEFT: ***Thirty-six weeks post attack.*** BOTTOM RIGHT: ***Forty-seven weeks post attack.***

A treatment plan was then agreed with the vet. The guiding principle was that if Bridie couldn't be kept reasonably comfortable, then it wouldn't be ethical to carry on. Given the extent of damage to the solar corium, it was clear that there was little structure forward of the centre of the foot that could support weight without doing more damage. However, the padding system already in place provided appropriate support to the foot (supporting the front half of the foot only via the wall). The fact that this system had been in place already for two weeks may well have contributed to the good quality of new sole growth under the tip of the pedal bone. As such it was decided to continue the booting and padding arrangements already in place.

The other main issue was infection control. With the vet's agreement, both front feet were soaked in CleanTrax®, a proprietary penetrating disinfectant originally developed for orthopaedic work but now marketed for serious equine foot infections. The damaged areas of sole were then dressed with non-adherent dressings, held in place with cohesive bandage. It is important to use non-adherent dressings in this situation to prevent any fragile, newly formed tissue from adhering to the dressing and hence

being damaged when the dressing is removed. Each foot was then wrapped in a plastic bag (to keep the dressings clean and dry), and placed back in the Old Mac boots. Bridie remained fairly comfortable in her boots and pads, despite the degree of damage to her feet. It was this level of comfort that convinced the author and the vet that it was ethical at this point to continue trying to recover her feet.

Bridie's owner was advised to continue keeping her in the small fenced-off area of the field, and to continue with the same diet. The dressings were left on for ten days in the hope that leaving things well alone would minimize the risk of reinfection. They were then reapplied for a further ten days. After that, the dressings were removed and only the pads and boots were used.

After a few weeks there were some problems with the boots rubbing Bridie's heels. This could have been a serious issue, given that Bridie was not yet recovered enough to cope without the boots and pads. The owner improvised by sliding the sleeves of a worn-out cashmere jumper over her feet to act as socks, and this allowed the heel bulbs to recover.

As Bridie recovered and became more and more comfortable, it became clear that she was exhibiting 'stressy' behaviour consistent with stomach pain. This was contributing to the behaviour issues that prevented it being possible to examine her hind feet. Digestive pain, often associated with equine gastric ulcer syndrome, is common in horses with severe laminitis – possibly because of the stress of the foot pain, as well as the use of pain relief drugs, which are known to cause acidity in the stomach. It was felt that using an endoscope to examine her for the possibility of gastric ulcers would be problematic given her lack of trust, so instead, she was trialled with Hilton Herbs® GastriX, a product aimed at supporting digestion in stressful situations. The Danilon® was also phased out at this point, as it was not felt to be necessary any more.

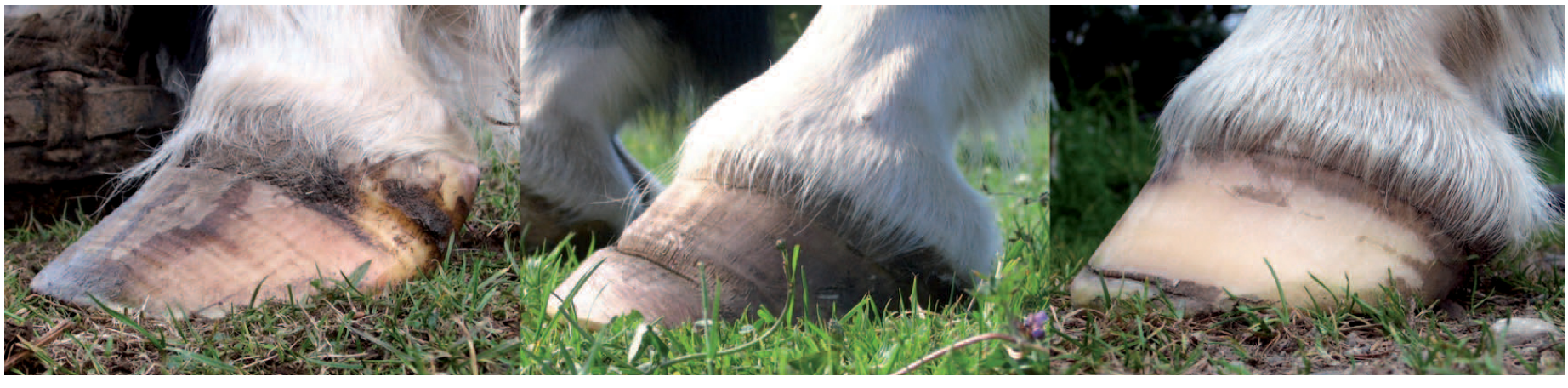


Fig. 124 A side-on view of Bridie's left fore during recovery. LEFT: ***Twelve weeks after the initial attack.*** CENTRE: ***Thirty-six weeks post attack.*** RIGHT: ***Forty-seven weeks post attack.***

The owner noticed a marked improvement in Bridie's behaviour within a couple of weeks. At the next visit, the author was able to pick up and trim her hind feet, albeit with some difficulty. It was not possible to safely photograph the hind feet, but they showed evidence of rotation and sinker, albeit milder than the front feet.

By twenty-five weeks after the initial attack, Bridie's soles had healed well enough that the boots and pads were no longer needed. From that point, Bridie was fully sound barefoot, even though the cosmetic damage to the feet took significantly longer to grow out.

Bridie went on to make a full recovery. After twelve months from the initial attack, it wasn't possible to tell from her feet that she had ever had laminitis. She was also by that point sound on all surfaces, including a stone track. She clearly had no underlying medical conditions pre-disposing her to laminitis, and her one attack was caused by a feeding mistake and hence was an isolated incident. Her case is a good example of how well equine feet can recover from even the most extreme damage so long as the underlying laminitis is fully controlled. Bridie went on to live a happy life without any signs of laminitis for a further seven years before dying of an unrelated illness.

