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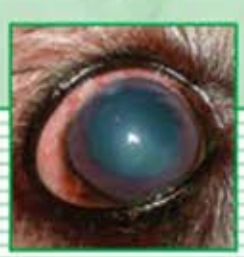
SALLY M. **TURNER**

Series Editor: **Fred Nind**









SMALL ANIMAL OPHTHALMOLOGY

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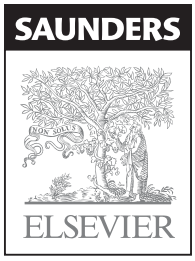
SAUNDERS SOLUTIONS IN
VETERINARY PRACTICE

SMALL ANIMAL
OPHTHALMOLOGY

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Dedication

To Jeff
Thank you again for your
love and support

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Introduction

Saunders Solutions in Veterinary Practice series is a new range of veterinary textbooks which will grow into a mini library over the next few years, covering all the main disciplines of companion animal practice.

Readers should realize that it is not the intention of authors to cover all that is known about each topic. As such the books in the *Saunders Series* are not standard reference works. Instead they are intended to provide practical information on the more frequently encountered conditions in an easily accessible form based on real life case studies. They cover that range of cases which fall between the boringly routine and the referral. The books will help practitioners with a particular interest in a topic or those preparing for a specialist qualification. The cases are arranged by presenting sign rather than by the underlying pathology as this is how veterinary surgeons will see them in practice.

Each case also includes descriptions of underlying pathology and details of the nursing required both in the veterinary clinic and at home. It is hoped that the books will also, therefore, be of interest to veterinary students in the later parts of their course and to veterinary nurses.

Continuing professional development (CPD) is mandatory for many veterinarians and a recommended practice for others. The *Saunders Series* will provide a CPD resource which can be accessed economically, shared with colleagues and used anywhere. They will also provide busy veterinary practitioners with quick access to authoritative information on the diagnosis and treatment of interesting and challenging cases. The robust cover has been made resistant to some of the more gruesome contaminants found in a veterinary clinic because this is where we hope these books will be used.

Joyce Rodenhuis and Mary Seager were the inspiration for the Series and both the Series editor and the individual authors are grateful for their foresight in commissioning the Series and their unfailing support and guidance during their production.

OPHTHALMOLOGY

Ophthalmologic cases can be challenging. The proliferation of veterinary ophthalmology societies and study groups testify to the rapid expansion in the knowledge and skill base in this area. Incorrect or inadequate treatment can compromise what is an important sense for all companion animal species or lead to debilitating pain and discomfort. Indeed, the failure to accurately diagnose some ophthalmic conditions, and then treat them appropriately, can result in loss of the eye, which could have been avoided had the correct approach to the case been followed. Most of the cases discussed can be dealt with in general practice, but where referral to a suitably experienced colleague is recommended, this is mentioned within the text. The book includes a section on the ophthalmic examination – such a fundamental part of the subject that its omission would be negligent! The choice of cases discussed covers selected areas of the subject, particularly common or potentially frustrating cases, as well as the occasional less frequently encountered diseases which should, nonetheless, be recognized by general practitioners. Each section is fully illustrated – ophthalmology is clearly such a visual subject that colour photographs are essential! Several useful appendices are included, covering for example ophthalmic emergencies, conditions which should be checked for in young animals and commonly used pharmacological agents. It is hoped that this book will help practitioners to keep up to date in this discipline, rise to the challenge that eye cases pose and develop confidence in dealing with ophthalmologic cases.

Fred Nind
Series Editor

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1 The ophthalmic examination

A full ophthalmic examination should be performed on all animals presenting with an ocular complaint. To perform this properly it is important to have a standard approach to all patients together with appropriate facilities and equipment.

The basic parts of the ophthalmic examination are:

- History taking
- Hands-off examination
- Hands-on examination
  - Schirmer tear test readings
  - Vision testing and neurological testing
  - Ophthalmoscopy.

Further tests include:

- Ophthalmic dyes
- Nasolacrimal duct investigation
- Tonometry
- Laboratory investigation.

Table 1.1 Facilities and equipment required for the ophthalmic examination

General practice	Those with an interest in ophthalmology
• Room capable of being fully darkened • Table of adjustable height • Pen torch • Direct ophthalmoscope • Magnifying lens • Magnifying loupe • Tonometer (Shiotz)	All items listed left plus: • Slit lamp biomicrosope • Finhoff transilluminator • Indirect ophthalmoscope • Goniolens • Tonopen or Tonovet tonometer

Disposable items required for ophthalmic examination are:

- Schirmer tear test papers
- Fluorescein paper test strips
- Rose Bengal dye (for specialty ophthalmology practice)
- Cotton wool and gauze swabs
- Tropicamide (Mydriacyl 1% Alcon)
- Topical anaesthetic (proxymetacaine)
- Sterile saline
- Lacrimal cannulae
- Bacterial swabs and transport media
- Slides and laboratory equipment.

HISTORY

History taking can be divided into general history and that specifically pertaining to the eyes. A general history should consider the following points:

- *Genetics*: As breed pedigree dogs and cats can suffer from inherited eye diseases, the breed is of importance. Even in crossbred dogs the general type of dog might be relevant – for example, terriers are prone to lens luxation which is seen in pedigree wire haired fox terriers and miniature bull terriers among others, but is common in all types of Jack Russell from the pedigree Parson terrier to the general terrier type. In cats, corneal sequestrum is a common condition in Persians and Burmese while retinal degeneration is recognized in Siamese and Abyssinian breeds. Clearly we should not assume that just because a patient is a particular breed it will definitely be suffering from a certain condition – but breed-related diseases are frequently encountered in veterinary ophthalmology.
- *Age*: This is always a factor to be considered. Conditions such as entropion and prolapse of the nictitans gland (cherry eye) are common in young animals (3–12 months) while neoplasia is more common in older patients. Congenital problems such as cataract and collie eye anomaly should be considered when patients come in for their initial vaccinations (see Appendix 4).
- *Sex*: Although of less importance with regard to ophthalmology than for some other disciplines, the sex

of the patient might have some influence on its ocular disease. For example, young male dogs are more prone to entropion while keratoconjunctivitis sicca is seen in neutered females more commonly than entire males.

- *General health*: Many ocular conditions can be manifestations of systemic disease and questions related to appetite, general demeanour and concurrent illness are extremely important. Patients might be presented with sudden blindness as the owner's complaint, which turns out to be due to diabetes mellitus and the owner has failed to appreciate that the polydipsia, weight loss and so on were actually due to a serious medical condition.
- *Medications administered*: Previous or ongoing medication may be the cause of some ophthalmic conditions. An example is the dog with colitis which is treated with sulfasalazine (Salazopyrin) and develops an acute, severe bilateral keratoconjunctivitis sicca. Some ophthalmic drugs can cause a localized hypersensitivity which exacerbates the ocular disease. Owners are notorious for using left-over medication from a previous problem – which might not even have been for the pet! Such actions can be deleterious to the patient.
- *Presence of other pets in the house*: This is particularly relevant when considering infectious diseases such as feline herpes virus where repeated infection can be a problem. If the owner has more than one pet it is important to find out if others are affected. The presence of a new puppy in a home with a cat might be relevant if the puppy is brought to the surgery with an acutely painful eye – it is quite likely that the cat has scratched it!
- *Source of the pet*: Cats from rescue centres and/or breeders may have pre-existing diseases such as herpes or *Chlamydophila* infections, while dogs from puppy farms may be prone to inherited ocular disease since unscrupulous breeders pay no attention to such things.
- *Travel history*: This is becoming more and more relevant in the UK since the introduction of the Pets Passports schemes but is relevant in all countries. Several Mediterranean diseases such as leishmania and ehrlichia can have ocular involvement.

Once a thorough general history has been taken we can move on to the more specific ophthalmic history. This will include asking the owners the following questions:

- What was the first thing the owner noticed wrong with the eye(s)? Were they concerned about discharge, pain, redness or other change in colour, an alteration in appearance of the eye (e.g. bulging or sunken) or perhaps they noted a decrease in vision.
- When did they notice something wrong? The time span of the disease is relevant – some owners will bring a pet along to the surgery immediately while there are those owners who leave the pet for weeks or even months before presenting it.
- Progression of the problem? Has the problem got worse, better or stayed the same since it was first noticed? Has it been present continuously or has it been intermittent? If the latter, has the owner noticed any 'trigger factor' which brought on a return of the symptoms? For example, did the atopic Labrador always have itchy eyes when he returned from a walk through certain fields? (This could suggest an allergic component.)
- Is the problem unilateral or bilateral? As mentioned in the general history taking, systemic disease may be the underlying problem and this is much more likely with bilateral conditions. With infectious disease we might also expect a bilateral presentation although it is not unusual for one eye to be affected before the other – *Chlamydophila* in cats is an example where a delay of a few days can be seen before the second eye develops symptoms.
- Treatment – has the owner given any medication? If so, what, and was it beneficial or detrimental?
- Previous ocular history? It is important to establish whether the pet has had any ocular problem in the past – and, if so, was the presentation the same and was it the same eye which was affected? Also, response to treatment given previously is relevant.

THE OPHTHALMIC EXAMINATION

Hands-off examination

While the history is being taken the patient should be observed. Dogs are allowed to wander off the lead and investigate the consulting room while cats are encouraged out of their baskets and then watched from a distance. It is important to look at the behaviour of the pet, along with the gross appearance of the eyes and face. Signs of ocular discomfort – blepharospasm, increased lacrimation or other discharge, symmetry of the eyes and

face – sunken or small eyes, enlargement of the globe, periorbital swellings or squints for example. Clues might inform us that the animal has been rubbing – periorbital hair loss and erythema or saliva staining on the front legs where the animal licks then rubs the face might be evidence of self-trauma. Basic assessment of the visual ability of the patient can be undertaken at this time – blind animals will often stay close to the owner and not move around the room, while if they do move they are often very cautious, sniffing the environment and exhibiting a high stepping gait.

Hands-on examination

The first part of the hands-on examination takes place in a well-lit room. The patient should be gently restrained – one hand under the chin and the other behind the back of the head is usually sufficient. A trained nurse is invaluable during ocular examinations – most owners are not very good at restraining their pets in general, and when you are very close to the biting end it is particularly important that the patient is properly held by a confident adult!

A close inspection of the gross appearance of the eyes and face is performed with illumination such as a pen torch. Particular things to consider include the presence of any ocular discharge – the nature of it, whether unilateral or bilateral and the amount of it. Some dogs commonly have a small amount of mucoid discharge at the medial canthus, especially those with doliocephalic conformation such as Dobermanns, and it can be considered a normal finding in such animals. The size of both eyes should be compared – they should be symmetrical but if one is larger than the other it is necessary to establish whether one is enlarged (hydrophthalmic) or if one is shrunken (microphthalmic). In addition to size, the actual position of the eyes should be noted – looking from above the patient directly down on the head can assist in establishing the presence of exophthalmos, enophthalmos or strabismus. General head symmetry and the presence of periorbital swellings should also be noted.

It might be necessary to take samples for laboratory analysis at this stage if indicated. Swabs for bacterial culture and isolation should be taken before any discharges are cleaned away.

Schirmer tear test readings

Schirmer tear test readings should be taken before the eyes are cleaned or handled further. Commercial tear testing strips are used (colour bar calibrated strips are the easiest to use and are available from Schering-Plough). The strips come in sterile plastic wallets. The strips should be bent at the notch while still in their plastic wallet (to prevent sweat and grease from hands interfering with the readings). The packet is opened and each test paper is held at the distal end. The shorter piece is placed in the ventral conjunctival sac half to two-thirds along from the medial canthus (i.e. out of the way of the third eyelid) (Figure 1.1). The strip is left in position for 1 minute before removing it and immediately reading the level of wetting on the scale (see Table 1.2). It is easier to hold the patient's eye closed to prevent the strip from falling out prematurely. Topical anaesthetics are not used prior to measurement.

Basic vision testing and neurological tests

1. Menace response

A threatening gesture will cause the animal to blink and pull away slightly. The reflex tests the visual pathway (optic nerve, cranial nerve II) and the ability to close the lids (facial nerve, cranial nerve VII), i.e. can the animal see you and, if so, can it react and blink normally? It is



Figure 1.1 Schirmer tear test in a cat showing placement of paper strip.

Table 1.2 Schirmer tear test readings in dogs and cats

	Dog	Cat
Normal	15–25 mm/min	Readings variable and often lower than in the dog, especially in stressed cats
Borderline	10–15 mm/min	
Keratoconjunctivitis sicca	<10 mm/min	

important that the stimulus is visual only, and does not generate air currents which would trigger sensory nerve endings on the cornea and skin (trigeminal nerve – cranial nerve V). For this reason some people advocate testing from behind a clear Perspex screen, but in reality this is not really necessary. So long as the hand movements involve just a couple of fingers rather than waving the whole hand in front of the eyes there should not be any confusion over the actual reflex being tested. It is important to stimulate both from directly in front of the eyes and also from other angles – above and below plus medial and lateral. The fellow eye can be covered with your other hand to assess each eye separately.

CLINICAL TIP

If the animal is visual but cannot blink, for example with facial nerve paralysis, the third eyelid will move across the globe and the animal will pull back from the stimulus. A palpebral reflex can then be performed to confirm that the animal cannot blink (see below). However, it is clear that it could see, and react to, the menacing gesture. Blind animals will not react at all.

CLINICAL TIP

The menace response is a learned response and is not present in very young animals (one reason why young puppies are often scratched on the cornea by the family cat – they have not learned to blink for protection!).

2. Dazzle reflex

A very bright light source is needed for this test – a Finhoff transilluminator is ideal. This is a focussed light source which can be fitted to the handle of the direct ophthalmoscope. Often a pen torch is not bright enough. The animal is seen to blink and sometimes pull away when the bright light is shone into each eye in turn. This tests the same two nerves as the menace response – optic and facial nerves – but unlike the menace response it does not require involvement of the cerebral cortex for the reflex to occur. It is a useful test if the retina and optic nerve cannot be examined due to opacity in the eye – cataract for example. A positive reflex suggests that the retina and optic nerve are functional.

3. Tracking responses (cotton wool balls)

Cotton wool is used because it does not smell strongly and makes no sound when it lands. Thus we can be sure that we are testing vision and not smell or hearing instead! Several small pieces are used and dropped from above the animal's face once you have gained their attention. Dogs and cats have better movement detection than us and should follow the path of the falling cotton wool. Each eye can be tested separately and the test can be performed in bright and dim lighting conditions. False-negative results are seen when the animals are bored or disinterested – more of a problem in cats. Sometimes dangling a loose piece of bandage will encourage the cat to 'track' the movement.

4. Maze testing

An unfamiliar room is used – a large consulting room or empty waiting room for example. A mixture of solid objects (e.g. bins) and open objects (e.g. chairs) is chosen and randomly placed in the room. The animal is held at one corner of the room while the owner stands at the other side of the room and then calls their pet. Normally sighted animals will negotiate the room confidently but visually impaired ones will be hesitant and slow, and may bump into objects. This test should be performed in both bright and dim lighting levels and the objects should be moved about in between tests.

It can be difficult to get cats to cooperate. The test can be adapted for them by placing their basket on the floor behind a chair and watching to see how they manage to get to the safety of their basket!

5. Palpebral (blink) reflex

It is important to check that the animal can blink normally. Sensory stimulation of the trigeminal nerve (cranial nerve V) by tapping the medial and lateral canthal skin should result in a brisk closing of the eyelids (facial nerve – cranial nerve VII). The animal may also try to move away. If the animal cannot feel the stimulation then it will not blink (but should have a normal menace response assuming that it can see). If the animal can feel the stimulus but cannot blink due to a facial nerve paralysis it is likely to move away from the stimulus, and would have an abnormal menace response where the third eyelid crosses the cornea but no blink occurs, as mentioned previously. Corneal sensitivity can also be tested by touching the corneal surface with a wisp of cotton wool and watching a normal blink. It is usually very dif-

ficult to touch the cornea in this way without the animal trying to blink.

6. *Pupillary light reflexes*

Pupillary light reflexes should be assessed both in normal room light and in the dark. They are more sensitive in the dark.

CLINICAL TIP

Pupillary light reflexes are not an assessment of vision but an indication of retinal, optic nerve and some central pathway function.

A bright pen torch or Finhoff transilluminator is required. The light is shone into one eye and a normal response is seen as the pupil rapidly constricts. This is the direct reflex. The opposite eye is examined (with the light still shining in the first eye) and constriction of the fellow pupil should have occurred – this is the consensual or indirect reflex. The light should then be moved to the second eye which is tested in the same way. The afferent nerve involved is the optic nerve (cranial nerve II) while the pupillary constriction is mediated by the parasympathetic fibres running in the oculomotor nerve (cranial nerve III).

The light source is then alternated between the two eyes in the swinging flashlight test. The light is shone into the first eye for a couple of seconds and then rapidly moved across to the second eye. A note is made of the response of the second pupil – it could constrict further, stay the same or dilate. A normal response would show both pupils staying miotic, often with further direct constriction of the newly illuminated pupil. If the second pupil suddenly dilates under direct illumination this is an abnormal response (called a positive swinging flashlight test) and indicates a lesion in the retina or optic nerve in this eye.

In addition to examining for the ability to constrict, the speed and degree of constriction should also be evaluated.

CLINICAL TIP

Animals which are very excited or aggressive will have slow and incomplete pupillary constriction as a result of increased sympathetic tone.

Additionally, the resting position of both pupils should be assessed. They should be symmetrical and semi-dilated in dim light, with moderate constriction in room light.

Ophthalmoscopy

Once vision has been assessed and the neurological testing performed, the systematic examination of all sections of the eye follows. The adnexa and anterior structures are examined both in the light and in the dark with the use of magnification. Specialists use a slit lamp biomicroscope which gives excellent illumination and magnification but due to the cost they are not routinely available in general practice. A good compromise is the use of magnifying loupes (Figure 1.2) and illumination, although a direct ophthalmoscope can also be used – settings of +20 are best for the lids and cornea while +8 to +12 lenses are selected to examine the lens.

The eyelids are examined for position, the presence of swellings, eyelash abnormalities and the position and size of nasolacrimal punctae. The third eyelid is checked for colour, position, prolapse of the nictitans gland and abnormalities of cartilage. The conjunctival surfaces are examined for colour, swelling (chemosis), haemorrhage, moisture, masses and foreign bodies. The cornea is checked for transparency, vascularity, cellular infiltration, ulceration and alterations in shape. The anterior chamber is normally optically clear. In the presence of inflammation (uveitis) there will be leakage of proteins, cells and



Figure 1.2 Magnifying loupes are useful to examine the adnexa, anterior chamber and lens as well as being indispensable for many ocular surgical procedures.

sometimes red blood cells into the anterior chamber. This makes the aqueous cloudy and is termed aqueous flare. Frank blood in the anterior chamber is called hyphaema while an abundant accumulation of white cells is termed hypopyon (and often looks like pus in the anterior chamber although usually hypopyon is sterile). The anterior chamber is also assessed for depth – it might be deeper than normal if the lens is luxated into the posterior segment or shallow if there are adhesions between the iris and the cornea (anterior synechia). Colour, adhesions, masses and persistent pupillary membrane remnants should be considered on iris examination. The lens is examined for optical clarity. The presence of an opacity in the lens is a cataract. Examination of the lens is best performed once the pupil has been dilated but before that distant direct ophthalmoscopy should be performed.

Distant direct ophthalmoscopy

This technique is used to examine the eye for a clear visual axis and to assess pupil symmetry. It should be performed in the dark. A setting of 0 is used and the instrument is held to the examiner's eye at arm's length from the dog or cat (Figure 1.3). The tapetal reflex is obtained. Any opacity in the visual axis will block the fundic reflex – either totally or partially. This will be seen as a dark shadow within the bright tapetal reflex. Opacities can be on the cornea, in the aqueous, lens or vitreous. By moving from side to side one can assess the depth of the opacity. For example, a cataract on the anterior lens capsule will not move as you move your head because it is in the same plane as the pupil. A corneal opacity, which is in front of the plane of the pupil, will move in the same direction as you while a cataract at the back of the lens, behind the pupil, will move away. This uses the phenomenon of parallax.



Figure 1.3 Distant direct ophthalmoscopy.

Distant direct ophthalmoscopy is also used to assess pupil size. Anisocoria (a difference in size between the two pupils) is detected more easily using this technique than by looking with the naked eye. The observer looks through the instrument while the light from the ophthalmoscope is shone from one eye to the other and the size of pupils is compared.

Another useful aspect of distant direct ophthalmoscopy is that it can differentiate between a true cataract and the ageing change of nuclear sclerosis. Since a cataract is an opacity in the lens, it will appear as a dark shadow against the tapetal reflex as mentioned above. However, nuclear sclerosis is an age-related hardening of the lens, which although it renders the lens cloudy when viewed in the reflected light of a room, the direct light of the ophthalmoscope will pass right through it, with just a circular ring visible at the junction of the lens nucleus and cortex. Many owners, and unfortunately some veterinary surgeons, mistakenly think a dog has cataracts whereas in fact it only has nuclear sclerosis – if the method of distant direct ophthalmoscopy were used more frequently, then owners would be saved the expense and time involved in referral to a specialist veterinary ophthalmologist only to be told that their pet has normal eyes for an older dog!

CLINICAL TIP

Use distant direct ophthalmoscopy to differentiate between nuclear sclerosis and a true cataract.

Complete examination of the lens, vitreous and fundus can only be achieved following mydriasis (pupil dilation). One drop of tropicamide 1.0% (Mydriacyl, Alcon) is applied to each eye. After 15–20 minutes the pupil should be fully dilated, allowing examination of the intraocular structures.

Close direct ophthalmoscopy

The lens can be examined, after pupil dilation, using a setting of +8 to +12 dioptres. The position of the lens should be assessed and any opacity within it should be localized (discussed further in Section 6).

To examine the fundus a setting of 0 is chosen. If the observer has any known refractive errors (e.g. short

sightedness) it will be necessary to adjust this setting in order to have a focussed image of the retina. One needs to be close to the patient – only 2–3 cm away from their eye. Resting one finger on the bridge of the animal's nose will assist stabilization and alert the observer to any change in position of the animal. It is important to be systematic when examining the retina. Firstly the optic disc is located. It is examined for colour, size and appearance of the blood vessels. The retina is then examined section by section. This can initially be challenging but with practice it becomes second nature and is most easily done by mentally splitting the back of the eye into quarters and, starting from the optic disc, examining each quarter in turn. If you get disoriented by the patient moving its eyes, finding the optic disc again will allow you to continue the systematic examination. It is important to look right out to the periphery although this can be difficult laterally in dogs with long noses. Using your right eye to examine the patient's right eye and vice versa will keep the nose out of the way but most people manage using just their dominant side. By adding positive dioptre lenses in the ophthalmoscope it is possible to examine the vitreous.

You need to be close to the patient to see properly with direct ophthalmoscopy. Use the comparison of looking through a keyhole (the pupil): the closer you are, the more you will be able to see!

It is sensible to start examining with the light on the ophthalmoscope quite dim, so that the animal gets used to your presence, before gradually increasing the rheostat brightness. This will be more comfortable for the patient rather than diving straight in with the light on full brightness!

Indirect ophthalmoscopy

This technique has advantages over direct ophthalmoscopy in that a large field of view is obtained allowing a quick overview of the fundus and binocular indirect ophthalmoscopy allows an appreciation of the fundus three-dimensionally (therefore depressions and raised areas are more easily imaged). Indirect ophthalmoscopy also provides an improved image through opaque corneas and lenses. However, the image is inverted and back to front and the magnification is less than with direct ophthalmoscopy. Ideally both techniques should be employed.

A condensing lens is required – usually 20D – although panretinal lenses offer a wider field of view (at greater cost!). Monocular indirect ophthalmoscopy uses a bright light source (e.g. Finhoff transilluminator) held at the examiner's ear, while binocular indirect ophthalmoscopy utilizes a special head or spectacle-mounted light source. The examiner shines the light into the dilated pupil from approximately 50 cm away. Once the tapetal reflex is obtained, the condensing lens is moved into the line of vision just in front of the animal's eye. An upside down and back to front image of the fundus is obtained. It is often necessary to move both the position and the angle of the lens to achieve the best image. This technique is more difficult to master than direct ophthalmoscopy but is certainly the preferred method of fundus examination among ophthalmologists. In addition, a teaching mirror can be fitted to most indirect headsets, allowing observers to view the same image as the ophthalmologist.

CLINICAL TIP

The Panoptic ophthalmoscope is a hybrid with the ease of use of a direct ophthalmoscope but a wider field of view (but still smaller than with the indirect). It is useful in general practice, although most people with an interest in ophthalmoscopy prefer the indirect instrument.

FURTHER EXAMINATION TECHNIQUES

Ophthalmic dyes

Fluorescein is an orange-coloured, water-soluble dye which stains corneal ulcers green. The intact cornea is lipophilic and as such it does not allow uptake of fluorescein but once the epithelium is damaged the dye will adhere to the exposed stroma (which is hydrophilic).

Impregnated strips (Fluorets, Chauvin) or single-use vials (Minims Fluorescein, Chauvin) can be used. If the strips are used they should be moistened with sterile saline before being lightly touched onto the conjunctival surface. Alternatively, a drop can be allowed to fall into the eye, as is done with the single-use vial. Multi-use preparations should be avoided as they are easily contaminated by potentially pathogenic bacteria. It is important to flush excess dye away with sterile saline to prevent false positives (e.g. irregularity of the corneal surface will allow pooling of the dye).

Although corneal ulcers with exposed stroma will stain bright green, very deep ulcers might not – Descemet's membrane does not take up the dye (because it is lipid-rich like the epithelium). Thus, if a pattern of staining around the edge but with a clear centre is

observed, this is likely to be a very deep ulcer and is potentially extremely serious. Fluorescein can be safely used even in ruptured ulcers or corneal perforations as sterile fluorescein does not harm intraocular contents. Sometimes if the cornea has been perforated a small stream of clear aqueous can be seen flowing through the fluorescein on the cornea (Seidel test). Staining is enhanced under blue and ultraviolet light (e.g. Wood's lamp).

CLINICAL TIP

The results of fluorescein testing are immediate and obvious and it is always useful to demonstrate ulcers to owners – it helps them to understand the nature of their pet's problem much more easily.

A second corneal dye, Rose Bengal, is sometimes used, mainly to detect ulcers due to herpes virus (FHV-1) in cats. It stains dead and devitalized epithelium a bright magenta colour and is more sensitive than fluorescein for the fine epithelial defects seen in herpetic keratitis. However, it is more irritant than fluorescein.

In addition to flushing excess dye away before interpreting the results of the test, it is sensible to flush again at the end of the ophthalmic examination so that the patient is less likely to rub due to discomfort.

Nasolacrimal duct system investigation

Initial assessment of the nasolacrimal drainage system can be made using fluorescein dye. A drop is placed in each eye and the nostrils are observed for the appearance of the bright green colour (Figure 1.4). Although the dye usually appears quickly, it can take up to 5 minutes. If no colour is seen, the mouth should be checked, since sometimes the nasolacrimal ducts have openings in the mouth – especially in brachycephalic breeds (both dogs and cats). This test is quite unreliable and false negatives are common. For this reason nasolacrimal flushing is often performed.

Under topical anaesthesia it is possible to cannulate the nasolacrimal punctae in dogs and rabbits, although cats often need sedation. The ducts are assessed for patency using sterile saline solution and this is more accurate than the fluorescein test mentioned above. Pliable plastic cannulae are recommended over metal ones in conscious animals. It is easier to cannulate the upper punctum in cats and dogs (but rabbits only have one large lower punctum!). A Nettleship's dilator, a pencil-like metal or plastic instrument, can be used to help to locate the punctae. In a normal patient, gentle flushing through the upper punctum, using sterile saline in a 5 ml syringe, will result in saline flowing through the lower punctum. This can be occluded with a finger and saline will drip from the nose as the syringe is gently depressed. Some dogs will swallow or cough as the solution drains into the mouth as well. The procedure is then repeated with the lower punctum cannulated – saline is initially allowed to flow through the upper punctum before occluding this and allowing the solution to drain down to the nasal ostium. If there is a blockage, then further investigation (including contrast radiography) might be indicated. Samples of the flushed material can be collected for bacterial (or occasionally fungal) culture and sensitivity.

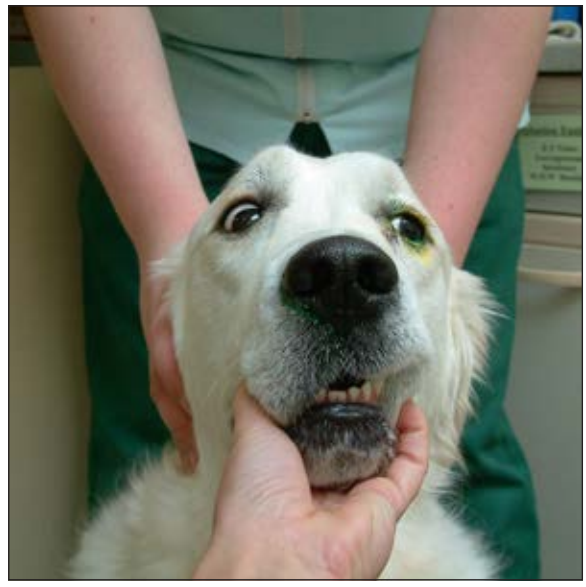


Figure 1.4 Positive nasolacrimal duct patency on the right side showing fluorescein at the nostril while none appears on the left, while excess dye is present around the eye. This side should now be cannulated and flushed to see if a physical blockage is present, as is discussed further in Chapter 18.

Tonometry

Tonometry is the measurement of intraocular pressure (IOP). Normal IOP in dogs and cats is 10–25 mmHg. IOP can vary according to several factors, including breed (often higher in terriers), age (reduces with ageing) and the degree of restraint needed to measure the pressure – holding tightly around the neck will compress the

jugular veins and could result in falsely elevated readings. Glaucoma is present if the IOP is clinically raised. Intraocular inflammation, i.e. uveitis, causes the pressure to drop below normal.

The instrument used to measure IOP is called a tonometer. Different types are available, but the most common ones in general use are the Schiotz tonometer (which measures by indenting the cornea – indentation tonometry) and the more expensive Tonopen (which measures by flattening the cornea – applanation tonometry). To measure IOP animals should be unsedated but topical anaesthesia should be instilled into the eyes. Readings should be taken from both eyes. With both types of instrument minimal restraint is required.

To use a Schiotz tonometer the patient must have its eyes in the horizontal plane. This usually means pointing their nose up to the ceiling – but without putting too much pressure around the neck! The footplate of the tonometer is allowed to rest on the cornea and the reading is taken from the scale. Several readings should be taken from each eye. A conversion chart comes with the instrument such that the reading on the scale can be converted to mmHg. This tonometer should not be used in very painful eyes or those with deep ulcers which could rupture. It is accurate to use but is cumbersome and some patients resent it. After each use the instrument should be dismantled and thoroughly cleaned.

The Tonopen is a more user-friendly tonometer. It should be calibrated prior to use each day – instructions are easy to follow and come with the instrument. A new tip-cover is used for each patient and after the application of topical anaesthesia the tip of the Tonopen is held perpendicular to the cornea and gently touched against it – the machine makes a beeping noise each time the cornea is touched and after three to five touches a different beep is heard and the pressure appears as a digital reading in mmHg. The small screen also records the degree of error in the reading, which should be less than 5%. If it is higher than this, the reading is not accurate and the pressure should be measured again. The Tonopen is small and light to use and the cornea does not need to be horizontal for measurements. This means that the patient can have their head in a normal position, thus making them less resentful to the procedure. It can also be used in very small patients such as kittens or rodents, as well as horses and farm animals. As with the Schiotz tonometer, care must be exercised with deep ulcers or corneal lacerations.

Another type of tonometer – the TonoVet tonometer – has recently been introduced into the veterinary market. This is a rebound tonometer which seems to be easy to use and accurate. It is likely to become more popular in the near future, although since it costs the same as the Tonopen, this could limit its appeal!

Gonioscopy

Gonioscopy is the examination of the iridocorneal (drainage) angle and is normally only performed by specialists or those with an interest in ophthalmology. However, an understanding of the concept is essential to the interpretation of types of glaucoma.

It is not possible to see the iridocorneal angle in dogs without the use of special contact lenses (goniolenses). Various different types of lens are available and personal preference usually dictates the type chosen. Koeppe and Barkan lenses are the most frequently encountered. These are placed on the anaesthetized cornea and allow the light to be bent (refracted) such that the drainage angle can be evaluated. The lenses basically allow the observer to look around the corner between the cornea and the iris. The technique is used mainly in dogs to check for abnormalities in this angle (goniodysgenesis) which predispose to the development of primary glaucoma. Figure 1.5 illustrates the normal appearance of the drainage angle and of goniodysgenesis in diagram form while Figure 43.2 is a photograph of a normal

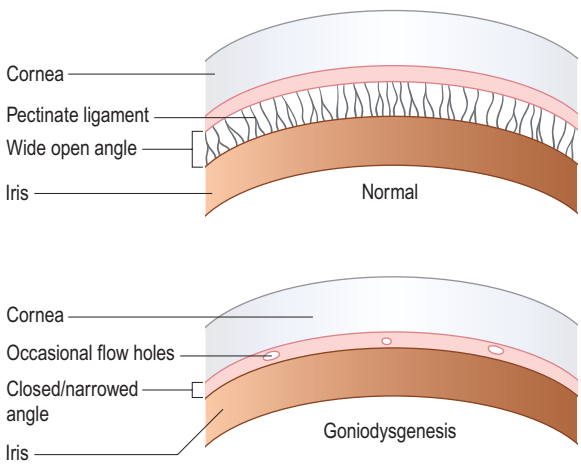


Figure 1.5 Diagram of the gonioscopic appearance of a normal drainage (iridocorneal) angle and one affected with goniodysgenesis.

drainage angle. If one eye is glaucomatous, the fellow eye is examined for evidence of goniodysgenesis.

Gonioscopy is also used to screen dogs for goniodysgenesis in breeds known to suffer from primary inherited glaucoma – for example, Basset hounds, flat coated retrievers and Welsh springer spaniels among others. The technique takes a lot of practice, and interpretation of the findings can prove difficult – even among ophthalmologists there can be a difference of opinion as to the significance of any abnormalities and as such it is perhaps best left for specialists to argue about!

Electroretinography

Electroretinography is not normally available in general practice. However, an understanding of its use is important which is why it is included in this section. The electroretinogram (ERG) is the electrical response recorded when the retina is stimulated with light. It is not a measure of vision (ERG will be normal in central blindness) but measures the integrity of the outer retinal layers, including the retinal pigment epithelium. It is used mainly to establish retinal function when opacification (usually cataract) prevents visualization of the fundus. In this situation it is very important to establish that the retina is working normally if cataract surgery is being considered.

Inherited retinal degenerations are common in pedigree dogs and cataracts frequently develop along with the degeneration – cataract surgery would not restore vision if the retina is atrophied which is why the test is so important. ERGs are also used to investigate cases of sudden blindness where the eye looks normal, in order to differentiate sudden acquired retinal degeneration (SARD) from optic neuritis or central blindness.

Patients are usually lightly sedated to perform an ERG, although in some animals general anaesthesia is preferred. A special contact lens electrode filled with a viscous gel (such as 2% hypromellose or carbomer gel) is placed on the cornea, while ground and reference needle electrodes are placed subcutaneously, usually at the nuchal crest and lateral canthus respectively. These are connected to a computer system and light source. Animals usually need to spend about 20 minutes in the dark (called dark adaptation) prior to ERG testing so that the retina is maximally sensitive to the stimulating light. Different protocols of light stimulation are used – single flashes of differing light intensity, flickers of light and so on – which can stimulate rods and cones separately. The response of the retina is recorded graphically and various measurements can be taken – for example, the amplitude of the response. If no response is recorded this indicates no functional retina and is found in end-stage progressive retinal atrophy and SARD.

Blood pressure measurement

The measurement of blood pressure is routinely performed in ophthalmic patients since hypertension is a common cause of ocular disease in both dogs and especially cats. Indirect measurement is routinely employed and two main methods are available – the Doppler and oscillometric systems. Thankfully these are becoming routine in general practice and details of how to measure blood pressure accurately in both dogs and cats are readily available elsewhere. Suffice to say that readings should be obtained with patients which present with hyphaema, sudden onset blindness due to retinal detachments, retinal haemorrhages and unexplained chorioretinitis lesions.

2 Laboratory investigation

The collection and handling of samples from the eyes is commonly performed in veterinary ophthalmology. Samples collected include swabs, scrapings and other samples for cytology, aspirates and biopsy specimens. They are useful for the investigation of suspected infectious disease, as well as immune-mediated and neoplastic processes.

Swabs can be taken from the conjunctiva, cornea or discharges. It is important to tell the laboratory exactly where the sample has been taken from. A sterile swab is gently rolled across the appropriate surface and topical anaesthesia is not usually required (it can affect interpretation of the results and can have a bacteriostatic effect which is not desirable when trying to isolate such pathogens!). If the sample is for cytology, then a dry swab is used and cellular integrity is usually preserved; however, there are better methods of sample collection for cytology, as discussed below.

The majority of swabs taken are for microbial analysis, and in this instance it is better to use a damp swab (moistened either with sterile saline or the appropriate culture medium). This gives a better yield of pathogens. It is important not to touch the swab against other parts of the eye since it could become contaminated with bacteria which are normally present on the ocular surface. Swabs should be labelled and dated prior to being dispatched. Previous and current medication should be detailed since this can affect interpretation of the results. If there is any delay between sample collection and processing, then the swab should be refrigerated (but not frozen). The culture requirements for bacteria, chlamydophila, mycoplasma, fungi and viruses can all vary and if there is any uncertainty about the best medium for collection and so on, then it is important to discuss this with the laboratory. In addition to culture, the sample should undergo susceptibility testing for aerobic bacteria.

Swabs taken for viral isolation or polymerase chain reaction (PCR) are usually for feline herpes virus (FHV-1) and special culture media are required. Combined media for FHV-1 and chlamydophila are routinely used. Swabs are usually obtained from both the conjunctiva and the oropharynx. These samples can be frozen if there is any delay in dispatch to the laboratory (over the weekend for example) but it is always sensible to check this with the laboratory first.

Scrapings are usually used for cytological examination. They are useful for inflammatory and neoplastic conditions mainly, but are of use in infectious processes as well. Samples can be taken from eyelids in exactly the same way as from other skin sites – a scalpel blade is used and the tissue gently squeezed during the scraping to express material from the glands and hair follicles. It is important that the eyelid margins are not damaged, and since the procedure can result in some swelling and discomfort afterwards, the animal must be monitored to prevent self-trauma. Scrapings from the conjunctiva and cornea require topical anaesthesia, and sometimes sedation of the patient. Holding a sterile cotton bud moistened with topical anaesthesia against the area to be sampled for 30–60 seconds will achieve a deeper numbing. A specialized instrument is available for scraping the cornea and conjunctiva – the Kimura spatula. However, most people use the blunt end of a scalpel blade for convenience. The material obtained is gently spread across a dry microscope slide.

Another way of collecting samples for cytology is to use a *cytobrush*. These are small nylon-bristled brushes that look like mascara-wands. They were initially designed for gynaecological use but are popular for ocular cytology since they are safe to use and maintain good cellular integrity as well as distributing the cells evenly. Once the material is collected by gently rolling the brush over the surface of the tissue, it is then rolled again over a dry microscope slide.

A final technique for sample collection for cytology is the *impression smear*. This is again very easy to perform – a dry microscope slide is pressed firmly against the

tissue to be sampled – any cells which readily exfoliate are transferred to the slide.

Whichever method of sample collection is used, the principles for processing the specimens are the same. Samples are gently spread on a clean microscope slide and air dried. In-house assessment using Diff-quik stains and Gram stains can be useful for bacterial identification, the presence of fungi and evaluation of cell types, but samples should also be sent to a cytologist for professional interpretation; it is useful to send unstained samples as well since further specialist stains can be used if indicated – for example, immunofluorescence stains for feline herpes virus and chlamydophila.

Aspirates can be taken from ocular tissue in the same way as sampling from elsewhere in the body. They are useful for investigating masses involving the adnexa, orbit or the eye itself. Abscesses, granulomas, tumours such as lymphomas, mast cell tumours and cysts can be differentiated. Inadvertent globe damage can be avoided by making sure that the animal is suitably restrained (with light sedation if necessary) and always directing the needle away from the globe. The area to be sampled is stabilized if possible, and the needle is inserted without the syringe attached. Suction is then applied, and the needle is redirected several times to get a representative sample. The syringe is removed, filled with air, and then reconnected to the needle. The sample obtained is then expressed onto a microscope slide. A second slide is used to gently spread the material. Occasionally it is necessary to centrifuge the sample prior to smear preparation if cellular yield is likely to be low (e.g. samples taken from the anterior chamber). In addition to cytology, microbial analysis can be performed on aspirates by placing the material on a moistened swab as detailed above.

Biopsy samples can be taken where the pathological process is too deep for scrapes and smears to be of use, or when the actual architecture of the tissues, rather than just the cells involved, needs to be examined. Samples should be placed in their correct orientation on card before putting in the fixative to make interpretation easier. If necessary, samples can be sent for microbial investigation as well as histology. Conjunctival biopsies are taken after the area has been desensitized with a cotton bud soaked in topical anaesthetic as mentioned above. An area of conjunctiva is tented up using a fine pair of rat-toothed forceps, and a piece is snipped off from below using small sharp scissors. No sutures are needed as the area heals rapidly. Some haemorrhage can be expected but this soon stops if gentle pressure is applied.

Eyelid biopsies are taken under general anaesthesia if wedge sections including the eyelid margin are needed. Sometimes punch biopsies can be taken with the animal sedated. Histology can be performed on biopsy samples from the cornea – these are obtained by surgical keratectomy under general anaesthesia.

A complete histological examination is frequently performed on enucleated globes. Examples include painful blind eyes due to refractive uveitis or hyphaema, glaucoma or endophthalmitis. Histology can help to understand the pathological processes which lead to loss of the eye and can be useful for ongoing case management – typing tumours for example. Correct handling of the specimen is required prior to submission to the laboratory. All tissue apart from the optic nerve should be trimmed from the globe – the histopathologist does not require eyelids and muscle if the pathology is contained within the eye. If there are concerns about the periocular tissues (e.g. tumour spread), then these can be submitted separately if necessary – leaving them on the enucleated globe will only reduce penetration of the fixative. Most globes will be fixed in formalin, but several specialist media are available and consultation with the pathologist might be useful.

All fixatives are potentially dangerous and so due attention to health and safety issues must be paid if handling them. It is important to give the pathologist as much information as possible – in addition to the animal's details and which eye is submitted, information about the clinician's findings should be included – a copy of their ophthalmic examination report together with diagrams of any focal lesions will be of great assistance. Details of any medical or surgical treatments plus any findings in the fellow eye should be submitted.

SECTION 1

EYELIDS

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3 Eyelids – introduction

An understanding of the basic anatomy and physiology of the eyelids is important since so many common ophthalmic problems are encountered in this area, especially in dogs, but less frequently in cats and rabbits as well. The tremendous variation in conformation exacerbated by selective breeding for specific appearance has contributed to the numerous pathological conditions in the area. These can be primarily ocular, such as entropion (rolling in of the eyelids) or distichiasis (extra eyelashes along the eyelid margin) or can be secondary to skin diseases or immune-mediated conditions for example.

The eyelids are dorsal and ventral folds of skin lined with palpebral conjunctiva and they form the palpebral fissure in which the globe is situated (Figure 3.1). The normal outline of the palpebral fissure is almond shaped (Figure 3.2) but many different variations occur with pedigree animals. The skin should be only loosely adherent to underlying structures but again this can vary from being excessively loose (e.g. in the Saint Bernard) to being very tight (e.g. the miniature poodle). The upper eyelid is more mobile than the lower in most species. A fibrous layer for support, the tarsal plate, is present within the eyelids. This is less well developed in dogs than cats.

In addition to providing support, the eyelid folds also contain both smooth and striated muscles and a good vascular and nerve supply. The most important muscle is the orbicularis oculi. The orbicularis oculi is mainly involved with eyelid closure and acts as a sphincter. It is located immediately below the skin and is innervated by the palpebral branch of the facial nerve (cranial nerve VII). Eyelid sensation is provided by branches of the trigeminal nerve (cranial nerve V). The usual rapid action of this muscle is important for globe protection and in the distribution of the tear film. Ocular pain can result in severe blepharospasm as the orbicularis oculi remains tightly contracted which can lead to further ocular pain with entropion and ulceration.

The normal position of the eyelids is resting against the ocular surface, and they slide over it during blinking. Many breeds of dogs and cats have poor lid–globe apposition and this results in ocular problems such as entropion or ectropion.

Palpebral ligaments are present medially and laterally and these fibrous bands stabilize the lids and help to maintain the shape of the palpebral fissure. The medial canthal ligament is well developed and having originated from the medial fibres of the orbicularis oculi muscle it inserts on the orbital rim of the lacrimal bone. In most dogs the lateral canthal ligament is poorly developed and really consists of lateral fascial attachments only. However, in some breeds, particularly those with broad skulls and relatively deep-set eyes such as the Rottweiler and mastiff types, the lateral canthal ligament is more defined but misdirected medially, resulting in some lateral canthal involution. This needs to be addressed when considering entropion surgery in these breeds and is mentioned in the case on complicated entropion later in this section.

Along the free margin of the lids (especially the upper lid) are a row of cilia (not true eyelashes). These are located close to, but not actually on, the eyelid margin. Within the eyelid margins are meibomian glands (also called tarsal glands) which secrete the oily (lipid) portion of the tear film. Their openings can be seen as a series of small grey dots (the grey line) along the eyelid margin. The eyelid margin itself is normally totally free from hairs and can be pigmented. The eyelid skin is very thin and easily becomes inflamed and oedematous (numerous mast cells within it contribute to this) – thus many clinical problems can occur in this area.

The eyelids have several functions. Clearly protection is important, along with the distribution of the tear film (assisting corneal nutrition) and propulsion of tears towards the nasolacrimal punctae for drainage. Foreign bodies such as dust and debris are removed via blinking.

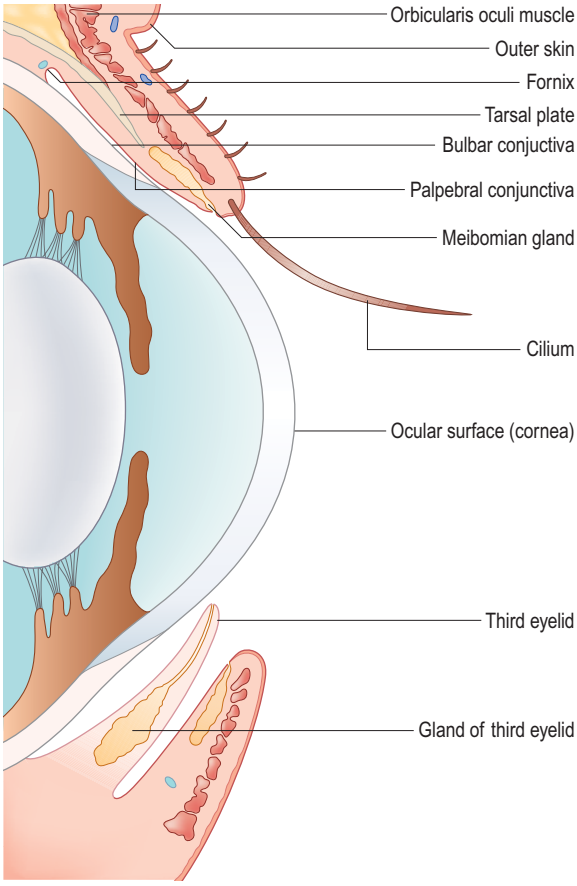


Figure 3.1 Anatomy of the eyelids.

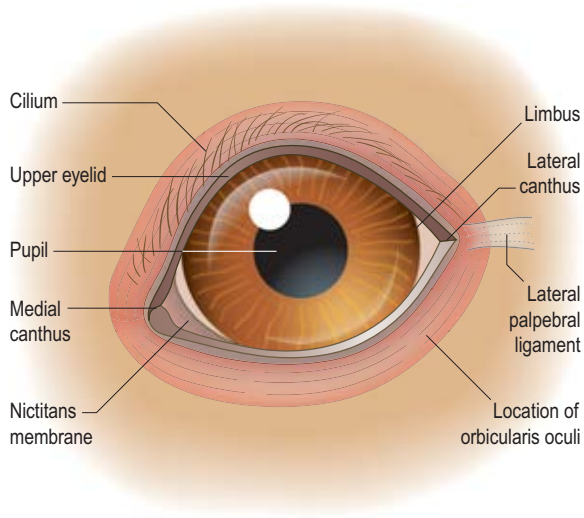


Figure 3.2 Normal canine palpebral outline and adnexal structures.

Closure of the eyelids reduces visual stimuli and assists sleeping while the cilia around the eyelids are important tactile organs.

The third eyelid or nictitating membrane (membrana nictitans) is a ventromedial fold of conjunctiva containing a T-shaped cartilage for support and the nictitans gland at its base (which together with the lacrimal gland produces the aqueous portion of the tear film). In addition to the nictitans gland, a true Harderian gland is present in rabbits, which secretes a more oily contribution to the tear film. The nictitans gland is attached to the cartilage of the third eyelid and the ventral periorbita by loose connective tissue. The leading edge of the third eyelid is often pigmented; however, in some dogs, particularly those with parti-coloured coats, one eye might have a pigmented border while the other is non-pigmented. This is a completely normal variation and should not be confused with inflammation (the pink third eyelid is more noticeable against the darker colour of the iris than the pigmented one and owners often think that the dog has conjunctivitis in one eye!). The third eyelid sweeps across the globe during blinking and assists in spreading the tear film and removing debris. It can be likened to a windscreen wiper in this respect. Unlike dogs, cats have some striated muscle fibres present within their third eyelids, which allow for active protrusion of the membrane.

The most common conditions affecting the eyelids include abnormalities in eyelid position, inappropriate eyelashes and prolapse of the gland of the third eyelid, all of which will be discussed together with blepharitis, since the latter can be overlooked in general practice or misdiagnosed as purely conjunctivitis with understandably limited response to treatment.

4 Blepharitis

INITIAL PRESENTATION

Colour change red/swollen, pain (irritation), abnormal appearance

PRESENTING SIGNS

Blepharitis, inflammation of the eyelids, is seen relatively commonly in general practice but is not always immediately diagnosed as such. Patients are more commonly dogs than cats and the condition is more frequently bilateral than unilateral. Owners report that the dog is irritated by its eyes with frequent attempts at rubbing. Some mucoid or mucopurulent discharge is likely and the owner is also likely to mention that the eyes look red. This can be due to a combination of conjunctival hyperaemia, eyelid hyperaemia and eyelid erosions or areas of ulceration. The condition is most likely to be insidious and gradually progressive in onset, although occasionally acute presentations do occur. Although focal forms, such as chalazia and styes, do occur, we will concentrate on the diffuse cases since the former are typically straightforward to diagnose and treat.

CASE HISTORY

The history will vary somewhat according to the underlying aetiology for the blepharitis. Thus there might be a history of slight conjunctivitis for a few days followed by a marked eyelid reddening and swelling together with a purulent ocular discharge in the case of a staphylococcal blepharitis. There might be a young puppy, less than 3–4 months old, or more than one in a litter, which has swollen eyelids then develops ulceration and crusting around the lids and nose, together with general malaise, pyrexia, inappetance and clearly has developed a juvenile pyoderma. A young Labrador retriever might present seasonally with itchy eyes and foot-chewing associated with atopy. A dog which has recently travelled to Mediterranean countries could present with blepharitis and eyelid crusting together with uveitis, and *Leishmania* would be strongly suspected in such an example. In these instances the definitive diagnosis can be reached from the history and clinical examination, with perhaps response to treatment, or just a single diagnostic test to confirm the diagnosis. However, other, often more chronic presentations are less straightforward, perhaps with a several week history of bilateral swelling and redness with intermittent rubbing, or simply a change in appearance of the periorbital area. Clearly further work-up is required in these cases.

CLINICAL EXAMINATION

Since many forms of blepharitis are part of a generalized dermatological condition, careful general clinical examination with specific concentration on the skin is required. The causes of blepharitis can be as varied as those for generalized dermatitis and sometimes the ocular symptoms can be the first signs of a more general involvement. Secondary bacterial infection is common and can mask the underlying aetiology. Careful examination of the feet, external ear canals and all mucocutaneous junctions is advised.

Ophthalmic examination will reveal eyelid swelling and hyperaemia, possibly with crusting or exudative erosions close to the eyelid margins (Figure 4.1). Some lesions are erosive and a serosanguinous discharge can be present (Figure 4.2). Periorbital hyperaemia or alopecia could be signs of self-trauma. Close examination of the eyelids themselves might reveal multiple swellings along the lid margin in the location of the meibomian



Figure 4.1 Six-year-old male spaniel with severe blepharitis. Note the lid swelling, hyperaemia and exudative lesions but marked lack of actual ocular involvement. This was due to a staphylococcal infection and responded well to systemic cefalexin.



Figure 4.2 Five-year-old rough collie with ulcerative blepharitis at the medial canthus. The condition was bilateral and similar lesions were noted at the lip commissures. Skin biopsy confirmed immune-mediated disease.

glands – suggestive of a meibomianitis (Figure 4.3). Schirmer tear test readings should be checked in all cases.

Conjunctival hyperaemia will accompany most cases of eyelid inflammation and can be incorrectly assumed to be the primary problem. Sometimes the eyelid swelling can distort the palpebral fissure, resulting in such secondary problems as entropion, ectropion and trichiasis. Corneal ulceration can also develop which will further exacerbate the blepharospasm and a vicious cycle can



Figure 4.3 Two-year-old Labrador retriever with meibomianitis – note the multiple small lid swellings along the palpebral conjunctiva.

be established. Normally there is no intraocular involvement with cases of blepharitis, but if any uveitis, for example, is present it might suggest either an immunological condition such as uveodermatological syndrome, or a protozoal infection such as Leishmania.

DIFFERENTIAL DIAGNOSES

These are outlined in Tables 4.1 and 4.2.

CASE WORK-UP

Once a thorough general clinical and ophthalmic examination has been performed the appropriate diagnostic tests can be initiated. In many cases swabs for bacterial culture and sensitivity should be taken – both from the ocular discharge and from exudations from the lid margins themselves. Samples for parasitic isolation should also be considered so several swabs might be required in different transport media. Impression smears of the lid margins can be useful, and scrapes and hair plucks to look for parasites can be rewarding. If *Leishmania* is suspected then serological testing is advised. Additionally, many cases benefit from biopsy – especially since the commonly found bacterial infection might not be the underlying problem – with allergic reactions and immune-mediated disease high on the list of potential underlying conditions. Biopsy specimens should include the eyelid margin whenever possible – a small wedge resection is removed under general anaesthesia and sutured with 6/0–4/0 soft material (such as polyglactin 910, although care should be exercised using braided suture in the face of infection). If meibomian gland involvement is present, samples of inspissated matter

Table 4.1 Causes of blepharitis in dogs

		Clinical signs	Diagnosis
Parasitic	Demodectic mange Sarcoptic mange	Lesions not confirmed to eyelids Sarcoptic mange intensely pruritic	Skin scrapes Occasionally biopsy
Bacterial	Staphylococcal infection Juvenile pyoderma	Exudative eyelid lesions Moderate irritation Purulent discharge	Culture and sensitivity Cytology (Gram stain)
Fungal	Ringworm *Microsporum canis* or *Trichophyton mentagrophytes*	Young dogs Alopecia, crusting and scaling Commonly periorbital	Hair plucks Fungal culture
Protozoal	*Leishmania*	Scaly periorbital lesions, uveitis, ulcerative dermatitis	Travel to endemic regions Serology Skin biopsy
Photosensitization		Non-pigmented hair and skin affected	Skin biopsy Improves when kept out of sunlight
Immune mediated	Atopy Drug induced Food allergy Uveodermatological syndrome Pemphigous complex	Various presentations Usually some pruritus and other skin lesions	History Skin biopsy Allergy testing Exclusion diet
Neoplastic	Any large lid mass	Mass or ulcerative lesion	Cytology Skin biopsy

Table 4.2 Causes of blepharitis in cats

		Clinical signs	Diagnosis
Parasitic	*Notoedres cati*	Eyelids, ears, periorbital skin Intense pruritus	Skin scrapes
Bacterial	Rare in cats Facial pyoderma in Persians Mycobacterium – feline leprosy	Ulcerated nodules Feet also affected Risk in immunosuppressed cats	Cytology Culture Demonstration of acid-fast organisms
Fungal	Ringworm	Alopecia, crusting and scaling Commonly periorbital	Hair plucks Fungal culture
Protozoal	*Leishmania* rare in cats	Scaly periorbital lesions, uveitis, ulcerative dermatitis	Travel to endemic regions Serology Skin biopsy
Viral	Feline pox virus (rare)	Ulcerated crusty papules and nodules	Skin biopsy and viral isolation
Photosensitization		White cats predisposed	Skin biopsy Improves when kept out of sunlight
Immune mediated	Atopy Drug induced Food allergy Pemphigous complex	Various presentations Usually some pruritus and other skin lesions especially around face	History Skin biopsy Allergy testing Exclusion diet
Neoplastic	Squamous cell carcinoma most common	Mass or ulcerative lesion	Cytology Skin biopsy

can be curetted from the swellings and sent for both bacterial culture and cytology. Biopsy samples from other skin lesions should be considered. If atopy is suspected, then intradermal skin tests can be performed.

NURSING ASPECTS

These patients are often quite uncomfortable and try to rub the eyes – this should be prevented and the judicious use of Elizabethan collars can help. Cleaning away of the discharge can be painful, especially if the lids are ulcerated, and should be done very gently using soft swabs soaked in warm saline solution. Hot and cold compresses are useful, particularly in cases of meibomianitis, where they can help to loosen the inspissated discharge. The use of diluted povidone–iodine solution (1 : 10 for the eyelids but 1 : 50 if any is used near the conjunctival sac) can be beneficial as an antiseptic wash. Some cases will require the application of ointments or creams to the lids – if these are not ophthalmic preparations then care is required to prevent inadvertently allowing conjunctival or corneal contact. Placing a bland ophthalmic lubricant such as soft paraffin ointment into the eye before the application of the skin treatment can prevent this. In addition to topical therapy, many patients will also be on systemic drugs as well.

EPIDEMIOLOGY

For details regarding generalized skin complaints with eyelid involvement such as atopy, juvenile pyoderma and the pemphigus group of diseases, the reader is referred to general dermatology texts. Many cases of blepharitis involve a bacterial infection, most often with staphylococcal species, followed by an inappropriate allergic or sensitivity reaction to the bacterial toxins. This is the case for chronic blepharitis and meibomianitis in dogs. With the latter it is proposed that keratin plugs block the openings to the meibomian glands which provide an ideal environment for bacterial growth. Bacterial lipases are produced which hydrolyse the meibomian secretions to free fatty acids, which in turn cause a toxic reaction exacerbating the localized inflammatory process. Some cases of bacterial blepharitis are simply an extension of bacterial conjunctivitis but bite wounds in both dogs and cats are also frequently implicated. In addition to hypersensitivity reactions to bacterial toxins, some patients develop similar responses to topical antibiotics, or the preservatives therein (Figure 4.4).

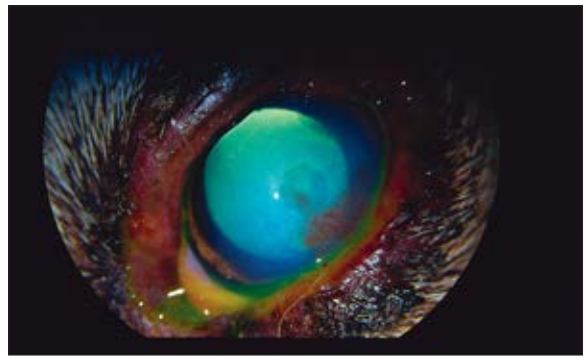


Figure 4.4 Fifteen-year-old Jack Russell terrier with severe blepharitis. A non-healing corneal ulcer had been treated with multiple different topical antibiotics, including gentamicin and chlortetracycline, and the lid inflammation developed several weeks after the ulcer started to heal. The corneal scarring and oedema are clearly visible. The lid inflammation was suspected to be a hypersensitivity reaction to the topical medication and resolved completely on cessation of all topical medication, although a week of oral cefalexin and carprofen were given.

Parasitic infections such as demodectic mange do have a predilection for the head and eyelid region. Many cases are self-limiting in young dogs.

Leishmania spp. are transmitted by sandflies and the protozoal infection is endemic in the Mediterranean region. Travel to the area is clearly necessary for the condition to develop.

CLINICAL TIPS

- Take samples for bacterial culture and sensitivity both from ocular discharge and the lid lesions themselves
- Do not forget Schirmer tear tests – some cases also have keratoconjunctivitis sicca
- Check the rest of the animal carefully for any evidence of skin disease
- Check travel history
- If initial response to treatment is not as expected, re-evaluate and consider biopsy

TREATMENT OPTIONS – MEDICAL

Clearly the specific treatment regime will depend upon the underlying aetiology for the blepharitis. General hygiene and preventing self-trauma will apply to all cases.

For a primary bacterial eyelid infection a broad spectrum systemic antibiotic such as cefalexin is advised, often for 2–4 weeks. Remember that the eyelids are part of the skin, and require systemic medication. Topical antibiotics are not always necessary – some patients will benefit but others might develop a hypersensitivity reaction, exacerbating their clinical signs. If any patient worsens on treatment a drug sensitivity should be considered – all treatment is withdrawn for 48 hours and the patient re-evaluated.

Conjunctivitis is a feature of many cases of blepharitis and tear film abnormalities are frequently unrecognized – involvement of the meibomian glands will affect the lipid layer of the tear film and evaporation can lead to qualitative tear film abnormalities. Thus the use of a topical lubricant such as a carbomer gel should be considered while more specific treatment is implemented. If the eyelids are particularly inflamed a systemic anti-inflammatory agent is required. A non-steroidal drug is chosen unless an immune-mediated component is suspected or confirmed on biopsy results. Then systemic prednisolone is normally used at immunosuppressive doses which are reduced as soon as a clinical improvement is seen, although often several weeks at a tapering dose are required. An alternate treatment regimen for blepharitis and meibomianitis involves doxycycline and

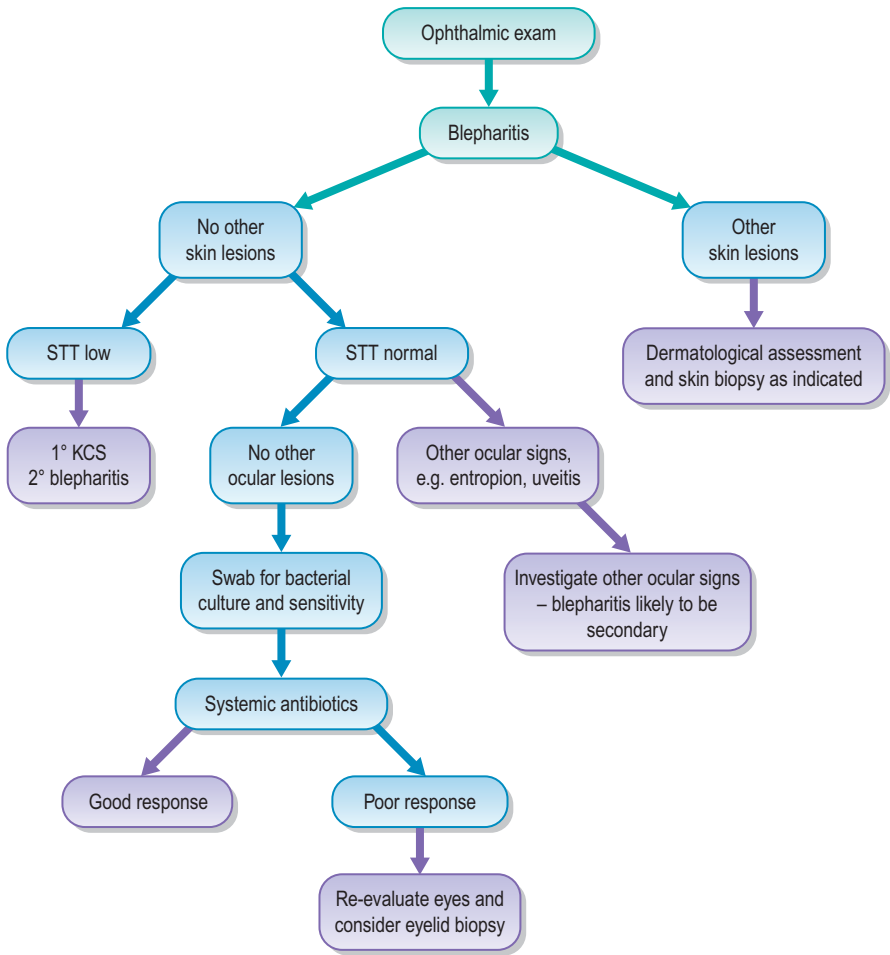


Figure 4.5 Decision-making tree – blepharitis.

niacinamide, both at 250–500 mg three times daily per dog, for several weeks. This avoids the need for systemic steroids.

Parasitic blepharitis will respond to the usual systemic medications and washes but care must be exercised to prevent ocular damage with the latter (see nursing section above). Many dermatophyte infections are self-limiting but occasionally require treating if the crusting and scaling are excessive or there are concerns regarding the zoonotic potential. The environment should also be disinfected.

TREATMENT OPTIONS – SURGICAL

Surgery is rarely required for cases of blepharitis. Occasionally granuloma type lesions along the lids can be surgically excised and curetted. For chronic meibomianitis, surgical debridement and cryosurgery have been advocated but since the eyelid skin is very delicate and surgical damage to the lid margins could have serious long-term repercussions for the patient, these techniques are best left in the hands of specialists.

PROGNOSIS

Generally the prognosis for cases of blepharitis is good, although a protracted treatment course lasting several weeks might be required. Providing owners are aware of this from the outset then compliance is usually good. The zoonotic potential for dermatophytes and *Leishmania* should be pointed out to owners. The prognosis for the latter is guarded. If the blepharitis is associated with a general dermatological condition such as atopy or pemphigus then clearly the prognosis for a complete cure is less good but long-term control can be achieved.

CASE EXAMPLE 4.1

Patient details: Black Labrador retriever, 13 months old, male

PRESENTATION AND HISTORY

The patient was presented with red, swollen eyes. He was rubbing the eyes with his paws and on the carpet and occasionally this resulted in bleeding. A moderate mucopurulent discharge was present. The dog had suffered from a short episode of presumed bacterial conjunctivitis 2 months previously which had responded well to a week of twice daily fusidic acid. One month later the eyelids became red and inflamed but there was no response to the same treatment and the condition had gradually progressed over the ensuing weeks.

CLINICAL SIGNS

The Labrador was typically bright and bouncy with no systemic problems on initial presentation. Both eyes were similarly affected although the left was slightly worse. Although the eyes were held open most of the time, the dog did try to rub them during the clinical examination and was clearly uncomfortable when the periorbital area was touched. Multiple swellings were present along the lid margin extending a few millimetres into the surrounding skin, and some of these were ulcerated and scabbed (Figure 4.6). There was an obvious 'grey line' along the eyelid margins marking the openings of the meibomian glands. The palpebral conjunctiva was markedly hyperaemic; the bulbar conjunctiva was not inflamed. A moderate mucopurulent discharge was noted. No corneal ulceration was noted and intraocular contents were normal.



Figure 4.6 Left eye of Labrador on initial presentation.

WORK-UP

Swabs were taken from the ocular discharge in both eyes, and from an ulcerated skin lesion in the left. These revealed a mixed growth of mainly commensal bacteria but a moderate growth of coagulase-positive *Staphylococcus* spp. was cultured with no significant antibiotic resistance.

TREATMENT AND OUTCOME

The dog was assumed to have meibomianitis and blepharitis secondary to bacterial infection and possibly also a hypersensitivity component. The details of treatment and the response to it are outlined in Table 4.3. As can be seen it was a long and involved case – the dog responded very well to the immunosuppressive doses of prednisolone along with the cefalexin but did not tolerate the higher doses of steroids and the eyes worsened each time the steroids were reduced. Swapping to the combination of doxycycline and niacinamide was successful but the improvement in the lids took much longer and was less dramatic than on the steroids. However, the dog was well in himself on this new regimen and the owner was very compliant with the management. Thankfully the dog did make a full recovery, although this took 6 months to achieve! No recurrence has been reported since.

This case highlights the fact that the management of cases of meibomianitis/blepharitis can be complicated and good owner compliance is required from the outset.

Table 4.3 Treatment and outcome for case example

Treatment	Duration	Response
Cefalexin 20 mg/kg twice daily Dexamethasone ointment twice daily	2 weeks	Slight improvement with less exudation of lesions but swelling still significant
As above but also carprofen orally 2 mg/kg/day	2 weeks	Minimal improvement
Cefalexin continued carprofen stopped Prednisolone 1 mg/kg twice daily	1 week	Dramatic improvement with minimal swelling remaining and no irritation at all
Cefalexin continued Prednisolone reduced to 0.5 mg/kg twice daily	1 week	Continued improvement Dog becoming PUPD and lethargic
Cefalexin continued Prednisolone gradually reduced to 0.5 mg/kg every other day	3 weeks	Lids minimally swollen Dog well in himself
Continue just prednisolone as above	2 weeks	Eyelid swelling returned with multiple nodules, exudation and self-excoriation again. Discussion with owner – surgical curettage of lesions and skin biopsy
Skin biopsy under GA and lesions debulked Repeat samples for bacterial culture and sensitivity Cefalexin restarted Prednisolone increased to 0.5 mg/kg twice daily again Dexamethasone ointment restarted twice daily	2 weeks	Biopsy showed a pyogranulomatous reaction centred around meibomian glands Scant growth of *Corynebacterium* sensitive to cefalexin Eyelids dramatically improved again Dog not tolerating high dose steroids very well
Prednisolone reduced as per previous regimen	3 weeks	Eyes worsened again Dog still lethargic, PUPD, dry coat, etc.
Doxycycline 500 mg and niacinamide 500 mg both three times daily Prednisolone reduced to 0.5 mg/kg every other day and then stopped	2 weeks	Slower improvement than with the steroids but definite gradual amelioration of signs Dog's general demeanour improved
Continue doxycycline and niacinamide as above	6 weeks	Good resolution with minimal lid scarring Dog well in himself

PUPD, polyuric/polydipsic.

5 Complicated entropion

INITIAL PRESENTATION

Discharge, pain

PRESENTING SIGNS

Entropion is an in-rolling of the lid margin such that hairs rub against the cornea. Cases typically present young, 4–12 months of age, and although sometimes unilateral the potential for both eyes to become affected is high. Normally the patient will have a painful watering eye and this is why the owner will come to see you. Signs of irritation are usually present with attempts at rubbing and sometimes the animal will be head shy. Pedigree dogs are most commonly affected and a wide variety of types of entropion is encountered depending on the anatomical weakness with each particular conformation. Thus, in sporting breeds such as the Labrador retriever, Weimaraner and other gundogs, the lateral half of the lower lids is typically affected. In giant breeds such as the Great Dane and St Bernard a combination of upper lid entropion laterally plus medial and lateral entropion with central ectropion of the lower lid is common – so-called diamond eye. The Shar Pei often has 360 degree entropion and can present with problems at 2–3 weeks of age, immediately after the eyelids have opened. Older cocker spaniels suffer from facial droop with an upper lid entropion and trichiasis (atonic entropion). Brachycephalic breeds, including Persian cats, suffer from a medial entropion of the lower lid. The list could go on and on! Secondary entropion should not be overlooked – any trigeminal pain can result in blepharospasm which can progress to entropion.

CASE HISTORY

Typically the history is of mild ocular discomfort and wetness for several weeks, possibly intermittently initially (worse when the animal is tired) but with progression to quite obvious blepharospasm such that the dog does not hold the eye fully open at any time. Occasionally owners will have already tried treating the dog which might have become very head shy and resent any further intervention.

CLINICAL EXAMINATION

No abnormalities are present on general clinical examination but before doing this, and prior to handling the patient at all, it is important to assess the eyes from a distance. This will reveal the true degree of blepharospasm and discomfort before it is exacerbated by the hands-on examination. Evidence of bilaterality, the nature of any discharge and self-trauma should be visible before holding the animal for examination. The discharge is typically serous in nature and present both at the classic medial canthal area but also extending along the area of eyelid in-turning. During the ophthalmic examination care must be taken not to pull the facial skin out of position too much as eyelid position can be dramatically altered, especially in breeds with loose skin on their faces.

Menace responses should be normal providing the dog is able to open its eyes sufficiently to be assessed! However, they might trigger an intense blepharospasm such that further examination is difficult. Providing there is no evidence of keratoconjunctivitis sicca (in which case a Schirmer tear test should be performed first), a drop of topical anaesthetic can be applied to the cornea to relieve the trigeminal pain and ease further examination. This will also remove any spastic component to the entropion such that the degree of anatomical entropion

can be assessed. The lid margin will typically be wet, and is often slightly depigmented along the area of inversion (Figures 5.1 and 5.2). It can be gently rolled out but will immediately start to roll inwards again. Many dogs will have elevation of their nictitans membranes and possibly some degree of enophthalmos as a result of chronic corneal pain and this should also be reduced following the application of topical anaesthesia. Conjunctival hyperaemia will be present and varying degrees of corneal damage will be noted. This can range from very slight irregularity of the corneal epithelium, fine superficial vascularization, overt ulceration of differing depths and scarring and granulation tissue. In very severe cases of entropion, particularly in young Shar Pei puppies, the rubbing from the eyelid hair can have been so severe as to cause almost total corneal granulation or sometimes corneal rupture (see Case example 5.1).

In addition to assessing the position of the eyelids it is important to consider the canthi, especially in large breed dogs. Normally the medial and the lateral canthus should be in the same horizontal plane, but with laxity at the lateral canthus – for example, in giant breeds with 'diamond eye' – this can slip ventrally giving a sloping appearance to the palpebral fissure (Figure 5.3). The skin at the lateral canthus can be rolled in (lateral canthal involution) which is common in breeds with large skulls such as Rottweilers and retrievers, especially male dogs, and is due to a tight and abnormally positioned lateral canthal ligament. This sometimes needs addressing along with the entropion, discussed in the case below (see Case example 5.2).

Other ocular abnormalities such as distichia might accompany the entropion and it is essential to determine whether they are significant in the pathogenesis. Similarly, if a corneal ulcer is present this might have been the primary problem, with entropion developing secondary to the trigeminal pain. Secondary entropion can follow from corneal sequestrum formation in cats (Figure 5.4). A mild reflex uveitis might be noted on intraocular examination but often the rest of the examination is unremarkable.



Figure 5.1 Ten-month-old Rhodesian ridgeback with typical lower lid entropion.



Figure 5.2 Same dog having gently pulled down the lower lid. Note the wetness along the hairs and the depigmentation of the lid margin.



Figure 5.3 Eight-month-old Neapolitan mastiff with severe 'diamond eye'.

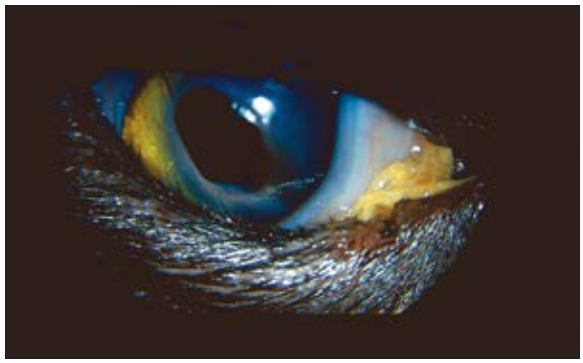


Figure 5.4 Large corneal sequestrum with lower lid entropion in an 8-year-old domestic short haired cat. Did the sequestrum cause the entropion or vice versa?

DIFFERENTIAL DIAGNOSES

- Primary entropion – conformational and breed related
- Spastic entropion – secondary to other ocular pain such as ulceration, ectopic cilia, severe uveitis, enophthalmos, blepharitis, phthisis bulbi and so on

CASE WORK-UP

Minimal work-up is required since the entropion can be easily diagnosed on clinical examination and the use of topical anaesthesia will assist in determining the degree of spastic versus anatomical entropion. If the lid inversion is purely due to trigeminal pain, then this will be resolved fully following the application of two drops of topical anaesthetic 30 seconds apart. If it is still not clear how significant the entropion is, then temporary everting sutures can be placed under general anaesthesia (see below).

NURSING ASPECTS

Other than the application of any topical and systemic medication which has been prescribed and the prevention of self-trauma, no specific nursing instructions apply to cases of entropion.

EPIDEMIOLOGY

Most cases of primary entropion are breed related, with an inherited component. It is not uncommon for several related animals to be affected with entropion and breeding from such individuals is to be discouraged. Unfortunately some breeders, particularly those of the Shar Pei, seem to consider it 'normal' for puppies to require eyelid tacking at only a few weeks of age followed by sharp surgery when only a few months old. This attitude – that the condition is not severe and easily managed by surgery with few long-term sequelae, such that it is not really important – is a worrying trend among some breeders who deliberately choose animals with poor eyelid anatomy for breeding simply because that is the way they like the animals to look. Although the specific inherited factors contributing to entropion have not been identified, and a multifactorial aetiology is likely (as with hip dysplasia), the strong breed predisposition justifies advising against breeding from affected individuals and breed clubs should be encouraged to try to reduce the incidence of the condition in their particular breed.

The wide variation in skull shape, eye size, skin thickness and actual globe positioning all contribute to the development of entropion. There is a complex relationship between the relatively weak tarsal plate (which should provide support and eyelid rigidity), pressure on the lids from the globe, tone within the orbicularis oculi muscle and the length of the palpebral fissure, all of which interact and result in eyelid involution. Some patients have a very mild, borderline entropion, which is only intermittent and causes minimal clinical signs until they develop ocular pain for another reason such as a corneal ulcer, which then causes blepharospasm and 'tips the balance', such that the entropion then becomes more severe and needs addressing as well as the ulcer.

Secondary entropion due to spasm in the orbicularis oculi muscle results from many painful ocular conditions, particularly if chronic or not correctly treated. In such cases surgical repair of the entropion will only result in temporary improvement and careful re-examination will be necessary to elucidate the underlying problem.

CLINICAL TIPS

- Examine the patient from a distance (e.g. in the waiting room) before any hands-on examination since they can be head shy and difficult to assess once the face has been touched
- Examine the patient at their level – if you only examine from above, making them look up at you, the true lid alignment will be altered,

especially in breeds with heavy brow folds – these fall forwards each time it looks down and will exacerbate any upper lid entropion
- Do not delay surgery until the patient reaches maturity – modern anaesthetic agents allow safe general anaesthesia even in young puppies and postponing surgery can result in corneal disease, more severe entropion and chronic discomfort for the patient
- Use topical anaesthesia to assess how much lid inversion is due to trigeminal pain (spastic component) and how much is truly anatomical
- Use the appearance of the patient post-topical anaesthesia to make the surgical plan and do not alter this once the patient has been premedicated or anaesthetized

TREATMENT OPTIONS – MEDICAL

Medical management alone is not sufficient in cases of primary entropion, and unfortunately many cases of secondary entropion (such as due to corneal ulceration) also require surgery, since once the blepharospasm–trigeminal pain cycle has been established the entropion rapidly becomes permanent and not just spastic secondary to the pain.

The use of topical medication in complicated cases of entropion should be undertaken with caution. As mentioned above, many patients are extremely head shy and resent any attempts at examination, cleaning or medication of their sore eyes. If medicating is becoming a fight each time the owner attempts to do so, then most patients are better off with minimal intervention. Although systemic antibiotics are not usually required following entropion surgery, in complicated cases their judicious use can be beneficial over topical preparations. Systemic NSAIDs are often used as well.

TREATMENT OPTIONS – SURGICAL

For uncomplicated cases of entropion a simple Hotz-Celsus procedure under general anaesthesia, removing an ellipse of skin parallel to the in-turning, is very successful. The steps for how to do this are detailed in Table 5.1. However, we will concentrate on the more complicated cases – and only the more commonly encountered types of these. Some very complex lid abnormalities, such as diamond eye with lateral canthal laxity, might be better referred to an experienced ophthalmic surgeon.

Rottweiler type (Case example 5.1)

Entropion in large-headed breeds, such as the Rottweiler, can be challenging to treat – although on initial

Table 5.1 Steps for performing a Hotz-Celsus procedure

	Procedure	Effect
1	Decide on area of skin requiring excision prior to sedation but after the application of topical anaesthetic	Removes the spastic component of the entropion leaving purely the anatomical abnormality
2	Perform initial skin incision parallel to lid margin, 1–2 mm away	Close as possible to lid margin for most everting effect but sufficient space to place sutures
3	Incision depth to include orbicularis oculi	Sufficiently deep to have effect
4	Second incision shaped to provide exact out-rolling of abnormal lid margin	Two incisions meet at gentle point, not abruptly
5	Excise skin between two incisions	Use rat-toothed forceps and fine scissors such as tenotomy scissors
6	Suture carefully with 6/0 material in simple interrupted pattern	Place first suture centrally and work out to prevent uneven placement
7	Ensure knots are away from lid margin and cut suture ends short	Ensure nothing can rub to irritate the cornea



Figure 5.5 Fifteen-month-old male Rottweiler. Severe lower lid entropion with marked lateral canthal involution and large ventrolateral corneal ulcer. This photograph was taken after the application of topical anaesthesia. See Case example 5.1.

examination it might appear as a straightforward lower lid entropion. Unfortunately, this breed, and others with similar anatomy, frequently develops very intense blepharospasm resulting in corneal ulceration in the ventrolateral quadrant (Figure 5.5). This can develop prior to your intervention, or sometimes a few days following a simple Hotz-Celsus surgery. It is not uncommon for the patient to look fine on a 2-day post-surgery check-up, only to return 48 hours later with a very sore eye with ulceration, marked blepharospasm and further entropion – even with the sutures from the initial surgery now rubbing and exacerbating things. Obviously owners are never happy if this situation develops!

To try to minimize such complications it is important to assess the true entropion prior to surgery. As mentioned previously, examination after the application of topical anaesthesia, but before any sedation, is the time to choose your surgical approach, and not to change this plan later since once sedated or under general anaesthesia the eyelid position will change dramatically. However, once anaesthetized it is important to check the lateral canthal ligament in these breeds. Normally when placing a finger between the globe and lid at the lateral canthus it is easy to extend the finger a little way laterally with no resistance. If a tight lateral canthal ligament is present, this will feel like a taut band preventing you from extending your finger further. In addition to performing the normal Hotz-Celsus entropion procedure it might be necessary to address this ligament. The simplest way to do this is to continue the excision of the ellipse of skin

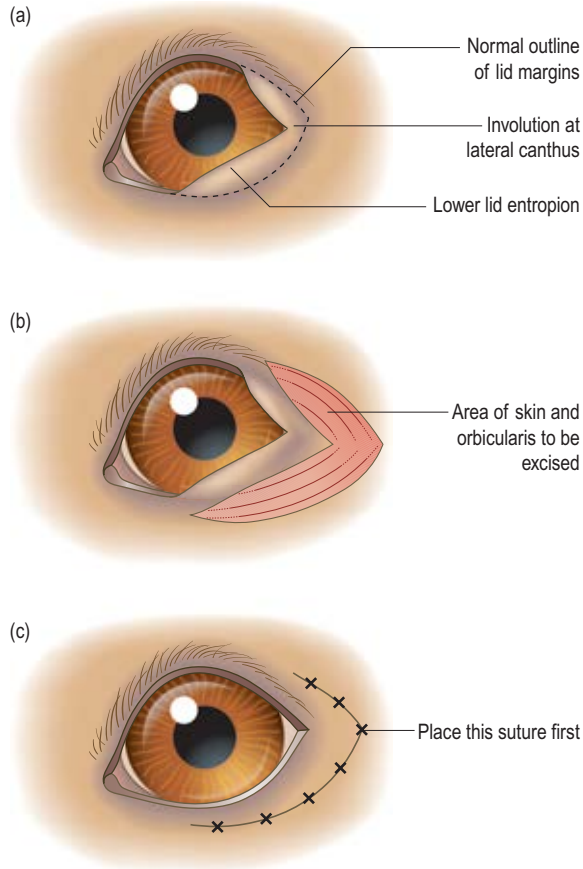


Figure 5.6

(a) Schematic diagram of lower lid entropion with lateral canthal involution.

(b) Modified Hotz-Celsus in arrowhead shape removing skin and some orbicularis oculi muscle.

(c) Surgical outcome.

around the canthus, in an arrowhead shape (Figure 5.6). This has the effect of pulling out the canthal skin and reducing the involution.