Conclusion

Laminitis is an extremely complex family of conditions, and whilst we still only have a small proportion of the pieces of the jigsaw, tantalizing glimpses are starting to emerge of how those pieces might fit together. We appear to be reaching a tipping point, where research has the potential to accelerate and provide real answers in a fairly short timeframe – but for that to happen, we need to be prepared to let go of outdated ideas and attitudes to this condition, and open our minds.

In terms of the biomechanics of the foot itself, letting go of the human-centred view and instead reframing the problem from the horse's viewpoint allows for a far better understanding of both how the foot fails and the potential ways in which it can heal. When it comes to the underlying causes, we need to see laminitis as a manifestation of a range of underlying health problems, rather than a disease in its own right.

Perhaps the thing that we have most lost sight of is that the horse, like any animal, has an innate ability to heal itself – an ability that evolution has perfected over hundreds of thousands of years. If we continue to take the typical human approach of thinking that we can do better than nature, then we are likely doomed to failure. A more productive attitude is to see our role as attempting to create an environment that optimizes the horse's own ability to heal itself. That might be by creating a mechanical distribution of pressure on the foot that allows for healing, by creating a space in which the horse can promote recovery through appropriate levels of movement, and by creating a diet that is sufficiently close to the diet the horse evolved to thrive on. Whatever solutions we attempt to put in place, we need to consider how evolution has equipped the horse to solve its own problems, and try to use that to our advantage.

What is becoming increasingly clear is that, once a horse-centred approach is taken, the mechanical recovery of the foot is the easier part of the problem, the difficult part being understanding and controlling the underlying causes of the laminitis. Much more research is needed, particularly into the mechanisms by which laminitis occurs in the first place, and that research needs to focus on the whole horse, not just on the laminae.

The recent identification of LGL has the potential to be a tipping point in research. On the one hand, the possibility of LGL means that existing research needs to be carefully re-evaluated. Where research has compared laminitic horses with non-laminitic control horses, the possibility should be considered that the control horses were not entirely laminitis free after all. This might mean that some existing research is no longer valid, and other research may need to be reinterpreted.

On the other hand, LGL has the potential to be an invaluable research tool in the future. Up to now, much research has been focused on deliberately inducing severe acute laminitis and then studying the outcome for the horse. Quite apart from the ethical problems with this approach, such research risks missing subtle aspects of the onset and development of the disease. And, of course, this research is typically performed using the carbohydrate overload model, and hence it is of less relevance to the pasture-induced cases predominantly seen in the UK. In contrast, LGL cases can be easily studied in everyday practice. New interventions can be trialled in the LGL population, with far fewer ethical challenges and at a far larger scale. A lot can even be learnt from just observing LGL cases without targeting specific interventions at them. 'Big data' may finally become the tool that allows us to spot patterns in the complex variability of the disease processes we group together under the heading of laminitis.

We are also starting to see more cooperation between different groups and specialisms when it comes to laminitis. With individual cases, the goal of a vet-led, multi-disciplinary team that includes non-veterinary experts such as equine podiatrists, osteopaths and dentists, is just starting to become a

reality. The progress that can be made in even the most complex cases where such an approach is used is hugely encouraging. It is to be hoped that this new era of cooperation will now spill over into the area of laminitis research. Perhaps then we can truly start to understand this distressing condition, and find a way to reduce the suffering it currently causes.

Esme (the horse that started the author researching into low grade laminitis) being hacked out by the author's son, Lucas, with hoof boots on all four feet.

Abbreviations Used in This Book

ACP acetylpromazine

ACTH adrenocorticotropic hormone

AVA arterio-venous anastomosis

CDET common digital extensor tendon

DDFT deep digital flexor tendon

DIP joint distal interphalangeal joint = coffin joint

ECIR Equine Cushing's and Insulin Resistance Group

EMS equine metabolic syndrome

EP equine podiatrist

EPA Equine Podiatry Association

EPSPS 5-enolpyruvylshikimate-3-phosphate (EPSP) synthase

ESCs ethanol-soluble carbohydrates

IBS irritable bowel syndrome

LDET long digital extensor tendon

LGL low-grade laminitis

MMP matrix metalloproteinase

MOS mannan oligosaccharides

NPK nitrogen/potassium/phosphate fertilizer

NSAID non-steroidal anti-inflammatory drug

P1 first phalanx = proximal phalanx = long pastern bone

P2 second phalanx = middle phalanx = short pastern bone

P3 third phalanx = distal phalanx = coffin bone = pedal bone

pH potential of hydrogen – a measure of acidity or alkalinity

PIP joint proximal interphalangeal joint = pastern joint

POMC pro-opiomelanocortin

PPID pituitary pars intermedia dysfunction

SDFT superficial digital flexor tendon

SIBO small intestine bacterial overgrowth

TRH thyrotropin-releasing hormone

VFA volatile fatty acid

WSCs water-soluble carbohydrates

Glossary

abscess A localized infection that encapsulates away from the main blood supply.
acute Happening over a short period of time.
anastomosis A port allowing connectivity between two adjacent structures.
avascular Containing no blood vessels.
basal cells The single-cell thick layer of cells on the surface of the basement membrane, responsible for creating external skin/horn.
basement membrane (also known as the basal membrane) Membrane that sits between the dermal and epidermal layers of the skin (including the hoof).
bursa A sac around a joint that contains synovial fluid.
caecum A blind-ended fermentation chamber between the small intestine and the colon.
central sulcus The depression in the centre of the back portion of the frog.
chronic Happening over an extended period of time.
circumflex artery (more accurately, **circumflex artery of the sole**) Artery situated around the bottom of the pedal bone, which supplies circulation to the solar corium.
club foot A condition in which the foot is too upright as a result of damage to the deep digital flexor tendon.
collateral ligaments Ligaments on each side of a joint that hold the joint together.
colon The final part of the digestive tract before the rectum.
corium (plural **coria**) Vascular tissue responsible for creating a part of the horny hoof.
corn Area of bruising in the sole or frog that results in an artificial thickening of that area.
cryotherapy Using cold (especially the use of ice) to treat a condition.
Cushing's disease A tumour in the pituitary gland that produces ATCH and hence raises cortisol levels.
Cushing's syndrome A condition in which cortisol levels are excessive.
dermal layer (dermis) Vascular layer of the skin.
desmosome A structure that creates a bond between two adjacent horn or skin cells.
digit Equivalent to the finger or toe of a human, so relating to the pastern and foot.
digital Related to the digit.
digital cushion The firm pad between the heels that aids shock absorption when the foot impacts a hard surface.
disaccharide A carbohydrate consisting of two monosaccharide molecules joined together.
distal Towards the end of the limb.
dorsal Towards the front of the limb.
duodenum The first part of the small intestine.
endocrinopathic Relating to failures in the endocrine (hormone) system.
endotoxaemia Poisoning of the blood by endotoxins.
endotoxin Toxin originating from the cell membrane of a bacterium.
epidermal layer (epidermis) Avascular layer of the skin.
exotoxin Toxin originating from the contents of a bacterium cell.
extension Bending of the leg in a forward direction.
flexion Bending of the leg in a backward direction.
foregut The stomach and small intestine.
founder A term relating to rotation or sinker.
fructan A polymer carbohydrate made up of multiple fructose molecules.

gas pocket A region on a radiograph (x-ray) that is less dense, possibly indicating the presence of gas or fluid.
glycaemic index A measure of the peak level of glucose released into the bloodstream as a food is digested.
haematoma A blood blister.
hemidesmosome A structure that creates a bond between a horn/skin cell and the basement membrane.
hindgut The caecum and colon.
hinny The offspring of a female donkey and a male horse.
hirsutism The presence of excessively long hair (usually also associated with a failure in normal seasonal coat shedding).
hypothalamus A glandular structure at the base of the brain responsible for sensing the levels of chemicals in the blood, and for providing an interface between the brain and the endocrine system.
ileum The final section of the small intestine.
impar ligament Ligament attaching the navicular bone to the pedal bone.
internal arch Arch formed by the pedal bone and the lateral cartilages (also with help from the digital cushion, coronary band and sensitive frog).
ischemia Loss of blood circulation to a tissue.
jejunum The middle section of the small intestine.
keratinocyte An epidermal (skin) cell capable of producing keratin.
keratoma A tumour (usually benign) of the corium, which produces keratinocytes abnormally.
laminar wedge A diseased, wedge-shaped tissue produced by the laminae in response to rotation.
lateral cartilages The cartilaginous structures that provide the framework that supports the heels.
latero-medial From one side to the other.
leaky gut syndrome A condition in which the lining of the gut becomes more permeable than normal, allowing toxins from the gut to pass into the bloodstream.
lignin A structural carbohydrate that gives plants mechanical strength.
long pastern The first phalanx.
matrix metalloproteinase An enzyme capable of breaking down the proteins that bind cells together in connective tissue.
metabolism The chemical processes within the body (not within the gut) that break down food and manufacture the resources needed for life.
monosaccharide A simple sugar (one that cannot be broken down into smaller sugars).
mule The offspring of a male donkey and a female horse.
oedema The build-up of excessive fluid in a tissue, resulting in swelling.
palmar Towards the back of the limb.
papillae Spikes on the surface of a corium that increase its surface area and hence the rate at which horn is produced.
perfusion The passage of blood through blood vessels.
periople The layer of skin covering the top of the hoof wall, which allows the wall to harden after production at the coronary corium.
phalanx (plural **phalanges**) One of the lowest three main bones in the leg.
phenylbutazone (bute) A non-steroidal anti-inflammatory drug (painkiller).
pituitary Gland at the base of the brain responsible for controlling the endocrine system.
polysaccharide Polymer carbohydrate formed from multiple monosaccharides.
prebiotic Food substance that helps to promote the growth of beneficial bacteria in the gut.
probiotic Food that supplies additional bacteria to assist the gut flora.
proximal Away from the end of the limb.
radiograph Image taken using x-rays.
remodelling Change in the shape of a tissue (especially bone) in response to mechanical pressure.

repair horn Soft horn (often incorporating blood products) that is rapidly produced by a corium in response to injury.
reperfusion The return of circulation to a tissue that has been starved of blood.
rotation Angular deviation of the alignment of the front surface of the hoof wall from the underlying surface of the pedal bone.
septicaemia Widespread dissemination of bacterial infection via the bloodstream.
serum Portion of the blood after all blood cells and clotting factors have been removed.
short pastern Middle phalanx.
sinker Vertical displacement of the pedal bone (especially the front portion) downwards in relation to the hoof capsule.
squamous cell Flattened cell in the outermost layer of skin/horn.
sub-solar Relating to the tissue immediately underneath the solar horn (the solar corium).
supra-orbital fossa Natural depression just above a horse's eye.
synovial fluid Fluid that lubricates joints.
terminal arch Artery situated in the middle of the pedal bone.
vascular Containing blood vessels.
vasoconstriction Contraction of blood vessels so as to reduce blood flow.
vasodilation Expansion of blood vessels so as to increase blood flow.
venogram Radiograph (x-ray) taken with a contrast dye injected into the veins so that they are easily visible.
villi Finger-like protrusions on the surface of the gut lining that act to increase the surface area capable of absorbing nutrients.
volatile fatty acid Small-molecule fatty acids used as an energy source by the horse and produced as a by-product of bacterial fermentation of fibre in the hindgut.

References

CHAPTER 1

Back, W., and Clayton, H. M. (2001) *Equine Locomotion* (W. B. Saunders).

Denoix, J. M. (2000) *The Equine Distal Limb: An Atlas of Clinical Anatomy and Comparative Imaging* (CRC Press).