In some patients this is not sufficient and resection of the ligament is also required – this can be performed by gentle blunt dissection using small sharp scissors (Figure 5.7). Once the ligament is transected, the canthus will be released and the eyelid fissure will seem much bigger. The remaining entropion can then be assessed – a Hotz-Celsus resection will probably still be required. However, if the eyelid now appears very long, it might be necessary to also remove a full thickness wedge of lower eyelid to shorten the palpebral fissure and prevent the lid from 'flopping' back inwards again, despite your removal of an ellipse of tissue to evert the lid.

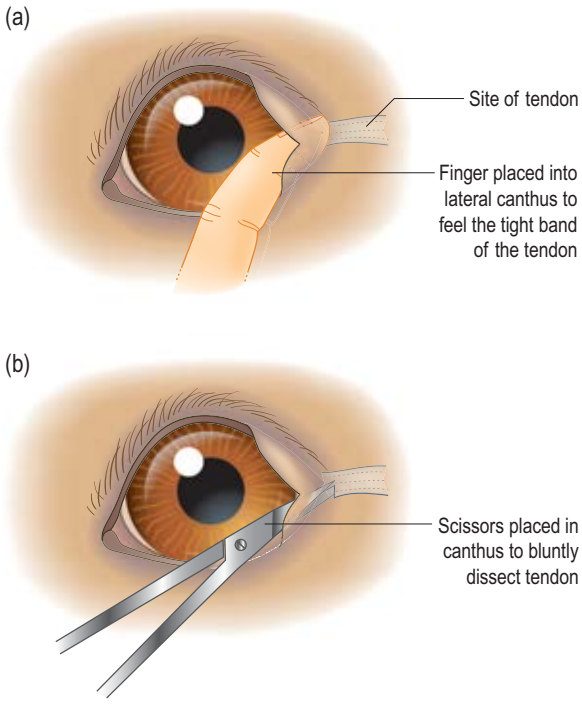


Figure 5.7 Lateral canthal tendonotomy to release the tension the tight, misdirected tendon places at the lateral canthus.

Standard ophthalmology textbooks have a wide range of similar procedures, all with complicated names, to tackle this problem. And now we are considering changing our surgical plan during the procedure, something which we have warned against! Clearly this is no longer a 'simple procedure'. It might be best to follow the original plan, but warn the owner that a second, more minor procedure might be required as a fine adjustment once the initial surgical site has fully healed.

The choice of suture material is also important – it should be soft and non-irritating. Thus silk or polyglactin 910 are the normal choice – the latter has the advantage of not usually requiring removal. Sizes of 6/0 or 5/0 are normally used.

In addition to the sharp surgery there are two other procedures to consider with these complicated lids. If there is any corneal ulceration present, the dog will start to exhibit blepharospasm immediately postoperatively; this can rapidly reverse the corrected lid position, triggering another episode of entropion and so the vicious cycle continues. To reduce this risk a bandage contact lens can be placed at the cessation of surgery. This will alleviate the trigeminal pain so that the dog no longer squints, and will allow the ulcer to heal beneath. Also, if the dog does start to show any blepharospasm the lids will not rub on the already sore cornea, and as such there will be no stimulus for the spastic entropion to return, leading to a permanent in-rolling and us being back at square one again.

In addition to placing a contact lens, temporary everting sutures can also be placed – normally between two and four along the lower lid, one at the lateral canthus and one at the upper lateral lid. These aim to cause a slight ectropion and again abolish the squint–rub cycle. Either 4/0 polyglactin 910 or silk is chosen for these sutures which can be removed after 14 days (although they often pull out before then). Topical anaesthesia should not be used as this will delay the epithelialization of any ulcers – it should only be used as a diagnostic tool and never as treatment.

Avoid rubbing with the use of systemic NSAIDs to promote comfort; an Elizabethan collar or foot bandages are all important in the postoperative period.

Shar Pei type (Case example 5.2)

The Shar Pei, as well as some Chow Chows, frequently suffers from a severe entropion affecting all four eyelids. Temporary tacking sutures placed soon after the eyelids open can reduce this risk but in patients over 12–16 weeks of age they are rarely effective and surgical intervention is required. It is not uncommon for the Shar Pei to develop severe corneal ulceration with marked granulation tissue, sometimes covering almost the entire corneal surface (Figures 5.8 and 5.9). So the poor animals cannot see both due to the entropion and the corneal pathology. However, the prognosis can be very good with accurate surgery. Assessing the patient can be difficult – they are not always the most cooperative dogs to examine, and even after the application of topical anaesthesia it can be virtually impossible to see their eyes!

A surgical plan should be drawn up which will normally entail a 360 degree Hotz-Celsus procedure, with a larger amount of skin removed from the upper lids than the lower ones (Figure 5.10). Indeed, sometimes up to 1 cm width needs excising from the upper lids. One difficulty with the Shar Pei, encountered during the surgery, is the phenomenon of mucinosis, where the excessive subcutaneous mucin which accumulates due



Figure 5.8 Nine-month-old male Shar Pei with upper lid entropion.

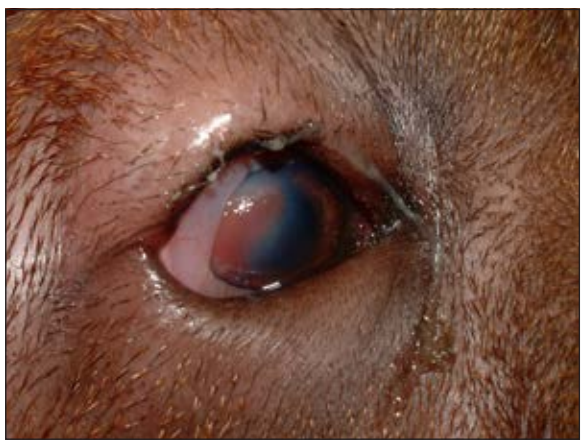


Figure 5.9 Same dog as in Figure 5.8 with the upper lid pulled out to reveal large dorsolateral corneal ulcer with marked granulation tissue reaction.

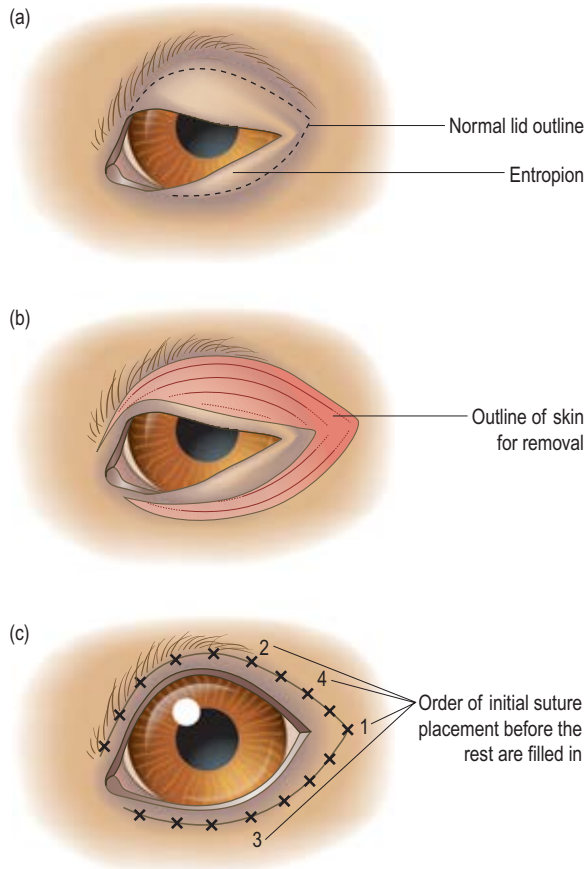


Figure 5.10

(a) Schematic outline of typical Shar Pei entropion.

(b) Outline of skin for removal.

(c) Final outcome with initial order in which to place sutures outlined.

to a connective tissue abnormality gives the false impression of oedema of the eyelid skin. However, during the surgery the sticky nature of the mucin is readily apparent, making the actual surgery quite difficult as the strands of mucus adhere to everything. Meticulous surgical closure using 6/0 suture material is necessary as with any entropion surgery. Contact lenses can be placed as with the previous case, but if the corneal surface is very irregular from the granulating ulcers, then retention of them could be difficult. Alternatively, temporary everting sutures can be placed following surgery.

Postoperatively minimal handling of the eyes is advised to reduce the risk of further spastic entropion. Normally the patient is much more comfortable and the ulceration heals remarkably quickly. If the surgery is performed in a very young dog, the owners should be warned that another procedure might be required as the animal reaches maturity, depending on how its head shape changes. It is not advised to delay surgery, however, since the patient will suffer unnecessary pain and severe corneal damage could develop.

Mastiff/Great Dane type

Many of the giant breeds suffer from complicated entropion mainly due to a macropalpebral fissure (over-long eyelids), compounded with a deep-set eye and lax lateral canthus. If the so-called 'diamond eye' is excessive, with entropion medially and laterally on the lower lids, ectro-



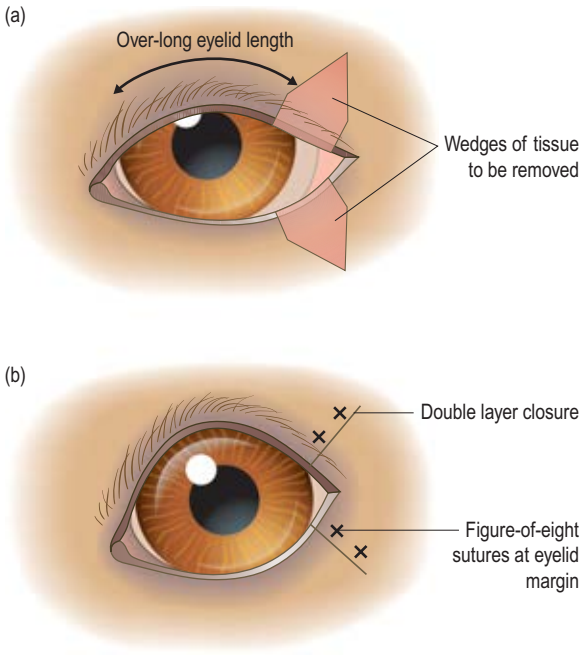
Figure 5.11 Nine-month-old male Dogue de Bordeaux with severe 'diamond eye' and a prolapsed nictitans gland in the left eye.

pion in the centre of the lower lids, and entropion of the upper lids laterally, then referral should be considered (Figure 5.11). There are a whole host of different surgical procedures described to correct these abnormalities and the choice rests with surgeon preference as well as the individual characteristics of the eyelid dynamics in the patient. Some cases are not quite so complicated, however, and can be tackled successfully from general practice.

Dogs with over-long lids which result in an upper lid entropion laterally and ectropion in the same location on the lower lids can benefit from simple lid shortening (Figure 5.12). Thus, a full thickness wedge of skin can be removed from both upper and lower lids close to, but not actually including, the lateral canthus. Up to half lid length can be removed in this way. It is important to use a double layer closure, with a figure of eight at the lid margin. This will achieve perfect lid alignment (no step along the margin) and ensures that there is no suture knot very close to the lid which could rub. In giant breeds a slightly larger suture size – 5/0 not 6/0 – might be necessary to provide sufficient strength in the thick eyelids.

Medial entropion in brachycephalics

Medial entropion in brachycephalic breeds – cats as well as dogs – can result in chronic epiphora and corneal ulcers and scarring ventromedially. Discussion of this topic is found in the cases on pigmentary keratitis and epiphora and will not be addressed further in this section.



NB. This technique is not suitable if the lateral canthus is dropped ventrally below the level of the medial canthus. Such cases should be referred

Figure 5.12 Wedge resection for over-long eyelids and simple diamond eye:
(a) outline of tissue for resection
(b) surgical outcome.

PROGNOSIS

The prognosis is generally good for most cases of entropion, even those complicated with ulceration. Certain things can be done to reduce the risk of complication. For example, make sure that the incisions are in the right place! Removing a strip of tissue from far away from the lid margin will not be successful (Figure 5.14). Ensuring that the suture material is sufficiently strong, and that the patient is not too active immediately after the surgery, will reduce the risk of wound breakdown (Figure 5.15). Using contact lenses if there is ulceration so that the cornea is no longer painful after the surgery, thus reducing the risk of further blepharospasm, will help. In addition, owners should be educated from the outset, such that they understand why the condition has developed and how its management can sometimes be more complicated than we would like. They should also be made aware that more than one surgical procedure is sometimes required to achieve a successful outcome.

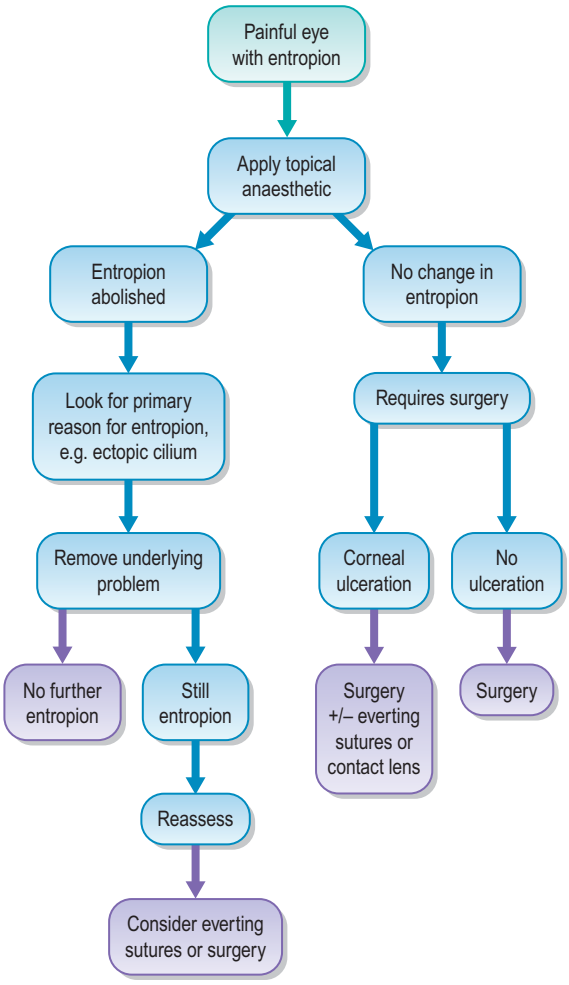


Figure 5.13 Decision-making tree – complicated entropion.



Figure 5.14 Fifteen-month-old bloodhound with large scar dorsolateral to the eye. It is no wonder that this previous surgery was not successful in correcting the entropion!



Figure 5.15 Breakdown of wound following wedge resection for over-long lower eyelid. A combination of overactivity of the adolescent dog immediately after surgery and a too-fine suture with insufficient strength in the relatively thick eyelid contributed to this.

CASE EXAMPLE 5.1

Patient details: Rottweiler, 15 months old, male

PRESENTATION AND HISTORY

The dog was presented with a wet, painful left eye which was held closed most of the time. The dog had already undergone two surgical procedures to correct the entropion which was causing the clinical signs, one at 9 months and one at 14 months. The initial surgery was on both eyes and no problems recurred with the right eye. The left was successful for a while, but the dog started to hold the eye closed again a few weeks after the surgery and was given topical antibiotic drops which did not help. After the second operation he held the eye wide open for 48 hours but then it started to discharge (serous) and the dog started to close it, especially when the owners tried to medicate the eye. He was starting to resent it whenever anybody approached his head and the owners were now unable to even clean the eye.

CLINICAL SIGNS

General clinical examination was unremarkable. The right eye was comfortable but a tendency to lateral canthal involution was noted and the lower lid was slightly long. The dog showed no symptoms with this eye. The left was held closed and it was difficult to examine. A copious serous ocular discharge was present. After several drops of topical anaesthesia had been placed in the eye over a 10-minute period it was possible to evaluate the patient. A lower lid entropion was present along the lateral two-thirds of the lid margin, together with in-turning of the lateral canthus. A large granulating corneal ulcer was present ventrolaterally, and conjunctival hyperaemia was apparent (Figure 5.5). A mild miosis was noted but no other intraocular changes.

WORK-UP

No further work-up was undertaken but immediate surgery was advised.

TREATMENT

Under general anaesthesia the lateral canthal tendon was palpated and found to be a very tight band pulling the skin at the lateral canthus inwards. The conjunctiva over it was incised with a scalpel and the tendon was bluntly dissected with tenotomy scissors (see Figure 5.7). Once the tendon was resected the eyelid fissure appeared larger but there was still slight involution at the canthus. A Hotz-Celsus procedure was performed along the lateral two-thirds of the lower eyelid and continued in an arrowhead shape around the lateral canthus. Sutures were 5/0 polyglactin 910 (Vicryl, Ethicon). Two everting mattress sutures of 4/0 polyglactin 910 were placed ventrolaterally. A bandage contact lens was placed in the eye. Postoperative medication consisted of a week of systemic amoxicillin/clavulanic acid and carprofen together with an Elizabethan collar. No topical medication was dispensed.

OUTCOME

The dog was immediately much more comfortable. However, the owner was concerned that the lower lid was very 'baggy'. After 7 days the everting sutures were removed and the eyelid margin was in a normal position. The contact lens fell out after another 5 days but by this time the corneal ulcer had fully healed and the dog has suffered no further problems.

This case illustrates that some dogs require more than one procedure to effect a cure, and that sometimes quite complicated management is required. Although we would not normally recommend not using topical antibiotics in the face of corneal ulceration, especially if a contact lens is placed, in some animals the struggle to apply drops can encourage further blepharospasm and actually make matters worse. It is not possible to determine whether if a lateral canthal tendonectomy had been performed at the first operation this might have prevented further problems. The fact that the right eye had not required additional surgery might suggest that the tendonectomy was not required at that time, but this eye did show a tendency to lateral canthal involution and the owner was warned that should the dog develop any ocular pain in future this could tip over the borderline anatomy into full-blown entropion again.

CASE EXAMPLE 5.2

Patient details: Shar Pei, 9 months old, male

PRESENTATION AND HISTORY

The dog was presented with severe bilateral blepharospasm and a copious serous discharge and persistent rubbing. He had had his eyelids tacked open as a young puppy and the owner reported that his eyes had always been fully open and comfortable until a week ago when he was bitten by an insect and his whole face swelled dramatically. This reduced rapidly after corticosteroid and antihistamine injections but the eyes remained closed and painful. The previous veterinary surgeon prescribed antibiotic/steroid drops which the owner felt were not helping.

CLINICAL SIGNS

Both eyes were held closed such that it was impossible to see the corneas at all when the dog was on the floor. It bumped into the consulting room table and was very nervous. Once on the table it was possible to see the ventral corneas by squatting down on the floor and looking up at the dog. Marked upper lid entropion was apparent and the dog had heavy brow folds (Figure 5.8). A seromucoid discharge was present bilaterally. On gently everting the upper lids large areas of granulation tissue and surrounding corneal ulceration were noted (Figure 5.9). No significant lower eyelid entropion was noted. After the application of topical anaesthesia the eyes were held open and the upper lid entropion resolved. Intraocular examination was limited but no gross abnormalities were detected. No abnormalities were detected on general clinical examination apart from otitis externa.

TREATMENT

Since the dog had had no previous problems with his eyes until the allergic reaction to the insect bite it was assumed that the entropion had occurred as a result of the eyelid swelling. The fact that the lid alignment was good after the application of topical anaesthesia reinforced this. The owner was reluctant for surgery, and so under sedation four everting sutures of 4/0 polyglactin 910 were placed in the upper eyelids of each eye. Oral carprofen and cefalexin were dispensed for a week plus an Elizabethan collar.

Initially the dog did very well for 5 days – the granulation tissue reduced and the eyes were open. Unfortunately, the Elizabethan collar was removed and the dog immediately started to rub the eyes. Some of the everting sutures were removed. On presentation 2 days later (a week after the initial presentation) the areas of granulation tissue were very pronounced and it could be seen that the remaining everting sutures were rubbing on the cornea (Figure 5.16). At this stage the owner consented to surgery.

Under general anaesthesia a Hotz-Celsus procedure was performed along both upper eyelids with sutures of 5/0 polyglactin 910. Two everting sutures of 4/0 polyglactin 910 were also placed, with long ends which would be less likely to rub should they become misdirected towards the cornea (Figure 5.17). Contact lenses were not placed due to the extreme irregularity of the corneal surface – retention times would be minimal. The dog was sent home with oral treatment as before and instructions not to remove the Elizabethan collar at all!



Figure 5.16 Nine-month-old Shar Pei with severe granulation tissue reaction resulting from rubbing of an upper eyelid suture.

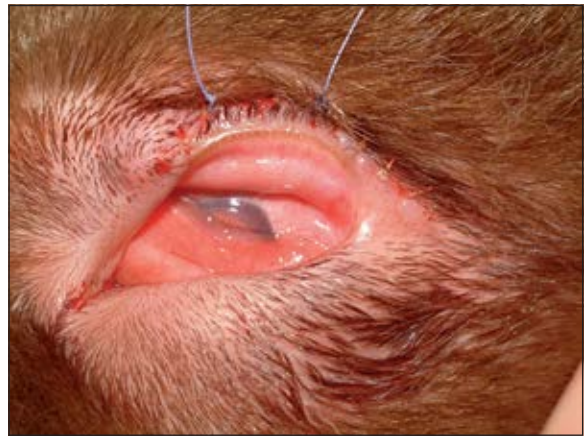


Figure 5.17 Same Shar Pei immediately post-surgery with both entropion wound sutures (white) and everting sutures (lilac) visible.

OUTCOME

The dog did very well. None of the sutures was removed and they gradually dissolved or fell out. The corneal granulation tissue reduced quickly and after 3 weeks only mild vascularization and scarring were left. Vision was fine. The eyes were fully open all the time and the owner was happy.

This case illustrates the borderline nature of the adnexal anatomy of many Shar Peis. It appears that the

entropion was triggered by the facial oedema due to the insect bite. In most dogs this degree of facial swelling would subside without incident, but the heavy brows and tendency to entropion innate in the breed triggered problems. The use of steroid-containing drops could have contributed to the ulceration which then exacerbated the blepharospasm and entropion, and a vicious cycle was set up. It was hoped that simply everting the lids to remove the continual corneal abrasion they were causing would allow things to settle but unfortunately this was not successful and sharp surgery was required.

CASE EXAMPLE 5.3

Patient details: Domestic short haired cat, 3 years old, male (neutered)

PRESENTATION AND HISTORY

The cat had been presented to another veterinary surgeon because the right eye was closed and some mucoid discharge was present. This had developed over 4 days. The veterinary surgeon noted the partially closed eyelids, third eyelid protrusion and slight miosis of the affected eye and made a diagnosis of Horner's syndrome (see Figure 54.4 for a case of Horner's syndrome in a cat). He examined the cat carefully and discovered moderate otitis externa and surmised that middle ear disease might also be present. Antibiotic and steroid-containing ear drops were prescribed together with a course of amoxicillin/clavulanic acid orally. The owner was unhappy that there was no improvement after 3 weeks and sought a second opinion.

CLINICAL SIGNS

Apart from slightly waxy ears, general examination was unremarkable. The right eye was held closed, with a copious mucopurulent discharge and third eyelid protrusion (Figure 5.18). Closer examination of the right eye showed a marked lower eyelid entropion. When the eyelid was pulled down, discharge adhered to it and the lid margin itself was slightly depigmented (a classic indication of chronic entropion). Superficial corneal vascularization was present but no ulceration was noted. The pupil was slightly miotic. The left eye was normal.

WORK-UP

No further work-up was necessary and the cat was taken to surgery.

TREATMENT

Under general anaesthesia a straightforward Hotz-Celsus procedure was performed on the lower lid. Sutures were 6/0 polyglactin 910 (Figure 5.19). One drop of atropine was placed in the eye at the time of surgery. Chloramphenicol ointment was dispensed twice daily for 5 days.

OUTCOME

The cat was immediately more comfortable and held the eye fully open. The pupil remained dilated for 72 hours and then gradually returned to normal. The



Figure 5.18 Three-year-old domestic short haired cat with partially closed right eye, ocular discharge, third eyelid protrusion and mild miosis (anisocoria not clear in photograph).



Figure 5.19 Same cat as Figure 5.18 immediately post-surgery.

corneal scarring faded completely and no complications ensued.

This case highlights the importance of a correct diagnosis! Although the cat did have clinical signs similar to Horner's syndrome, this clearly was not the problem. It is most likely that there was initial corneal ulceration which triggered the blepharospasm and secondary entropion since primary entropion is very rare in cats. Once any spastic entropion becomes chronic it can easily remain even if the inciting cause is removed. The reflex uveitis was minimal and the fact that the atropine drop lasted for several days is typical if there is only slight intraocular inflammation.

6 Large eyelid mass

INITIAL PRESENTATION

Abnormal appearance – change in shape/position/swelling

PRESENTING SIGNS

Dogs, and less frequently cats, will be presented due to a change in appearance and possibly a red eye. There is also likely to be some ocular discharge. The onset is often gradual – the owner has probably been aware of a problem for a few weeks but the lesion increases in size such that they present the patient. Alternatively, the lid mass might have been present for months but has suddenly changed – growing rapidly, bleeding or itching such that the patient rubs and further traumatizes the lesion. No other symptoms are generally reported. Most patients are older adults (8 years and over) and no breed or sex predilection is apparent with the exception of squamous cell carcinoma which is more common in white furred animals, especially cats.

CASE HISTORY

In general there is little in the way of previous history. However, some dogs will have had an eyelid mass or other skin masses in the past, which frequently have been removed, often without histopathological examination. In some cases regrowth can occur very quickly – within weeks – of the previous excision. In other examples owners initially suspect that the current problem is similar to small benign warty growths from which the dog suffers, but the lesion either suddenly enlarges quickly, or changes in appearance – becomes friable and starts to bleed, or causes the pet to rub for example – and then they present the animal.

CLINICAL EXAMINATION

General clinical examination is normally unremarkable, although occasionally a localized lymphadenopathy is present and should be checked for. On ophthalmic examination a large mass can be seen on either the upper or lower lid margin. The periorbital skin might be thickened and inflamed. The mass itself will probably involve the lid margin, but not invariably so (Figure 6.1). The mass might be smooth or lobulated and can vary in colour from pale pink through red to darkly pigmented. The palpebral conjunctiva will be inflamed and the lid margin should always be everted to check the internal extent of the mass – frequently what can be seen from the outside is just the 'tip of the iceberg' (Figure 6.2). Some ocular discharge is normally present – this might be serous but is commonly mucopurulent. In some patients it will be excessive and can stick the eyelids together. Schirmer tear test is usually normal. The cornea should be carefully examined – some keratitis is common where the lid mass rubs on the cornea during blinking, and ulceration is not uncommon. Intraocular examination is usually unremarkable, although there might be slight miosis, representing a reflex uveitis if significant trigeminal pain is present.

DIFFERENTIAL DIAGNOSES

- Neoplastic lid mass (Table 6.1)
- Chalazion
- Dermoid
- Granuloma
- Meibomianitis
- Meibomian gland hyperplasia (rare)



Figure 6.1 Lid mass affecting the palpebral conjunctiva rather than the lid margin which presented as a variable lid swelling rather than an obvious mass. Mast cell tumour.



Figure 6.2 Upper eyelid mass in a 10-year-old terrier. The grey papillomatous growth at the lid margin was all that could be seen with the lid in its normal position, but on everting it a much larger pink lesion can be seen extending both medially and posteriorly.

Table 6.1 Eyelid tumour types in dogs

Tumour type	Typical appearance
Papilloma	Warty, elevated, cauliflower-like Superficial Rapidly growing Younger dogs – frequently viral Oral papillomata can also be present
Adenoma/ adenocarcinoma	Most frequent lid neoplasm in dogs Arises from meibomian glands Can extend into lid or be pedunculated Metastasis very rare
Histiocytoma	Rapidly growing Smooth, pink and broad based Mainly younger dogs May regress spontaneously Caution in Bernese mountain dog re systemic histiocytosis
Melanoma	Common Usually but not invariably pigmented Based at eyelid margin Benign
Mast cell tumour	Very variable presentation Intermittent pruritus common Variation in size or swelling frequent Consider referral
Squamous cell carcinoma	Usually on non-pigmented skin/ white fur Minimal pigment Papillary or ulcerative appearance Locally invasive
Other rare types include fibroma, fibrosarcoma, lymphosarcoma and aplastic sarcoma.	

CASE WORK-UP

For simple lid masses minimal work-up is necessary as they are usually surgically excised and the tissue sent for histological typing. However, for atypical masses, where rapid growth has occurred for example, or if a lesion has recurred, then further investigation is warranted before opting for surgery. Fine needle aspirates are frequently diagnostic and can be performed without sedation in most patients. Impression smears can be similarly obtained and are very useful in erosive or ulcerated lesions. Punch biopsies can be taken under sedation and a normal 4–6 mm punch biopsy will provide adequate samples. The lids are highly vascular and will bleed copiously following such sampling. Suturing is rarely necessary however. Occasionally it is sensible to perform fine needle aspirates of draining lymph nodes – particularly if the histology reveals a potentially malignant mass such as a squamous cell carcinoma or lymphosarcoma.

NURSING ASPECTS

Apart from preventing self-trauma both pre- and postsurgically, and the application of topical medication, there is minimal nursing required.

EPIDEMIOLOGY

Eyelid neoplasia is common in the dog, where thankfully 75% of neoplasms are benign although even the histologically malignant forms rarely metastasize – local recurrence is the main problem. The prognosis for mast cell tumours is significantly worse than for any of the other histological types. Some reports suggest that certain breeds – including the boxer, collie, poodle, and English springer spaniel – have a higher incidence than the general population but any breed can be presented. The average age at presentation is 8 years. If a Bernese mountain dog is presented with a strange lid mass which is determined to be a histiocytoma, then careful examination for evidence of systemic involvement should be considered since the breed is predisposed to systemic histiocytosis.

In the cat, malignant lid tumours are more common than in dogs, with squamous cell carcinoma the most common. These are seen particularly in white furred cats (even if only the periorbital fur is white) and are exacerbated by UV light. The ventromedial canthus is most frequently affected and most lesions are erosive rather than presenting as a discrete mass. Local regrowth frequently occurs and spread to regional lymph nodes occurs later in the course of the disease. The other eye can be affected as well and frequently the aim of treatment is good control rather than a complete cure.

CLINICAL TIPS

- Evert the eyelid margin to see the extent of the mass
- Consider fine needle aspiration and cytology in atypical or large lesions
- Eyelid neoplasia in cats is more frequently malignant than in dogs

TREATMENT OPTIONS – SURGICAL

Surgical excision is the treatment of choice for all lid masses, but is more complicated in larger lesions. All standard ophthalmology textbooks illustrate various surgical approaches. Histological typing prior to removal in atypical cases is recommended as this will provide information regarding the width of clear margins necessary – a 2 cm margin is recommended for mast cell tumours for example, and since the eyelids are essential to normal ocular health they cannot be simply cut away – reconstructive blepharoplasty might be necessary and referral should be considered in such cases. If the mass involves the medial canthus or is located close to the nasolacrimal punctae, referral is recommended.

In general, excision will involve full thickness lid resection and is simply done in lesions up to one-third of the lid length. If the lesion does approach the nasolacrimal punctae, then these should be cannulated with coloured nylon prior to the first incision so that their location is highlighted throughout the surgery. This will avoid inadvertent transection of the canaliculi which would result in permanent epiphora. Closure should always be in two layers, with close attention to the lid margin, ensuring that there is no step or notch between the two sides of the wound. A figure-of-eight suture is recommended at the lid margin. Suture ends should either be cut very short or left long, and soft so that they cannot rub on the cornea. Soft absorbable suture material such as 6/0-4/0 polyglactin 910 (Vicryl, Ethicon) is ideal.

Larger masses will necessitate some form of additional surgery to provide a sufficiently large palpebral fissure which matches the opposite lid. A simple lateral canthotomy can be sufficient in many cases – this can be lined with palpebral conjunctiva if necessary to reduce the risk of trichiasis causing ulceration of the cornea. Cryosurgery can be performed later on the offending hairs if this is a problem. Sliding or advancement flaps are easily performed but care should be taken to ensure that the fur growth is directed away from, and not toward, the cornea. Some techniques involve rotating part of the unaffected eyelid margin into the defect created by the removal of the mass to ensure a normal lid margin. Lower lid defects can benefit from skin flaps rotated from the mouth such that lip margin replaces the lost lid tissue. Frequently these are complicated, two-stage procedures requiring at least two anaesthetics. Referral should be considered if such a technique is necessary.

Some tumours are sensitive to cryosurgery – especially squamous cell carcinomas in both dogs and cats. Normally tumours are debulked surgically prior to the application of a double freeze–thaw cycle of liquid nitrogen or nitrous oxide. Frequently the lid margin can be spared on debulking, and can recover well from the cryotherapy. Significant postoperative swelling occurs and the lesions appear red and scabbed prior to sloughing so the owners should be aware of the undesirable cosmetic appearance for the first couple of weeks after surgery.

Other procedures such as brachytherapy for mast cell tumours and squamous cell carcinomas can be a suc-

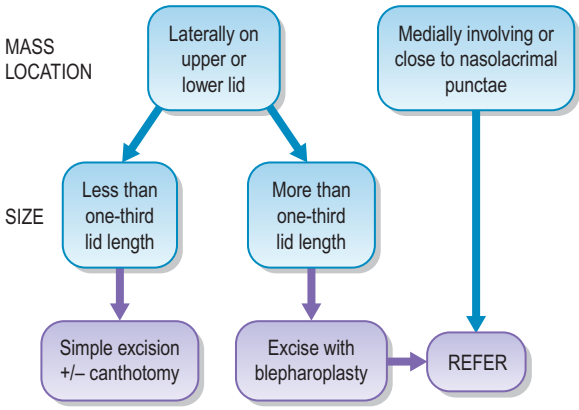


Figure 6.3 Decision-making tree – eyelid mass.

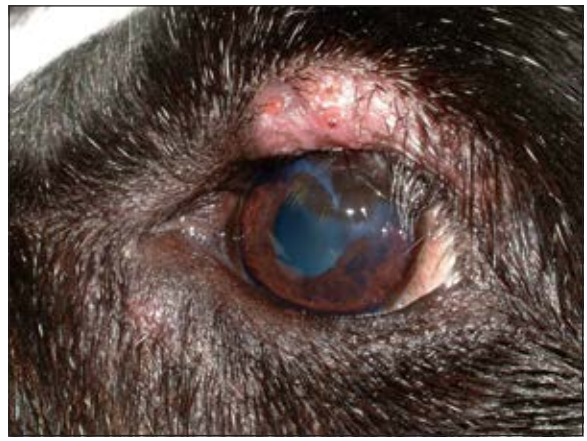


Figure 6.4 Appearance following poor surgical removal of lid mass – blepharitis, trichiasis and pigmentary keratitis are present.

cessful alternative for aggressive cases but clearly referral is necessary for this.

PROGNOSIS

The prognosis for benign lid tumours is generally good, even if they are very large. Complications following surgical excision are usually related to inadequate lid function such that some degree of lagophthalmos and exposure keratitis develop, or to trichiasis with hairs rubbing causing corneal irritation and possibly ulceration (Figure 6.4). In some patients both of these can be managed with ophthalmic lubricants. Ointments applied to the offending hairs – coating and softening them, while also encouraging them to stick to fur away from the eye – can be sufficient. Cryosurgery can also be successful in permanently removing these hairs. Severe cases of exposure keratitis might benefit from a permanent partial tarsorrhaphy. Local regrowth of the mass can be a problem, especially in malignant cases such as squamous cell carcinoma. In a few cases enucleation proves necessary since there is no normal lid margin left to function. Rarely euthanasia is required if the patient's quality of life is seriously compromised.

CASE EXAMPLE 6.1

Patient details: Beagle, 15 years old, male (entire)

PRESENTATION AND HISTORY

The dog was presented with a history of a rapidly growing eyelid mass on the left upper lid. This had been removed 3 months previously but no histology had been performed. The swelling had returned within a few weeks and had become very large. The dog was rubbing the eye occasionally but, although elderly, was otherwise well.



Figure 6.5 Large ulcerated eyelid mass in a 15-year-old beagle.

CLINICAL SIGNS

On ophthalmic examination a very large, ulcerated warty mass was noted affecting the lateral portion of the upper eyelid (Figure 6.5). Some bloodstained discharge

was present. The eye itself was fine with mild conjunctival hyperaemia only. No other significant abnormalities were detected.

WORK-UP

The owner was keen to have the mass removed, since it was clearly bothering the dog, but did not want to 'mess him around too much at his age'. Impression smears were taken from the mass after gently removing any surface contamination. Fine needle aspirates were also taken. Both of these confirmed squamous cell carcinoma. Various surgical options were discussed with the owner but she did not want complicated surgery and so a relatively simple procedure was chosen – the owner was warned that the cosmetic result might not be as good as with a two-stage procedure but she was reluctant to agree to multiple surgeries. Routine haematology and biochemistry were unremarkable, as was chest radiography.

TREATMENT

Under general anaesthesia the mass was removed with 1 cm margins. A lateral canthotomy was made and this incision extended in a curvilinear fashion to reduce the distortion of the eyelid margin as much as possible. The surgical technique is outlined in Figure 6.6. The lateral canthotomy was slid up to create the outer part of the upper eyelid. This was lined with bulbar conjunctiva and sutured to the raw skin edge with a simple continuous suture of 8/0 polyglactin 910, while the rest of the incisions were closed with a double layer of simple interrupted 6/0 polyglactin 910. A figure-of-eight suture was placed at the lid margin. A fine needle aspirate was taken from the submandibular lymph node at the time of surgery.

OUTCOME

The wounds healed well. Histology confirmed the squamous cell carcinoma with clear margins at the edges of the tissue. No neoplastic cells were found on the lymph node aspirate. Figure 6.7 is the dog 1 week after surgery and Figure 6.8 is 3 months after surgery. The absence of lid margin laterally did not result in corneal damage or discomfort. The owner had been warned that further regrowth could occur, and cryosurgery had been discussed but this was not required.

This case illustrates that although many complicated techniques are described for recreating lid margins following removal of large masses, sometimes simple procedures are sufficient. Had the owner been willing to allow more than one general anaesthetic the recommended surgery would have involved moving a section of lower lid into the defect created by removing the

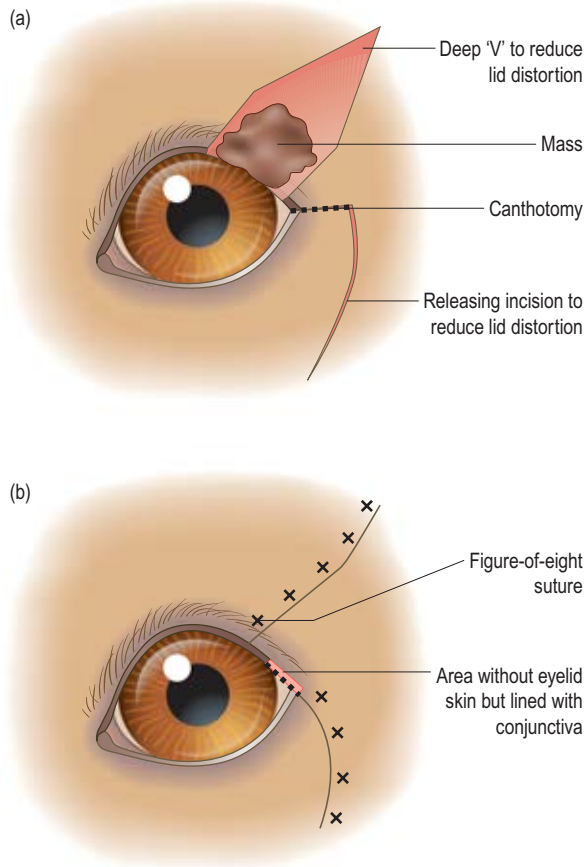


Figure 6.6 Diagram outlining the approach to the removal of a large upper eyelid mass.



Figure 6.7 Appearance 1 week after surgery.



Figure 6.8 Good outcome 3 months postoperatively.

upper lid mass, followed by a second operation to replace the lower lid with lip margin. The main concerns with the technique followed in this case were excessive distortion of the remaining eyelid tissue, decreased blinking ability and trichiasis from the hair in the lateral lid. Thankfully none of these complications developed.

7 Eyelash problems – distichiasis and ectopic cilia

INITIAL PRESENTATION

Abnormal appearance – red; discharge

PRESENTING SIGNS

Eyelash abnormalities are very common in dogs but are rarely encountered in cats. Distichia are extra eyelashes along the eyelid margin which originate from slightly abnormal meibomian glands. Ectopic cilia are lashes which grow through the palpebral conjunctiva towards the cornea (Figure 7.1). The typical presentation, if the distichia are clinically relevant, is of wet, itchy eyes. Thus the owner reports the dog's eyes run constantly and that it rubs them frequently. Some redness is reported but unless corneal ulceration has occurred the eyes are not usually overtly painful. Ectopic cilia more commonly result in corneal ulcers than do distichia. Some breeds such as the Staffordshire bull terrier, English bulldog, boxer, miniature long haired dachshund and cocker spaniel commonly develop distichia, while ectopic cilia are seen in flat coated retrievers and Staffordshire bull terriers more commonly than other breeds. An inherited incidence is clearly present but the exact inheritance has not been evaluated. Typically young dogs are presented from 6 to 18 months.

CASE HISTORY

The typical history is of a young dog which has suffered from intermittent watery, itchy eyes for several weeks prior to presentation. The owners might report redness as well but most cases are not particularly painful. In cases with ulceration the affected eye is obviously more uncomfortable with blepharospasm, markedly increased lacrimation and some attempts at self-trauma. There might be a history of such irritation for a few days which then resolved, but recurred 4–6 weeks later. This would correspond to natural epilation followed by regrowth of the offending lash. Some owners will have tried treating the dog with topical antibiotics with minimal effect.

CLINICAL EXAMINATION

General clinical examination is unremarkable. On ophthalmic examination the aberrant cilia on the eyelid margin can often be seen with the naked eye against the white of the sclera, particularly if they are pigmented. Lashes along the lower lid margin are not immediately so obvious. Increased lacrimation is a feature and Schirmer tear test readings are usually towards the upper end of normal. Some conjunctival hyperaemia is normally present but overt conjunctivitis is not a feature unless ulceration is present. Mild secondary entropion can be present in long-standing cases following chronic blepharospasm and must be differentiated from primary entropion with incidental distichiasis. The application of a drop of topical anaesthesia will alleviate any spastic component to the entropion.