Ellis, A. D., and Hill, J. (2005) 'Nutritional physiology of the horse', *Journal of Equine Veterinary Science*, *25*(12), 524.

Floyd, A. E., and Mansmann, R. A. (2007) *Equine Podiatry* (Elsevier Health Sciences).

Macleod, C. (2007) *The Truth about Feeding your Horse* (J. A. Allen).

Pollitt, C. C. (1992) 'Clinical anatomy and physiology of the normal equine foot', *Equine Veterinary Education*, *4*(5), 219–224.

Savoldi, M. T., and Rosenberg, G. F. (2003) 'Uniform sole thickness', *American Farriers Journal*, *35*, 21.

Wyche, S. (2002) *The Horse's Muscles in Motion* (The Crowood Press Ltd).

CHAPTER 2

Cripps, P. J., and Eustace, R. A. (1999) 'Factors involved in the prognosis of equine laminitis in the UK', *Equine Veterinary Journal*, *31*(5), 433–442.

Cripps, P. J., and Eustace, R. A. (1999) 'Radiological measurements from the feet of normal horses with relevance to laminitis', *Equine Veterinary Journal*, *31*(5), 427–432.

Dik, K. J., and Gunsser, I. (1988) *Atlas of Diagnostic Radiology of the Horse. Part 1: Diseases of the Front Limbs* (Wolfe Publishing Ltd).

Eastman, T. G., Honnas, C. M., Hague, B. A., and Moyer, W. (1998) 'Deep digital flexor tenotomy as a treatment for chronic laminitis in horses: 37 cases', in *AAEP Proc* (Vol. 44, pp. 265–266).

Engiles, J. B. (2010) 'Pathology of the distal phalanx in equine laminitis: more than just skin deep', *Veterinary Clinics: Equine Practice*, *26*(1), 155–165.

Hood, D. M. (1999) 'Laminitis in the horse', *Veterinary Clinics: Equine Practice*, *15*(2), 287–294.

Morrison, S. (2011) 'Long-term prognosis using deep digital flexor tenotomy and realignment shoeing for the treatment of chronic laminitis', *Journal of Equine Veterinary Science*, *31*(2), 89–96.

O'Grady, S. E. (2010) 'Farriery for chronic laminitis', *Veterinary Clinics: Equine Practice*, *26*(2), 407–423.

Pollitt, C. C. (2008) *Equine Laminitis: Current Concepts* (Rural Industries Research and Development Corporation).

Redden, R. F. (2001) 'A technique for performing digital venography in the standing horse', *Equine Veterinary Education*, *13*(3), 128–134.

Redden, R. F. (2003) 'Radiographic imaging of the equine foot', *Veterinary Clinics: Equine Practice*, 19(2), 379–392.

CHAPTER 3

Agne, B. (2010) 'Diagnosis and treatment of foot infections', *Journal of Equine Veterinary Science*, *30*(9), 510–512.

McGuigan, M. P., Walsh, T. C., Pardoe, C. H., Day, P. S., and Wilson, A. M. (2005) 'Deep digital flexor tendon force and digital mechanics in normal ponies and ponies with rotation of the distal phalanx as a sequel to laminitis', *Equine Veterinary Journal*, *37*(2), 161–165.

Ramey, P. (2011). *Care and Rehabilitation of the Equine Foot* (Hoof Rehabilitation Publishing, LLC).

CHAPTER 4

Colles, C. M. (1989) 'The relationship of frog pressure to heel expansion', *Equine Veterinary Journal*, *21*(1), 13–16.

Wylie, C. E., Shaw, D. J., Verheyen, K. L., and Newton, J. R. (2016) 'Decision-tree analysis of clinical data to aid diagnostic reasoning for equine laminitis: a cross-sectional study', *The Veterinary Record*, *178*(17), 420–420.

CHAPTER 5

Asplin, K. E., Sillence, M. N., Pollitt, C. C., and McGowan, C. M. (2007) 'Induction of laminitis by prolonged hyperinsulinaemia in clinically normal ponies', *The Veterinary Journal*, *174*(3), 530–535.

Asplin, K. E., Patterson-Kane, J. C., Sillence, M. N., Pollitt, C. C., and Mc Gowan, C. M. (2010) 'Histopathology of insulin-induced laminitis in ponies', *Equine Veterinary Journal*, *42*(8), 700–706.

Asplin, K. E., *et al.* (2011) 'Glucose transport in the equine hoof', *Equine Veterinary Journal* 43.2, 196–201.

Attia, P. (2013) 'Is the obesity crisis hiding a bigger problem?' *TEDMED 2013*.

Bäckhed, F., Manchester, J. K., Semenkovich, C. F., and Gordon, J. I. (2007) 'Mechanisms underlying the resistance to diet-induced obesity in germ-free mice', *Proceedings of the National Academy of Science*s, *104*(3), 979–984.

Bailey, S. R., and Elliott, J. (1998) 'Plasma 5-hydroxytryptamine constricts equine digital blood vessels in vitro: implications for pathogenesis of acute laminitis', *Equine Veterinary Journal*, *30*(2), 124–130.

Bailey, S. R., and Elliott, J. (2007) 'The corticosteroid laminitis story: 2. Science of if, when and how', *Equine Veterinary Journal*, *39*(1), 7–11.

Bailey, S. R., Menzies-Gow, N. J., Marr, C. M., and Elliott, J. (2004) 'The effects of vasoactive amines found in the equine hindgut on digital blood flow in the normal horse', *Equine Veterinary Journal*, *36*(3), 267–272.

Bajza, Á, *et al*. (2004) 'Development of insulin resistance by nitrate tolerance in conscious rabbits', *Journal of Cardiovascular Pharmacology*, *43*(3), 471–476.