Sometimes the presence of the distichia might be masked by small globules of mucus along the lid margin. Gently bathing the eyes should clear these. Careful examination with good illumination and magnification (+20D on the direct ophthalmoscope is adequate) is required, paying attention to all four eyelid margins. Placing a drop of fluorescein in the eye might highlight the lashes further. Identification of the distichia is not normally difficult – it is deciding whether they are the cause for the dog's symptoms that is more challenging. Many cases are subclinical, incidental findings, and a

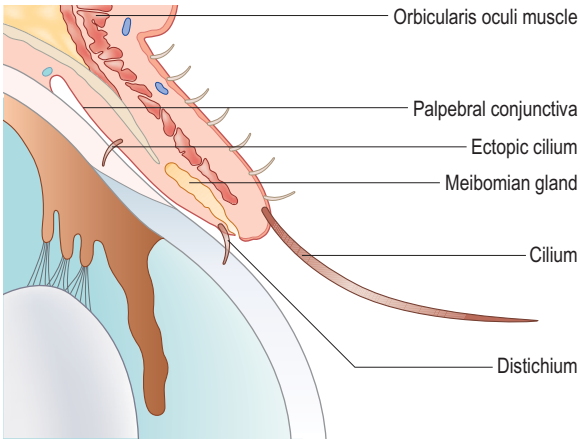


Figure 7.1 Illustration showing the location of abnormal eyelashes.



Figure 7.2 Ectopic cilium on the upper palpebral conjunctiva of a flat coated retriever.

thorough examination of the rest of the eyes is required to see if there is another explanation for the lacrimation and irritation. The lashes should be evaluated for position, thickness and number, along with their direction. Even multiple fine lashes might be insignificant if they are not in direct contact with the corneal tear film, whereas one thick, stubby lash directed towards the cornea can be very significant.

In addition to identifying the lashes, the fluorescein will obviously highlight any corneal ulceration. If this is present, its location should be checked against the position of the distichia. If none are parallel to the ulceration, then they are unlikely to have caused it. Everting the upper eyelid, and checking carefully for any ectopic cilia, is essential if any corneal ulceration is present (Figure 7.2). One or two ectopic cilia can be present, normally close to 12 o'clock on the upper lid, 4–6 mm from the eyelid margin. Their presence is almost always clinically significant. If ulceration is a feature, then a mild reflex uveitis might be noted, but normally there is no intraocular involvement and thus the rest of the ocular examination is unremarkable.

DIFFERENTIAL DIAGNOSES

- Distichiasis
- Ectopic cilia
- Trichiasis
- Entropion
- Other causes of conjunctivitis, e.g. allergy, bacterial infection
- Other causes of ulceration, e.g. trauma, entropion

CASE WORK-UP

As mentioned above, the identification of distichia is not particularly challenging, but establishing their relevance can be. As such, a straightforward way to determine their significance is simply to pluck them and see if this alleviates the dog's symptoms. There should be immediate relief if the lashes are relevant and the owner should report reduced lacrimation and a resolution of the irritation. If this does not occur, the patient requires further detailed ophthalmic evaluation since clearly the distichia were not the cause of the dog's clinical signs. If the dog is much more comfortable following plucking then when they grow back, usually within 4–6 weeks, one would expect the clinical symptoms to return, further confirming the diagnosis. More permanent treatment options can then be evaluated.

Some dogs will have multiple distichia which cause no problems at all until the dog develops an unrelated painful ocular complaint – perhaps a corneal ulcer as a result of scrapping with another dog or shampoo accidentally splashing in its eye during a bath. The painful eye triggers blepharospasm which changes the angle at which the distichia touch the cornea, causing them to rub and exacerbate the corneal pain. In these instances it is extremely important to decide how relevant the lashes actually are, and plucking them to relieve this secondary discomfort while treating the other eye problem is sensible.

NURSING ASPECTS

Apart from preventing self-trauma and applying medication as prescribed, there are no specific nursing requirements with distichiasis. After-care following cryosurgical removal is discussed below under the surgical treatment section.

EPIDEMIOLOGY

Distichiasis and ectopic cilia occur on an individual basis in any breed of dog, but some breeds are more commonly affected, such as the Staffordshire bull terrier, English bulldog, cocker spaniel (both English and American), miniature long haired dachshund, boxer and flat coated retriever. The incidence is very high, but the percentage of dogs with ocular disease attributed to the distichia is low. Distichia erupt from meibomian glands. Since these are modified hair follicles anyway the lashes tend to develop from undifferentiated meibomian tissue. Normally the hairs emerge individually but several can come out from one opening (termed districhiasis). They emerge from the meibomian gland orifices as the route of least resistance, but can emerge as ectopic cilia through the palpebral conjunctiva, normally of the upper lid, and this positioning is clinically more severe. The ectopic cilium grows out perpendicular to the cornea and scrapes against it each time the animal blinks, causing a typical linear ulcer in the dorsal cornea.

CLINICAL TIPS

- Are the distichia really significant? Just because they are present does not mean that they are the cause for the symptoms – check the eyes thoroughly!
- Place a drop of fluorescein in the eye to help highlight the lashes
- Always look for ectopic cilia if ulceration is present, especially if it is a vertically linear ulcer in the upper cornea at 12 o'clock
- If in doubt, pluck the lashes and see if the symptoms are alleviated

TREATMENT OPTIONS – MEDICAL

A variety of treatment options is available for treating distichia but it cannot be overemphasized that the majority of cases are subclinical and do not require any form of treatment! However, apart from regularly plucking the offending lashes, which owners can be taught to do if their pet is cooperative, all more permanent solutions are surgical.

TREATMENT OPTIONS – SURGICAL

Various surgical options are available for treating clinically relevant distichia and ectopic cilia. For the former, various lid splitting techniques have been described in the past, but none of these is currently recommended due to the significant lid scarring and damage to the meibomian glands which can occur, even in patients with relatively thick eyelids. Similarly, electrocautery can lead to scarring due to excessive current damaging adjacent tissue. Alternatives that are successful and do not result in scarring include electrolysis/catholysis and cryosurgery.

Electrolysis and catholysis are essentially the same and involve inserting a fine electrode into each orifice though which a lash emerges and passing a small electric current through the needle to destroy the follicle. Although effective, the procedure can be very time consuming and long general anaesthetics can be required. Accurate electrode positioning is essential for surgical success and magnification is required. Ideally this should be with an operating microscope and as such the procedure is normally only undertaken in referral situations.

Cryosurgery is a more available technique in general practice. Again general anaesthesia is required and the lid margins are normally stabilized with a chalazion clamp. Either liquid nitrogen or nitrous oxide units can be used. A small (3 mm) probe is applied to the palpebral conjunctiva about 2–3 mm from the eyelid margin and the iceball is allowed to reach the lid margin before removal. The area is allowed to thaw and a second freeze applied. If the distichia are localized then only affected areas of lid are treated but it is not unusual to have to go along the whole of the lid margin. The lashes should be plucked at the same time – it can take several days for them to fall out otherwise. Swelling following cryosurgery can be significant. Premedication with a suitable anti-inflammatory will help but owners need to be warned of the fact that the postoperative appearance will be worse than prior to treatment!

Nursing with cold compresses immediately after performing will reduce the swelling – ice packs of frozen peas are ideal as they will mould to the shape of the patient! Postoperatively an antibiotic/steroid combination drop or ointment is used for several days (providing no ulceration was noted) and systemic NSAIDs are

advised until the lid swelling is fully resolved. Depigmentation of the eyelid margin, and occasionally vitiligo, can occur and owners must be warned of this (especially in show animals!). Repigmentation occurs within a few weeks in most patients. A small percentage of regrowth can occur but this does not always require further attention since the lashes are commonly sparse and fine.

If only one or two lashes close together are present then surgical excision using a wedge resection of the eyelid margin can be performed. This is very successful with no regrowth occurring but is only applicable in a small number of patients.

Ectopic cilia almost always require surgical excision. Under general anaesthesia the eyelid is everted and stabilized with a chalazion clamp and the lash is excised together with its follicle. A triangular- or square-shaped sharp incision made around the lash using a pointed blade (e.g. No. 11) is followed by removal of the triangle or square of tissue with fine pointed scissors (Figure 7.3). The area is left to heal by secondary intention and topical

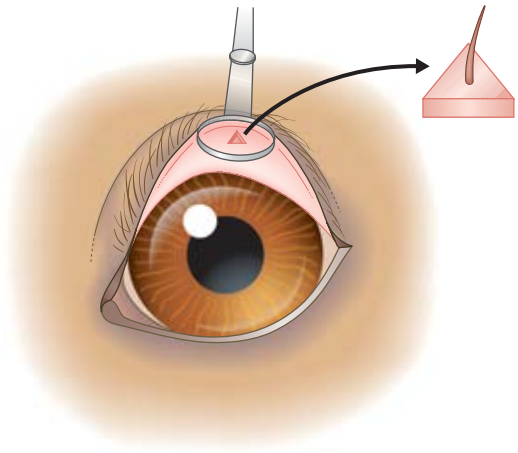


Figure 7.3 Diagram illustrating surgical removal of ectopic cilium.

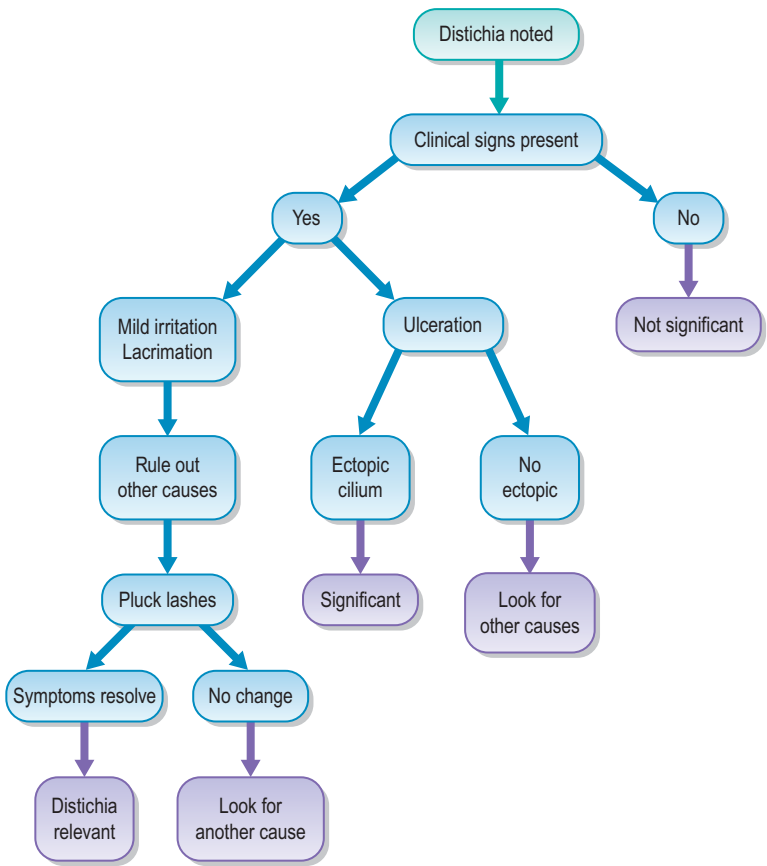


Figure 7.4 Decision-making tree – eyelashes.

antibiotic drops are used until the wound and probably the corneal ulcer have fully healed. Cryosurgery can also be used for ectopic cilia but is not usually warranted.

PROGNOSIS

The prognosis for most eyelash abnormalities is very good, although a surgical procedure (or sometimes two in cases of severe distichiasis) might be required to effect a cure. Getting the correct diagnosis can be the most challenging part of the case, since the presence of distichia is easily identified and might be more obvious than the actual cause for the pet's symptoms (such as a subtle entropion or atopic conjunctivitis for example). Unfortunately, some dogs undergo surgery for distichia which was not required, and can even be detrimental if inappropriate techniques were employed such that the resultant lid scarring further exacerbated the dog's ocular discomfort (see Case example 7.2 below). As such, if there is any doubt regarding the significance of the lashes, then a specialist opinion should be sought before embarking on unnecessary surgical procedures.

CASE EXAMPLE 7.1

Patient details: Approximately 6-month-old (rescue) terrier cross, male

PRESENTATION AND HISTORY

The young dog was a rescue in a welfare sanctuary and no previous history was available. He was uncomfortable with both eyes, rubbing them with his paws and on his bedding and significant serous ocular discharge was present. He would hold both eyes partially closed most of the time and the kennel assistant thought that his eyes were abnormally small.

CLINICAL SIGNS

Moderate blepharospasm was present in both eyes together with a slight lower lid entropion, especially laterally in the left eye. A bilateral serous ocular discharge was noted. Multiple distichia were noted on all four eyelids, especially the upper lid in the right and in clumps on the medial side of the upper left lid and the centre of its lower lid (Figure 7.5). Conjunctival hyperaemia was surprisingly absent. No corneal ulceration was noted but some scarring was present ventrally in the left eye. No ectopic cilia were noted. It did look as though the dog's eyes were small, as the kennel assistant had commented, but after the application of topical anaesthesia the eyes opened normally and full ophthalmic examination revealed no further abnormalities.

WORK-UP

It was suspected that the distichia were the reason for the dog's discomfort, given the fact that the lashes were quite thick and were growing in various directions. The dog was anaesthetized for castration and the distichia were plucked at the same time. The palpebral conjunctiva was checked again for any ectopic cilia and none was seen. The dog was much more comfortable and held both eyes fully open with no remaining lateral canthal entropion after the lashes were removed. The dog was clinically normal for 3 weeks and then the discharge, blepharospasm and rubbing began again. By this time the dog had been rehomed and the new owners were aware that more permanent surgery would be necessary.



Figure 7.5 Distichiasis in a young terrier cross, right eye, mainly affecting upper lid. Note the ocular discharge but lack of conjunctival hyperaemia.

TREATMENT

Four weeks after the lashes were plucked they had regrown in a similar pattern. Since there were so many, and he had quite thick eyelids, cryosurgery was recommended over electrolysis. Under general anaesthesia this was performed using a double freeze–thaw cycle of liquid nitrogen as described in the treatment section above. The lashes were again plucked. Postoperatively antibiotic/steroid ointment (Maxitrol, Alcon) was dispensed twice daily and carprofen orally, both for a week.

OUTCOME

Within hours of surgery the eyelids were very swollen and marked chemosis was present. Ice packs were used until the dog would no longer allow their application! The following day the swelling had reduced but it still took 2 weeks for it to fully resolve. The above medication was continued for this time. The eyelid margins became very pink but the dog was quite comfortable. Six weeks after the surgery the eyelid margins had started to repigment and three lashes had regrown medially in the left upper lid. However, these were causing the dog no discomfort and so no further treatment was required. The corneal scarring had fully resolved.

This case illustrates that in some dogs distichia are significant and treatment can be straightforward. The lack of conjunctival hyperaemia was surprising, especially given that the dog did rub his eyes and hold them partially closed. However, the immediate resolution of signs on plucking the lashes confirmed their relevance.

CASE EXAMPLE 7.2

Patient details: Flat coated retriever, 2½ years old, female

PRESENTATION AND HISTORY

The dog had a long history of recurrent ocular problems since 6 months of age. These had affected both eyes, but the right was always worse and had had several ulcers. On current presentation the owner was concerned that both eyes were slightly closed, some irritation was present in the left, both third eyelids were elevated and the right lower lid was 'baggy'. The owner felt that the eyes always looked sore, but only a slight mucoid discharge was present. Previous treatments had included various antibiotic drops to treat recurrent ulceration of the right eye, diathermy for distichia in both eyes and lower lid entropion surgery in the right eye. None of these had effected any long-term cure.

CLINICAL SIGNS

The dog was in good general health. Menace responses and pupillary light reflexes were normal. Mild blepharospasm was present in the right eye and the nictitans membrane was elevated in this eye. A lower lid ectropion was noted along with depigmented patches on the lower eyelid. Several distichia were present on the upper eyelid in both eyes, worse in the right. A small linear corneal ulcer with surrounding corneal pigment and vascularization was present close to the limbus at 12 o'clock in the right eye. Eversion of the upper eyelid revealed a single ectopic cilium in the centre of the lid about 3 mm from the eyelid margin. The left eye was comfortable but the lower eyelid was very scarred with several notches and puckering along its length (Figures 7.6 and 7.7). Multiple distichia were present on the upper lid.

WORK-UP

It was clear from the clinical examination that the ectopic cilium in the right eye was the dog's main problem and no further work-up was undertaken.

TREATMENT

Under general anaesthesia the ectopic cilium was excised using a triangular incision around the lash and then removing the section of palpebral conjunctiva together with the root of the lash. In addition, a small wedge resection was performed laterally on the lower lid to correct the iatrogenic ectropion. Postoperatively fusidic



Figure 7.6 Flat coated retriever right eye – lower lid ectropion with some scarring along lid margin and upper lid distichia. A small dorsal corneal ulcer was present.



Figure 7.7 Left eye of the same dog as Figure 7.6 with severe lower eyelid scarring from previous diathermy surgery.

acid gel (Fucithalmic, Vet XX) was used twice daily for a week, together with 3 days of oral carprofen.

OUTCOME

The dog did very well. The right eye was immediately comfortable and the third eyelid protrusion settled. The corneal ulcer healed within 72 hours and did not recur. The owner was happy that the eye was no longer 'red and baggy' in appearance. Both eyes were cleaned with sterile saline on a daily basis to prevent any build up of discharge along the irregular lid margins which could contribute to recurrent conjunctivitis. The distichia on the upper lids were left untreated.

This case was clearly misdiagnosed and the dog underwent several unnecessary procedures. The history of recurrent ulceration in one eye, while both have similar distichia, should alert one to the fact that there is likely to be another explanation for the ulcer. An ectopic cilium had never been diagnosed. Had the lashes simply been plucked in the first instance it is likely that it would have been immediately clear that there was another problem present. The distichia on the upper lids had regrown despite diathermy – although the upper lids were not scarred in the same way as the lower ones, and so one assumes that lower energy settings were used. The treatment for the previously noted distichia on the lower lids was clearly quite aggressive and it is unlikely that these lashes ever caused the dog any clinical symptoms. The entropion which was reportedly present is likely to have been purely spastic as a result of the ocular pain from the ulcer. The fact that it was surgically corrected yet left a moderate ectropion corroborates this. There is little that can be done to salvage the marked scarring which was present on the left lower lid in particular. Thankfully this dog did not develop corneal damage as a result of poor lid function (due to uneven tear film spreading and poor debris removal for example).

8 Prolapsed nictitans gland

INITIAL PRESENTATION

Abnormal appearance – red

PRESENTING SIGNS

The patient will be presented due to a sudden change in appearance of the eye and the owner will have noticed a pink fleshy mass at the medial canthus (Figure 8.1). Patients are normally young dogs, 3–12 months old, although sometimes cats (e.g. Burmese) can be affected. Very occasionally an older dog will present which does not fit into the normal signalment. In this instance it is necessary to look for an underlying predisposing cause such as keratoconjunctivitis sicca which will be discussed briefly later. The condition is initially unilateral but has a definite bilateral risk. The patient is normally unconcerned about the appearance of the lump, with minimal discomfort and usually only a very mild serous ocular discharge. The mass can become very inflamed in appearance and slightly swollen within a couple of days and the discharge can become mucopurulent due to secondary bacterial infection.

CASE HISTORY

Usually there is no previous history prior to the appearance of the mass. The owners typically are alarmed by the sudden appearance and seek veterinary attention quickly. However, a few do leave the dog for a week or more since the pet does not show any symptoms of pain or visual disturbance. By this time a mucopurulent ocular discharge has often developed. The mass can appear overnight or sometimes the dog will have been out playing and returns with the strange appearance, making the owners think that the pup has been injured.

CLINICAL EXAMINATION

General clinical examination is typically normal. On ophthalmic examination the most obvious abnormality is the presence of a smooth, pink mass at the medial canthus. Mild conjunctival hyperaemia and a slight serous discharge might be present but no overt blepharospasm or discomfort is usually noted.

On close examination the mass can be seen extending from the bulbar aspect of the third eyelid, and the leading edge of the nictitans membrane cannot be seen. Sometimes there is kinking, or scrolling, of the cartilage of the nictitans membrane (Figure 8.2), although this might not be appreciated in the conscious patient when the gland is fully prolapsed. No ulceration is usually present. Schirmer tear tests should be performed and are usually normal or marginally reduced (presumably due to inflammation in the gland restricting the secretion of aqueous tears). Intraocular contents are also typically unremarkable.

The fellow eye should be fully evaluated since the condition has a strong tendency to be bilateral (although not necessarily at the same time).

The globe should be retropulsed to examine the nictitans membrane. Any scrolling or weakness of the cartilage can be examined. The bulbar aspect of the membrane should be examined if there is any concern about the gland's position. A cotton bud soaked in topical anaesthetic is held against the third eyelid for 30–60 seconds and then it can be grasped with fine fixating forceps (e.g. Von Graefe forceps, Figure 13.1) and the underside examined. In addition to checking the position of the gland, its size is evaluated and the presence of any follicles or inflammatory lesions on the bulbar aspect noted.



Figure 8.1 Prolapsed nictitans gland of the left eye in an Old English (or Victorian) bulldog.



Figure 8.2 Scrolled cartilage in a young Labrador retriever – this can be mistaken for a prolapsed nictitans gland so careful ophthalmic examination is essential.

DIFFERENTIAL DIAGNOSES

- Prolapsed nictitans gland
- Scrolled cartilage of the nictitans membrane
- Plasmacytic conjunctivitis (plasmoma)
- Neoplasia (rare)
- Nictitans cyst (rare)
- Protrusion of membrane (enophthalmos, Horner's syndrome, tetanus, ocular pain, etc.)

CASE WORK-UP

Since the condition is readily diagnosed on clinical examination there is little requirement for case work-up. The Schirmer tear test should be recorded in the notes since many of the breeds predisposed to prolapsed nictitans glands are also those which suffer from keratoconjunctivitis sicca later in life. The fellow eye should be fully evaluated and discussions with the owners over the risk to the second eye, and whether it should undergo prophylactic surgery at the same time as correction on the affected eye, should be considered.

NURSING ASPECTS

Apart from general nursing care pre- and postoperatively, and the application of topical medication as prescribed, there is little specific nursing requirement for this condition.

EPIDEMIOLOGY

Prolapse of the nictitans gland is a breed-related condition. Thus, there must be some genetic aspects, although as with so many adnexal diseases in dogs the exact genetic abnormalities have not been established. Predisposed breeds are many, and include the English bulldog, great Dane, Neapolitan mastiff and other mastiff breeds, English and American cocker spaniels, Lhasa Apso and Shih Tsu, and Pekingese among others. Burmese cats are also affected but it is rare in other feline breeds. Typically dogs are a few months old when presented – bulldogs are frequently very young, sometimes less than 3 months – while the larger breeds such as the mastiffs are often 6–9 months before the prolapse develops.

Although the condition is very common, the actual pathogenesis remains poorly understood. The standard wisdom is that there are anatomical anchoring abnormalities such that the gland is not properly attached to the orbital fascia, allowing it to pop out of position. A local adenitis is often present, but this is probably secondary to the abnormal anatomy rather than being the primary underlying problem. However, once the gland becomes displaced it rapidly becomes swollen and inflamed, which further affects its positioning and can result in lowered aqueous tearing as well as predisposing to secondary bacterial infections.

CLINICAL TIPS

- Check the fellow eye – the condition is frequently bilateral
- Try to replace the gland manually – if not too enlarged it might remain in situ for a day or two, protecting it from further hypertrophy and damage while surgery is scheduled (but surgery will be required – once fully prolapsed the gland will not remain in situ)
- Check for scrolling of the cartilage – this can occur before the gland fully prolapses. Indeed some breeds can present just with the scrolled cartilage, which is successfully resected, to re-present shortly afterwards with a prolapsed nictitans gland. Thus always check the gland if presented with what appears to be a purely cartilage problem
- Perform Schirmer tear tests – especially in predisposed breeds and older dogs
- DO NOT EXCISE THE GLAND!

ANATOMY AND PHYSIOLOGY REFRESHER

See introduction. The nictitans gland is found deep within the ventromedial third eyelid and is attached loosely to the periorbital tissue and base of the nictitans cartilage. It secretes the aqueous portion of the tear film, contributing up to one-third of this (the remainder comes from the main lacrimal gland).

TREATMENT OPTIONS – MEDICAL

Medical management is not a long-term option for the treatment of prolapsed nictitans glands. However, if the gland has become hypertrophied and inflamed, then a combination of topical steroids and sometimes systemic NSAIDs will help to reduce the swelling and facilitate surgery. If there has been a delay between prolapse and presentation, allowing a secondary bacterial infection to develop, then topical antibacterial medication is advised for a few days before surgical repositioning is undertaken.

Atypical presentations in older dogs can be associated with keratoconjunctivitis sicca and if tear production is restored in these patients, and the gland manually replaced, occasionally permanent surgical replacement is not required. The increased friction caused by the lack of aqueous tears causes the nictitans membrane to drag more as it moves and this can result in gland prolapse (an analogy is to think of windscreen wipers moving on a dry windscreen and you can picture how the gland can be dragged out of position). Hence correcting the tear abnormalities and manually replacing the gland can be sufficient in some patients.

TREATMENT OPTIONS – SURGICAL

Surgical repositioning of the nictitans gland is the treatment of choice and two main approaches are routinely used – pocketing the gland into the nictitans membrane, or anchoring it to the orbital rim. These will be discussed shortly. The surgical excision of the gland cannot be condoned, despite unfortunately still being performed in general practice. Since the breeds of dog which suffer from prolapse of the gland also tend to suffer from keratoconjunctivitis sicca later in life, and removal of the gland has clearly been demonstrated to result in a reduction in Schirmer tear test readings, excision of the gland cannot be recommended.

Many breeders still favour removal – it is a quick, relatively cheap procedure and certainly provides an immediate cure. However, the keratoconjunctivitis sicca which can develop later in life can be more severe than if the gland had not been removed, and frequent, expensive, life-long medication is necessary. Unfortunately, although we know that this link between gland removal and lower tear production occurs, we have no way of predicting which dogs will develop keratoconjunctivitis sicca and which will not. If owners are still adamant that they want the gland removed, even after these risks are fully explained, such that they realize that by performing a quick-fix gland excision at this stage they could jeopardize the health of their dog's eye in the long term, yet they still insist on gland removal, then sometimes practitioners are coerced into performing a procedure which they do not consider is in the best interests of the patient. It would be sensible to insist that owners sign a waiver form to the effect that they realize that surgical removal is not recommended by ophthalmologists and long-term complications could arise.

Hopefully owners will agree to surgical repositioning immediately! Currently the most widely performed method of replacement is the so-called pocket technique, with some modifications (Figure 8.3). Under general anaesthesia the nictitans membrane is reflected using stay sutures or fine fixation forceps – ensuring that these are not placed through the leading edge of the third eyelid or its cartilage – both of which could cause

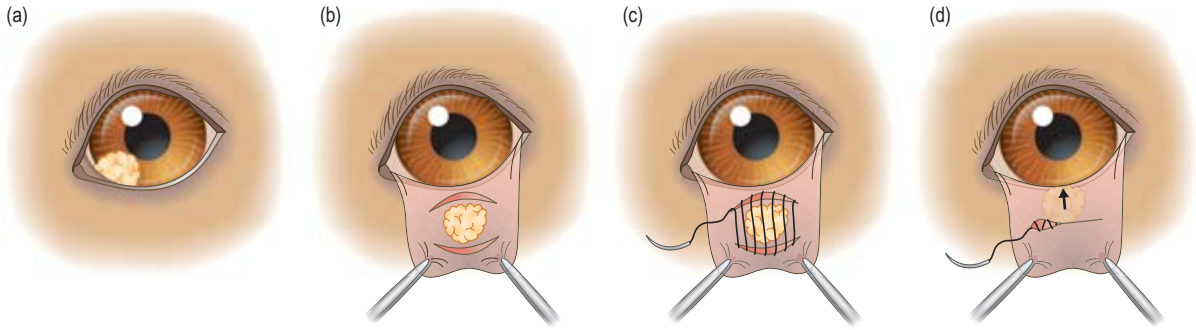


Figure 8.3 Pocket technique to replace a prolapsed nictitans gland.
(a) prolapsed gland
(b) elliptical incisions either side of gland on bulbar aspect of nictitans membrane
(c) continuous suture
(d) gland replaced into pocket and suture tied on palpebral aspect of nictitans membrane.

damage and affect the 'windscreen wiper' effect of the leading edge of the nictitans membrane. Elliptical incisions are made either side of the prolapsed gland and blunt dissection away from the gland is performed. The incisions should be through the conjunctiva and underlying tissue – deep enough for the sutures to have sufficient holding strength (too shallow and they will cheese-wire through the thin conjunctiva). The gland itself should not be incised. If there is any scrolling of the cartilage of the third eyelid the abnormal section can be resected at this time.

Closure of the incisions involves a continuous suture from the outside to outside of each incision across the gland which results in it being 'pocketed' into a fold of conjunctiva. The ends of the incision are left open which is thought to allow the lacrimal secretions to escape. The knots of the suture can be buried in the bulbar surface of the membrane, as was originally described, but a more satisfactory method is to place them on the palpebral side well away from the cornea, thus removing any risk of corneal irritation or even ulceration. The suture material of choice is 6/0 polyglactin 910 although occasionally a slightly larger size can be employed if the gland is particularly hypertrophied or in large breeds. Postoperative medication is normally topical antibiotics and systemic NSAIDs.

The owners should be warned that the third eyelid will appear red and swollen after surgery and it can take 2–4 weeks for this to fully resolve. About 3–4 weeks after surgery is the time of the highest risk of surgical failure as the sutures start to dissolve. Thus it is very important to keep a close check on patients in the first month after surgery.

SURGICAL TIPS

- The surgery is much easier if performed with the aid of magnification.
- If the third eyelid cartilage is scrolled, remove the affected portion through the incision closest to the free margin of the third eyelid before repositioning the gland.
- Place the knot on the palpebral surface of the third eyelid.
- Suture in the direction from the globe to the outside of the third eyelid – this way the needle tip is always facing away from the cornea, removing the risk of inadvertent ulceration.
- Avoid topical steroids postoperatively – the sutures could be weakened early, resulting in an increased risk of gland re-prolapse.

The second surgical technique aims to anchor the gland back down to the orbital rim. This technique is technically slightly more difficult. Previously described anchoring techniques included fixing the gland to the sclera but the risk of globe perforation makes this technique no longer recommended. An advantage to the anchoring technique is that the gland and ductules are left mainly untouched and thus tear production should not be affected in any way. However, in addition to anchoring the gland, the third eyelid itself is anchored, resulting in very restricted movement. Since brachycephalic breeds are commonly affected, which suffer some degree of lagophthalmos anyway, reducing nictitans function can result in exposure keratitis and ulceration and so should be undertaken only with care in such breeds.

The initially described technique involved an incision in the palpebral conjunctiva at the ventral fornix through which the suture was passed down to the periosteum of the ventral orbital rim and up through the gland before burying the tied suture in the conjunctival incision. Rather than feeling blindly for the periorbita, it is easier to perform the surgery via a skin incision over the orbital rim and then pass the suture up to the gland and back down (Figure 8.4). The suture should be monofilament non-absorbable of 2/0–4/0 size depending on the breed. In addition to the risk of reduced third eyelid movement, there can be infection around the retained suture (see Case example 8.1 below) and if the placement has not been adequate (i.e. not fully in the periosteum) the suture can pull through, resulting in further prolapse.

Some breeds of dog, most notably the Neapolitan mastiff, are renowned for surgical failure (see Case example 8.1). To increase the success rate a combined surgical approach using both techniques can be considered but referral is also recommended in mastiff types.

PROGNOSIS

The long-term success rate is high but there is a risk of re-prolapse of the gland (Figure 8.6). This is most common in certain breeds such as the Neapolitan and other mastiff varieties as mentioned above and in English bulldogs – perhaps with the latter purely as a result of the high numbers of affected dogs which are seen rather than due to a true predisposition. Owners should be warned that a second surgical procedure might be required. If this is the case then every effort should be made to ensure that it is successful – the surgery is always more difficult second time around. Referral should be considered.

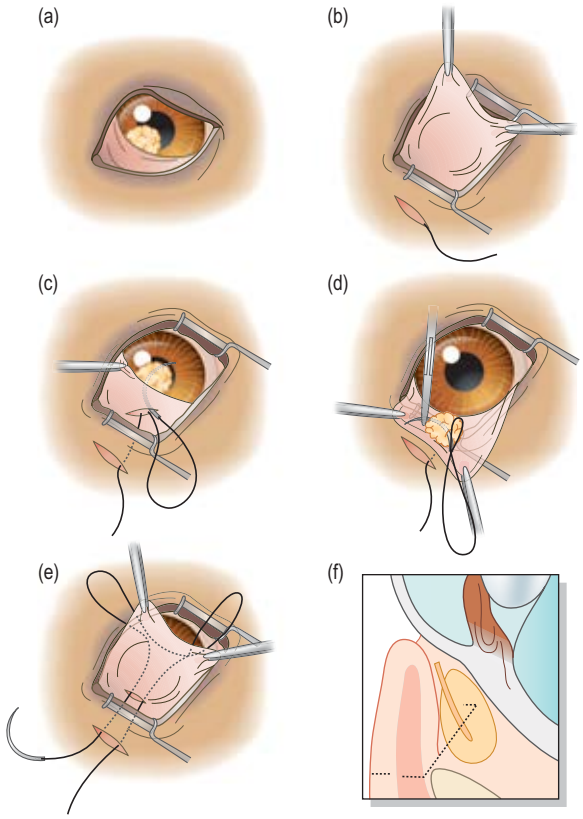


Figure 8.4 Anchoring of prolapsed nictitans gland to orbital rim:
(a) prolapsed gland
(b) skin incision ventromedial to globe. Third eyelid elevated
(c) suture passed into third eyelid after bite into periosteum
(d) suture looped through gland
(e) looping pattern of suture passing through gland before taking second bite of periosteum
(f) cross-section of final result.

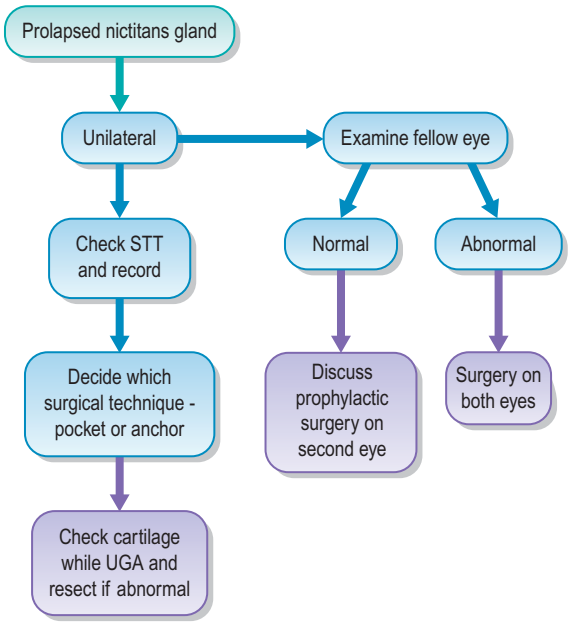


Figure 8.5 Decision-making tree – prolapsed nictitans gland. UGA = under general anaesthesia.



Figure 8.6 Partial re-prolapse of the nictitans gland – the conjunctiva is very thickened and hyperaemic and the swollen gland can be seen protruding behind the third eyelid. Treating with topical steroids for a week before attempting further surgery is advised.

Tips to try to improve success rates include the use of topical steroids to help to reduce glandular inflammation prior to the surgery, but not afterwards when they could slow wound healing and increase suture failure. A larger size of suture material can be chosen, but as this will result in more tissue reaction and swelling, and possibly increase the rate of bacterial infection, a balance must be reached. Additional sutures with the pocket technique – placing them closer together or using a second row on top of the first one to doubly fold down the gland – can be considered, but again further tissue reaction can be expected from their use. Some authors suggest placing a third eyelid flap at the completion of surgery to keep the third eyelid flat and assist normal healing. However, this is not usually necessary.

Once the surgery has been completed the nictitans membrane will be prolapsed and swollen with some hyperaemia for 2–4 weeks afterwards. However, it gradually settles and tear production should remain normal. Thus owners can be given a generally good prognosis.

CASE EXAMPLE 8.1

Patient details: Neapolitan mastiff, 8 months old, male

PRESENTATION AND HISTORY

The dog was first presented at 6 months of age with a sudden onset mass at the medial canthus in the right eye. This was correctly diagnosed as a prolapsed nictitans gland and was surgically repositioned using the pocket technique. Three weeks later the gland re-prolapsed. Topical steroid drops were started and surgery was arranged for the following week, but the left eye subsequently prolapsed as well. Surgery was then performed bilaterally using an orbital anchoring technique. The third eyelids remained inflamed and swollen although the glands remained in position. However, they gradually seemed to move anteriorly and a purulent discharge developed bilaterally which did not improve with fusidic acid or chloramphenicol drops (Figure 8.7).



Figure 8.7 Young Neapolitan mastiff with partial prolapse of right nictitans gland, bilateral third eyelid protrusion and marked purulent ocular discharge. Note also the macropalpebral fissure.



Figure 8.8 Close-up of right eye showing gland visible between nictitans membrane and cornea.

CLINICAL SIGNS

The nictitans membranes were prolapsed bilaterally and were markedly inflamed. A partial prolapse of the nictitans gland was present in the right eye (Figure 8.8). A marked mucopurulent discharge was present in both eyes with what appeared to be an abscess in the ventral nictitans conjunctiva in the left. His eyelids were too long, resulting in some lower lid lateral ectropion. Intraocular contents were unremarkable.



Figure 8.9 Swollen nictitans membrane in left eye with ventral abscess.

WORK-UP

Swabs were taken from both eyes. A moderate growth of mixed bacteria was isolated, most notably a beta-haemolytic streptococcus which was resistant to fusidic acid but sensitive to everything else tested. Closer examination of the left eye after the discharge had been bathed revealed an abscess was based around a suture (Figure 8.9). Further surgery was advised.

TREATMENT

Under general anaesthesia the eyes were prepped for surgery and the abscess was found to be associated with the large suture which had been used to anchor the gland to the orbital rim (Figure 8.10). The suture was removed easily and was not attached anywhere apart from in the nictitans membrane. The right had a similar suture. The glands were both partially prolapsed and in addition there was scrolling of the nictitans cartilage which further exacerbated the abnormal position of the glands. The weakened portions of cartilage were resected and the glands were surgically replaced using a combined technique of the pocket method (using 5/0 polyglactin 910) and an anchoring suture (of 3/0 prolene) placed via a skin incision into the periosteum of the ventral orbital rim. Postoperatively topical chloramphenicol ointment plus oral carprofen and cefalexin were dispensed for a week.



Figure 8.10 Left eye under GA showing suture knot as cause of abscess.



Figure 8.11 Ineffective sutures removed showing excessive knots.

OUTCOME

Both nictitans membranes remained inflamed for 3 weeks before gradually settling down. After 5 weeks topical neomycin, polymyxin B and dexamethasone drops (Maxitrol, Alcon) were dispensed for 2 weeks and the third eyelid swelling reduced and the glands remained in situ. Eyelid surgery was recommended but declined by the owner.

This case illustrates how some patients with prolapse of the nictitans glands are not always straightforward! The initial surgeries failed to identify the scrolling of the cartilages. The first pocket technique failed and so an orbital anchoring procedure was performed. The suture knots for this were excessive (Figure 8.11) and it is hardly surprising that an infection developed. In addition, the sutures did not pass through the periorbitum. They were probably placed too shallowly which is a common mistake when 'blindly' trying to feel for the orbital rim through the conjunctiva. The large size of needle makes it difficult to pass the needle accurately while smaller needles break on the orbital rim! Thus the approach via a skin incision is recommended, particularly in large breeds.

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SECTION 2

CONJUNCTIVA

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9 Conjunctiva – introduction

The conjunctiva is a very important, but often overlooked, part of the eye. Surface disease is very common in general practice and it is essential to understand the relevance of conjunctival involvement with regard to a purely superficial problem, such as a primary bacterial conjunctivitis, or as part of a more serious intraocular condition such as uveitis. We will consider a few common but often frustrating conditions which affect the conjunctiva – by no means a complete overview of all conjunctival diseases but a selection of those which are perhaps overlooked or difficult to manage.

ANATOMY/PHYSIOLOGY REFRESHER

The conjunctiva is the thin mucous membrane which lines the eyelids and the exposed portion of sclera. It can be divided into three anatomic sections which are continuous with each other. These are the palpebral conjunctiva which lines the eyelids, the bulbar conjunctiva which covers the globe, and the nictitans conjunctiva which covers the third eyelid. The palpebral conjunctiva starts at the eyelid margin and lines both upper and lower lids. It meets the bulbar conjunctiva at the fornix where the conjunctiva is reflected over the sclera of the globe. The bulbar conjunctiva continues to the limbus.

The conjunctiva is composed of a non-keratinized, stratified squamous epithelium and substantia propria. It contains goblet cells in its outer epithelium, which produce the mucoid portion of the tear film, lymphoid tissue in its middle layer, and connective tissue, blood vessels and nerves in the deepest layer. The bright red branching blood vessels can be easily seen, especially in the bulbar conjunctiva. Sensory nerves are from various branches of the trigeminal nerve (cranial nerve V). Variable amounts of pigment are also present. The conjunctiva is freely mobile except at the limbus and eyelid margins. Functions of the conjunctiva include preventing corneal desiccation, increasing eyelid mobility and providing a barrier to microorganisms and foreign bodies.

Like most parts of the eye, the conjunctiva can only respond to noxious insults, be these endogenous or exogenous, in a limited number of ways.

Conjunctivitis, inflammation of the conjunctiva, is the most commonly diagnosed ocular condition encountered in general practice and reaching a definitive diagnosis can be challenging in some cases. The three most common reactions of the conjunctiva are hyperaemia, chemosis and ocular discharge. Pain is a variable symptom, while follicle formation is encountered in many cases of chronic conjunctivitis and is not pathognomonic for any particular aetiology.

Hyperaemia varies in severity but should be distinguished from episcleral or scleral congestion. Involvement of these deeper vessels normally indicates some form of intraocular disease, although the conjunctival blood vessels will also be hyperaemic in this instance. Thus the anatomical location of the hyperaemia is very important in differentiating a purely superficial conjunctivitis from a potentially more serious intraocular inflammatory process. Remember that the conjunctival vessels are narrow, branch frequently and are often tortuous in nature. They will move as the eyeball position changes. Deeper vessels tend to be thicker, straighter, perpendicular to the limbus and rarely branch. They are also normally a darker red in colour.