Carter, R. A., Treiber, K. H., Geor, R. J., Douglass, L., and Harris, P. A. (2009) 'Prediction of incipient pasture-associated laminitis from hyperinsulinaemia, hyperleptinaemia and generalised and localised obesity in a cohort of ponies', *Equine Veterinary Journal*, *41*(2), 171–178.

Daradka, M., and Pollitt, C. C. (2004) 'Epidermal cell proliferation in the equine hoof wall', *Equine Veterinary Journal*, *36*(3), 236–241.

French, K. R., and Pollitt, C. C. (2004) 'Equine laminitis: loss of hemidesmosomes in hoof secondary epidermal lamellae correlates to dose in an oligofructose induction model: an ultrastructural study', *Equine Veterinary Journal*, 36(3), 230–235.

Gard, P. R. (2002) *Human endocrinology* (CRC Press).

Hisano, H., Kanazawa, A., Kawakami, A., Yoshida, M., Shimamoto, Y., and Yamada, T. (2004) 'Transgenic perennial ryegrass plants expressing wheat fructosyltransferase genes accumulate increased amounts of fructan and acquire increased tolerance on a cellular level to freezing', *Plant Science*, *167*(4), 861–868.

Howieson, M. J., and Christians, N. E. (2008) 'Carbohydrate metabolism and efficiency of photosystem II in mown creeping bentgrass (*Agrostis stolonifera L.*)', *HortScience*, *43*(2), 525–531.

Johnson, P. J. (2002) 'The equine metabolic syndrome: peripheral Cushing's syndrome', *Veterinary Clinics of North America: Equine Practice*, *18*(2), 271–293.

Johnson, P. J., Messer, N. T., and Ganjam, V. K. (2004) 'Cushing's syndromes, insulin resistance and endocrinopathic laminitis', *Equine Veterinary Journal*, *36*(3), 194–198.

Johnson, P. J., Messer, N. T., Slight, S. H., Wiedmeyer, C., Buff, P., and Ganjam, V. K. (2004) 'Endocrinopathic laminitis in the horse', *Clinical Techniques in Equine Practice*, *3*(1), 45–56.

de Laat, M. A., McGowan, C. M., Sillence, M. N., and Pollitt, C. C. (2010) 'Equine laminitis: induced by 48 h hyperinsulinaemia in Standardbred horses', *Equine Veterinary Journal*, *42*(2), 129–135.

Livingston, D. P., Hincha, D. K., and Heyer, A. G. (2009) 'Fructan and its relationship to abiotic stress tolerance in plants', *Cellular and Molecular Life Sciences*, *66*(13), 2007–2023.

Longland, A., and Cairns, A. (1998, September) 'Sugars in grass – an overview of sucrose and fructan accumulation in temperate grasses', in *Proceedings of the Dodson and Horrell International Research Conference on Laminitis, Stoneleigh, Warwickshire, England* (pp.1–3).

Longland, A. C., and Byrd, B. M. (2006) 'Pasture nonstructural carbohydrates and equine laminitis', *The Journal of Nutrition*, *136*(7), 2099S–2102S.

McGowan, C., Cooper, D., and Ireland, J. (2016) 'No evidence that therapeutic systemic corticosteroid administration is associated with laminitis in adult horses without underlying endocrine or severe systemic disease', *Veterinary Evidence*, *1*(1).

Mungall, B. A., Kyaw-Tanner, M., and Pollitt, C. C. (2001) 'In vitro evidence for a bacterial pathogenesis of equine laminitis', *Veterinary Microbiology*, *79*(3), 209–223.

Nourian, A. R., Baldwin, G. I., Eps, A. V., and Pollitt, C. C. (2007), 'Equine laminitis: ultrastructural lesions detected 24–30 hours after induction with oligofructose', *Equine Veterinary Journal*, *39*(4), 360–364.

Peroni, J. F., Moore, J. N., Noschka, E., Grafton, M. E., Aceves–Avila, M., Lewis, S. J., and Robertson, T. P. (2006) 'Predisposition for venoconstriction in the equine laminar dermis: implications in equine laminitis' *Journal of Applied Physiology*, *100*(3), 759–763.

Pollitt, C. C. (1996) 'Basement membrane pathology: a feature of acute equine laminitis', *Equine Veterinary Journal*, *28*(1), 38–46.

Pollock, C. J., and Jones, T. (1979) 'Seasonal patterns of fructan metabolism in forage grasses', *New Phytologist*, *83*(1), 9–15.

Rendle, D. I. (2005) 'Equine Cushing's: a predisposing factor in infectious disease', *Equine Veterinary Education*, *17*(4), 184–186.

De Roover, J., Vandenbranden, K., Van Laere, A., and Van den Ende, W. (2000) 'Drought induces fructan synthesis and 1-SST (sucrose: sucrose fructosyltransferase) in roots and leaves of chicory seedlings (*Cichorium intybus L.*). *Planta*, *210*(5), 808–814.

Rowe, J. B., Lees, M. J., and Pethick, A. W. (1994) 'Prevention of acidosis and laminitis associated with grain feeding in horses', *The Journal of Nutrition*, *124*(12), 2742S.

Schott II, H. C. (2002) 'Pituitary pars intermedia dysfunction: equine Cushing's disease', *Veterinary Clinics of North America: Equine Practice*, *18*(2), 237–270.

Siciliano, P. D. (2015) '123 Effect of sward height on grazing preference and pasture NSC concentration', *Journal of Equine Veterinary Science*, *35*(5), 435.

Siciliano, P. D., Gill, J. C., and Bowman, M. A. (2017) 'Effect of Sward Height on Pasture Nonstructural Carbohydrate Concentrations and Blood Glucose/ Insulin Profiles in Grazing Horses', *Journal of Equine Veterinary Science*, *57*, 29–34.

Treiber, K. H., Kronfeld, D. S., and Geor, R. J. (2006) 'Insulin resistance in equids: possible role in laminitis', *The Journal of Nutrition*, *136*(7), 2094S–2098S.

Turnbaugh, P. J., Ley, R. E., Mahowald, M. A., Magrini, V., Mardis, E. R., and Gordon, J. I. (2006) 'An obesity-associated gut microbiome with increased capacity for energy harvest', *Nature*, *444*(7122), 1027.

Valluru, R., and Van den Ende, W. (2008) 'Plant fructans in stress environments: emerging concepts and future prospects', *Journal of Experimental Botany*, 59(11), 2905–2916.

Visser, M. B., and Pollitt, C. C. (2012) 'The timeline of metalloprotease events during oligofructose-induced equine laminitis development', *Equine Veterinary Journal*, 44(1), 88–93.

CHAPTER 6

Bezdekova, B., and Futas, J. (2009) 'Helicobacter species and gastric ulceration in horses: a clinical study', *Veterinarni Medicina*, *54*(12), 577–582.

Borer, K. E., Bailey, S. R., Menzies-Gow, N. J., Harris, P. A., and Elliott, J. (2012) 'Use of proxy measurements of insulin sensitivity and insulin secretory response to distinguish between normal and previously laminitic ponies', *Equine Veterinary Journal, 44*(4), 444–448.

Contreras, M., Morales, A., Garcia Amado, M. A., De Vera, M., Bermúdez, V., and Gueneau, P. (2007) 'Detection of Helicobacter-like DNA in the gastric mucosa of Thoroughbred horses', *Letters in Applied Microbiology*, *45*(5), 553–557.

Dakshinamurti, K. (2015) 'Vitamins and their derivatives in the prevention and treatment of metabolic syndrome diseases (diabetes)', *Canadian Journal of Physiology and Pharmacology*, *93*(5), 355–362.

De Laat, M. A., Kyaw-Tanner, M. T., Nourian, A. R., McGowan, C. M., Sillence, M. N., and Pollitt, C. C. (2011) 'The developmental and acute phases of insulin-induced laminitis involve minimal metalloproteinase activity', *Veterinary Immunology and immunopathology*, *140*(3–4), 275–281.

Graillot, V., Takakura, N., Hegarat, L. L., Fessard, V., Audebert, M., and Cravedi, J. P. (2012) 'Genotoxicity of pesticide mixtures present in the diet of the French population', *Environmental and Molecular Mutagenesis*, *53*(3), 173–184.

Hashemipour, M. *et al*. (2009) 'Effect of zinc supplementation on insulin resistance and components of the metabolic syndrome in prepubertal obese children', *Hormones (Athens)*, *8*(4), 279–285.

Henderson, L., *et al*. (2003) 'The National Diet and Nutrition Survey: adults aged 19 to 64 years: vitamin and mineral intake and urinary analytes' (The Stationery Office, London, UK).

Hernandez, J. A., Garbarino, E. J., Shearer, J. K., Risco, C. A., and Thatcher, W. W. (2005) 'Comparison of milk yield in dairy cows with different degrees of lameness', *Journal of the American Veterinary Medical Association, 227*(8), 1292–1296.

Katz, L. M., and Bailey, S. R. (2012) ‘A review of recent advances and current hypotheses on the pathogenesis of acute laminitis’, *Equine Veterinary Journal*, *44*(6), 752–761.

Kempson, S. (2009) *Laminitis – from the inside out!* Author’s personal lecture notes from presentation to students of Equine Podiatry Training Ltd.

Khan, A. R., and Awan, F. R. (2014) ‘Metals in the pathogenesis of Type 2 diabetes’, *Journal of Diabetes and Metabolic Disorders*, *13*(1), 16.

Kortenkamp, A. (2007) ‘Ten years of mixing cocktails: a review of combination effects of endocrine-disrupting chemicals’, *Environmental Health Perspectives*, *115*(Suppl 1), 98.

Morales, A., Garcia, F., and Bermudez, V. (2010) ‘Detection of Helicobacter-like organisms in Thoroughbred horses from Venezuela’, *Braz J Vet Path*, *3*, 52–55.

Moyaert, H., Pasmans, F., Decostere, A., Ducatelle, R., and Haesebrouck, F. (2009) ‘Helicobacter equorum: prevalence and significance for horses and humans’, *FEMS Immunology and Medical Microbiology*, *57*(1), 14–16.

Paolisso, G., Scheen, A., d’Onofrio, F., and Lefebvre, P. (1990) ‘Magnesium and glucose homeostasis’, *Diabetologia*, *33*(9), 511–514.

Paolisso, G., Sgambato, S., Gambardella, A., Pizza, G., Tesauro, P., Varricchio, M., and d’Onofrio, F. (1992) ‘Daily magnesium supplements improve glucose handling in elderly subjects’, *The American Journal of Clinical Nutrition*, *55*(6), 1161–1167.

Reeves, P. G., and O’Dell, B. L. (1983) ‘The effect of zinc deficiency on glucose metabolism in meal-fed rats’, *British Journal of Nutrition*, *49*(3), 441–452.

Rodriguez-Moran, M., and Guerrero-Romero, F. (2003) ‘Oral magnesium supplementation improves insulin sensitivity and metabolic control in Type 2 diabetic subjects: a randomized double-blind controlled trial’, *Diabetes Care*, *26*(4), 1147–1152.

Samsel, A., and Seneff, S. (2013) ‘Glyphosate’s suppression of cytochrome P450 enzymes and amino acid biosynthesis by the gut microbiome: pathways to modern diseases’, *Entropy*, *15*(4), 1416–1463.

Shepherd, S. J., and Gibson, P. R. (2006) ‘Fructose malabsorption and symptoms of irritable bowel syndrome: guidelines for effective dietary management’, *Journal of the American Dietetic Association*, *106*(10), 1631–1639.

Steinrücken, H. C., and Amrhein, N. (1980) ‘The herbicide glyphosate is a potent inhibitor of 5-enolpyruvylshikimic acid-3-phosphate synthase’, *Biochemical and Biophysical Research Communications*, *94*(4), 1207–1212.