Chemosis is most noticeable in acute conjunctivitis, where swelling or oedema of the conjunctiva can be dramatic. The loose arrangement of cells within the conjunctival stroma allows the rapid and extensive oedema to develop. The bulbar conjunctiva is normally the most likely to be involved and may be so swollen as to prevent visualization of the globe. Hyperaemia may or may not be present. The Shar Pei breed often has the appearance of chemosis which is actually due to an accumulation of

mucin in the conjunctiva in the same way as mucinosis is seen in the skin. It is not an indication of conjunctivitis in the breed unless accompanied by hyperaemia and since ocular problems are so common in the breed it is useful to remember this!

An *ocular discharge* is often the presenting complaint with cases of conjunctivitis. This can be serous, mucoid, mucopurulent or purulent in nature. Consideration of sample collection for viral or bacterial culture should be made and such samples should be taken before the discharge is cleaned away or any topical agents (such as topical anaesthetics or fluorescein dye) are applied to the eye.

Normal commensal bacteria are present in the conjunctival sac – indeed they can be isolated from 70–90% of normal dogs (lower percentages in cats) and thus a positive culture does not necessarily correlate to significant infection. The most common isolates from normal canine eyes are Gram-positive organisms such as *Staphylococcus*, *Bacillus* and *Corynebacterium* spp. The normal flora will vary with the climate (both geographic and seasonal) and the breed. Normal cytology of the conjunctiva (via scrapings for example) will reveal sheets of epithelial cells and occasional bacteria but white blood cells are rare (unlike in disease processes). Examination of pathological conjunctival scrapes can help identify the underlying disease process. For example, keratinized cells can be seen in cases of keratoconjunctivitis sicca, chronic irritation from entropion, and vitamin A deficiency, while goblet cell proliferation is commonly encountered in cases of keratoconjunctivitis sicca. However, many chronic changes in the conjunctiva are non-specific and thus both cytology and biopsy samples can be disappointing.

10 Acute conjunctivitis in dogs

INITIAL PRESENTATION

Colour change – red

PRESENTING SIGNS

Acute conjunctivitis is very common in dogs and normally presents as a suddenly inflamed, red eye, with some discharge. This is initially serous in nature but can become mucopurulent after 1–2 days. In addition to the colour change, owners will report various degrees of irritation or discomfort. This can range from slight blepharospasm to severe self-trauma and secondary eyelid or periorbital involvement. One or both eyes can be affected. Occasionally more than one dog in the same household will be affected, which obviously suggests an infective aetiology.

CASE HISTORY

Owners will have normally been aware that the dog has had red eye(s) for a day or two before presentation. The conjunctival hyperaemia can be marked and usually it is the change in appearance of the eye(s) rather than the discharge or irritation about which the owner is concerned. The condition could have developed suddenly, after a walk for example, or been slightly more insidious, taking 48 hours to manifest. Sometimes owners might have tried treating the dog themselves with some ocular medication present in the house. It is not uncommon for them to have tried chloramphenicol drops, since these are available over the counter in the UK, prior to bringing the pet to the surgery.

CLINICAL EXAMINATION

Even if the patient seems to be suffering from a straightforward conjunctivitis on initial presentation it is important to perform a full general clinical examination as well as an ophthalmic one, since conjunctivitis can be a manifestation of some systemic diseases (coagulopathies, toxicities and neoplastic processes for example). Failure to appreciate this can be detrimental to the patient. However, assuming that the clinical examination is normal, attention can be focussed on the ocular abnormalities present. Conjunctival hyperaemia is the hallmark of conjunctivitis, with variable degrees of chemosis and some increased lacrimation.

Clinical signs of conjunctivitis

- Hyperaemia
- Chemosis
- Ocular discharge
- Haemorrhage
- Follicle formation
- Pruritus
- Abnormal swellings
- Subconjunctival haemorrhage.

It is very important to establish whether these signs are due to purely conjunctivitis or whether other ocular involvement is present. Intraocular disease will normally be accompanied by conjunctivitis. Thus attention to examining menace responses, pupillary light reflexes, evidence of deeper episcleral congestion and intraocular examination are necessary (Table 10.1). Intraocular pressure should be measured if there is any evidence of intraocular disease.

If the ophthalmic examination reveals no intraocular disease, the next step is to determine whether the conjunctivitis is primary or secondary. The latter is common,

Table 10.1 Differential diagnosis of ocular inflammation

	Conjunctivitis	Superficial keratitis	Anterior uveitis	Glaucoma
Conjunctiva	Hyperaemic Thickened Sometimes folded	Hyperaemic Not thickened	Not thickened, vessels easily seen	Not thickened, vessels easily seen
Conjunctival vessels	Superficial, injected, diffuse and branching with both palpebral and bulbar vessels affected	Superficial bulbar vessels injected, especially close to limbus	Mild injection of superficial vessels but deeper, darker, straighter vessels engorged	Mild injection of superficial vessels but deeper, darker, straighter vessels engorged
Discharge	Serous to purulent, often copious	Serous to purulent; can be copious	Serous	Serous
Pain	Minimal	Minimal (unless ulcerated when moderate)	Moderate to severe, especially if acute	Moderate to severe, especially if acute
Photophobia	Minimal	Minimal to moderate	Severe if acute, otherwise moderate	Moderate
Cornea	Unaffected	Opaque and possibly ulcerated	Oedematous	Oedematous
Pupil size	Normal	Normal	Small	Dilated
Pupillary light reflexes	Normal	Normal	Sluggish	Absent or sluggish
Intraocular pressure	Normal	Normal	Low	High

with corneal ulceration, entropion, eyelash abnormalities, foreign bodies and keratoconjunctivitis sicca being most frequently involved. Once these have been ruled out we can, by a process of elimination, surmise a primary conjunctival disease (Figure 10.1).

DIFFERENTIAL DIAGNOSES

- Primary conjunctivitis:
  - Viral
  - Bacterial
  - Allergic
  - Toxic or chemical

  (Fungal and parasitic diseases are rare in the UK)
- Secondary conjunctivitis:
  - To adnexal disease
  - To corneal disease (e.g. keratoconjunctivitis sicca or ulceration)
  - To retrobulbar disease
  - To systemic disease

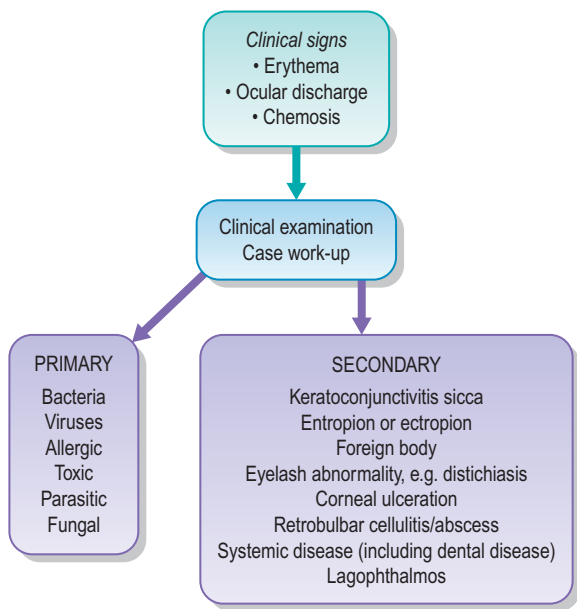


Figure 10.1 Steps in the diagnosis of conjunctivitis.

CASE WORK-UP

Once a tentative diagnosis of primary conjunctivitis is reached, a decision is made regarding whether to treat empirically initially or to embark on a full investigation. Often the latter is only undertaken should initial treatment be ineffective. Primary viral conjunctivitis is rare in dogs. Swabs for bacterial culture and sensitivity can be taken, while cytology scrapes can be informative regarding cell type (e.g. multiple neutrophils noted in an acute bacterial infection, eosinophils in an allergic reaction). If an allergic aetiology is suspected, careful examination of the patient should be repeated to look for signs of atopy – conjunctivitis frequently accompanies the skin disease. Careful questioning of the owner is also important if an allergic conjunctivitis is suspected – has a smoker moved into the house, have they put some new plants in the garden and so on. Conjunctival biopsy specimens are often less helpful than cytology samples.

NURSING ASPECTS

Other than routine hygiene, preventing self-trauma and topical medication, there are no specific nursing requirements for primary conjunctivitis in the dog.

EPIDEMIOLOGY

Primary bacterial conjunctivitis is not particularly common in dogs – secondary infections due to corneal ulcers, eyelid disease or keratoconjunctivitis sicca are more likely. However, occasional primary infections do occur, and can be contagious – several dogs at a show or working event might become affected or more than one dog in the same household. *Escherichia coli* and staphylococcal, streptococcal and *Corynebacterium* spp. are all frequently involved, but care should be taken when interpreting the result of bacterial culture since several of the isolates are also normally present in low numbers in many dogs. The patient's ocular conformation can affect the course of the infection – for example, cocker spaniels and those with deep set eyes (see medial canthal pocket syndrome) might be more prone to repeated primary bacterial infections.

Allergic conjunctivitis is encountered regularly. Hypersensitivity reactions to plant allergens can occur through direct contact or more commonly as a manifestation of atopy. Young golden and Labrador retrievers are particularly susceptible to the latter and an inherited component is definitely present. Food allergies can also cause conjunctivitis so should not be overlooked. Allergies to bacterial toxins can occur, with staphylococcal and streptococcal infections being most frequently to blame. Additionally, hypersensitivity reactions to topical antibiotics, most notably aminoglycosides, can occur and a total cessation of treatment can result in spontaneous resolution should this be the case. Sometimes simple irritants can cause primary conjunctivitis and these should not be overlooked (Table 10.2).

Table 10.2 Irritants which can cause primary conjunctivitis

Exogenous	Endogenous
• Dust	• Keratoconjunctivitis sicca
• Sand	• Entropion
• Foreign body	• Ectropion
• Smoke	• Cilia abnormalities (e.g. distichia)
• Wind	• Trichiasis
• General air pollution	• Lagophthalmos

CLINICAL TIPS

- Check thoroughly for any evidence of other ocular disease – secondary conjunctivitis is more common than primary in dogs
- Perform Schirmer tear test
- Perform fluorescein dye
- Check under nictitans membrane and in ventral fornix for foreign body
- Repeat full ophthalmic examination if response to treatment is not as expected

TREATMENT OPTIONS – MEDICAL

In the case of a suspected bacterial infection a broad spectrum topical drop or ointment is advised. Since most bacterial infections are Gram-positive, fusidic acid is a good choice, as is chloramphenicol. A significant improvement should be expected within 48 hours. Thus the patient should be recalled then. If there is no meaningful improvement then the full ophthalmic examination should be repeated to ensure that an underlying problem (perhaps a grass seed awn in the fornix or a ventromedial ulcer) has not been missed. If a primary cause is still not apparent, then a swab should be taken for culture and sensitivity testing and cytology scrapes considered. It is

not appropriate to simply change antibiotic preparations without good reason. Topical anti-inflammatory agents are not usually necessary in cases of primary bacterial conjunctivitis and their routine use is discouraged.

Cases of allergic conjunctivitis can be frustrating to treat if the inciting agent is not immediately apparent. Thus, if a topical hypersensitivity to a plant in the garden has occurred, or to an applied medication, avoidance of the culprit should result in resolution of symptoms. Unfortunately, the inciting allergen is often unclear. Since atopic conjunctivitis is so common, attention to generalized skin disease with appropriate intradermal testing as indicated should be considered. In addition to treating the dermatitis, empirical treatment with topical steroid drops will relieve the irritation and conjunctival hyperaemia, but clearly does not address the cause. Topical mast cell stabilizers have been used in dogs, but they are not licensed for such use and their effects can be variable. Since they are most effective prior to the onset of the inflammation, they are really only of use in cases of seasonal allergic disease where the initiating allergens are known and treatment can start before these pollens or similar start to trouble the dog.

PROGNOSIS

The prognosis for primary bacterial conjunctivitis is good and most cases resolve fully within 5–7 days.

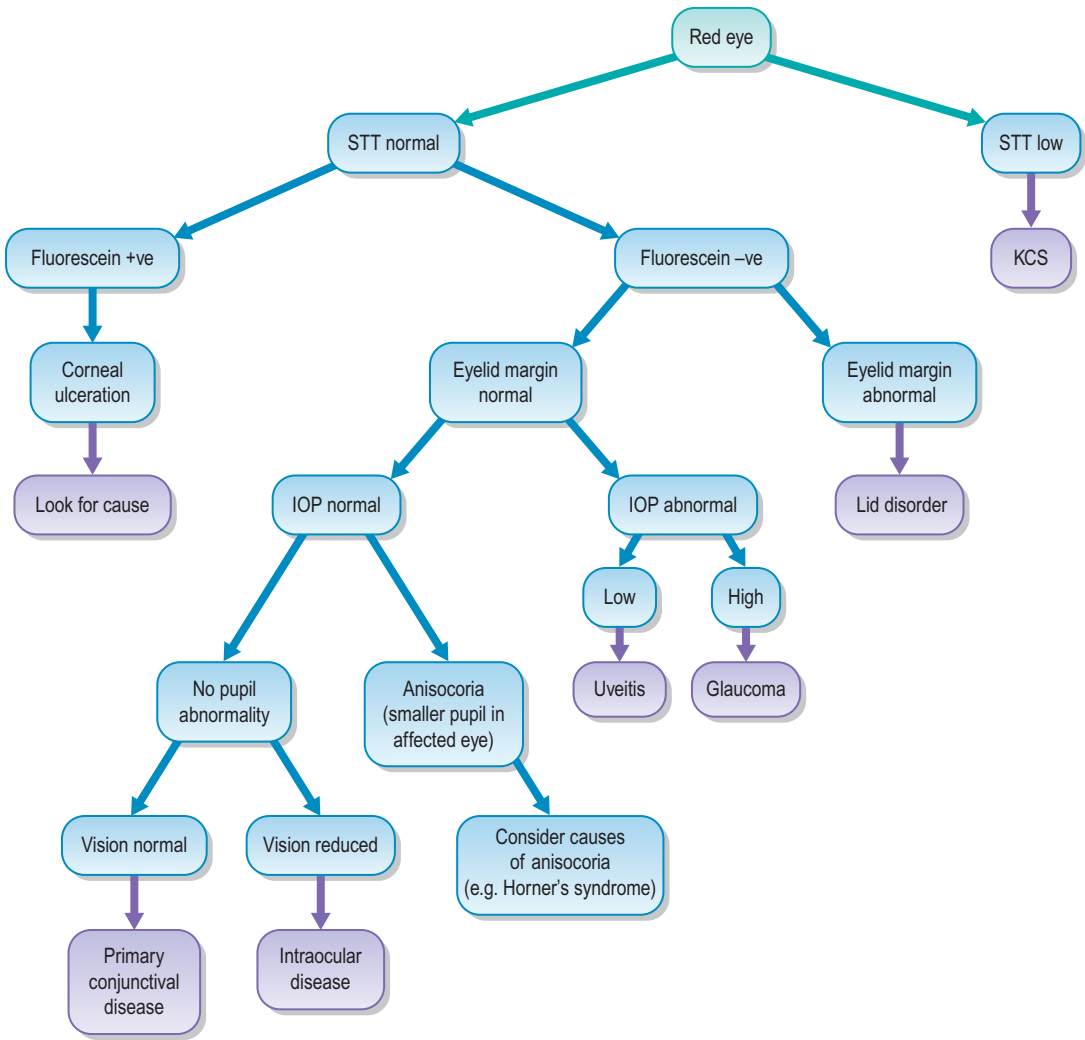


Figure 10.2 Decision-making tree – acute conjunctivitis.

Failure to detect an underlying abnormality such as keratoconjunctivitis sicca will result in recurrence of the symptoms and therefore repeated, thorough general and ophthalmic examinations are required should any relapses occur.

Allergic conjunctivitis can be more frustrating to treat. Although the condition is not particularly severe – causing redness and mild irritation rather than being sight threatening for example – it does cause low grade debilitation for the patient and owners can become despondent at the poor response or repeated attacks of conjunctivitis. Careful client education and addressing any underlying systemic allergies are important for successful resolution.

CASE EXAMPLE 10.1

Patient details: Jack Russell terrier, 4 years old, female (neutered)

PRESENTATION AND HISTORY

- Bilateral red eyes
- Serous ocular discharge
- Rubs both eyes from time to time
- Occurred previously at same time of year (June)
- Occasionally chews her feet

CLINICAL SIGNS

General clinical examination unremarkable apart from saliva staining of all four feet.

Ophthalmic abnormalities were limited to mild conjunctival hyperaemia affecting both bulbar and palpebral conjunctiva (Figure 10.3).

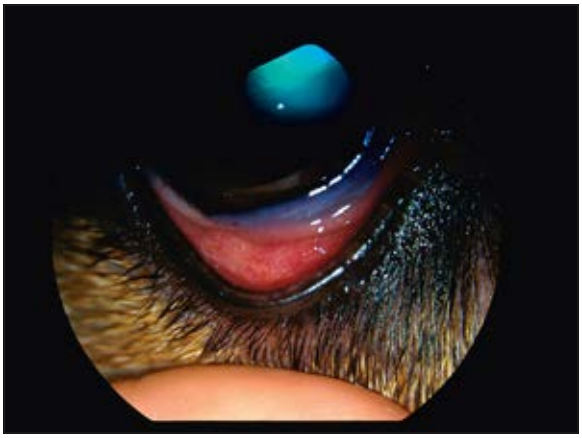


Figure 10.3 Allergic conjunctivitis in a 4-year-old Jack Russell terrier.

WORK-UP

Full ophthalmic examination, checking for corneal ulceration, adnexal disease and so on revealed no abnormalities. Cytology considered but owner not keen on further work-up initially. Suspected to be allergic – either local irritation from something in the environment or associated with mild atopy.

TREATMENT

Twice daily dexamethasone drops relieved all conjunctival redness and the discharge was fully resolved. However, on stopping medication after 2 weeks the symptoms quickly returned.

OUTCOME

Further investigation was advised and the owner agreed. Full dermatological assessment was very suggestive of atopy but the owner felt that symptoms were not sufficiently severe to warrant the proper work-up. The steroid drops were restarted and then changed to lodoxamide (mast cell stabilizer) twice daily once conjunctivitis settled again. The owner stopped the drops after 4 weeks and the symptoms returned. On restarting dexamethasone, the signs cleared up again and treatment was continued once daily throughout the summer. Treatment was stopped in the autumn and the eyes remained asymptomatic. The owner was warned that this was most likely a seasonal allergy and similar symptoms could recur the following year.

11 Feline conjunctivitis

INITIAL PRESENTATION

Colour change – red

PRESENTING SIGNS

Conjunctivitis can present in a number of ways in cats but conjunctival hyperaemia, a red eye, will always be a main owner concern. Since the presentation is similar regardless of cause, careful questioning of the owner regarding the progress of the disease, vaccination status, previous episodes and so on is required. In general, it is sensible to rule out the infectious causes, by far the most frequent cause for feline conjunctivitis, before considering other aetiologies such as allergy, neoplasia or tear film abnormalities.

Conjunctivitis can be seen as part of an upper respiratory disease in young kittens, with whole litters (plus sometimes the dam) being affected to varying degrees. When present as part of the cat 'flu complex like this, due to feline herpes virus infection, corneal involvement is common, with ulceration and even perforation occurring. Older kittens and adults can present with a unilateral conjunctivitis which later becomes chronic and can affect both eyes. Although this is also associated with feline herpes virus (FHV-1), respiratory involvement is much less common. *Chlamydophila felis* is also frequently implicated in feline conjunctivitis and corneal involvement is very unusual. Both kittens (usually over 4 weeks of age) and adults can be affected and the conjunctivitis can be both acute and chronic, affecting one or both eyes.

CASE HISTORY

Normally the history is one of an acutely red, wet eye. The discharge is initially serous but becomes mucopurulent within a few days. Involvement can be unilateral or bilateral. If upper respiratory signs are present, then FHV-1 must be considered the most likely causal agent but other infectious processes could coexist. Checking the vaccination status of affected animals, including the dam if young kittens are presented, is important, but the condition can still develop in vaccinated cats, although usually in much milder forms.

Slightly older kittens and adults will normally present with a unilateral conjunctivitis and may have a history of previous episodes. The recurrences are most likely when the cat is stressed – for example, placed in a cattery, introduction of new cat(s) to the home, given a general anaesthetic, immunosuppressed for another reason (feline leukaemia virus or systemic steroid administration) and so on. The owner should be asked if any of these stress factors have occurred to their cat recently. Occasional sneezing might be reported but recrudescent FHV-1 is typified by chronic conjunctivitis, sometimes with corneal involvement, but not with full blown respiratory disease. Respiratory signs are not usually a feature of *Chlamydophila* infection.

Although less common than infectious causes, allergic conjunctivitis is recognized in cats, most frequently due to a topical hypersensitivity to prescribed medication. Chlortetracycline is notorious for this so if the history is of a conjunctivitis which initially responded to this drug, but then the cat developed a marked conjunctival hyperaemia, possibly with some attempts at rubbing or eyelid involvement, then an allergic component should be considered. Stopping all topical medication for several days can sometimes be curative!

CLINICAL EXAMINATION

General clinical examination is important for all cases of feline conjunctivitis. Acute infections with FHV-1 mani-

fest as the 'cat 'flu' complex in young kittens and older, unvaccinated cats, and thus both respiratory signs and a pyrexia are common. If no systemic involvement is noted, then careful ophthalmic examination is required. This should rule out secondary causes of conjunctivitis such as entropion or a conjunctival foreign body, as well as keratoconjunctivitis sicca (see Figure 10.1). The cardinal signs of conjunctival hyperaemia, chemosis and increased lacrimation with a serous or mucopurulent discharge are non-specific for the different causes of conjunctivitis and therefore further case work-up is necessary to reach a definitive diagnosis.

Careful corneal evaluation is required since ulceration or keratitis with evidence of vascularization and scarring could suggest FHV-1 over *Chlamydophila*. Any symblepharon or limbal damage could also point to FHV-1 as described in later sections. Any chronic conjunctivitis can lead to follicle formation and this is not pathognomonic for any particular aetiology. The use of rose Bengal, rather than fluorescein, as a corneal dye is recommended in suspected cases of FHV-1 conjunctivitis since this might highlight areas of corneal epithelial devitalization which manifest as dendritic ulcers – pathognomonic for FHV-1 – which are rarely highlighted with fluorescein alone.

Intraocular involvement is not a feature of feline conjunctivitis but clearly if any evidence of uveitis for example is noted, then the tentative diagnosis should be reviewed and further ocular evaluation undertaken. As always, both eyes should be thoroughly examined, even if only one appears affected on initial presentation.

DIFFERENTIAL DIAGNOSES

- FHV-1 – the most frequently encountered infectious cause of ocular disease in cats
- *Chlamydophila felis* – very common cause of acute and chronic conjunctivitis
- *Mycoplasma felis*
- Calicivirus infection (rarely causes ocular symptoms alone)
- Bacterial conjunctivitis (rarely primary in cats)
- Allergic conjunctivitis
- Foreign body
- Keratoconjunctivitis sicca
- Proliferative (eosinophilic) keratoconjunctivitis
- Neoplasia (e.g. lymphoma)
- Secondary to other ocular disease – entropion, uveitis, glaucoma, etc.

CASE WORK-UP

Once the ophthalmic examination has confirmed that conjunctivitis is the clinical diagnosis, rather than any other ocular disease in which conjunctival involvement is a feature, then investigation of the possible underlying cause is required. However, this can be frustrating and inconclusive on occasion. Swabs should be taken of both conjunctiva and oropharynx and placed in combined viral and chlamydial transport medium (VCTM). These can be used for both virus isolation and the more sensitive polymerase chain reaction (PCR) which is available for *Chlamydophila* as well as FHV-1. Unfortunately, intermittent excretion of both agents can lead to false negatives and repeated sampling might be required in some cases. Serology is of no use in vaccinated cats and only of limited use for *Chlamydophila* since high titres are common and can remain for many months post-infection.

Cytology can be useful for *C. felis* by demonstrating intracytoplasmic inclusion bodies in the epithelial cells during the acute phase of the disease. It can also help identify allergic components or if secondary bacterial infections are present.

Conjunctival biopsy can be considered in atypical cases, particularly if a neoplastic process such as lymphoma or squamous cell carcinoma is suspected.

NURSING ASPECTS

Nursing of kittens affected with the cat 'flu complex can be time consuming but is also very rewarding. Barrier nursing to prevent cross-infection must be maintained. The condition is highly contagious and spread is via direct contact (discharges and fomites) and sneezing. Specific ophthalmic nursing will include frequent removal of ocular discharges by gentle bathing, application of broad spectrum antibiotic agents topically, along with ocular lubricants and sometimes topical antiviral drops. General nursing support with cleansing of nasal discharges, supplementary feeding and intravenous fluids in severe cases will be required. Systemic antibiotics are usually necessary, along with decongestants in some cats.

Older animals which present with just conjunctivitis but no systemic involvement also need regular attention with removal of discharges and frequent application of both topical and systemic medications. Nurses should ensure that owners are able to medicate their cat effectively.

EPIDEMIOLOGY

Since the two most common causes of conjunctivitis in cats – FHV-1 and *C. felis* – are infectious in nature, outbreaks can occur in multi-cat households and in outdoor cats which come into contact with infected cats. Both agents are ubiquitous in the feline population. Other infectious causes such as *M. felis* and calicivirus are less common, and primary bacterial infections are uncommon in cats (although secondary infections do occur). Less common causes such as topical hypersensitivity or neoplasia will only occur on an individual cat basis.

Initial infection with FHV-1 will cause respiratory signs – rhinitis, tracheitis and bronchopneumonia – as well as ocular signs, with conjunctivitis and corneal involvement particularly in young kittens and unvaccinated cats. The disease is caused by the direct cytopathic effect of viral replication in the relevant epithelium. Incubation periods are typically 2–10 days, but vary according to the amount of virus present and its pathogenicity, and the acute phase of infection will last about 2 weeks. Up to 80% of infected cats become carriers, and the latent infection can reactivate under conditions of stress – new cats in the neighbourhood, building work at home, other illnesses or anaesthesia, immunosuppression, prescribed medication such as systemic steroids and so on. Even treating these cats can stress them sufficiently to exacerbate viral replication and so a balance often has to be met between the ideal treatment for the condition and the individual temperament of the cat and its severity of symptoms. The reactivation of latent virus can result in chronic conjunctivitis or many of the more severe sequelae of FHV such as symblepharon, corneal ulceration, sequestrum formation, keratoconjunctivitis sicca, stromal keratitis and chronic epiphora (due to punctal occlusion). Table 11.1 summarizes the ocular manifestations of FHV-1 infection.

Table 11.1 Ocular manifestations of feline herpes virus

Age	Clinical signs
2–4 weeks – primary infection	Upper respiratory disease Sometimes life threatening Severe bilateral conjunctivitis Marked serous to purulent ocular discharge Severe corneal ulceration common Symblepharon formation
3–6 months – primary infection	Upper respiratory disease Acute bilateral conjunctivitis Corneal ulceration mild to moderate Resolves in 14 days
Older than 6 months – secondary infection or recurrence	Minimal respiratory involvement Chronic conjunctivitis Chronic epiphora Usually unilateral Stromal keratitis Recurrent ulceration Sequestrum formation Keratoconjunctivitis sicca

C. felis is an obligate intracellular bacterium and is a primary conjunctival pathogen. Neonatal infections are rare – kittens are normally at least 4 weeks old before symptoms develop which is another difference from FHV-1, as is the lack of both corneal and respiratory involvement. Untreated, the infection can drag on for weeks or months in some patients with a chronically thickened hyperaemic conjunctiva and persistent ocular discharge. The combination of a short-lived immunity, repeated infections and carrier status (with the agent remaining in the gastrointestinal tract) all contribute to the recurrent nature of the conjunctivitis in some cats. In a cattery or multi-cat household, treatment of the whole population followed by PCR to check for carriers, with isolation of those still affected, is advised prior to vaccination of the stock. This can certainly help control the condition but vaccination is not usually warranted on an individual basis.

Since both FHV-1 and *C. felis* can result in carrier status, such that recurrence is likely, owners need to be fully appraised of this to prevent frustration and therefore reduced compliance with medication, which can be both time consuming and expensive.

Other agents which can be implicated in infectious feline conjunctivitis, such as *M. felis* and calicivirus, are often found in conjunction with the two main infectious agents. Table 11.2 lists the similarities and differences between the main infectious causes of conjunctivitis in cats.

CLINICAL TIPS

- Meticulous ophthalmic examination to check for other causes for conjunctivitis
- Suspect infectious agents – the most common cause of feline conjunctivitis
- If there is corneal involvement, suspect FHV-1 rather than *Chlamydophila*
- If chemosis is severe, suspect *Chlamydophila* over FHV-1

Table 11.2 Differential diagnosis of feline conjunctivitis

	Feline herpes virus (FHV-1)	*Chlamydophila felis*	*Mycoplasma felis*	Feline calicivirus
Ocular signs	Conjunctivitis and keratitis	Conjunctivitis	Conjunctivitis	Increased lacrimation
Systemic signs	URTI in young cats	None	None	Mild URTI Oral ulcers
Incubation period	2–10 days	6–15 days	4–10 days	1–9 days
Course of infection	2–4 weeks Can be chronic	Often chronic	Sometimes chronic	7–10 days
Immunity	Usually weak and transient but can be persistent	Weak and transient	Weak and transient	Good protection to some strains
Carrier status	Latent phase with periodic excretion	Yes	Yes	No
Morbidity	High	Variable	Variable	High
Mortality	High in kittens, low in adults	Very low	Very low	Moderate in young kittens
Diagnosis	PCR and virus isolation	PCR and cytology (inclusion bodies)	Culture	Virus isolation
Treatment	Symptomatic and supportive, topical antivirals	Topical tetracyclines, systemic doxycycline	Systemic doxycycline	Supportive
Vaccination	Yes	Yes	No	Yes

PCR, polymerase chain reaction; URTI, upper respiratory tract infection.

- Chronic follicular conjunctivitis can occur with both agents but is more common with *Chlamydophila*
- Take swabs and samples before the application of any dyes
- Rose Bengal is more sensitive than fluorescein for herpetic (dendritic) ulcers

TREATMENT OPTIONS – MEDICAL

The treatment for feline conjunctivitis is medical rather than surgical. Obviously ideally the causative agent should be identified by PCR, virus isolation or cytology samples but this is not always feasible, particularly in initial infections.

For young kittens with both respiratory and ocular involvement, symptomatic treatment with broad spectrum antibiotics to treat or prevent secondary bacterial infection is required along with supportive nursing. Specific antiviral medication is not usually necessary since the ocular signs are often self-limiting in 10–14 days, providing ulceration is not a serious feature. If severe ulcers are present then the risks of sequelae such as corneal perforation, chronic keratitis, symblepharon and keratoconjunctivitis sicca are all much higher. Thus topical antiviral agents such as trifluorothymidine drops (available from the pharmacy at Moorfields Eye Hospital) are indicated if severe ulceration is present. Clearly these need to be used alongside antibacterial agents. Unfortunately, frequent medication – up to eight times daily – is required initially and the cost is also considerable. This is one reason why topical antivirals are often reserved for chronic herpetic keratitis cases. As the topical antiviral agents can also trigger a hypersensitivity reaction, careful, repeated examination and evaluation are required if they are employed. If should be noted that no antiviral preparations are currently licensed for use in cats in the UK.

The choice of antibacterial agent will depend to some extent on whether *Chlamydophila* is suspected and the age of the kittens affected. Since tetracyclines are not indicated in young kittens, a better choice would be combined amoxicillin/clavulanic acid orally which can be safely given to lactating queens as well. A broad spectrum topical agent such as chloramphenicol is useful – ointments should be avoided as they can contribute to the thick discharge and encourage the eyelids to stick together, increasing the discomfort for the kitten.

Chronic herpetic conjunctivitis in older cats can be frustrating to treat. The use of topical antivirals is again controversial in these cases and they tend to be reserved for cats with concurrent corneal disease. In mildly affected cats the conjunctivitis can be short lived and self-limiting, and the stresses of medication can be worse than the disease itself in nervous cats for example. If secondary bacterial infection is suspected, a short course of topical antibiotics might help, but topical steroids should be avoided – even if the conjunctiva appears very inflamed – since their use can predispose to further viral replication and corneal ulceration, making the condition much worse.

Oral L-lysine has been shown to decrease the severity of FHV-1 conjunctivitis in cats and, since it is a natural amino acid, it is safe to use. However, controlled clinical trials are lacking – and this is somewhat reflected in the various treatment regimens reported. These range from 250 mg once daily during the acute phase of the infection up to 500 mg twice daily indefinitely! It might be of use in catteries or prophylactically prior to the cat undergoing a stressful experience (e.g. the introduction of another cat to the household). The L-lysine is thought to work by competing with arginine, which is required for viral replication, and thus higher levels of lysine will slow viral replication.

Other treatments for FHV-1 conjunctivitis include both topical or oral interferons, and systemic antiviral agents such as aciclovir and famciclovir. However, clinical trials regarding their efficacy have not, to date, been published and since their use is not licensed in cats they cannot be recommended as a first line of treatment in general practice at this time. Table 11.3 summarizes the rationale for treating FHV-1.

Conjunctivitis due to *C. felis* is, in theory, more straightforward to treat since it is sensitive to tetracyclines. Topical chlortetracycline needs applying four times daily for up to several weeks and care should be

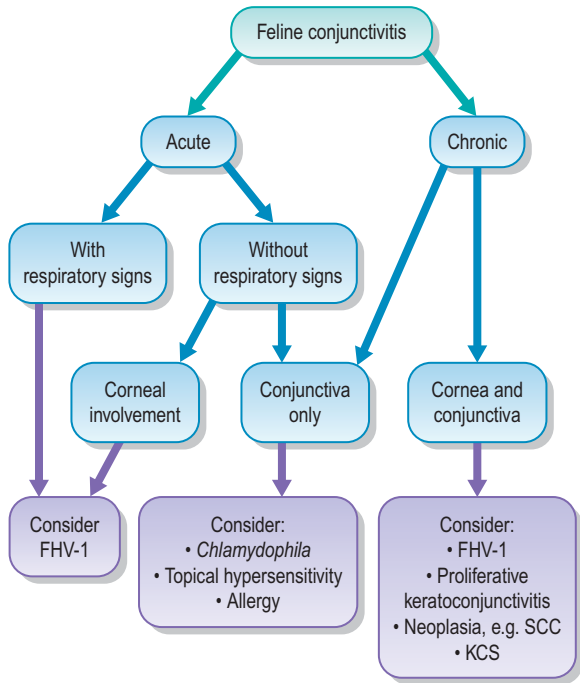


Figure 11.1 Decision-making tree – feline conjunctivitis.

Table 11.3 Treatment options for FHV-1 conjunctivitis

Primary infection in kittens	Recurrent conjunctivitis	Recurrent keratitis
• Supportive care • Systemic amoxicillin/clavulanic acid • Topical chloramphenicol drops • Consider ocular lubricant	• Might run its course within 2 weeks without treatment • Consider short course of topical antibiotics (chloramphenicol or fusidic acid) • Consider topical antiviral if severe (trifluorothymidine) • Oral L-lysine	• Topical antiviral drops (trifluorothymidine) • Oral L-lysine • Topical antibiotic drops • Consider ocular lubricant • Consider interferon (topically) • Consider oral famciclovir

exercised since hypersensitivity reactions are not uncommon. Fusidic acid has been shown to have some efficacy against *C. felis* but perhaps not at therapeutic doses. However, it is a sensible choice if secondary bacterial contaminants are suspected or in conjunction with oral medication. Topical anti-inflammatory agents are not indicated. The systemic drug of choice is doxycycline, at a dose rate of 5 mg/kg twice daily for 3–4 weeks. Oxytetracycline and combined amoxicillin/clavulanic acid can also be used as alternatives. The carrier status and short-lived immunity both contribute to recurrent infections and owners should be aware of this.

TREATMENT OPTIONS – SURGICAL

Surgery is not necessary for cases of feline conjunctivitis unless severe corneal ulceration is also present.

PROGNOSIS

The prognosis as far as preservation of vision is concerned is very good. However, with both FHV-1 and *C. felis* chronic recurrent disease is not uncommon and this can be debilitating for the cat and frustrating for owners. Careful owner education from the outset will help with long-term management, and a holistic approach – taking into account not only the ocular complaint but also the considerations of frequent, possibly expensive, medication and the cat's stress level – is sensible. Relatively simple changes in the cat's environment, such as locking the cat flap to prevent unwanted feline visitors or having someone come round and feed the cat at home rather than placing it in a cattery while the owners are on holiday, can reduce both the frequency and severity of relapses. Some cats will become less seriously affected with time.

CASE EXAMPLE 11.1

Patient details: Domestic short haired cat, 6 months old, male (neutered). Fully vaccinated, no previous ocular or systemic problems

PRESENTATION AND HISTORY

At a cattery for 2 weeks. After 10 days started to sneeze slightly and right eye developed acute conjunctivitis and serous ocular discharge. Left eye became similarly, but less severely, affected.

CLINICAL SIGNS

Clinical examination showed mild pyrexia, occasional sneezing, mild submandibular lymphadenopathy and an inflamed oropharynx. No nasal discharge was present. Severe conjunctival hyperaemia and chemosis of the right eye (Figure 11.2) and similar, but more mild changes in the left eye were noted. A bilateral serous ocular discharge was present. No corneal ulceration was seen.

WORK-UP

No initial work-up was performed.



Figure 11.2 Acute conjunctivitis, right eye.

TREATMENT

Topical chloramphenicol drops four times daily to both eyes for 10 days and a week of oral amoxicillin/clavulanic acid tablets.

OUTCOME

Initial complete resolution of signs within 14 days. However, 4 months later the cat developed a superficial corneal ulcer in the left eye (Figure 11.3) which responded to a week of chloramphenicol drops and healed without complication. Another month later the cat started to sneeze (as did the other cat in the house) and this time the right eye developed a large corneal ulcer and the initial serous discharge became mucopurulent (Figure 11.4). Further work-up was instigated. Conjunctival and oropharyngeal swabs were taken and placed in VCTM. Topical fusidic acid was started twice daily. PCR tests were negative for *Chlamydophila* but positive for FHV-1. Treatment was continued and oral L-lysine was started at 250 mg twice daily in food. Topical antiviral drops were considered, but as the cat resented treatment twice daily, six to eight times daily was considered too stressful for it.

After 10 days the corneal ulcer had healed with some scarring (Figure 11.5). Some permanent epiphora was present (due to occlusion of the nasolacrimal punctae). Topical medication was discontinued but the lysine was continued for 3 months. The cat continued to have occasional conjunctivitis every few months, in one or both eyes, but no further ulceration occurred. The owner was aware that the eyes could get worse each time the cat became stressed and catteries were avoided.

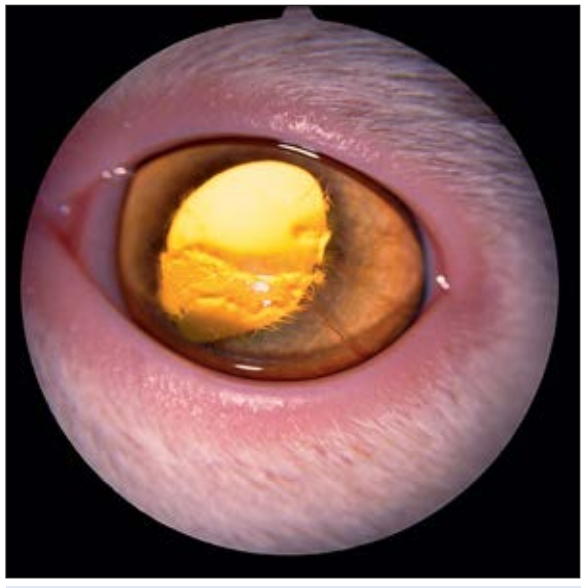


Figure 11.3 Superficial corneal ulcer in left eye.



Figure 11.4 Large corneal ulcer in right eye.



Figure 11.5 Healed right eye with some vascularization and epiphora.

CASE EXAMPLE 11.2

Patient details: Domestic short haired cat, 7 years old, female (neutered)

PRESENTATION AND HISTORY

Presented with an acutely red, watery right eye. No history of previous ocular problems and no systemic illnesses. A new cat had recently moved in next door and the two cats had started to fight.

CLINICAL SIGNS

General clinical examination was normal. Only the right eye was affected. Moderate conjunctival hyperaemia and chemosis were noted, together with a serous ocular discharge. No corneal ulceration was present. The fornix and behind the nictitans membrane were carefully examined but no foreign body was located. Intraocular contents were normal.

WORK-UP

The owner was offered diagnostic tests but elected to treat symptomatically to start with.

TREATMENT

Topical fusidic acid was started twice daily. After 4 days there was no improvement and the owner agreed to diagnostic testing. A swab was taken for bacterial culture and sensitivity testing, together with another for virus and *Chlamydophila* isolation. All proved negative. PCR was not performed. Treatment was changed to topical chlortetracycline three times daily and oral doxycycline (5 mg/kg) for 3 weeks.

OUTCOME

The conjunctivitis quickly resolved on the new treatment regimen and no recurrences were reported. The presumed diagnosis was *C. felis*, despite the lack of laboratory proof, and the cat could have become infected from the new neighbour. Shedding is not continuous and isolation is far less sensitive than PCR for detecting this. If the cat were to develop another bout of conjunctivitis, early sampling for PCR would be advised, and cytology can also be considered early in the course of disease since intracytoplasmic inclusion bodies are most likely to be detected then.

12 Medial canthal pocket syndrome

INITIAL PRESENTATION

Discharge

PRESENTING SIGNS

Medial canthal pocket syndrome (MCPS) is a very common, but rarely diagnosed, conjunctival condition. Typically it presents as a chronic or recurrent conjunctivitis, manifested by a thick mucoid or mucopurulent discharge and moderate conjunctival hyperaemia. Owners complain that the discharge is most noticeable in the morning but can recur later in the day, especially after sleeping, and that it is thick and tenacious. The dog rarely shows any signs of discomfort, despite the 'red eye', although occasionally can rub the eyes on waking – presumably to dislodge the discharge. The most frequently presented breeds include the Dobermann, standard poodle, Afghan hound, rough collie and lurchers. Other breeds with broad heads and deep set eyes such as the Weimaraner, golden and Labrador retriever, great Dane and Rottweiler can be affected.

CASE HISTORY

Typically the patient will be a young (6–18 months old), clinically well dog of dolichocephalic conformation. A chronic mucoid or mucopurulent discharge will be reported, centred medially, which does not seem to bother the dog unless it becomes copious. The patient normally improves on topical antibiotic medication, although the discharge is quick to reform as soon as the course of treatment is finished. Often the patient undergoes several treatment periods before careful examination leads to the diagnosis.

CLINICAL EXAMINATION

General clinical examination is normal. On ophthalmic examination all neurological tests are normal and the eyes are open and comfortable. Schirmer tear test readings are normal. The most obvious feature is a discharge at the medial canthus and some localized conjunctival hyperaemia (Figure 12.1). The bulbar conjunctiva is normally only very slightly hyperaemic whereas the ventral palpebral and nictitans conjunctiva can appear very inflamed. The discharge is bilateral but not necessarily totally symmetrical. Some discharge is seen within the ventral fornix, in a pocket formed by the gap between the eyeball and the lids (due to the relative enophthalmos inherent in the breed conformation). Follicles can be noted within the conjunctiva close to the discharge on cleaning it away. No corneal pathology is normally encountered but a mild ventral entropion is sometimes seen. Intraocular examination is unremarkable.

DIFFERENTIAL DIAGNOSES

- Conformational – medial canthal pocket syndrome
- Primary bacterial conjunctivitis
- Entropion
- Ectropion
- Dacryocystitis
- Keratoconjunctivitis sicca
- Conjunctival foreign body
- Allergic conjunctivitis



Figure 12.1 Typical grey–white mucoid accumulation at the medial canthus in a collie/German shepherd cross (XGSD).

CASE WORK-UP

A thorough ophthalmic examination is essential, and normally this will lead to the diagnosis of MCPS. Careful evaluation of the globe – lid positioning, together with an appreciation of the exact location of the discharge – will help. If the discharge appears purely mucoid in nature then sample collection is not warranted; however, if any purulent component is noted then a swab should be sent for culture and sensitivity testing. Cytology samples are not usually required. It is sensible to flush the nasolacrimal ducts – normally this can be done under topical anaesthesia or light sedation – since a dacryocystitis will be one main differential (although the discharge is likely to be purulent, some discomfort is likely and the distribution is more commonly unilateral than bilateral in the case of dacryocystitis).

NURSING ASPECTS

Regular bathing and cleaning away of the discharge is the mainstay for this condition. Thus owners should be shown how to do this and demonstrate their proficiency to enable good management of these affected patients.

EPIDEMIOLOGY

Breeds affected with medial canthal pocket syndrome include:

- Afghan hound
- Briard
- Dobermann
- Great Dane
- Giant schnauzer
- Golden retriever
- Lurcher types
- Rottweiler
- Rough collie
- Standard poodle
- Weimaraner.

Medial canthal pocket syndrome is caused by anatomical/conformational abnormalities. The common feature of affected breeds is a relatively small globe in a deep set orbit – specific breeding has led to these dogs being slightly enophthalmic. This leads to a gap between the eyeball and the eyelids ventrally – a pocket in the ventral fornix. In addition, mild entropion might be present together with poor tear dynamics. All of this results in a build up of normal ocular secretions at the medial canthus. This is purely mucoid in nature initially, but quickly becomes contaminated by secondary bacterial opportunists. The warm, moist, protected fornix provides an ideal medium for bacterial overgrowth. Thus isolates are normally Gram-positive agents such as *Staphylococcus*, *Bacillus* and *Corynebacterium* spp. Gram-negative bacteria and primary pathogens are rarely isolated. Dust, dirt and general debris can also accumulate in the medial pocket, exacerbating the condition and contributing to the conjunctival hyperaemia and follicle formation. In some young male dogs which are neutered young, the skull never grows to its full maturation. Since broadening of the skull occurs quite late in development, they can be left with a slightly narrower shape which on occasion can exacerbate the tendency for MCPS to develop. The condition is not contagious.

CLINICAL TIPS

- Consider the breed and age of the dog
- Examine the eye carefully for location of discharge
- Question the owner regarding the frequency of discharge build up
- Is the dog irritated by the discharge?
- Check Schirmer tear test readings
- Swab for culture and sensitivity before using topical anaesthesia or fluorescein dye

ANATOMY AND PHYSIOLOGY REFRESHER

Normally the globe should sit snugly in the orbit and the lids glide smoothly over the cornea on blinking. However, pedigree dogs have been bred for a specific appearance over generations and this has led to many conformational abnormalities in numerous breeds. Thus the breeds of dog with a long nose, narrow skull and relatively small eyes, together with those with a broad skull and deep set eyes, are prone to MCPS. The presence of a pocket at the medial canthus – the gap between the cornea and lower eyelid – is abnormal, and allows ocular secretions to build up. The palpebral and bulbar conjunctiva should be adjacent such that there is no gap to allow this accumulation. A relative entropion can develop either medially, functionally reducing nasolacrimal drainage, or laterally, contributing to increased tear production, and both of these alter the local tear dynamics. Failure to naturally remove the accumulated secretions leads to chronic, low grade irritation – manifested by localized conjunctival hyperaemia and follicle formation – together with opportunistic bacterial infection.

TREATMENT OPTIONS – MEDICAL

Hygiene is the key to managing this condition. Thus the owners are instructed to bathe the discharge, using sterile saline solution, removing it on a twice daily basis initially. Once under control they should continue with daily cleansing in the morning, plus additional bathing should the dog have been in an environment that could cause irritation – in a dusty area or on the beach for example. Working dogs should have their eyes bathed on returning from the day's work.

If a purulent discharge is present, then a topical antibiotic agent such as fusidic acid or chloramphenicol should be chosen, pending culture and sensitivity. In general, drops are advised rather than ointments since the latter can contribute to the accumulation of material. However, many cases do not require a topical antibiotic and, as such, several days of regular bathing are advised before opting for antimicrobial treatment. Topical steroids are not normally required. If significant conjunctival follicle formation (lymphoid follicles) has occurred with chronicity these normally resolve following frequent bathing.

Owners should be made aware that the condition is due to the conformation of their pet, and as such can be controlled but not fully cured. Failure to follow the hygiene guidelines will result in repeated bacterial infections, possibly involving eyelid as well as the nasolacrimal system; since these are more serious for the dog, it is important that they are avoided.

TREATMENT OPTIONS – SURGICAL

Unless a significant entropion or ectropion is present, surgery is not usually necessary for this condition.

PROGNOSIS

The prognosis for MCPS is good. Thankfully, corneal involvement is rare and with careful flushing and general ocular hygiene the condition is readily managed. Occasional bacterial infections can occur but these normally respond well to a few days of topical antibiotic drops. Thus, in most cases, it is just a question of owner education and reassurance that is required.

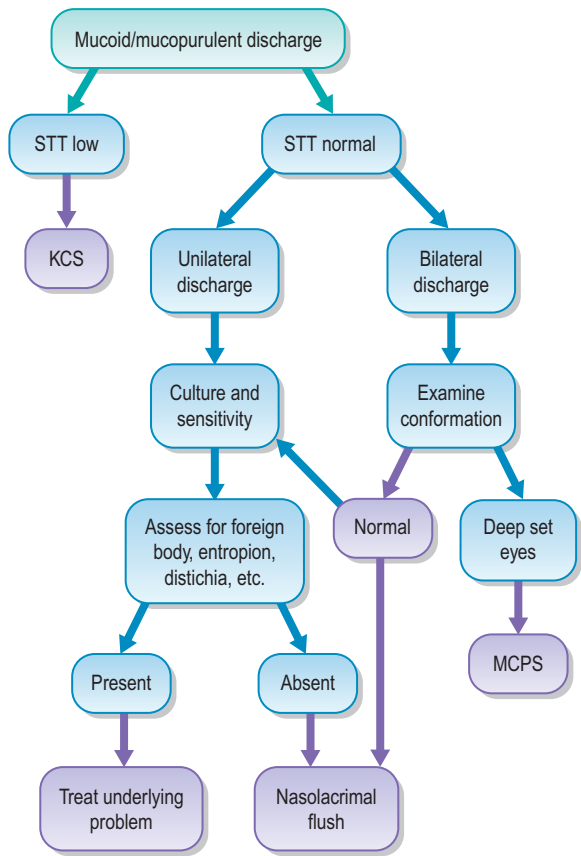


Figure 12.2 Decision-making tree – medial canthal pocket syndrome.

CASE EXAMPLE 12.1

Patient details: Chocolate Labrador retriever, 10 months old, male (neutered)

PRESENTATION AND HISTORY

The owner was very frustrated and presented the dog with a history of recurrent conjunctivitis. It was always bilateral, but sometimes one eye was worse than the other. She had treated the dog with a variety of topical antibiotics, both drops and ointments, on numerous occasions, and whilst the dog improved while on treatment, the problem recurred within a few days of cessation. Swabs had been taken for bacterial culture and sensitivity testing and a mixed population of normal commensals, sensitive to all antibiotics tested, had been isolated. The owner was most concerned about the stringy discharge followed by the redness of the eyes. The discharge was always worse in the morning, and sometimes accumulated during the day, especially after he had been asleep. The dog was not at all bothered by his eyes.



Figure 12.3 Mild mucoid discharge and conjunctival hyperaemia of the ventral fornix in a young Labrador retriever with non-pigmented nictitating membrane.

CLINICAL SIGNS

General examination was normal. On ophthalmic examination a mild mucoid ocular discharge was present bilaterally. This was centred at the medial canthus. The leading edges of both third eyelids were non-pigmented. Mild conjunctival hyperaemia was present affecting the ventral palpebral conjunctiva and that over the palpebral surface of the nictitans membrane only (Figure 12.3). Eyelid position was normal. No corneal ulceration was noted and intraocular contents were unremarkable.

WORK-UP

The history and clinical examination were typical for MCPS and so no work-up was undertaken.

TREATMENT

It was explained to the owner that the recurrent conjunctivitis present in her dog was very mild and a result of his conformation. She was reassured that the eyes were not particularly red – the non-pigmented third eyelid is a normal variation and she was shown that the colour was the same as the dog's gums – bright pink but not really inflamed. Once she understood that the discharge was purely a build up of what is normally produced, but that it was not draining normally because of the presence of the medial canthal pocket, she was happy to clean and bathe this twice daily and not to use any further topical medications.

OUTCOME

The outcome was good – only one episode of true bacterial conjunctivitis occurred 3 months later when the dog had been staying with friends who had another dog for a few days, and they did not clean the eyes at all. It was likely that the build up of mucus and change in environment allowed bacterial overgrowth which responded very quickly to chloramphenicol drops. As the dog matured, the amount of discharge present in the morning reduced – presumably as a result of a change in conformation affecting tear dynamics – and the owner only needed to clean the eyes every couple of days.

13 Rabbit conjunctivitis

INITIAL PRESENTATION

Colour change – red; discharge

PRESENTING SIGNS

The owner's usual complaint is of a red eye, but frequently it is also sore, and often quite significant discharge is present. Rabbits of any age or breed can be affected and one or both eyes can be involved. If the rabbit is housed with others these too could be affected. The rabbit is usually clinically well, although some might be eating less, and some will have a nasal discharge and the owner might report noisy breathing.

CASE HISTORY

The condition is usually sudden in onset, and often one eye is affected initially, closely followed by the other. There is frequently no relevant previous history, although sometimes the condition will coincide with a change in management – different bedding, feeding from different containers, a change in placement of the outdoor run, or the introduction of a new rabbit to the household. Some rabbits will have a history of previous ocular or respiratory infections.

CLINICAL EXAMINATION

General clinical examination can be variable. In many animals it will be normal but a significant number will have evidence of respiratory disease – nasal discharge, increased respiratory rate and effort, mild pyrexia and so on. Sometimes the rabbit will be thin. As with all rabbits which are presented to the surgery their teeth should be checked!

On ophthalmic examination the affected eye(s) will be obviously red and inflamed. Thus marked conjunctival hyperaemia will be present, usually affecting both bulbar and palpebral conjunctiva, and moderate chemosis is frequently also present. Sometimes the conjunctiva is so swollen that visualization of the eye itself is difficult. Moderate blepharospasm is present, frequently with significant lid swelling as well as hyperaemia. The lids should be checked for signs of entropion which can occur in young rabbits and will cause secondary conjunctival hyperaemia. An ocular discharge is almost always present but can be very variable in nature. Initially it is usually serous but rapidly becomes mucopurulent and can be very thick and sticky – adhering to the eyelids and medial canthal skin and sometimes causing local skin excoriation.

The cornea should always be stained with fluorescein to check for evidence of corneal ulceration but this can be difficult in very painful eyes with excessive chemosis. A slight miosis can be present but in general intraocular examination is unremarkable.

DIFFERENTIAL DIAGNOSES

- Conjunctivitis:
  - Traumatic
  - Infectious
- Dacryocystitis
- Corneal ulceration
- Entropion
- Aberrant conjunctival overgrowth
- Orbital fat prolapse
- Intraocular disease (e.g. uveitis, glaucoma)
- Retrobulbar disease

CASE WORK-UP

Although swabs for bacterial culture and sensitivity are not usually taken from rabbits on initial presentation – empirical treatment is usually tried first – it is certainly something to consider: they can always be discarded if necessary but cannot be retaken after the eye has been cleaned up! Cytology can also be considered.

It is important to check for any foreign body – fragments of hay, straw and sawdust frequently become trapped in the conjunctival fornix or behind the third eyelid. Topical anaesthetic drops are applied to the eye and to a cotton bud. The latter can be gently rolled in the lower and upper fornix and will attract any debris. Flush copiously with sterile saline – a 5 ml syringe with a cut-off nasolacrimal cannula on the end is ideal for flushing the fornix. The third eyelid can be grasped with atraumatic forceps – von Graefe forceps (Figure 13.1) are ideal since they grip well without damaging the conjunctiva – and it is gently elevated to check behind for any foreign matter.

Since the main differential for conjunctivitis in rabbits is dacryocystitis, it is sensible to flush the nasolacrimal duct on initial presentation. The procedure for doing this is described in the case on rabbit dacryocystitis (Chapter 19, p. 115). Flushing at initial presentation will not only differentiate conjunctivitis from the more serious nasolacrimal duct involvement but will also immediately establish drainage such that any build up of discharge is less likely to block the duct and actually cause a secondary dacryocystitis should the rabbit not respond to its initial treatment.



Figure 13.1 Von Graefe forceps.

NURSING ASPECTS

It is important to keep the rabbit comfortable and regular removal of any ocular discharge will be beneficial – especially if this is sticky and building up such that the rabbit cannot open its eye. Soft, gauze-free swabs soaked with sterile saline are ideal. The rabbit will be treated with either drops or ointment. If the latter, it is sensible to change the bedding to shredded paper rather than straw or sawdust to reduce the risk of further damage as debris adheres to the ointment as well as the ocular discharge!

EPIDEMIOLOGY

Management errors and bacterial infections are the most common causes for conjunctivitis in rabbits. Overhead hay nets should be avoided as debris can fall into the eyes, causing conjunctivitis. Poor quality straw bedding can have the same effect. If the rabbit's hutch is not cleaned frequently a build up of ammonia and so on can decrease air quality and trigger irritation and then conjunctivitis. Overcrowding can also be an issue in some cases.

Pasteurella spp. are considered the most frequent primary bacterial infection in rabbits, causing conjunctivitis, dacryocystitis, abscesses and respiratory infections. Staphylococcal spp. are also frequently isolated with occasional reports of *Haemophilus* spp. The underlying bacterial type cannot be distinguished clinically – hence the need for culture and sensitivity testing. Viral causes are also encountered – the myxoma virus has various strains and the actual presentation will be different according to the strain involved. Typically lid swelling (blepharoedema) is dramatic and considered pathognomonic for myxomatosis, but early symptoms might be just a conjunctivitis (unfortunately followed by a swift death in many animals).

CLINICAL TIPS

- Always test with fluorescein
- Always check for foreign body
- Ask about any in-contact rabbits – are they also affected?
- Flush the nasolacrimal duct on initial presentation – it is very easy to do in normal rabbits

- Although keratoconjunctivitis sicca is rare in rabbits, it can occur, so consider performing Schirmer tear tests – especially if the cornea looks lacklustre or the rabbit fails to respond to initial treatment

TREATMENT OPTIONS – MEDICAL

Most initial medication will consist of broad spectrum antibiotic drops or ointment. Gentamicin and fusidic acid are both licensed in rabbits. Drops have the advantage of not clogging up the lids and encouraging debris to stick, but have the disadvantage of requiring more frequent application. Even if the conjunctiva seems very inflamed, steroid-containing topical medication is not required as a primary medication. Systemic administration of enrofloxacin is sometimes also advocated. Most rabbits will respond within a few days – if not, then further thorough examination is required with sampling if necessary. Affected rabbits should always be isolated and kept in clean, warm, dry, dust-free surroundings until fully recovered.

PROGNOSIS

The prognosis for rabbits with conjunctivitis only is generally good, providing the underlying cause is identified and corrected quickly. However, if the condition is neglected, then a secondary dacryocystitis will rapidly develop and the long-term management of this can be challenging and frustrating. Similarly, if a foreign body is present but undetected, it can easily cause corneal ulceration and, if not removed, can even result in corneal perforation. Many rabbit owners are aware of these risks and so will present their pets timely but unfortunately some are ignorant of the potential seriousness of an untreated conjunctivitis.

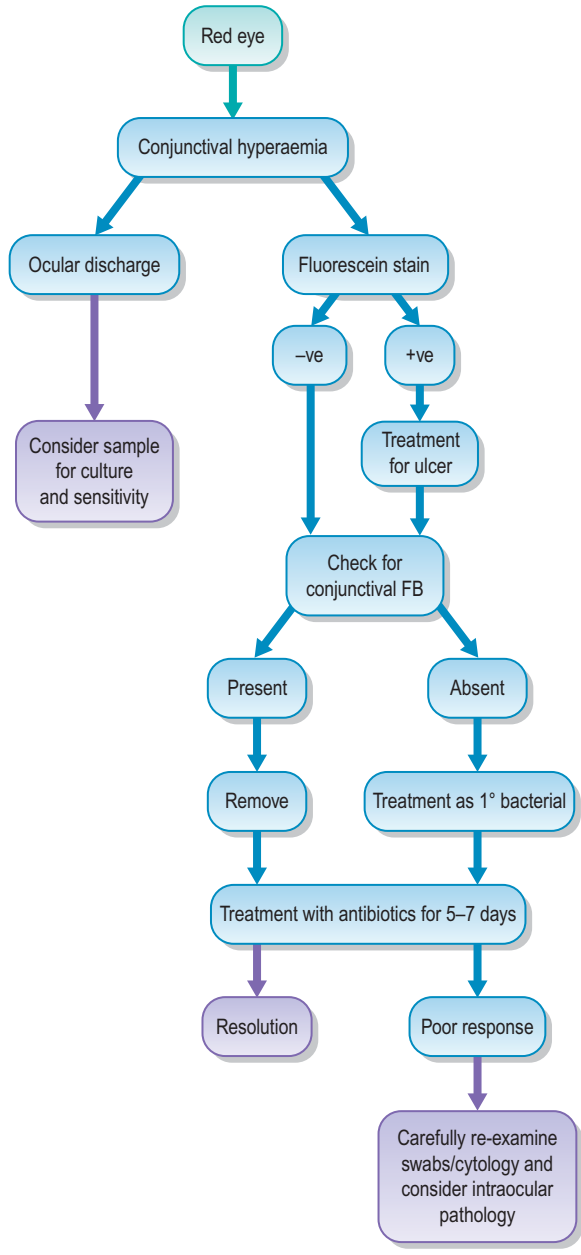


Figure 13.2 Decision-making tree – rabbit conjunctivitis.

CASE EXAMPLE 13.1

Patient details: Three-year-old entire female rabbit, dwarf cross

PRESENTATION AND HISTORY

The rabbit lived with a neutered male rabbit and a guinea-pig in a large outside hutch with access to grass. It was reportedly fit and well and had never suffered from ocular problems before. The owners noticed that the left eye was red and a watery discharge was present. They bathed it for 48 hours but there was no improvement and the right eye then started to look a bit red. Hence they sought veterinary attention. The in-contact animals were unaffected. There had been no change in the management of the animals.



Figure 13.3 Acute conjunctivitis in a dwarf rabbit – the palpebral conjunctiva is particularly inflamed. Photo courtesy of Alonso Dupuy Mateos.

CLINICAL SIGNS

General clinical examination was unremarkable – the rabbit was of a suitable weight and no noticeable dental disease was observed on a brief oral examination. The left eye was uncomfortable and held half closed. Marked tear overflow was present with some nasofacial dermatitis. The discharge was mainly serous but some mucopurulent matter was present in the conjunctival fornix ventromedially. The conjunctiva was extremely hyperaemic – particularly the palpebral rather than bulbar regions (Figure 13.3). The eyelid margins were thickened. No corneal ulceration was present and intraocular examination appeared normal. Mild conjunctival hyperaemia was present in the right eye but no significant discharge was noted.

WORK-UP

The left eye was gently bathed with sterile saline. A drop of topical anaesthesia (proxymetacaine) was applied to the eye and the fornix was carefully flushed again to ensure that no pieces of plant matter or similar foreign body were present – none were found. The bulbar surface of the nictitans membrane was also similarly checked with no foreign material identified. Since the rabbit was very good to handle, it was decided to flush the nasolacrimal duct. No obstruction was present and clear saline was obtained from the nose, ruling out dacryocystitis as a possible diagnosis. No work-up was undertaken for the right eye.

TREATMENT

The rabbit was treated with topical gentamicin drops three times daily bilaterally, and the owner instructed to gently bathe any discharge prior to applying the medication. Soft white paraffin (Vaseline) was advised to be applied once daily to the sore patch of skin ventromedial to the eye but no topical or systemic antibiosis was deemed necessary to treat the dermatitis. The rabbit was kept separate from its companions.

Five days later there had been no improvement in the left eye – still very inflamed with the mucopurulent discharge – while the right eye had deteriorated with more conjunctival hyperaemia and now a serous discharge from this eye. It was suggested that swabs were taken for culture and sensitivity, and the nasolacrimal duct flushing repeated bilaterally. However, the owners did not want any additional expense and so instead treatment was changed to fusidic acid twice daily bilaterally. Within 48 hours both eyes were much improved – minimal conjunctival hyperaemia, resolution of the lid swelling in the left eye and a significant reduction in the ocular discharge.

OUTCOME

The fusidic acid was continued for another 5 days (a full week in total), by which time both eyes appeared normal. It is assumed that a bacterial infection was present, with resistance to gentamicin. This could suggest a Gram-positive organism such as *Staphylococcus* spp. which have commonly been isolated from rabbits with conjunctivitis. However, without bacterial culture and sensitivity testing this is speculative. Thankfully the condition responded well and the rabbit did not develop dacryocystitis (the owners had been warned of this possibility right from the outset).

14 Aberrant conjunctival overgrowth in rabbits

INITIAL PRESENTATION

Colour change – red

PRESENTING SIGNS

Owners present the rabbit due to change in appearance of the affected eye(s) – it appears red. Normally they have noticed this for a few weeks and it is worsening. The eye is open and comfortable with no significant ocular discharge. Adult rabbits are presented and normally one, but occasionally both, eyes are affected. Dwarf rabbits and their crossbreds are possibly more commonly affected. No visual problems are reported and the rabbit is eating normally such that the owner has no other concerns.

CASE HISTORY

The history is minimal. Usually the rabbit has had no previous ocular problems and is in good general health. Occasionally the owner will have thought that the current ocular problem is conjunctivitis and tried treating with antibiotic drops with no improvement – this is particularly the case in multi-rabbit households where the owner has some medication for another pet.

CLINICAL EXAMINATION

General clinical examination is unremarkable – the rabbit is normally in good body condition with no dental problems. On ophthalmic examination the rabbit is normally visual (although menace responses are not always present in rabbits). No ocular discharge is present and the eye is open and comfortable. A pink, fleshy, vascular membrane is present over the cornea through a full 360 degrees extending centrally. It can extend only a couple of millimetres, but might cover virtually the whole of the cornea such that only a small central hole remains (see Figure 14.2). Intraocular examination, if possible, is normal. Rabbits have a merangiotic retina – a horizontally oval optic disc with vessels radiating from either end (laterally and medially) – and this is most easily examined by looking up at the rabbit from below, while its head is held over the end of the consulting table. No corneal ulceration is present. If a drop of topical anaesthetic is placed in the affected eye, the pink membrane can be lightly touched with a cotton bud and can be gently moved over the corneal surface – it is not attached centrally.

DIFFERENTIAL DIAGNOSES

- Aberrant conjunctival overgrowth
- Conjunctivitis
- Keratitis
- Vascularizing corneal ulcer
- Intraocular haemorrhage
- Prolapsed nictitans membrane

CASE WORK-UP

The clinical appearance is pathognomonic – once seen this condition is not forgotten and, as such, no further work-up is normally necessary.

NURSING ASPECTS

Apart from the postoperative application of topical medication, and the usual precautions necessary for hospitalization and anaesthesia of rabbits, there are no specific nursing issues.

EPIDEMIOLOGY

Aberrant conjunctival overgrowth, circumferential conjunctival hyperplasia and placation, epicorneal conjunctival membranes and pseudopterygium are all terms used to describe this abnormality. The condition is poorly understood and appears to be unique to the rabbit. The pink tissue is a fold of conjunctiva which grows centripetally from the bulbar conjunctiva at the limbus to obscure the cornea. It remains attached at the limbus but the central fold of tissue is freely mobile over the corneal surface. Vision is maintained until the central circular 'hole' is occluded by the progression of the membrane. Histologically the lesion consists of normal conjunctiva lining both the inner and outer surfaces of the membrane and is thought to result from a focal excess of conjunctival collagen. Any breed of rabbit can be affected, but dwarf and dwarf-crosses might be predisposed.

A similar condition is seen in humans – pterygium – where a fold of conjunctiva grows across the cornea from the nasal limbus. It does become attached to the cornea, unlike in rabbits, and is more common in warm, dry, sunny climates and in certain ethnic groups, especially black people. Exposure to UV light, wind, dust and trauma predispose people to developing pterygium but none of these factors appears applicable to rabbits. It is postulated that local immunity factors are involved but no research has been undertaken to date to prove this.

CLINICAL TIPS

- Once seen never forgotten (see Figure 14.2)
- The conjunctival membrane is not attached to the cornea, unlike true corneal vascularization or granulation tissue

TREATMENT OPTIONS – MEDICAL

Topical medication is neither effective at preventing further growth of the membrane nor at causing its regression. Both topical steroids and ciclosporin have been used unsuccessfully, although the latter can be of benefit post-surgery (see below). If the condition is unilateral and the reduced vision in the affected eye causes no behavioural changes in the rabbit, then it can be left untreated. The eye should be checked and bathed regularly since accumulations of mucus and debris could theoretically build up and predispose to infections, although clinically this does not appear to be a significant problem.

TREATMENT OPTIONS – SURGICAL

In severely affected animals, where they are having visual problems or not behaving normally with fellow rabbits, and certainly in bilateral cases, surgery is advised. This should be performed under general anaesthesia. Simply resecting the membrane back to the limbus is not sufficient – although it is the easiest approach! Unfortunately, the membrane simply regrows and the eye is back to square one within a matter of weeks. Instead, it is necessary to remove the membrane and then suture the cut edge to the bulbar conjunctiva and sclera just behind the limbus. Folding the cut surface back under the conjunctiva, away from the limbus, has been advocated to further reduce the risk of recurrence. The membrane is initially lifted up centrally and a radial cut made using small sharp scissors back to the limbus. This is then extended around the circumference of the globe just posterior to the limbus and the membrane can be excised in one piece (Figure 14.1a). Significant haemorrhage will occur but this can be controlled with gentle pressure, and will reduce further once the conjunctiva is sutured closed. Ideal suture material is 6/0 polyglactin 910 (Vicryl, Ethicon). Four simple interrupted cardinal sutures are placed at 12, 3, 6 and 9 o'clock and then a continuous suture can be placed around the full circumference (Figure 14.1b,c).

Postoperatively, topical ciclosporin ointment has been advised (twice daily for several weeks) to reduce further the risk of regrowth, although no trials comparing success rates with its use compared to no treatment have been undertaken following suturing the remaining flap of conjunctiva to the globe. Furthermore, there is no specific advice available regarding how long to continue treatment with ciclosporin: some recommend a few weeks while others suggest it should be continued indefinitely.

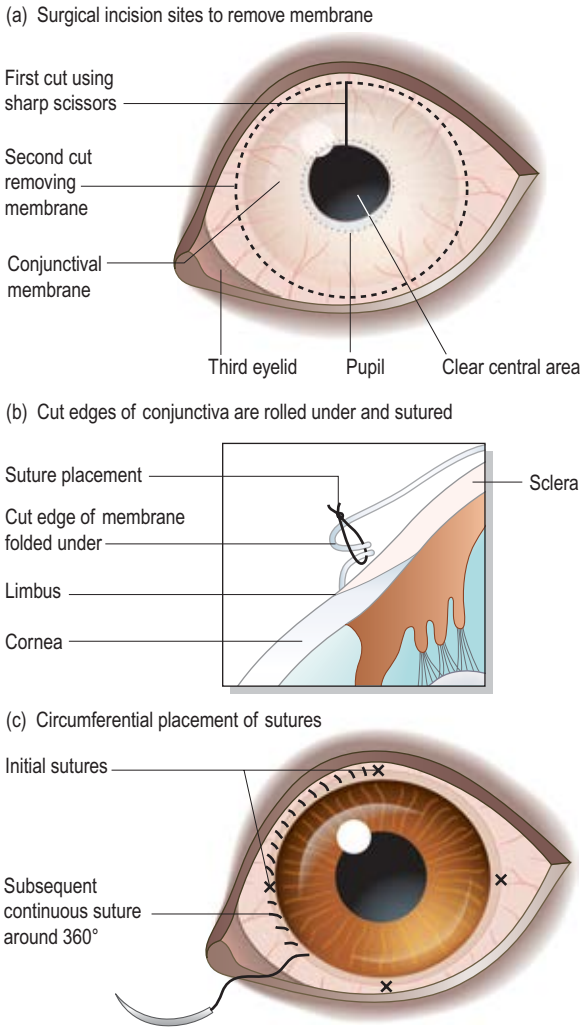


Figure 14.1 Approach to surgical removal:
(a) surgical incision sites to remove membrane
(b) cut edges of conjunctiva are rolled under and sutured
(c) circumferential placement of sutures.

PROGNOSIS

The prognosis for aberrant conjunctival overgrowth is reasonable. Some unilateral cases can be left untreated since they are non-painful and the visual impairment is frequently not debilitating. Simple surgical excision will result in regrowth within a few weeks, but this seems to be prevented by suturing the remaining conjunctiva to the episcleral tissue and using topical ciclosporin postoperatively.

CASE EXAMPLE 14.1

Patient details: Dwarf-cross rabbit, 3 years old, female

PRESENTATION AND HISTORY

The rabbit was presented because the owner had noticed that both eyes were gradually becoming red. There was a slight discharge reported but this was not the owner's main concern. The rabbit was comfortable with the eyes and seemed to see normally. No previous ocular problems had occurred and the rabbit was in good general health. She was housed with a guinea-pig, which was fine.

CLINICAL SIGNS

General clinical examination was unremarkable. Both eyes were open and comfortable. A scant mucoid discharge was noted. A thin, pink sheet of tissue extended across almost the entire cornea in both eyes (Figure 14.2). The left eye was slightly worse, with only a tiny central hole of clear cornea. Menace responses were difficult to assess but the rabbit hopped about the consulting room floor happily and did not bump into obstacles. Pupillary light reflexes could be determined through the thin corneal opacity and were normal; dazzle reflexes were brisk. Further intraocular examination was not possible.



Figure 14.2 Right eye of dwarf-cross rabbit showing conjunctival membrane across cornea.

WORK-UP

This was minimal – a drop of local anaesthetic was placed in each eye and the pink membrane was lightly touched with a cotton bud. It was moveable over the cornea and only appeared attached at the limbus. This, together with the classic clinical appearance, confirmed the diagnosis of aberrant conjunctival overgrowth.

TREATMENT

The options of surgery or doing nothing were discussed with the owner. The details of the surgery required (see above) were described, together with the fact that in some cases the membrane can regrow. The owner was not keen to subject the rabbit to general anaesthesia and since it did not appear to have any behavioural signs to suggest poor vision she decided not to treat it at all.

OUTCOME

The membrane in the right eye continued to grow such that both eyes looked like the left eye. No conjunctival hyperaemia or infection developed and the rabbit continued to eat well and appeared totally unconcerned by the strange ocular membranes!

SECTION 3

NASOLACRIMAL SYSTEM

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15 Nasolacrimal system – introduction

The nasolacrimal system is made up of a secretory component (the pre-ocular tear film) and an excretory component (nasolacrimal drainage system). Secretion of the tear film is from various glands – the lacrimal and third eyelid glands, meibomian glands and goblet cells – each contributing a different constituent as detailed below. The drainage system consists of the nasolacrimal punctae, canaliculi and duct.

THE PRE-OCULAR TEAR FILM

An adequate supply of tears is necessary to maintain ocular integrity and normal function. The pre-ocular tear film covers the corneal and conjunctival surfaces and its close proximity to these underlying structures means that it is important in both health and disease. The pre-ocular tear film has a pH of 6.8–8.0 (mean 7.5). This is important when considering topical medication – drops with a different pH are more likely to sting on application for example. Functions of the pre-ocular tear film include:

- maintaining an optically uniform corneal surface
- removing foreign material and debris from the conjunctiva and cornea
- permitting the passage of oxygen and nutrients
- possessing antibacterial properties.

Traditionally the pre-ocular tear film is considered to be made up of three layers, although the structure and interactions of these layers are probably much more complicated than this! However, for simplification, we will stay with this classification, which divides the tear film into outer, middle and inner layers as follows:

Outer oily layer

This thin layer is produced by the meibomian glands. The glands are present in the palpebral conjunctiva with their openings visible as a line of grey dots on the eyelid margin. This layer helps to decrease evaporation, improves the stability of the whole of the tear film and provides a protective oily barrier at the lid margins.

Middle aqueous layer

This is produced from lacrimal and nictitans glands and constitutes the bulk of the tear film. The lacrimal gland, located dorsolateral to the globe within the periorbita of the lacrimal bone, provides the majority of the aqueous tear production, with the nictitans gland producing approximately a quarter to one-third of the total amount. This aqueous layer allows oxygen and nutrient diffusion, facilitates smooth movement of the eyeball and contributes to corneal protection.

Inner mucin layer

This thin layer is produced by conjunctival goblet cells. It adheres to the cornea, allowing the aqueous layer to spread easily and evenly over the surface.

Blinking spreads the tear film and the blink rate is increased with pain or ocular discomfort (blepharospasm). An increased blink rate is also seen in restrained animals which is one reason why it is important to watch the animal on the floor/cat in the basket prior to handling in order to accurately assess their blinking.

NASOLACRIMAL DRAINAGE

In dogs and cats there is an upper and lower nasolacrimal punctum located on the palpebral conjunctiva a few millimetres from the medial canthus. Normally the openings are oval in shape. The punctae lead into the upper and lower canaliculi which join at the lacrimal sac. This structure is poorly developed in domestic species and is really just a dilation at the top of the nasolacrimal duct sitting in a depression in the lacrimal bone. The duct itself is narrow as it crosses the lacrimal bone and enters the maxillary bone (a reason for the frequent retention of foreign bodies in this area – and also why it is sometimes possible to flush them from one punctum to the

other). The duct ends in the nasal ostium close to the external nares, although a second opening into the hard palate at the level with the canine teeth can occur. In dolichocephalic and mesaticephalic breeds the duct is long and straight, while in brachycephalic dogs and cats the duct is short and sometimes tortuous. In rabbits there is a single, large nasolacrimal punctum only which is situated slightly more ventral to the lid margin in the lower palpebral conjunctiva than in dogs and cats. In rabbits the nasolacrimal duct is very tortuous and its diameter varies considerably during its passage between the eye and nose. This fact contributes to the frequency of drainage problems and dacryocystitis encountered in rabbits.

The nasolacrimal system is lined by pseudostratified columnar epithelium. Its sole purpose is to drain tears from the eye to the nasal passages. Some evaporation occurs before the tears reach the punctae, but about 60% are drained via the ventral punctum. A combination of gravity, local pressure changes on blinking causing a vacuum in the canaliculus drawing tears into it, together with capillary action, all contribute to tear flow down the ducts.

Abnormalities affecting the nasolacrimal system

Ophthalmic problems encountered in the nasolacrimal system can be broadly divided into abnormalities of production and of drainage. Thus, increased tear production (lacrimation) or decreased production (keratoconjunctivitis sicca) are both common in domestic species and Schirmer tear test readings should always be undertaken in any patient presenting with surface disease (see Table 1.2).

Increased tear production is normally associated with ocular pain, and careful ophthalmic examination should reveal the reason for this – entropion, a corneal ulcer or ectopic cilium for example. Correcting the underlying problem will resolve the increased lacrimation. Decreases in tear production can be qualitative or quantitative, affecting any part of the trilaminar tear film. The most frequently diagnosed condition is of a reduction in aqueous tear production which is termed keratoconjunctivitis sicca (KCS). This is easily diagnosed by performing Schirmer tear tests. Low aqueous production leads to hypertonicity and dehydration of the conjunctiva and cornea. Both epithelial and anterior stromal hypoxia occur, resulting in cellular compromise and corneal ulceration. Increased friction occurs during blinking, further compromising corneal health. The conjunctiva becomes hyperaemic and the surface epithelium of both conjunctiva and cornea undergoes squamous metaplasia. This results in a reduction in mucin adherence and further tear film destabilization.

Although quantitative tear film deficiencies are much more commonly encountered, qualitative problems should not be overlooked. Lipid abnormalities result from disease of the meibomian glands – for example, with severe blepharitis – and lead to early evaporation of the aqueous tears due to a lack of the oily outer layer. Generalized seborrhoea can also lead to abnormal lipid production from the meibomian glands. Problems with mucin production from the goblet cells also lead to tear film instability and chronic keratitis. Diagnosis of these types of tear production abnormality is more difficult since Schirmer tear test readings are usually normal if aqueous production is unaffected. Chronic conjunctivitis and keratitis are present but appear non-specific unless detailed evaluation is undertaken. Tear break-up times and careful examination of the meibomian glands and conjunctiva in the ventral fornix, which contains many goblet cells, can help reach a diagnosis. Histological examination of conjunctival goblet cells can be required and referral should be considered if a qualitative tear film abnormality is suspected.

Drainage problems normally present as epiphora, simple tear overflow, but the discharge can become infected and purulent in nature depending on the underlying aetiology. Various factors, such as imperforate or misplaced punctae, caruncular trichiasis and medial entropion, affect tear drainage dynamics, leading to epiphora. Some common examples of nasolacrimal problems are discussed in the following case selections.

16 Complicated keratoconjunctivitis sicca

INITIAL PRESENTATION

Discharge, abnormal appearance – red

PRESENTING SIGNS

The typical presenting signs of keratoconjunctivitis sicca (KCS) are well reported and generally the initial diagnosis is straightforward. Thus the dog presented with the slightly sticky mucoid or mucopurulent discharge and conjunctival hyperaemia should always have a Schirmer tear test (STT) performed. Readings of less than 10 mm are diagnostic for KCS but readings between 10 and 15 mm should be considered suspicious, particularly in large breed dogs (an association between higher STT readings in dogs of higher body weight has been demonstrated). Here we will discuss two complications of KCS: acute corneal ulceration and the poorly responsive case. The former will present with a suddenly painful eye – there will not be the increased lacrimation and 'wet eye' normally associated with an ulcer since the patient is unable to produce aqueous tears! Instead there will be a copious mucopurulent discharge, sometimes adhered to the cornea such that the patient cannot open its eye. Discomfort will be present and attempts at rubbing the eye are likely.

The presentation of the poorly responsive case of KCS is less dramatic. The dog will probably have been receiving treatment, but the owner might have discontinued this totally or become less meticulous with both medicating and cleaning the eye. Thus the tenacious ocular discharge will have gradually increased, some periorbital disease might have developed or the owner might present the dog again because they have become concerned that its vision has deteriorated.

CASE HISTORY

As outlined above there is typically a previous ocular history. Keratoconjunctivitis sicca has probably already been diagnosed and treatment instigated. However, the condition has not responded as anticipated, or did initially respond such that the owner felt that the dog was better and stopped treatment, and a serious relapse has occurred. The condition is normally bilateral but one eye is often affected more severely and this might be the reason that the owner has returned for a follow-up consultation.

CLINICAL EXAMINATION

Unless the dog has a systemic disease which can exacerbate KCS, such as diabetes mellitus, hypothyroidism or hyperadrenocorticism, general clinical examination is usually unremarkable. On initial ophthalmic assessment the most striking problem is the copious mucopurulent discharge. A sample should be taken for culture and sensitivity before gently wiping this away. Conjunctival hyperaemia and hyperplasia will be apparent. Schirmer tear test readings are expected to be bilaterally low – probably less than 2 mm of wetting in a minute. The periorbital skin might be inflamed and thickened, or even slightly ulcerated if a chronic blepharitis has been present or the patient has self-traumatized (Figure 16.1). The cornea is likely to be pigmented either across the dorsal half or sometimes covering most of its surface (Figure 16.2). Many dogs can still see and retain a

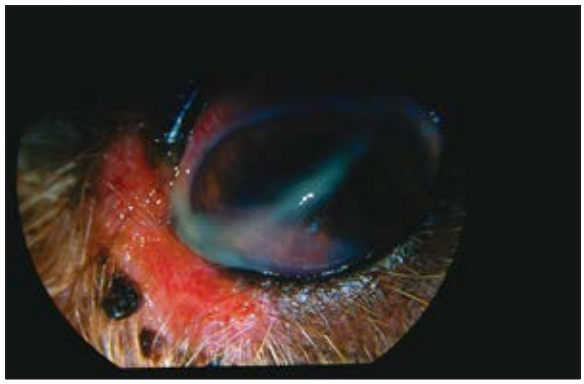


Figure 16.1 Chronic keratoconjunctivitis sicca in a cocker spaniel with copious mucopurulent discharge and medial canthal blepharitis.

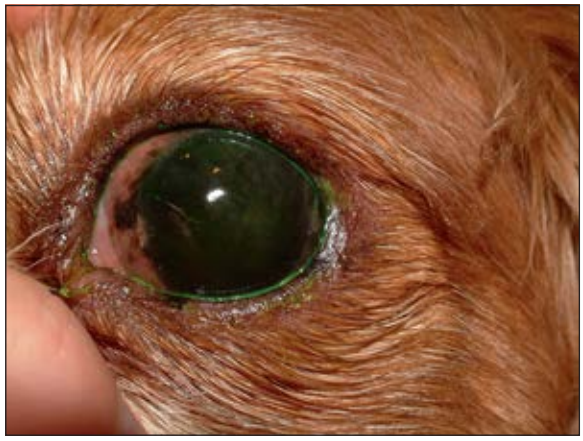


Figure 16.2 Chronic keratoconjunctivitis sicca in 6-year-old Cavalier King Charles spaniel (CKCS). Note the marked corneal pigmentation, vascularization and lacklustre corneal reflection.