Striffler, J. S., Polansky, M. M., and Anderson, R. A. (1998) ‘Dietary chromium decreases insulin resistance in rats fed a high-fat, mineral-imbalanced diet’, *Metabolism – Clinical and Experimental*, *47*(4), 396–400.

Takaya, J., Higashino, H., and Kobayashi, Y. (2004) ‘Intracellular magnesium and insulin resistance’, *Magnesium Research*, *17*(2), 126–136.

Teixeira, T. F., Collado, M. C., Ferreira, C. L., Bressan, J., and Maria do Carmo, G. P. (2012) ‘Potential mechanisms for the emerging link between obesity and increased intestinal permeability’, *Nutrition Research*, *32*(9), 637–647.

van Eps, A., Collins, S. N., and Pollitt, C. C. (2010) ‘Supporting limb laminitis’, *Veterinary Clinics: Equine Practice*, *26*(2), 287–302.

CHAPTER 7

Baxter, G. M., Tacklett, R. L., and Moore, J. N. (1989) ‘Reactivity of equine palmar digital arteries and veins to vasodilating agents’, *Veterinary Surgery*, *18*(3), 221–226.

Eps, A. W., and Pollitt, C. C. (2004) ‘Equine laminitis: cryotherapy reduces the severity of the acute lesion’, *Equine Veterinary Journal*, *36*(3), 255–260.

Eps, A. W., and Pollitt, C. C. (2009) ‘Equine laminitis model: cryotherapy reduces the severity of lesions evaluated seven days after induction with oligofructose’, *Equine Veterinary Journal*, *41*(8), 741–746.

Eustace, R. A., and Caldwell, M. N. (1989) ‘Treatment of solar prolapse using the heart-bar shoe and dorsal hoof wall resection technique’, *Equine Veterinary Journal*, *21*(5), 370–372.

Goetz, T. E. (1989) ‘The treatment of laminitis in horses’, *Veterinary Clinics of North America: Equine Practice, 5*(1), 73–108.

Kuwano, A., Katayama, Y., Kasashima, Y., Okada, K., and Reilly, J. D. (2002) ‘A gross and histopathological study of an ectopic white line development in equine laminitis’, *Journal of Veterinary Medical Science, 64*(10), 893–900.

Leise, B. S., Fugler, L. A., Stokes, A. M., Eades, S. C., and Moore, R. M. (2007) 'Effects of intramuscular administration of acepromazine on palmar digital blood flow, palmar digital arterial pressure, transverse facial arterial pressure, and packed cell volume in clinically healthy, conscious horses', *Veterinary Surgery, 36*(8), 717–723.

Morrison, S. (2010) 'Chronic laminitis: foot management', *Veterinary Clinics: Equine Practice, 26*(2), 425–446.

Taylor, D., Hood, D. M., and Wagner, I. P. (2002) 'Short-term effect of therapeutic shoeing on severity of lameness in horses with chronic laminitis', *American Journal of Veterinary Research, 63*(12), 1629–1633.

CHAPTER 8

Agne, B. (2010) 'Diagnosis and treatment of foot infections', *Journal of Equine Veterinary Science, 30*(9), 510–512.

DeBowes, R. M., and Yovich, J. V. (1989) 'Penetrating wounds, abscesses, gravel, and bruising of the equine foot', *Veterinary Clinics of North America: Equine Practice, 5*(1), 179–194.

Hickman, J., and Humphrey, M. (1988) *Hickman's Farriery* (J.A. Allen).

Powell, H. (2013) 'A preliminary study into the use of manual lymphatic drainage to support recovery from laminitis', *Journal of Equine Veterinary Science, 33*(10), 872.

Ramey, P. (2003) *Making Natural Hoof Care Work for You* (Star Ridge Publishing).

Taylor, D., Sperandeo, A., Schumacher, J., Passler, T., Wooldridge, A., Bell, R., and Ramey, P. (2014) 'Clinical Outcome of 14 Obese, Laminitic Horses Managed with the Same Rehabilitation Protocol', *Journal of Equine Veterinary Science, 34*(4), 556–564.

www.hoofrehab.com

CHAPTER 9

Beech, J., Donaldson, M. T., and Lindborg, S. (2002, December) 'Comparison of Vitex agnus castus extract and pergolide in treatment of equine Cushing's syndrome', in *AAEP Proceedings* (Vol. 48, pp. 175–177).

Donaldson, M. T., LaMonte, B. H., Morresey, P., Smith, G., and Beech, J. (2002) 'Treatment with pergolide or cyproheptadine of pituitary pars intermedia dysfunction (equine Cushing's disease) *Journal of Veterinary Internal Medicine, 16*(6), 742–746.

Dugdale, A. H. A., Curtis, G. C., Cripps, P., Harris, P. A., and Argo, C. M. (2010) 'Effect of dietary restriction on body condition, composition and welfare of overweight and obese pony mares', *Equine Veterinary Journal, 42*(7), 600–610.

Dugoua, J. J., Seely, D., Perri, D., Koren, G., and Mills, E. (2008) 'Safety and efficacy of chastetree (Vitex agnus-castus) during pregnancy and lactation', *Can J Clin Pharmacol, 15*(1), e74–e79.

Durham, A. E. (2013) 'Equine metabolic syndrome', *Equine Laminitis,* 329–333.

Durham, A. E., Rendle, D. I., and Newton, J. R. (2008) 'The effect of metformin on measurements of insulin sensitivity and cell response in 18 horses and ponies with insulin resistance*', Equine Veterinary Journal, 40*(5), 493–500.

Eagles, C. F. (1967) 'Variation in the soluble carbohydrate content of climatic races of Dactylis glomerata (cocksfoot) at different temperatures', *Annals of Botany, 31*(4), 645–651.

Freestone, J. F., Beadle, R., Shoemaker, K., Bessin, R. T., Wolfsheimer, K. J., and Church, C. (1992) 'Improved insulin sensitivity in hyperinsulinaemic ponies through physical conditioning and controlled feed intake', *Equine Veterinary Journal, 24*(3), 187–190

Harris, P., and Geor, R. J. (2009) 'Primer on dietary carbohydrates and utility of the glycemic index in equine nutrition', *Veterinary Clinics: Equine Practice, 25*(1), 23–37.

Kronfeld, D., Rodiek, A., and Stull, C. (2004) 'Glycemic indices, glycemic loads, and glycemic dietetics', *Journal of Equine Veterinary Science, 24*(9), 399–404.

Morvan–Bertrand, A., Boucaud, J., Le Saos, J., and Prud'homme, M. P. (2001) 'Roles of the fructans from leaf sheaths and from the elongating leaf bases in the regrowth following defoliation of Lolium perenne L', *Planta, 213*(1), 109–120.

Roemheld–Hamm, B. (2005) 'Chasteberry', *American Family Physician, 72*(5), 821–824.

Sliutz, G., Speiser, P., Schultz, A. M., Spona, J., and Zeillinger, R. (1993) 'Agnus castus extracts inhibit prolactin secretion of rat pituitary cells', *Hormone and Metabolic Research, 25*(05), 253–255.

Tinworth, K. D., Boston, R. C., Harris, P. A., Sillence, M. N., Raidal, S. L., and Noble, G. K. (2012) 'The effect of oral metformin on insulin sensitivity in insulin-resistant ponies', *The Veterinary Journal, 191*(1), 79–84.

www.nationalequinewelfarecouncil.co.uk. Body condition scoring.

CHAPTER 10

Albarracin, C. A., Fuqua, B. C., Evans, J. L., and Goldfine, I. D. (2008) 'Chromium picolinate and biotin combination improves glucose metabolism in treated, uncontrolled overweight to obese patients with Type 2 diabetes', *Diabetes/Metabolism Research and Reviews, 24*(1), 41–51.

Furukawa, Y. (1999) 'Enhancement of glucose-induced insulin secretion and modification of glucose metabolism by biotin', *Nihon rinsho. Japanese Journal of Clinical Medicine, 57*(10), 2261–2269.

Jackson, J. (2014) *Paddock paradise: A Guide to Natural Horse Boarding* (Star Ridge Publishing).

Johnson, P. J., Messer, N. T., and Kellon, E. (2004) 'Treatment of equine metabolic syndrome', *Compendium on Continuing Education for the Practicing Veterinarian. February*.

Kellon, E. M. (2006) 'Iron status of hyperinsulinemic/ insulin resistant horses', in *Horse Health Nutrition: Third European Equine Health and Nutrition Congress, Faculty of Veterinary Medicine, Ghent University, Merelbeke, Belgium, 17–18 March, 2006* (pp. 90–92) (The Equine Health and Nutrition Association).

Maebashi, M., Makino, Y., Furukawa, Y., Ohinata, K., Kimura, S., and Sato, T. (1993) 'Therapeutic evaluation of the effect of biotin on hyperglycemia in patients with non-insulin dependent diabetes mellitus', *Journal of Clinical Biochemistry and Nutrition, 14*(3), 211–218.

National Research Council (2007) *Nutrient Requirements of Horses (Ed. 6)* (The National Academies Press).

Reilly, J. D., Cottrell, D. F., Martin, R. J., and Cuddeford, D. J. (1998) 'Effect of supplementary dietary biotin on hoof growth and hoof growth rate in ponies: a controlled trial', *Equine Veterinary Journal, 30*(S26), 51–57.

Seelig, M. S., and Rosanoff, A. (2003) *The Magnesium Factor* (Penguin).

Self, H. P. (1996) *A Modern Horse Herbal* (Kenilworth Press).

CHAPTER 11

Jackson, J. (2001) *Founder: Prevention and Cure the Natural Way* (Star Ridge Publishing).

Further Information

The Equine Podiatry Association (EPA) exists as a self-regulating professional body for equine podiatrists (EPs) practising in the UK. The association is run as a democratic, not-for-profit organization, and is totally independent and self-governing. All EPA members are trained in the techniques and approaches discussed in this book. The EPA maintains a register of EPs who meet the training and continuing professional development requirements of the EPA. The EPA web site (www.epauk.org) has a map tool that helps UK clients find the nearest EPs to them.

Equine Podiatry Training Ltd is a UK company providing training courses leading to a professional qualification in equine podiatry (the entry requirement for the EPA). Anyone interested in training to become an EP should visit www.eptrain.co.uk

The Australian Equine Laminitis Research Unit (www.laminitisresearch.org), based at the University of Queensland, is perhaps the biggest research unit in the field of laminitis. Their web site has a range of useful information on the established view of laminitis.

The Laminitis Trust (www.laminitis.org) raises money for laminitis research, but also has some useful advice on its web site. It is a useful source of information on current established approaches.

The Laminitis Site (www.thelaminitissite.org) is a web site packed with the latest thinking (both mainstream and alternative) on laminitis. This is a very useful source of information.

The Equine Cushing's and Insulin Resistance Group (ECIR) (www.ecirhorse.org) is a field-trial database for cases of EMS and PPID run by Dr Eleanor Kellon, a US-based vet with a long-standing interest in the role of nutrition in laminitis.

Therapeutic memory-foam pads (as described in Chapter 8) can be obtained from www.equinepodiatrysupplies.co.uk – look for EPS pads.

Plantlife (www.plantlife.org.uk) is a UK conservation charity working to conserve threatened wild plants and habitats. It has a wealth of experience of the management of traditional grasslands, and is a useful source of information and advice for those wanting to create species-rich pastures to improve the diversity of their horses' diets.

The author's website (www.unshod.co.uk) has a number of useful articles and case studies.

Index

Table of Contents