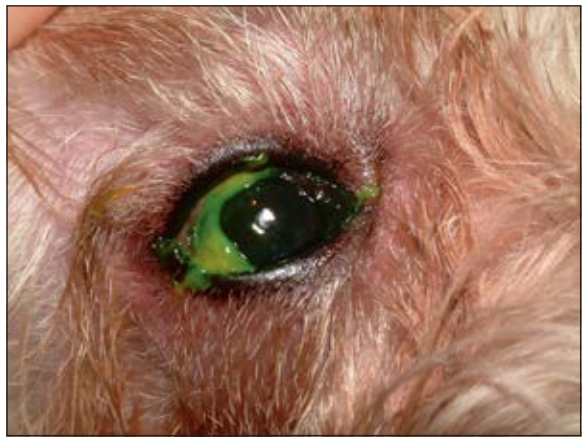


Figure 16.3 False-positive fluorescein test due to adherence of the dye to the copious mucoid discharge. This should be cleaned away before checking for ulceration.



Figure 16.4 Acute severe corneal ulceration in a CKCS. Schirmer tear test readings were 3 mm in this eye and 1 mm in the fellow, non-ulcerated, eye.

menace response despite significant pigmentary keratitis. Branching superficial blood vessels will also be present, and sometimes a slight granulation tissue reaction is also noted, particularly if previous ulceration has occurred. In poorly responsive or neglected cases of chronic KCS ulceration is not always a feature. Once the discharge is cleared away corneal examination will show a dull, lacklustre surface with some epithelial irregularity but not true ulceration. Careful irrigation of excess fluorescein dye is required to prevent false positives as the dye pools in the irregular surface or sticks to the mucus (Figure 16.3).

Acute ulceration associated with KCS is typically deep and central (Figure 16.4). Thus a facet is clearly visible in the central or subcentral cornea and surrounding corneal oedema can be present. If the secondary bacterial infection is severe then keratomalacia might be present such that the 'melting' cornea around the ulcer has a gelatinous appearance. Fluorescein staining will be positive, but it is not uncommon for a descemetocoele to be present such that only the sides of the ulcer retain dye with the central area remaining dark, representing Des-

cemet's membrane. Some peripheral corneal pigmentation and superficial vascularization might also be noted, reflecting the chronicity of the underlying KCS. A reflex uveitis and miotic pupil might also be noted.

DIFFERENTIAL DIAGNOSES

The definitive diagnosis of keratoconjunctivitis sicca has already been made. However, other exacerbating factors such as entropion, lagophthalmos, blepharitis and systemic disease might be present and should be considered. A list of the possible causes of KCS is provided in Table 16.1.

CASE WORK-UP

General clinical examination is important, and should any abnormalities be detected on this, further laboratory tests looking for diabetes mellitus, hypothyroidism and hyperadrenocorticism are indicated. All these conditions can exacerbate an underlying KCS and should be considered if the condition had previously been stable on medication but recently deteriorated.

Samples of the discharge should be taken for bacterial culture and sensitivity testing. In addition, conjunctival cytology or even eyelid biopsy in the case of suspected blepharitis might be warranted – particularly if the response to previous medical treatment has been poor.

NURSING ASPECTS

Specific nursing of complicated cases of keratoconjunctivitis sicca will vary depending on whether ulceration is present or not and whether surgery is indicated. However, careful medical attention is required regardless. Frequent and thorough cleansing of the discharge is required – both to promote patient comfort and to reduce the risk of further secondary bacterial contamination. The eyes should be gently bathed with soft gauze swabs or cotton wool pads soaked in sterile saline before the application of any medication. Since frequent medication is necessary, nurses should ensure that owners are properly able to carry out the treatment at the desired intervals. Patients should be prevented from self-traumatizing – this is most common immediately after the application of topical medication and, as such, the patient should be distracted for a few minutes afterwards.

EPIDEMIOLOGY

There are several causes for KCS in dogs (see Table 16.1) and its incidence is reported at about 1% of the population of dogs presented to North American Veterinary Colleges. Certainly a breed predisposition is noted with immune-mediated KCS, so a presumed inherited component is present, although details of this have not been elucidated, and many other factors are clearly involved.

Table 16.1 Possible causes for keratoconjunctivitis sicca (KCS)

Possible causes for KCS	Comment
Immune-mediated lacrimal gland disease (most common)	Most common
Congenital lacrimal gland aplasia	E.g. Cavalier King Charles spaniels, Jack Russell terriers
Drug suppression of lacrimal gland	E.g. atropine drops
Drug toxicity and damage to lacrimal gland	E.g. sulfasalazine and sulphonamides
Lacrimal gland adenitis	Distemper virus and some cases of conjunctivitis and blepharitis will have lower Schirmer tear test readings
Traumatic injury to lacrimal gland	Road traffic accident with head trauma
Neurogenic	Lack of parasympathetic supply to lacrimal glands Other neurological signs sometimes present
Prior removal of nictitans gland	Prolapsed nictitans glands are more common in the same breeds that are predisposed to KCS and gland removal is not recommended
Systemic disease	Hypothyroidism, hyperadrenocorticism, diabetes mellitus

English bulldogs, West Highland white terriers, Cavalier King Charles spaniels, American and English cocker spaniels and pugs are among the many breeds commonly seen with the condition. A sex predisposition to females has been reported in West Highland white terriers.

Although these chronic, insidious, immune-mediated causes are the most common form of KCS seen in practice, various other aetiologies exist. Viral lacrimal gland adenitis occurs with canine distemper infection leading to KCS, which clearly can occur as outbreaks in unvaccinated dogs; congenital hypoplasia of the lacrimal tissue is recognized, sometimes as a unilateral problem in the Cavalier King Charles spaniel and some miniature breeds; drug-induced KCS following systemic sulphonamides, sulfasalazine and topical atropine occurs, and neurogenic KCS due to loss of the parasympathetic innervation to the salivary glands occurs occasionally. Some systemic diseases can also lead to reduced tear production and so patients with diabetes mellitus, hypothyroidism and hyperadrenocorticism in particular should have Schirmer tear tests performed regularly.

CLINICAL TIPS

- Client education and cooperation right from the start when KCS is first diagnosed will reduce the risk of complications later on due to poor owner compliance
- Provide literature to the clients and ensure that they are aware that life-long treatment will be required – control not cure!
- Repeat Schirmer tear test readings regularly – every 3 months is advised for all KCS patients
- Ensure fluorescein dye is thoroughly flushed from the eye to prevent false positives where it adheres to mucus or pools in irregular patches on the corneal surface
- If significant corneal pigment is present, the use of indirect rather than direct ophthalmoscopy will enable more detailed intraocular examination
- Dogs with acute ulceration will not have increased lacrimation as a clinical sign, unlike dogs with normal tear production

TREATMENT OPTIONS – MEDICAL

If deep ulceration has occurred, as one of our possible complications to poorly controlled KCS, details of both medical and surgical options are included in the section on complicated ulcers.

Providing no ulceration is present it is sensible to try to 'kick start' tear production in conjunction with controlling the secondary infection and possibly reducing the inflammatory reaction as well. Frequent application of a broad spectrum antibiotic topically, such as chloramphenicol or fusidic acid, is advised pending the results of culture and sensitivity testing. Ciclosporin should be restarted if it had been stopped, or increased in frequency. If Schirmer tear test readings are less than 1 mm of wetting in a minute, then only 50% of cases respond to ciclosporin. Readings of >2 mm generate a response in approximately 80% of cases. Although twice daily administration is the recommended dose, it is sensible to increase this to three or even four times daily in cases which have previously responded then deteriorated and in neglected cases. Clients should be reminded not to apply too much – the size of a large grain of rice is sufficient. Despite the high cost of the drug they tend to apply too much, often with the false assumption that more will increase efficacy. More frequent dosing might, but more ointment will not! Also remember that excessive ciclosporin accumulation on the lid margins can trigger a hypersensitivity reaction and thus blepharitis – if this is present it is important to establish exactly how the owner applies the medication.

In addition to controlling infection, and trying to stimulate increased tear production, more frequent application of topical lubricants is indicated. There are many different formulations available over the counter for people suffering from dry eyes and some people benefit from one type of tear substitute over another. The same is true for our patients and changing lubricant can help. In general, the longer-acting and viscous agents, such as carbomer gel, hydroxypropyl methylcellulose and hylan, are more effective than the traditional polyvinyl alcohol drops. Unfortunately they tend to be more expensive! Ointments such as soft white paraffin are useful at night or if owners are unable to medicate sufficiently frequently during the day.

If, after 4 weeks of increased frequency of application, there is still no improvement in tear production, then it is acceptable to compound a stronger formulation of ciclosporin under the Cascade system. Tradition-

ally, a 2% solution is made by diluting the oral (not intravenous) human formulation in inert corn oil. This is applied two to three times daily and can significantly improve Schirmer tear test readings after 4 weeks. Owners must be made aware that this is an off-licence use of the drug and appropriate forms must be signed. Another recently discovered tear stimulant, tacrolimus, is also effective in poorly responsive cases but is not licensed at all and thus its use is probably best advised only by specialists.

The use of topical anti-inflammatory agents is controversial with refractory KCS. Certainly topical steroids do reduce the conjunctival hyperaemia and hyperplasia along with the corneal vascularization (which subsequently slows the deposition of pigment on the cornea since the blood vessels are the source of the pigment). However, the risk of ulceration is high anyway, and could be exacerbated by long-term topical steroids. The change in commensal bacteria induced by topical steroids can also predispose to more pathogenic bacterial infections. However, the potential benefits can outweigh the potential complications in some patients and their judicious use is sometimes necessary.

If any blepharitis is present, as is often the case with refractory KCS (especially in cocker spaniels!), then systemic antibiotics are indicated. A 2–3 week course of a cephalosporin is usually necessary. If the eyelid margins are very inflamed or ulcerated from self-trauma, a short course of oral corticosteroids (at an anti-inflammatory not immunosuppressive dose) may be beneficial but they should not be used in conjunction with topical steroids due to the high risk of ulceration.

TREATMENT OPTIONS – SURGICAL

If severe corneal ulceration is present, surgical intervention using a pedicle conjunctival graft is indicated as discussed in Case example 23.1 on complicated ulcers and also mentioned in Case example 16.2 below.

In cases with marked pigmentary keratitis, such that vision is impaired, superficial keratectomy to remove the pigmented corneal deposits has been advocated. However, it is not recommended in poorly controlled cases of KCS. Although the surgery is relatively straightforward, removing just the corneal epithelium and anterior stroma, the success is usually short lived. A large, albeit superficial, corneal ulcer is produced by the surgery and its healing is frequently not without complications. Corneal melting, infection, severe vascularization and scarring, and rapid repigmentation all occur. Even in cases of pigmentary keratitis due to other causes (e.g. brachycephalic dogs with lagophthalmos), this surgery is rarely advised, and treatment options aim to reduce the trigger to the pigment deposition rather than removing it after it has formed. Continued use of ciclosporin, even in the absence of increased tear production, can help to reduce the pigment via its anti-inflammatory action.

The main surgical option for non-responsive KCS remains parotid duct transposition. It is interesting to note that the frequency with which this surgery is performed in American Veterinary Colleges has dropped from more than 15% of KCS cases in the late 1970s and early 1980s to less than 2.5% in the late 1990s according to the American Veterinary Medical Data Program. This is mainly due to the advent of ciclosporin and increased earlier recognition of the disease, and clearly is good news for our patients.

However, parotid duct transposition surgery is still indicated in cases with minimal tear production, severe corneal ulceration (as well as surgery for the ulcer) and where frequent medical treatment is not feasible due to owner restraints or a lack of patient compliance. The surgery can be performed in general practice, but since it is no longer routinely required it might be best to consider referral to a specialist who has the necessary expertise and still undertakes this microsurgical procedure with some frequency! It is essential to check that the patient produces saliva normally prior to considering surgery – xerostomia (dry mouth) occasionally accompanies KCS. Placing a bitter substance on the tongue, such as lemon juice or a drop of atropine, should result in copious salivation and on careful inspection drops of saliva can be seen issuing from the parotid duct papilla just above the carnassial tooth on the buccal mucosa. If this does not occur, the patient will not benefit from parotid duct transposition surgery.

Owners need to be carefully informed of the reason for choosing the surgery and the potential complications which can occur. It is a salvage procedure for the eyes and although it is often very successful and of great benefit to the patient, it can be a bit of a poisoned chalice on occasion. Normally the surgery is successful in that the eye becomes wet with saliva almost immediately. However, saliva contains a much higher mineral concentration than tears, and salty deposits can accumulate both on the eyelids and the cornea itself. These

are often benign but some patients become uncomfortable and require removal either with EDTA drops or even superficial keratectomy. Lubricants to soothe the irritation can also help (soft liquid paraffin ointment for example). Bacterial infections can occur more commonly than in normal eyes – the usual conjunctival flora is changed considerably following surgery – and owners should be made aware that if there is any change in the nature of the discharge or patient comfort they should present the patient at the surgery.

Another potential problem following parotid duct transposition is epiphora – the degree of salivation into the eye cannot be controlled and in some dogs is excessive such that they have permanent tear overflow and resultant moist dermatitis on the face. This can be a particular problem in the 'slobbery' breeds such as Neapolitan mastiffs, such that surgery might even be contraindicated. Many dogs will 'cry' when fed or if food is being prepared, but providing this is not excessive it is an acceptable alternative to end-stage KCS.

PROGNOSIS

The prognosis for complicated KCS is somewhat guarded in a number of cases. If severe ulceration has developed, then both conjunctival grafting and parotid duct transposition might be indicated (sometimes at the same time) but even after this the eye will never be 'normal'. Occasionally enucleation is necessary. With chronic pigmentation vision can be impaired and even following parotid duct transposition complications with irritation and blepharitis from mineral deposition are not uncommon. Thus early diagnosis and regular monitoring, with good owner and patient compliance, are required to prevent such complications.

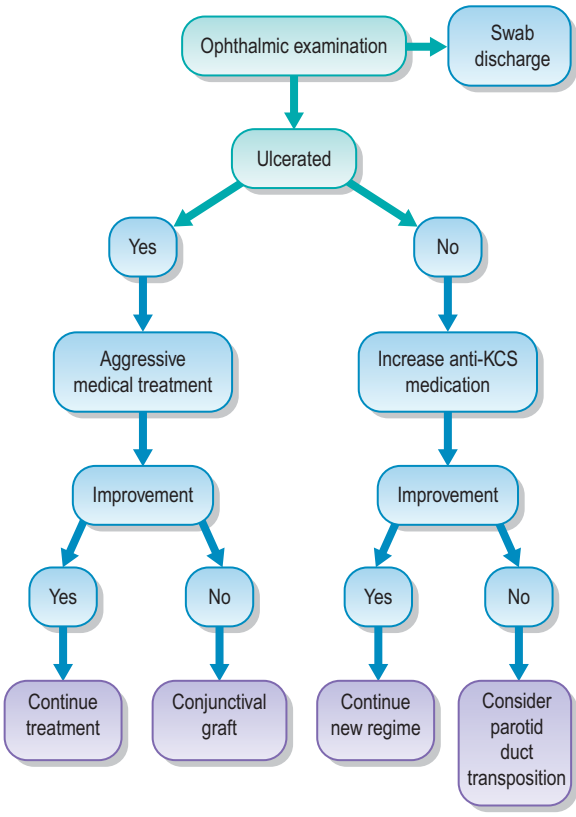


Figure 16.5 Decision-making tree – KCS.

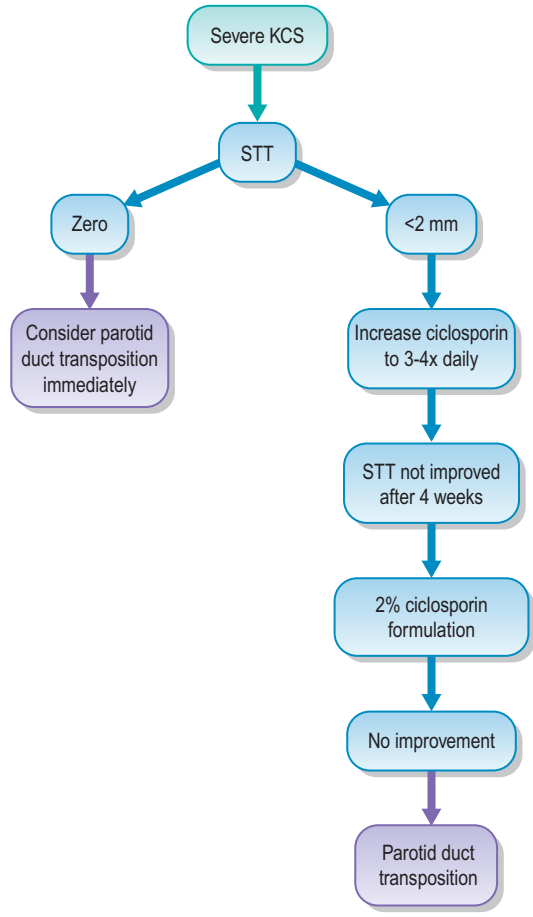


Figure 16.6 Decision-making tree – severe KCS.

CASE EXAMPLE 16.1

Patient details: Miniature long haired dachshund, 13 years old, male (neutered)

PRESENTATION AND HISTORY

The patient had a long history of keratoconjunctivitis sicca affecting both eyes, which had initially been diagnosed 3 years previously. It had been well controlled on twice daily ciclosporin and carbomer gel, and once daily soft liquid paraffin ointment at night. Schirmer tear test had been between 8 and 14 mm in each eye. However, 2 weeks before presentation the left eye started to discharge more, and the dog was unable to open its eye in the morning. The owner increased the frequency of carbomer gel to four times daily but little improvement occurred and so she presented the dog. He was also hypothyroid but this was well controlled with levothyroxine sodium.

CLINICAL SIGNS

General clinical examination was unremarkable apart from a slightly dry, scurfy coat. On ophthalmic examination a sticky mucopurulent discharge was present in the left eye, adherent to the cornea (Figure 16.7). Severe superficial corneal vascularization and pigment deposition were also noted. Schirmer tear test readings were 3 mm/min in the left eye and 11 mm/min in the right. Menace responses and pupillary light reflexes were normal and intraocular examination, although limited, showed no gross abnormalities. A swab was taken from the discharge before it was cleaned away and the cornea stained with fluorescein. This revealed no ulceration.

WORK-UP

The swab was submitted for bacterial culture and sensitivity, and showed a profuse growth of coagulase-positive *Staphylococcus* spp. with moderate resistance to fusidic acid and ciprofloxacin, although it was sensitive to chloramphenicol. Thyroid function was checked and found to be satisfactory.



Figure 16.7 Chronic KCS in a 13-year-old dachshund showing discharge adherent to cornea, pigmentation and marked superficial corneal vascularization. Note also the trichiasis dorsolaterally.

TREATMENT AND OUTCOME

The ciclosporin was increased to four times daily in the left eye, and chloramphenicol drops were started at the same frequency. Topical carbomer gel was applied every 3 hours during the day and the soft white paraffin continued at night. After a week the discharge had reduced but tear production was still only 5 mm/min and significant conjunctival hyperaemia remained. It was decided that anti-inflammatory medication was required, but the use of topical steroids was not advised given the risk of ulceration and so prednisolone was started at 0.5 mg/kg/day for 5 days then every other day for 10 days. The topical medication continued. The eye improved well, with Schirmer tear test readings of 10 mm in the left and 12 mm in the right after 3 weeks. The treatment was then continued with ciclosporin three times in the left (still twice in the right) and lubricants four times daily. After another month tear production was 14 mm in the left and the ciclosporin was reduced to twice daily.

This example illustrates that controlled cases of keratoconjunctivitis sicca can wax and wane such that the medication needs to be adjusted. The dog clearly developed a bacterial infection but it is difficult to know if this was the reason for the reduction in tear production or a result of it. Tear production remained low despite apparently controlling the infection, which suggested some lacrimal gland adenitis. The systemic steroids appeared to settle this and tear production returned to the pre-infection levels.

CASE EXAMPLE 16.2

Patient details: Cavalier King Charles spaniel, 11 years old, female (neutered)

PRESENTATION AND HISTORY

The dog had a 1-year history of bilateral KCS which was controlled on the standard medication of twice daily ciclosporin only. Tear production had increased from 8 mm/min to 16 mm/min (similar in both eyes) and no problems were reported. However, suddenly the right eye started to discharge excessively and the dog cried out when the owner tried to clean the eye. They started using some chloramphenicol drops twice daily which they had in the house. They presented the dog 5 days later.

CLINICAL SIGNS

The dog was slightly depressed but otherwise clinically unremarkable. The right eye was closed and the dog shied away and cried when attempts were made to examine it. A copious mucopurulent ocular discharge was present. Schirmer tear test readings were 8 mm/min in the right and 4 mm/min in the left. The latter had mild corneal vascularization and conjunctival hyperplasia typical of chronic KCS. After gently cleaning the right eye the severe corneal pathology became apparent. Marked corneal oedema and vascularization were present with what appeared to be intrastromal haemorrhage. A ventrolateral ruptured corneal ulcer was present, along with a deep ulcer more medially which was obscured by the third eyelid (Figure 16.8). A dazzle response was present but no menace response and a pupillary light reflex could not be assessed, although there was a consensual constriction of the left pupil when light was shone in the right.

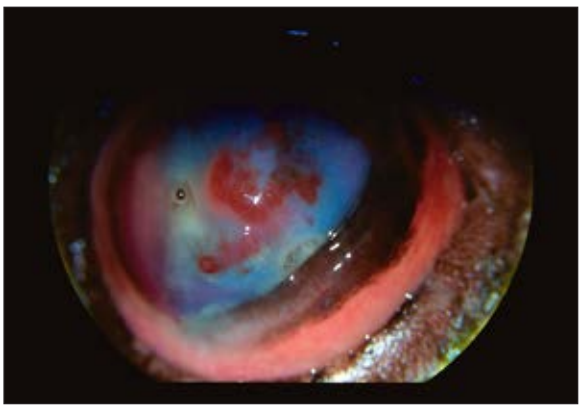


Figure 16.8 Eleven-year-old CKCS with chronic KCS and acute ulceration. Note corneal oedema, vascularization and ruptured pinpoint corneal ulcer (the bubble of aqueous can be seen in the defect). Copious mucopurulent discharge with some adherent to the cornea is typical of severe KCS.

WORK-UP

A swab was taken from the discharge but no significant growth was detected. It is difficult to know if this was as a result of the previous usage of chloramphenicol drops, although twice daily is a suboptimal level to achieve the bacteriostatic effect. The owners were given the option of attempting to salvage the eye or enucleation and opted for the former, and referral for this was arranged. Parotid duct transposition was discussed but the owners were reluctant to have this procedure as well. Pre-anaesthetic blood tests were unremarkable.

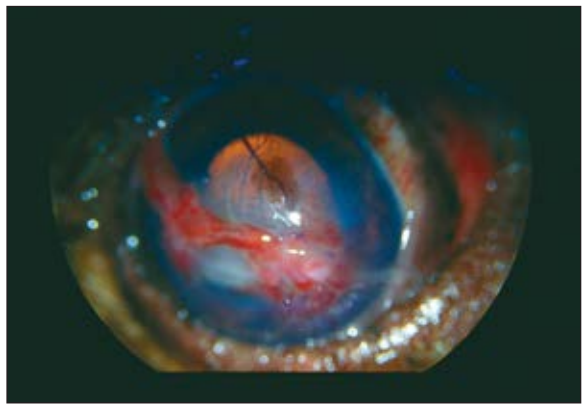


Figure 16.9 Same eye as Figure 16.6, 2 weeks after conjunctival graft.

Table 16.2 Postoperative medical treatment for Case example 16.2

Drug	Frequency	Reason
Ciclosporin	4×	Try to stimulate increased tearing
Ciprofloxacin	6×	Treat any infection (in spite of non-diagnostic swab)
Tropicamide	3×	Mydriasis to reduce reflex uveitis
Carbomer gel	6×	Topical lubrication
Cefalexin	125 mg twice daily	Reduce risk of secondary infection
Carprofen	20 mg twice daily	Analgesia and anti-inflammatory

TREATMENT

Under general anaesthesia the corneal ulcers were repaired using a long conjunctival pedicle graft sutured into the two ulcers (Figure 16.9). Postoperative medication was intensive (see Table 16.2) and continued for 2 weeks before reducing to just ciclosporin three times daily and frequent lubrication. Ciclosporin was increased to three times daily in the left at the time of surgery since tear production was also reduced in this eye.

OUTCOME

The dog did very well. Four weeks later the graft was incorporated into the cornea and the oedema and vascularization had reduced dramatically. Vision was good. The discharge was also minimal and tear production was back to 10 mm in the right and 8 mm in the left. The dog was continued on ciclosporin three times daily for another 2 months, at which time tear production was 13 mm in the right and 12 mm in the left, and it was reduced to twice daily again.

This case illustrates the sudden deterioration which occurs if ulceration develops. The dog could easily have lost the eye. If tear production had not improved again then parotid duct transposition would have been advised. The owners are aware that the left eye could ulcerate and check the dog very carefully.

17 Chronic epiphora

INITIAL PRESENTATION

Discharge

PRESENTING SIGNS

Epiphora is defined as tear overflow and thus these patients present with wet eyes! The discharge is typically serous in nature and can be unilateral or bilateral. It can be congenital (although the wetness does not often manifest until the animal is over 3–4 months of age) or acquired (e.g. following dacryocystitis or feline herpes virus infection). In most instances the patient is not uncomfortable but the owner is concerned about the wetness or the unsightly staining it causes (especially in pale coloured animals). Sometimes owners will also complain about the smell from the face.

CASE HISTORY

With congenital problems the owner may have noticed that the puppy had wet eyes right from when they first brought it home from the breeder. They might even have been made aware of the wetness by the breeder (who might have said that the puppy had 'a bit of a cold' or had been scratched by one of its littermates). However, the owners might not have been aware of any abnormalities until the puppy was 3–4 months old (or even older) when the epiphora and tear staining became more obvious with the growth of the animal. Sometimes the increased wetness is blamed on the puppy teething but it continues well beyond the full eruption of its adult teeth. The classic patient is the young miniature poodle with bilateral tear staining. With acquired cases the patient is usually older (perhaps with the exception of post-feline herpes virus infection in kittens). Acquired cases normally would have a history of a previous ocular problem – an infection or injury for example – which resolved with treatment but the pet was left with chronic tear overflow.

CLINICAL EXAMINATION

General clinical examination is typically unremarkable. On ophthalmic examination the obvious abnormality is a wet face – tear overflow from the medial canthus runs down the side of the nose (Figure 17.1). Often there is dark staining (as the presumed lactoferrin-type pigments in tears react with the atmosphere), and some localized skin inflammation and moist dermatitis can be present. It is essential to determine whether the tear overflow is purely due to poor drainage (i.e. epiphora) or to increased lacrimation. Sometimes a combination of both can be present. However, if Schirmer tear test readings are normal and there is no evidence of ocular discomfort, then epiphora can usually be assumed.

Careful examination with magnification and a good light source are required to evaluate potential causes for the epiphora. The nasolacrimal punctae should be evaluated for presence, size and position. Any medial entropion should be assessed together with lid apposition – some toy breeds have very tight skin–globe apposition which functionally reduces drainage. The caruncles in the medial canthi should be examined to see if any hairs are present on them which could be wicking tears away from the punctae and down the face. Other facial hair can contact the tear film and cause wicking as well – the fine curling fur of poodles is an important contributor to tear overflow.

Examination for conditions which could cause irritation and increase tear production slightly should be



Figure 17.1 Severe epiphora in a 6-month-old Cavalier King Charles spaniel (CKCS).

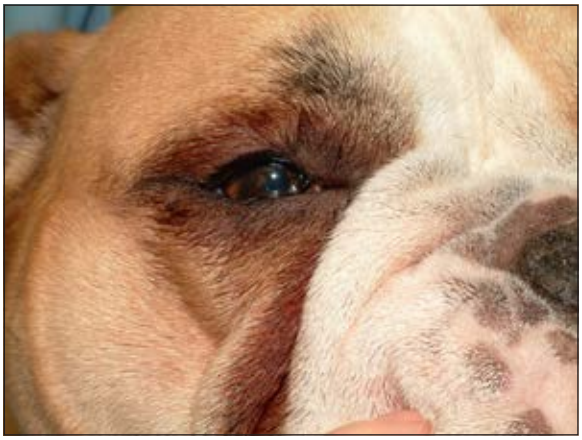


Figure 17.2 Epiphora and increased lacrimation in a young English bulldog. In addition to the tear staining at the medial canthus this dog is wet around the lateral canthus (unlike Figure 17.1) and some lower lid entropion is present causing increased tear production rather than the dog just having poor drainage. Schirmer tear test readings would be high in this dog, but normal in the CKCS in Figure 17.1.

undertaken. Thus the presence of nasal folds rubbing on the cornea or conjunctiva, distichia or ectopic cilia, overt entropion or corneal ulcers should be noted (Figure 17.2). In most cases of pure epiphora these latter abnormalities are not present and the rest of the ophthalmic examination is normal.

DIFFERENTIAL DIAGNOSES

- Epiphora:
  - Imperforate punctae
  - Micropunctae
  - Misplaced punctae
  - Medial entropion
  - Caruncular trichiasis
  - Tight lid–globe apposition
- Increased lacrimation:
  - Entropion
  - Distichiasis
  - Ectopic cilia
  - Corneal ulceration
  - Nasal fold trichiasis
  - Intraocular pain

CASE WORK-UP

The diagnosis should be reached from the ophthalmic examination since the reasons for poor drainage are usually noted on careful inspection of the medial canthal area in particular. Nasolacrimal flushing should be attempted. In the case of imperforate or micropunctae this might not be possible, although normally the upper punctum is of sufficient size for cannulation with a small nasolacrimal cannula (frequently only the lower one is imperforate). Gentle pressure on the syringe attached to the cannula (filled with sterile saline) will result in a small bleb of conjunctiva over the location of the ventral canaliculus pointing to the location for the punctum. This confirms the diagnosis of imperforate punctum and surgery can be performed (see below).

If the punctae are of normal size and nasolacrimal flushes are successful, then attention should be focused on tear dynamics and which of the anatomical abnormalities are most important regarding tear overflow such that the appropriate surgery can be performed as detailed in the following sections.

NURSING ASPECTS

No specific nursing instructions, other than the application of medication as required and bathing the discharge, are necessary.

EPIDEMIOLOGY

Once again, as is often the case for ophthalmic conditions, the selective breeding of pedigree dogs has led to the clinical problems encountered. Imperforate or micropunctae can occur in any breed, but golden retrievers and cocker spaniels (both English and American) are certainly over-represented, together perhaps with the Cavalier King Charles spaniel, miniature and toy poodle, Samoyed and Bedlington terrier.

The tear overflow syndrome seen in the miniature and toy poodle, Maltese terrier and so on is partly due to the head shape and ocular appearance of these dogs which has been specifically chosen by breeders. The small, tight eyelids can lead to medial entropion and physical closure of the punctae which are frequently already small. Unfortunately, the same problems are now seen in brachycephalic cats as well, where selection for the short nose and prominent eyes has led to medial entropion, tight medial lid–globe apposition and trichiasis in exactly the same way as in some dogs. Since in many patients the epiphora does not cause any clinical problem to the animal, and is therefore purely cosmetic, breeders do not see that this is still an abnormality which further selective breeding could reduce significantly.

CLINICAL TIPS

- Determine whether epiphora (STT normal) or increased lacrimation (STT high) is present
- Look carefully at where the wetness actually is (compare Figures 17.1 and 17.2)
- Apply fluorescein and see if drainage to nose/oropharynx occurs and watch where the dye goes (e.g. directly over the face on application)
- Check patency of nasolacrimal system
- Look carefully at the medial canthal area
- Rule out causes of ocular pain

TREATMENT OPTIONS – MEDICAL

In some patients minimal intervention is required. If the epiphora is not excessive, and no moist dermatitis is present, then all that is necessary is regular wiping of the discharge and drying the area. Sometimes the application of a bland ophthalmic lubricant ointment will both guide any hairs away from the canthal area such that the trichiasis is reduced, and will provide a barrier to the tears scalding the delicate skin in the area.

Some owners are concerned about the unsightly appearance of the tear staining together with the smell it creates. Systemic antibiotics such as oxytetracycline, doxycycline and metronidazole have been used orally for 2–3 weeks and certainly reduce the tear staining and smell (without lowering Schirmer tear test readings). However, the discoloration returns as soon as they are stopped and clearly they are not a long-term solution.

TREATMENT OPTIONS – SURGICAL

Surgical correction of imperforate or micropunctae is normally straightforward and successful. Complicated cases, where not only is the punctum absent but the canaliculus has also failed to form properly, are thankfully rare. Under general anaesthesia, ideally with some form of magnification, the upper punctum is cannulated and sterile saline gently injected to delineate the location of the lower punctum – seen as a raised bleb of conjunctiva (Figure 17.3). If the upper punctum is small or difficult to locate, a Nettleship's dilator, a fine pencil-like instrument, can be gently slid along the inner aspect of the upper lid towards the canthus which will catch on the opening of the punctum and thus can be introduced to slightly stretch it, making cannulation easier.

Once the bleb is identified the conjunctiva overlying it is removed – a triangular piece is normally excised and the correct location of the punctum is revealed as saline flows though. Once the newly created punctum has been tidied it should be cannulated and drainage to the nose/oropharynx established by flushing with sterile saline. Postoperatively topical mediation with an antibiotic/steroid combination is normally prescribed – the corticosteroid will both reduce any post-surgical inflammation and slow healing such that the punctum remains patent. Owners can be directed to gently massage the medial canthal area after the application of drops or ointment, which again will reduce the risk of the conjunctiva sealing the opening.

The success rate following opening or enlarging the punctae is normally good – the epiphora decreases within a couple of weeks. It is uncommon for the punctum to seal over again providing it has been created in the correct place! In the few instances where it does seal over, a second procedure is required and it is prudent to place an indwelling cannula for 2–3 weeks while maintaining the topical medication as above.

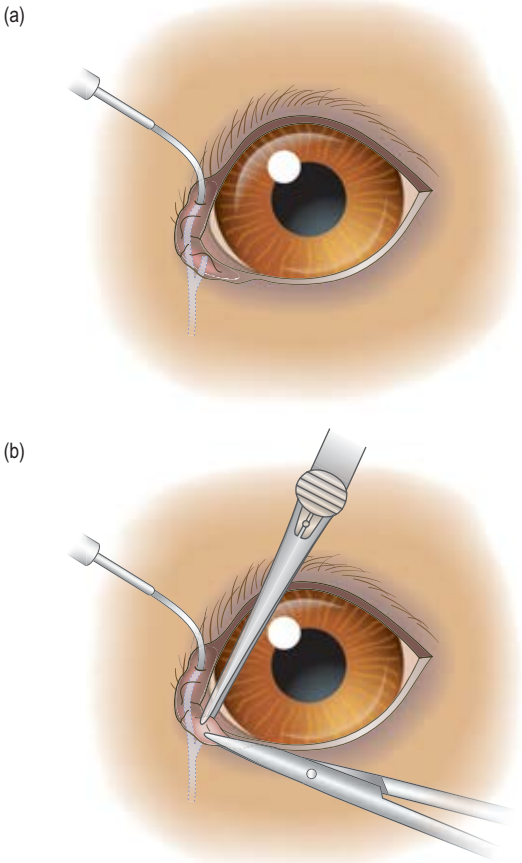


Figure 17.3 Surgery to create a punctum where the lower one is imperforate:

(a) cannulate upper punctum then gently inject saline

(b) locate 'bleb' of conjunctiva over canaliculus, grasp it with fine forceps and cut out the conjunctiva overlying it.

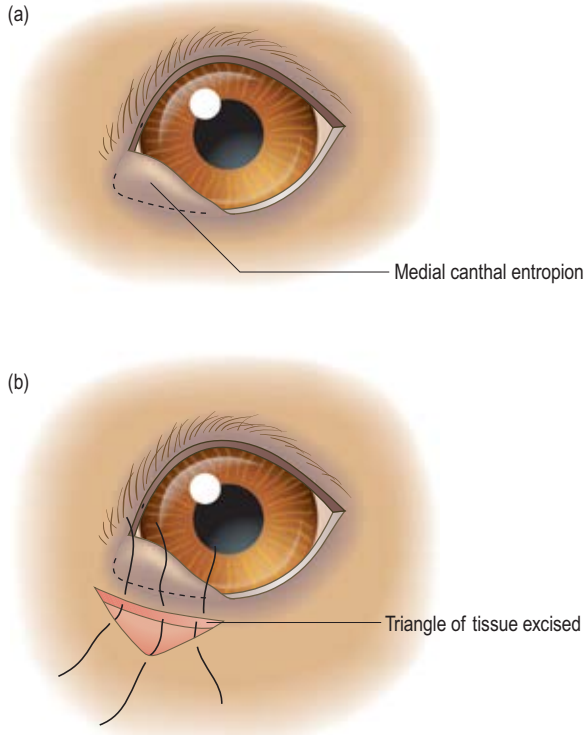


Figure 17.4 Modified Hotz-Celsus procedure for medial canthal entropion:

(a) skin at medial canthus is turned inwards closing punctum and allowing hairs to rub on cornea

(b) triangle-shaped piece of skin is removed to return the lid to its normal position.

If the nasolacrimal punctae are present and of normal size, but misplaced (normally more lateral than usual), they can be repositioned by microsurgical techniques which require referral to a suitably qualified colleague.

Medial entropion and caruncular trichiasis can be addressed in general practice providing some magnification is available. Surgery at this location is more complicated than laterally due to the tight medial canthal ligament making tissue manipulation more difficult, together with the less easy access to the surgical field. However, a simple Hotz-Celsus procedure along the ventromedial lid can be undertaken. Often the piece of skin removed needs to be almost triangular in shape, with the point of the triangle opposite the punctum, rather than the more usual ellipse (Figure 17.4). Sutures should be fine and soft – 6/0 polyglactin 910 is commonly utilized. If the caruncle is to be surgically removed, it is vital that the nasolacrimal punctae and canaliculi are not damaged. Catheterization of them with coloured nylon will assist in their delineation such that they are not inadvertently lacerated. The canaliculi pass quite superficially and careless surgery can easily damage them. Thus, although the procedure of cutting out the caruncles, including the hair follicles, seems very straightforward, one must be aware of the potential complications and referral should be considered, particularly if good magnification is not available.

An alternative to surgical excision of the caruncular trichiasis is cryosurgery. Nitrous oxide and liquid nitrogen systems are both suitable. A small probe is applied directly to the caruncle and a double freeze–thaw cycle

is employed. Postoperative swelling is to be expected – a topical antibiotic/steroid ointment is necessary for 14 days. The area will depigment and this can worry some owners. If there is no entropion present and the punctae are normal, such that it is only the caruncular trichiasis which needs addressing, then cryosurgery is a reasonable option; however, if sharp surgery is also required it seems sensible to address the caruncular problem via this route as well.

Acquired lesions (e.g. following trauma) are more complicated to manage and referral should be considered – particularly if lacerations to the canaliculi or duct have been incurred.

PROGNOSIS

The prognosis obviously depends on which of the various causes for epiphora are present on a patient-to-patient basis – for example, a simple imperforate punctum in a golden retriever carries a very good prognosis. A combination of medial entropion, tight lid–globe apposition and caruncular trichiasis in a miniature poodle carries only a fair prognosis since although the situation can be improved with surgery the tear dynamics will never be normal and some epiphora is likely to be permanent. However, since this is often cosmetic, with owner education the final outcome is often satisfactory.

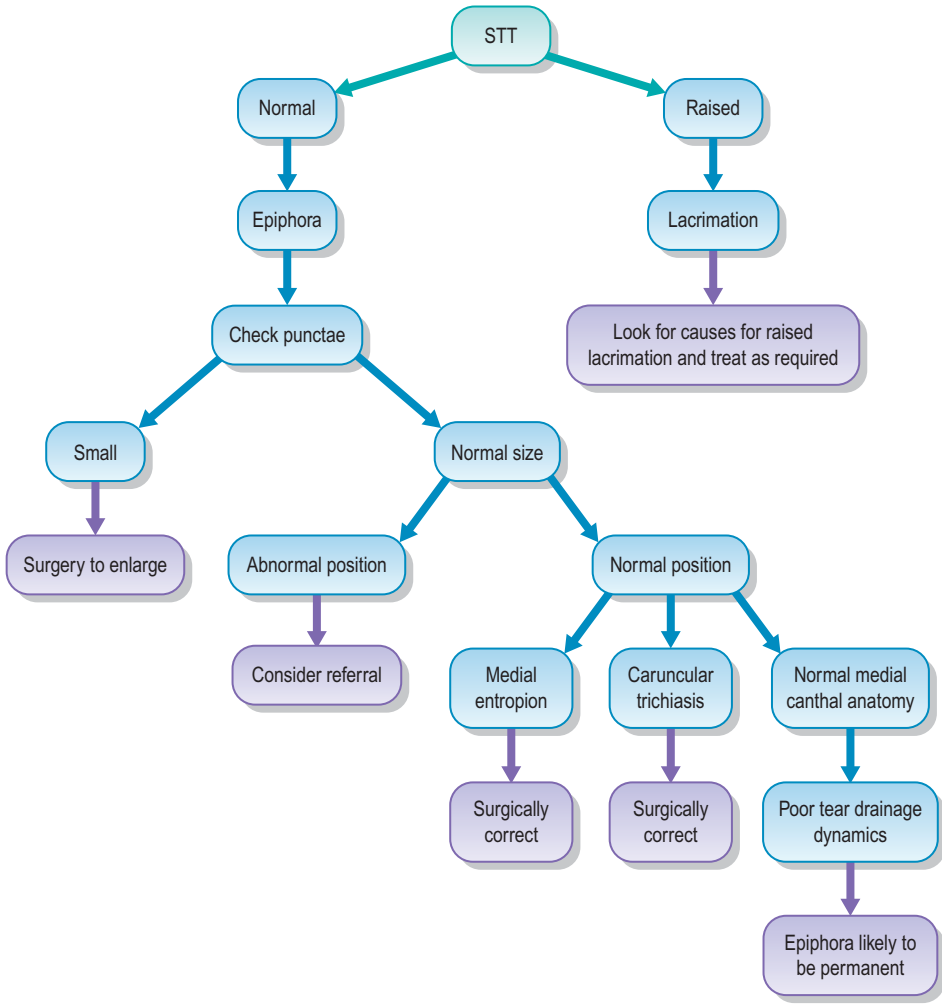


Figure 17.5 Decision-making tree – epiphora.

CASE EXAMPLE 17.1

Patient details: Cavalier King Charles spaniel, 6 months old, male

PRESENTATION AND HISTORY

The dog was presented with wet eyes and a smell from the face. This had been present since the owner acquired the puppy at 3 months and was getting worse. The dog showed no signs of discomfort and the eyes were not red.

CLINICAL SIGNS

On ophthalmic examination marked epiphora and tear staining were present bilaterally (see Figure 17.1). No conjunctival hyperaemia or blepharospasm were present and lid alignment was good. Schirmer tear test readings were 21 mm bilaterally, considered normal. No corneal ulceration was present. Gentle eversion of the lower eyelid medially revealed no nasolacrimal punctum ventrally in either eye, although the upper ones were visible (Figure 17.6). Intraocular examination revealed abnormalities on fundus examination – several dark vermiform streaks were present in the tapetal fundus of both eyes consistent with multifocal retinal dysplasia (Figure 17.7). Vision was unaffected. This condition was unrelated to the epiphora but is an inherited condition in the breed and some dogs can be more severely affected with areas of retinal detachment for example.

WORK-UP

Under topical anaesthesia the upper nasolacrimal punctum was cannulated and saline injected. This appeared at the ipsilateral nostril but none appeared in the eye, confirming an imperforate lower punctum. Close inspection of the lower eyelid at the medial canthus revealed a small bleb of conjunctiva when saline was flushed into the upper punctum. Both eyes were similar. Surgery was advised.

TREATMENT

Under general anaesthesia the upper punctum was cannulated as above and the bleb of conjunctiva snipped off as shown in Figure 17.3. Saline then flowed from the newly created punctum when the upper one was flushed. Postoperatively the dog was discharged with neomycin, polymyxin B and dexamethasone ointment (Maxitrol, Alcon) three times daily for 5 days then twice daily for another 10 days, with instructions to gently massage the medial canthus for 5 minutes after the application of ointment.



Figure 17.6 Same dog as in Figure 17.1 showing the absence of a ventral nasolacrimal punctum.

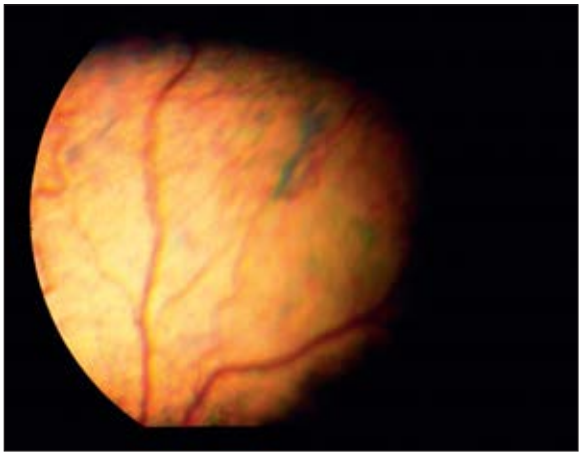


Figure 17.7 Fundus examination of 6-month-old CKCS showing dark vermiform streaks of multifocal retinal dysplasia in the tapetal fundus. Vision was unaffected.

OUTCOME

The dog did very well. The epiphora gradually reduced over the following 3 weeks and the unpleasant smell disappeared completely. The ventral punctae were easily visible on ophthalmic examination. Mild tear overflow did continue but this was not excessive and the owner was happy to accept that it was purely cosmetic.

This case illustrates that some cases of epiphora can be easily resolved with simple surgery. The unrelated multifocal retinal dysplasia is inherited and unfortunately neither the sire or dam nor the litter had been tested for this. Thankfully, the dog was mildly affected and was not intended for breeding, but it does show that owners should enquire about eye tests before purchasing puppies!

CASE EXAMPLE 17.2

Patient details: Miniature poodle, 8 months old, female

PRESENTATION AND HISTORY

The dog was presented with persistently wet eyes. The facial skin was slightly inflamed and the dog rubbed its face along the carpet and the settee several times daily. The eyes themselves did not appear uncomfortable. The dog had been treated with topical antibiotic/steroid drops with no improvement.

CLINICAL SIGNS

On ophthalmic examination severe epiphora was present bilaterally. The medial skin was wet and inflamed. The lid margins were tightly opposed to the cornea and lower lid medial entropion was present. Schirmer tear test readings were 25 mm/min bilaterally – at the upper end of normal. No corneal ulceration was present but fluorescein flowed straight over the face (Figure 17.8) and did not appear at the nostrils. The nasolacrimal punctae were visible but the lower ones were smaller than the upper ones. Some fine facial hairs were touching the corneal surface and a few fine hairs were also present on the caruncles in both eyes. Intraocular examination was normal.

WORK-UP

The dog was sedated and nasolacrimal flushing was performed. This was possible from upper to lower punctae and down the nose bilaterally, showing that anatomically the drainage system was in place, although clearly it was not functioning normally. Surgery was advised to improve tear dynamics.



Figure 17.8 Severe epiphora in an 8-month-old miniature poodle showing fluorescein dye flowing over the face. The eyelid margins are tightly opposed to the cornea with some medial lower lid entropion.

TREATMENT

Under general anaesthesia the lower nasolacrimal punctae were enlarged, the medial caruncles were excised and lower lid entropion surgery was performed as shown in Figure 17.4. Postoperatively topical neomycin, polymyxin B and dexamethasone ointment (Maxitrol, Alcon) was dispensed twice daily for 2 weeks and 5 days of carprofen 4 mg/kg/day were also supplied.

OUTCOME

The epiphora improved but did not fully resolve. The eyelid surgery healed well and the nasolacrimal punctae remained open. However, on blinking, some tears still drained over the face and not down the nasolacrimal

ducts. It was realized that as the facial fur grew longer the tear overflow got worse, and so the owners kept the fur very short around her eyes, and used soft white paraffin ointment to protect the corneas if they saw any curly hairs touching the cornea. Although the face was still slightly wet the skin inflammation subsided. The dog did improve further as she reached maturity but the eyes were never perfect!

This case shows how in some patients surgery can improve the situation but not completely cure the tear overflow. However, the owners understood the situation from the beginning and were happy that the skin was no longer inflamed and the dog had stopped rubbing her face. They had had poodles previously and thought that it was 'normal' for them to have some epiphora.

18 Dacryocystitis – chronic purulent conjunctivitis

INITIAL PRESENTATION

Discharge

PRESENTING SIGNS

Dacryocystitis is inflammation within the lacrimal sac and nasolacrimal duct. Affected animals are presented with ocular discharge – usually this is mucopurulent in nature. Alternatively, a unilateral red eye due to conjunctivitis with conjunctival hyperaemia and a variable discharge can be the initial presenting sign. It is quite common for the patient not to be brought to the surgery when the signs first develop, but only when they change and become persistent with a chronic mucopurulent discharge. The discharge is most copious at the medial canthus and reforms frequently following cleansing and removal by the owner. Sometimes the medial canthal skin is erythematous or swollen and the patient is uncomfortable. The condition is rarely encountered in cats but occurs occasionally in dogs and quite frequently in rabbits. The latter are discussed separately in Chapter 19.

CASE HISTORY

The patient may have history of a wet eye 1–3 weeks prior to the development of the chronic discharge and the discharge itself could have changed from serous to purulent in nature. A previous conjunctivitis or even removal of a conjunctival foreign body might have occurred. The patient can be any age or breed. Although normally unilateral in dogs, the condition can be bilateral on occasion (this is more common in rabbits, as discussed in Chapter 19).

CLINICAL EXAMINATION

General clinical examination is usually normal in dogs. Ophthalmic examination reveals a unilateral ocular discharge centred around the medial canthus (Figure 18.1). Evidence of pain with blepharospasm or self-trauma though rubbing is variable. Conjunctival hyperaemia is normally present in the nictitans conjunctiva but can be widespread. Schirmer tear test readings are normal. No corneal disease is usually present and the rest of the ocular examination is unremarkable. On cleaning the discharge (after taking a swab for culture and sensitivity testing and cytology for Gram stain) gentle pressure at the medial canthus will often result in the appearance of further purulent material at the nasolacrimal punctae (often just the lower one but sometimes the upper as well). Fluorescein passage to the nostril is absent on the affected side (Figure 18.2); remember to compare both sides, and also that false negatives are common.

DIFFERENTIAL DIAGNOSES

- Chronic bacterial conjunctivitis
- Entropion
- Distichiasis
- Foreign body in conjunctival sac
- Micropunctum/imperforate punctum
- Keratoconjunctivitis sicca
- Medial canthal pocket syndrome (compare Figure 18.1 with Figure 12.1)



Figure 18.1 Right eye of a 5-year-old golden retriever with mucopurulent discharge at medial canthus plus elevation and hyperaemia of nictitans membrane.

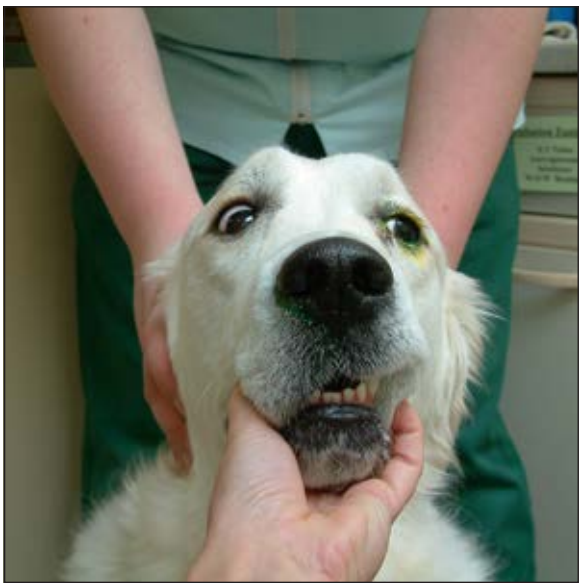


Figure 18.2 Another golden retriever with a blocked left nasolacrimal duct – notice the fluorescein at the right nostril but not the left, while it pools below the left eye.

CASE WORK-UP

Nasolacrimal duct flushing is necessary to reach a definitive diagnosis and to assist with establishing the underlying aetiology, if possible. Often this can be done conscious but sedation or general anaesthesia will be required if the condition is painful or if further diagnostic tests are to be undertaken. Some purulent material can be flushed between upper and lower punctae (depending which one is cannulated). Initially there is likely to be resistance to the passage of saline down the nasolacrimal duct to the nose; however, with persistence, and using steadily increasing pressure, it might be possible to establish drainage. Purulent material should be collected from the nasal ostium for bacterial culture and sensitivity testing. If the nasolacrimal flush is not successful under topical anaesthesia or sedation, then the patient should undergo general anaesthesia. Sometimes flushing from one punctum to the other in dogs will lead to the expulsion of fragments of foreign material (most commonly pieces of grass awn) which will suggest similar material down the nasolacrimal duct itself. Care should be exercised to prevent damage to the duct by overzealous flushing. If sharp pieces of foreign material are present, then exerting excessive pressure could result in rupture of the duct. If any bloodstained material is encountered, then further attempts at flushing should be discontinued.

If it is not possible to establish drainage, then there are several options available. The first is to attempt retrograde flushing. This is really only feasible in medium and large sized dogs. The nasal ostium of the duct is located on the ventrolateral nasal meatus approximately 1 cm inside the external meatus. An otoscope or small vaginal speculum is useful to provide magnification and illumination when trying to locate the opening. Initial cannulation with monofilament nylon (e.g. 2/0) is achieved and this is passed up the duct as far as possible – occasionally it is stiff enough to dislodge any inspissated material as the duct widens as it approaches the lacrimal sac. A narrow gauge catheter can be introduced over the nylon thread into the nasal ostium such that retrograde flushing can be attempted.

If retrograde cannulation or flushing is not feasible, then further case work-up will involve radiography – both plain and contrast studies (dacryocystorhinography). Plain radiographs might reveal bony fractures or neoplastic processes which were not clinically noted. Periodontal disease involving the upper premolars or canine teeth might be present, leading to a localized inflammation in the nasolacrimal duct and subsequent obstruction. Contrast studies, using water-soluble contrast agents, can locate the exact blockage and help identify whether surgical intervention is feasible. In dogs, small amounts (1–2 ml) of contrast are injected into the upper punctum while holding the lower one closed, such that the contrast material is forced down the duct. Care should be taken to ensure that no excess contrast material is spilled as this could easily confuse interpretation

of the radiographs. It is sensible to take a lateral radiograph after injecting the affected side, followed by dorsoventral or intraoral views after injecting both sides for comparison. Cystic dilations can be delineated by this method, as well as locating radiolucent foreign material. If the blockage is anterior – within the lacrimal bone for example – then referral for surgical removal can be considered.

NURSING ASPECTS

Frequent gentle bathing is required and owners should be shown how to do this effectively. Sterile saline on cotton wool pads or soft gauze swabs is advised. A combination of topical and systemic medication is normally necessary and again the nurse should ensure that the owner is able to medicate effectively.

EPIDEMIOLOGY

Dacryocystitis is usually an isolated unilateral finding in dogs and is not contagious. It is most commonly associated with infection following a conjunctival foreign body or extension of dental disease. Secondary bacterial infections are often due to mixed populations of bacteria, and culture and sensitivity testing is recommended. The lacrimal sac and nasolacrimal duct are small conduits which easily become blocked. Neoplasia spreading from adjacent structures can occasionally result in dacryocystitis and traumatic fractures in the area can also lead to problems, although other clinical signs would normally assist the diagnosis in this instance.

CLINICAL TIPS

- Temporary obstructions are easier to treat and as such early nasolacrimal duct flushing is advised
- Consider flushing the nasolacrimal duct immediately should a conjunctival foreign body be identified, or if the owner reports removing such an object prior to presentation with the ocular discharge
- Take swabs for culture and sensitivity testing from the discharge and subsequent flushings – the latter are likely to be more representative
- Repeat flushing every few days during treatment, especially if initial flushing is not successful

TREATMENT OPTIONS – MEDICAL

Initial medical treatment should involve both topical and systemic antibiotics. Broad spectrum agents are chosen such as topical chloramphenicol and systemic cefalexin until culture and sensitivity results are obtained. Repeated attempts at nasolacrimal flushing are advised every few days. If flushing is successful, then both topical and systemic treatment should be continued for 3–4 weeks – even if the clinical signs appear fully resolved before then. Failure to treat systemically, and to use too short a course of medication, are both common reasons for animals to have recurring problems. Some dogs need 6 weeks of oral antibiotics and owners should be advised that the full course of treatment should be completed to avoid relapses. If there is marked conjunctival inflammation at the medial canthus, then a short course of topical corticosteroid drops can be used alongside the antibiotics. Systemic anti-inflammatory agents such as carprofen or meloxicam can also be beneficial.

TREATMENT OPTIONS – SURGICAL

If medical management is not successful, then surgical intervention can be considered. Referral to a suitably qualified veterinary ophthalmologist is recommended. If the nasolacrimal duct is patent then a catheter can be sutured in place for 3–4 weeks while continuing with the medical treatment outlined above.

However, if a permanent blockage is present, attempts at removing this can be considered. It should be ascertained that the blockage is due to foreign material or a cystic lesion for example, rather than a neoplastic process, before surgery is undertaken. Magnetic resonance imaging might even be necessary. Surgical approaches include via the nasal chambers or from the lacrimal bone with gentle curettage of the bone over the duct. Clearly these are difficult approaches.

If the duct is totally blocked, but no foreign matter is present and any infection has been fully resolved, the patient will be left with chronic epiphora. This can occur following inflammation of the duct, such that the swelling incurred at the time sticks the duct together (like a straw collapsing on itself and remaining so). Although the permanent epiphora can be worrying for the owner, it rarely is so for the patient. In many cases it can be managed with regular wiping of the serous discharge and general hygiene. However, on occasions, the patient can suffer from serious epiphora such that

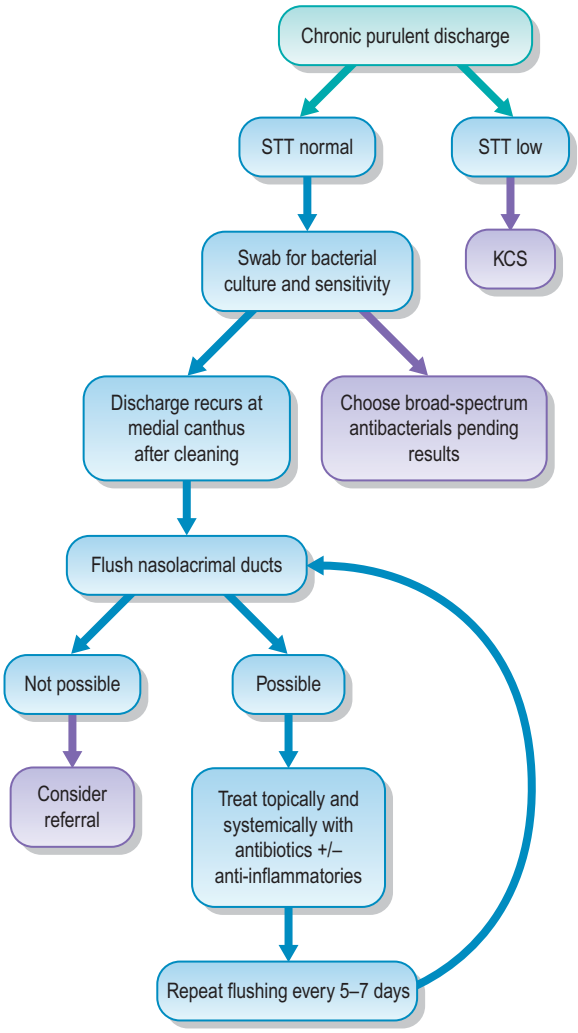


Figure 18.3 Decision-making tree – dacryocystitis. STT – Shirmer tear test.

the skin along the face becomes inflamed and infected with a type of moist dermatitis. In these rare cases it might be necessary to recreate an alternate pathway for tear drainage. Procedures such as a conjunctivorhinostomy or conjunctivobuccostomy, where alternate routes for tear drainage are established to the nasal chambers or mouth respectively, can be undertaken as specialist procedures.

PROGNOSIS

The prognosis for cases of dacryocystitis will depend upon the inciting cause and the duration of symptoms. Thus a relatively acute inflammation, where the duct is still patent and repeated flushing is possible, carries a good prognosis, providing treatment is continued for long enough. Stopping oral antibiotics too soon can result in recurrence. Clearly if a neoplastic process involving the nasal chambers is present, the prognosis is guarded. As with so many ophthalmic complaints, early diagnosis and appropriate treatment does offer the best outcome. Thus the dog with the purulent discharge which is sent away with inappropriate topical medication could re-present 2 weeks later with a permanently blocked duct, chronic infection and persistent epiphora, while simply flushing the nasolacrimal duct at the initial consultation could have resulted in a complete cure.

CASE EXAMPLE 18.1

Patient details: Golden retriever, 5 years old, male

PRESENTATION AND HISTORY

The dog presented with a purulent right ocular discharge which had been present for 2 months. The owner was unclear regarding its initial appearance but thought that the eye had been a bit red and weepy for a few days before she took the dog to the veterinary surgeon for an unrelated problem (grass seed in his left ear). The eye then became sticky and the owner mentioned this at the recheck for the ear. Treatment for the presumed conjunctivitis had included two different antibiotic drops and an antibiotic/steroid ointment. Although the eye improved slightly on all of these, within days of stopping the discharge returned as before. The other eye was fine and the dog was very well in himself.

CLINICAL SIGNS

General clinical examination was unremarkable. On ophthalmic examination the right eye had a mucopurulent ocular discharge at the medial canthus and the nictitans membrane was prolapsed and inflamed (see Figure 18.1). No corneal ulceration was noted and the bulbar surface of the third eyelid looked normal. No conjunctival foreign body was noted. Intraocular contents were normal. Fluorescein failed to flow to the nostril on the affected side, but did so rapidly on the normal side.

WORK-UP

A swab was taken for culture and sensitivity testing prior to the instillation of fluorescein dye. After cleaning the discharge away the medial canthus was palpated. The dog showed signs of discomfort when this was done, and more purulent material exited from the lower punctum. General anaesthesia for nasolacrimal flushing was advised – the dog would not tolerate this fully conscious and since the area was painful it was thought that sedation might not be sufficient.

TREATMENT

Under general anaesthesia the lower punctum was cannulated but no drainage could be established. The upper punctum was cannulated and copious amounts of purulent material were flushed out from the lower punctum. A swab was taken from this material and this was sent for bacterial culture while the previous swab from the conjunctival discharge was discarded. Small fragments of grass awn were present within the purulent material (Figure 18.4). It was not possible to flush to the nose initially. A piece of monofilament nylon was used as a gentle probe down the ventral nasolacrimal punctum, but this could only be extended 1.5 cm down the duct before stopping. Some bloodstained discharge was produced. The probe was removed and the upper punctum flushed again until only clear fluid drained from the lower punctum.

The patient was recovered from anaesthesia and discharged with oral cefalexin and carprofen, along with topical neomycin, polymyxin B and dexamethasone drops.

Five days later the dog was anaesthetized again and repeated flushing was performed. By this stage the discharge had reduced significantly and it was possible to flush down to the nose although with moderate resistance. No further fragments of plant matter were identified. A moderate grown of mixed bacteria was isolated which were sensitive to the antibiotics prescribed. These were continued for a further 3 weeks (both topically and systemically), together with the carprofen.



Figure 18.4 Several pieces of grass awn flushed from the lower punctum of Figure 18.1.

OUTCOME

The dog continued to improve. Two weeks later it was possible to flush the nasolacrimal duct under sedation and minimal resistance to this was encountered, with saline appearing at the nostril, but no mucopurulent material. Four weeks after initial presentation treatment was stopped and the dog remained clinically normal.

This case illustrates how grass seeds can become lodged in the nasolacrimal canaliculus and initiate a dacryocystitis. The fact that the dog had a grass seed in the opposite ear at the same time as the eye started to show symptoms should have alerted the veterinary surgeon in attendance that a foreign body could have been present in the eye. This was not looked for (it might have been in the ventral fornix at that stage and only tracked down the duct later, or might have been visible in the punctum). Identification and removal in the first place would have avoided the chronic problem and repeated anaesthesia. Thankfully the grass awn had started to break up and did not proceed down the bony part of the nasolacrimal duct where permanent obstruction is more likely to develop.

19 Rabbit dacryocystitis

INITIAL PRESENTATION

Discharge

PRESENTING SIGNS

Dacryocystitis, inflammation of the nasolacrimal system, is frequently encountered in rabbits and can be a frustrating condition to manage. Since the aetiology and prognosis are different from dogs (and cats) the condition is being discussed separately. Most rabbits present with unilateral disease but on occasion it can be bilateral. The owners bring the rabbit along because the eye has a nasty discharge. This is usually a thick, whitish, sticky material and can be copious. The medial canthal fur is typically wet and matted and sometimes the rabbit is unable to open the eye as the discharge sticks the lids closed. Sometimes the animal will also show evidence of dental disease, with excessive drooling, anorexia with weight loss and grooming problems (leading to caking of the faeces and so on).

CASE HISTORY

The condition is normally quite gradual in onset – the owners might have been aware that the eye was a bit wet or mucky for several days, or that the rabbit had been off its food for example. There is not normally any specific history suggesting a primary infectious agent (such as the introduction of a new rabbit to the environment). The condition may have occurred previously and if this is the case the owner is more likely to present the rabbit earlier in the course of the disease.

CLINICAL EXAMINATION

Ophthalmic examination will confirm the presence of a copious mucopurulent discharge centred around the medial canthus (Figure 19.1). Samples should be taken for bacterial culture and sensitivity (although samples from nasolacrimal flushings will be more representative – see below). The discharge can then be gently removed, including from the ventral fornix, with damp gauze swabs to facilitate examination. Conjunctival hyperaemia is normally present, often more severe at the medial canthus. Gently pressing the area will result in pus appearing from the single ventral nasolacrimal punctum. The cornea should be evaluated and fluorescein dye used – some patients will have ventromedial corneal ulceration present (presumably related to excoriation by the discharge). Intraocular contents are normally unremarkable although a mild reflex uveitis can be present.

General clinical examination should pay particular attention to the teeth. Overgrowth of both incisor and molar teeth can be the primary problem, as can generalized hyperparathyroidism due to imbalance in the calcium metabolism – often diet related and resulting in poor skull calcification. The rabbit's bodily condition should also be assessed.

DIFFERENTIAL DIAGNOSES

Dacryocystitis can normally be readily diagnosed from the clinical examination. However, other conditions which could appear similar include:

- bacterial conjunctivitis
- conjunctival foreign body with secondary infection
- allergic conjunctivitis
- primary dental disease with secondary ocular involvement.

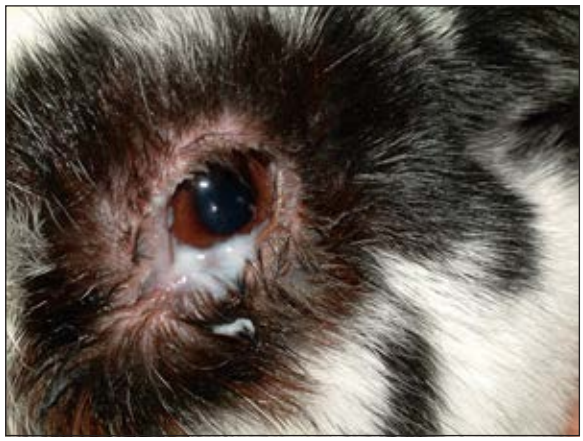


Figure 19.1 Rabbit with copious white purulent discharge and fur matting around the medial canthus.



Figure 19.2 Nasolacrimal cannula in place in the sole ventral punctum prior to flushing (the discharge has been bathed away to facilitate localization of the punctum).

CASE WORK-UP

The most important diagnostic test, which is also an important part of treatment, is to perform a nasolacrimal flush in exactly the same way as in dogs. Most rabbits tolerate this conscious (Figure 19.2). This will differentiate dacryocystitis from a solely conjunctival disease and samples can be obtained for bacterial culture and sensitivity. Once the discharge has been bathed away, including pressing at the medial canthus a couple of times and removing the extra discharge thus produced, topical anaesthetic is instilled into the conjunctival sac. The ventral punctum is cannulated (rabbits do not have an upper punctum) and sterile saline is gently injected. It is likely that initially there will be marked resistance and backflow of purulent discharge around the cannula. However, with repeated gentle attempts hopefully the discharge will be loosened and milky fluid will drip from the ipsilateral nasal punctum. Further flushing and treatment is described below.

In addition to flushing the nasolacrimal duct, careful dental examination is required, with skull radiography if indicated. The prognosis will vary depending on whether dental disease is also present and hence the importance of a complete oral examination.

NURSING ASPECTS

Frequent bathing of the accumulated discharge will be required and it is important to prevent matting of fur around the medial canthus. Thus clipping the fur in the area and the application of a protectant ointment such as soft petroleum jelly (Vaseline) can be useful – but it is important to ensure that this does not get in the eye. An ophthalmic formulation of soft white paraffin would be more suitable if this is a concern. General husbandry should be checked – for example, sawdust would not make suitable bedding as it could stick to the discharge and the eye, exacerbating the condition. Shredded paper might be more appropriate until the rabbit has recovered.

Topical and sometimes systemic medication will be prescribed. Drop formulations are normally preferred since they will not allow further clogging in the area and will enter the nasolacrimal duct more readily. Some owners find drops more difficult to apply in rabbits due to the lateral placement of their eyes and nurses should show owners the easiest way to medicate their pet.

EPIDEMIOLOGY

The development of dacryocystitis is multifactorial. The pathogens involved are typically opportunists – *Escherichia coli*, *Neisseria* and *Streptococcus* spp. and *Pasteurella multocida*. The last is frequently cited, and can be a primary pathogen in the rabbit, but in clinical practice

is not the most frequent isolate as is often quoted. Mixed infections are common. Normally individual rabbits are affected – it is not usually a contagious disease. Any age and breed can be affected, although it is uncommon in very young animals. Dwarf breeds seem to be particularly susceptible – probably due to their brachycephalic conformation.

Reduced tear drainage is necessary to allow a build-up of stagnant tears within the duct, which then becomes infected. Extension from a bacterial conjunctivitis is also possible, but less likely, and foreign bodies such as seeds and grass awns can also trigger dacryocystitis. The long, twisted nature of the nasolacrimal duct and its changes in diameter (see below) contribute to the frequency of infections. Additionally, the close association with the teeth roots (molar and incisor) and the presence of open tooth roots in rabbits are predisposing factors.

CLINICAL TIPS

- Rabbits only have one ventral nasolacrimal punctum
- Pressing on the medial canthus will produce pus from the punctum
- Consider dental examination and radiography

ANATOMY AND PHYSIOLOGY REFRESHER

An understanding of the anatomy of the nasolacrimal duct and its close relationship with the orbit and tooth roots (molar and incisors) is important in relation to oculodental disease. The rabbit has just one nasolacrimal punctum, situated ventromedially, and the nasolacrimal duct has a convoluted path through the lacrimal and maxillary bones. In addition to the tortuous route, the duct suddenly changes diameter several times and can easily become obstructed in its narrow sections. The duct passes close to the tooth roots of both molars and incisors and subsequently becomes involved with disease processes in the tooth roots (Figure 19.3).

TREATMENT OPTIONS – MEDICAL

Repeated nasolacrimal flushing and appropriate antibacterial medication is the mainstay for the treatment of dacryocystitis in rabbits. How to perform the nasolacrimal flush is described above in the section on case work-up. Normally this can be done in the conscious rabbit with the use of topical anaesthesia; however, if particularly painful, then sedation should be used. Sometimes it is not possible to flush initially. Several gentle attempts are made and if these do not result in any discharge appearing from the ipsilateral nostril, it is best to abandon attempts and treat symptomatically for 48 hours before trying again. A broad spectrum systemic antibiotic such as enrofloxacin and topical gentamicin or ofloxacin is advised. All being well, a second attempt at flushing will be more rewarding. Sterile saline should be used to flush initially and once patency has been established some authors advise a final flush with an antibiotic such as enrofloxacin. However, the pH of injectable forms can be inappropriate, causing stinging, and a 2% (1 : 50 dilution) solution of povidone–iodine might be more suitable. Always ensure that it is the solution and not the detergent formulation that is used. Catheterization of the duct, using fine nylon suture material, is very difficult due to the complicated anatomy and the risk of iatrogenic damage is high. As such it is not recommended.

After successful flushing, the patient is treated with topical and systemic medication for 1–2 weeks and the nasolacrimal duct should be flushed again every few days. Once there is no further discharge it is sensible to repeat a final flush 2–3 weeks later.

In some cases it is not possible to establish drainage at all. This is due to either inspissated pus completely blocking the duct or stenosis at one of the narrow or twisted regions. Dacryocystorhinography can be performed to locate the area of the blockage which might correspond to tooth root problems. Addressing the dental disease, and the rabbit's diet, might prove beneficial in such cases.

TREATMENT OPTIONS – SURGICAL

Surgery is rarely performed for cases of dacryocystitis. Dental extractions might occasionally be required. If permanently blocked, it is theoretically possible to create another drainage route into the nasal chambers (dacryocystorhinotomy) or mouth (dacryocystobuccostomy) but these procedures are rarely performed in dogs and cats let alone rabbits.

PROGNOSIS

The prognosis for cases of dacryocystitis in rabbits is poor, even if there is no underlying dental disease, but

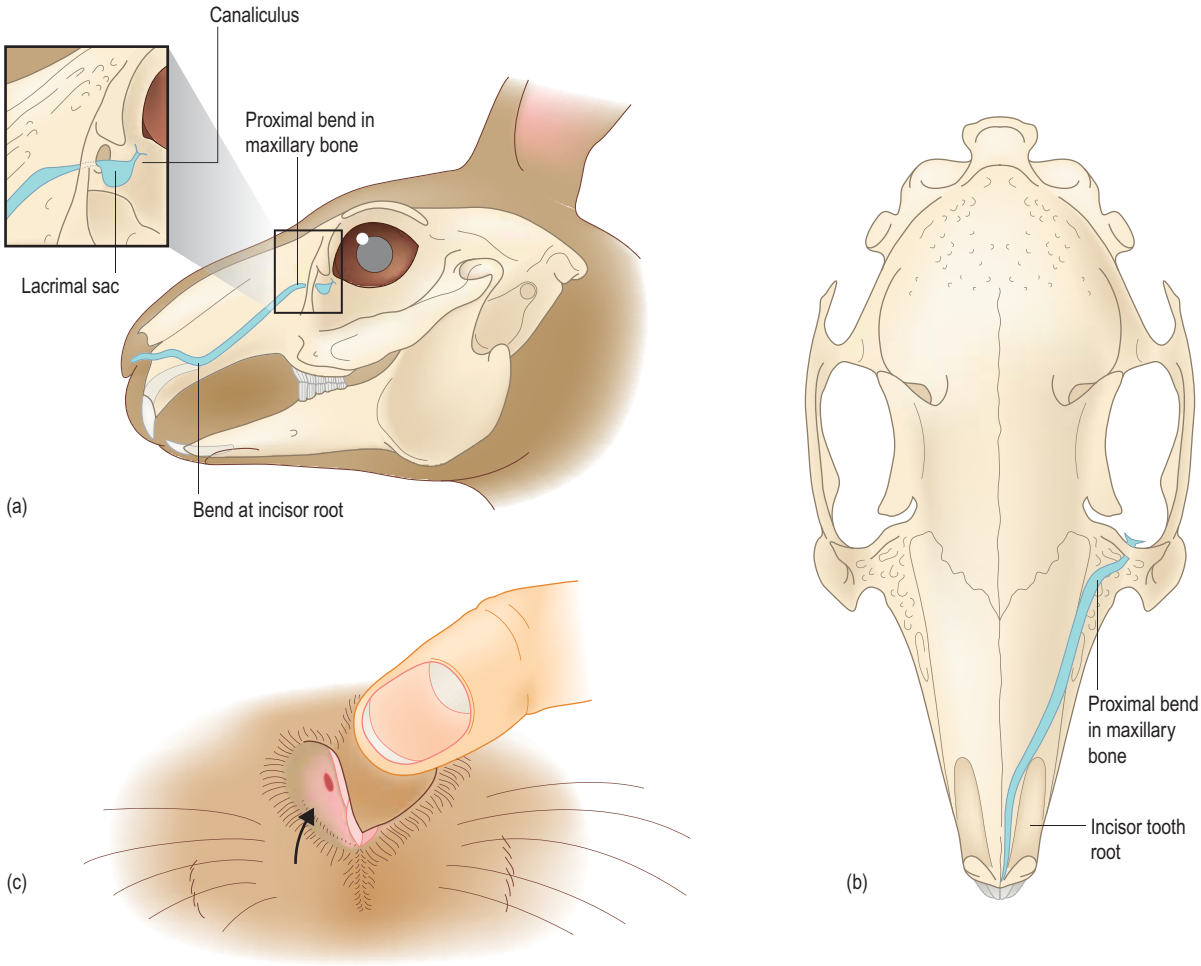


Figure 19.3 The anatomy of the nasolacrimal duct in rabbits:
(a) lateral view and enlarged inset
(b) dorsoventral view showing route of duct
(c) the nasal meatus opening of the duct (arrow).

guarded if the latter is present. For simple cases, repeated flushing with antibiotic cover can be curative. However, many cases can be recurrent and pose a frustrating challenge to manage successfully. Some rabbits require life-long flushing on a monthly basis to keep the condition at bay, but an outright cure is not possible. Long-term antibiosis can also be considered. Realizing that this is an oculodental disease, and addressing feeding and other general management issues, can be beneficial.

SECTION 4

CORNEA

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20 Cornea – introduction

Diseases of the cornea are frequently encountered in general practice and affect both dogs and cats (as well as rabbits). They can be broadly divided into those involving corneal ulceration and those which are non-ulcerative, with various subdivisions depending on cause, age at onset, appearance and so on (Table 20.1). Specific details of each disease and its management are discussed in the following sections – in this introductory part we will consider various anatomical and physiological factors which are applicable to most corneal disease.

ANATOMY/PHYSIOLOGY REFRESHER

The cornea is the transparent anterior portion of the fibrous outer layer of the globe. It is made up of several layers – the outer epithelium, the middle stroma and the inner endothelium. The acellular layer of Descemet's membrane lies between the stroma and endothelium (Figure 20.1). In dogs and cats the corneal thickness is approximately 0.45–0.55 mm. The epithelium itself is multi-layered and non-keratinized and produces its basement membrane. Cells are attached to each other and to the basement membrane via interdigitations of the cell membranes and hemidesmosomes. The stroma is the thickest layer of the cornea, made up of multiple layers of fine collagen-containing cells. Descemet's membrane is an elastic, acellular layer between the stroma and endothelium. The endothelium itself consists of just one layer of cells and therefore is the thinnest cellular layer. Despite its small size, the endothelium is critical for normal corneal functioning as will be seen later on. The corneal surface is bathed by the pre-ocular tear film. Further details of this can be found in the section on the nasolacrimal system (Chapter 15 and cases in Chapters 16 and 17).

Sensory innervation to the cornea is provided by the long ciliary nerves which derive from the ophthalmic branch of the trigeminal nerve (cranial nerve V). The nerves branch superficially – this means that more pain-sensitive nerve endings are present in the epithelium and anterior stroma compared to the deeper stroma and is particularly relevant when considering corneal ulcers: shallow ulcers are often more painful than deeper ones, and as such one must be slightly wary when owners return for a follow-up consultation and report that their pet is much more comfortable with its eye. Hopefully this means the ulcer has healed, but it could be a worrying sign that the ulcer has actually got much deeper!

The cornea has several functions. Along with the sclera, the posterior continuation of the outer fibrous coat of the globe, it provides structural support while protecting the intraocular contents. It also has a major role in both the refraction and transmission of light. It actually has more refractive power than the lens since the light rays pass from air to fluid (aqueous) as they traverse the cornea – crossing from aqueous through the lens has less refractive power. Corneal transparency is vital for normal functioning. This is maintained by the absence of blood vessels and pigment, the lattice organization of the cellular structures and the small size of the collagen fibrils. The endothelium has pumps to remove extra fluid from the cornea which renders it in a relatively dehydrated state and this also assists in maintaining transparency. If this pump fails, then dense corneal oedema can result, giving a blue–grey colouration to the cornea and increased thickness.

CORNEAL ULCERATION

There are many potential causes for corneal ulceration; the more common ones are listed in Table 20.2.

CORNEAL HEALING

If the corneal surface is damaged, the method of healing depends on the depth of the wound. Superficial defects, i.e. those involving just the epithelium, normally heal by

Table 20.1 Classification of corneal disease*

Ulcerative	Non-ulcerative
• Superficial corneal ulcer • **Recurrent epithelial erosion** • Mid-stromal ulcer • **Deep corneal ulcer** • Descemetocoele • **Melting corneal ulcer** • Ruptured corneal ulcer • **Corneal foreign body** • **Corneal sequestrum**	• **Chronic superficial keratitis (pannus)** • Interstitial keratitis • **Pigmentary keratitis** • **Proliferative (eosinophilic) keratoconjunctivitis** • **Keratoconjunctivitis sicca** • **Corneal endothelial dystrophy/degeneration** • Dermoid • Corneal lipidosis • Lipid keratopathy • Corneal neoplasia

*Bold type indicates cases discussed.

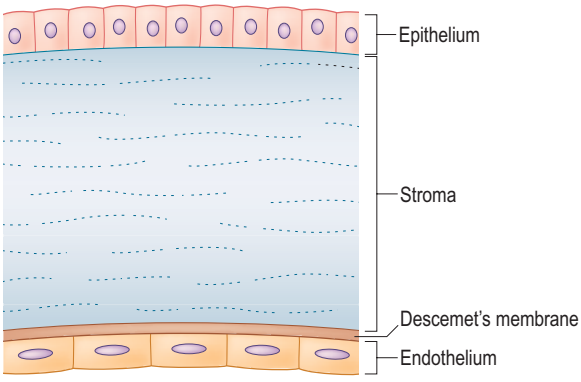


Figure 20.1 Cross-section of the normal cornea.

Table 20.2 Causes of corneal ulceration

Underlying aetiology	Examples
Eyelid abnormalities	Eyelid agenesis (coloboma) Entropion Blepharitis Eyelid neoplasia Lagophthalmos
Eyelash or hair abnormalities	Ectopic cilia Distichiasis Trichiasis Nasal fold trichiasis
Irritants	Shampoo Smoke Acids or alkalis UV light
Tear film abnormalities	Keratoconjunctivitis sicca Qualitative tear film abnormalities
Trauma	Cat scratch Road traffic accident Blunt trauma (blow to face) Foreign body
Infection	Primary bacterial Primary viral Fungal (rare)
Dystrophy/degeneration	Corneal endothelial dystrophy Corneal oedema with glaucoma

migration (epithelial sliding) and mitosis. Thus about an hour after the injury the epithelium at the edge of the lesion flattens and slides inwards to cover the gap. Mitosis of the epithelial cells then allows the structure to return to its normal thickness (Figure 20.2). If the whole of the epithelium is removed it takes only 48–72 hours for it to be replaced, although it remains thinner than normal for several weeks. Comparing how quickly a normal epithelium will heal with that in cases of refractory ulceration highlights the abnormalities present with the disease.

If the corneal damage is deeper and also involves the stroma, the epithelium fills the defect in the same way, by sliding and dividing, and this is followed by fibroblast proliferation in the stroma which slowly fills in the stromal defect and allows the then thickened epithelium to return to its normal depth. Thus when reassessing deeper corneal ulcers it is important to establish whether the epithelium has grown across the bottom of the defect and carefully flushing out any excess fluorescein will assist with this diagnostic test. Stromal injuries will result in more scarring than if the epithelium only is damaged. This is mainly due to the transformation of stromal keratocytes into fibroblasts. These interrupt the normal alignment of the fine stromal lamellae and result in permanent opacification.

Blood vessels also grow into the damaged cornea from the limbus. These are a mixed blessing, since although they bring leucocytes and various growth factors to the damaged area, they can also predispose to excessive inflammation, including granulation tissue formation, and further scarring can ensue. Obviously vascularization can also be a factor in non-ulcerative

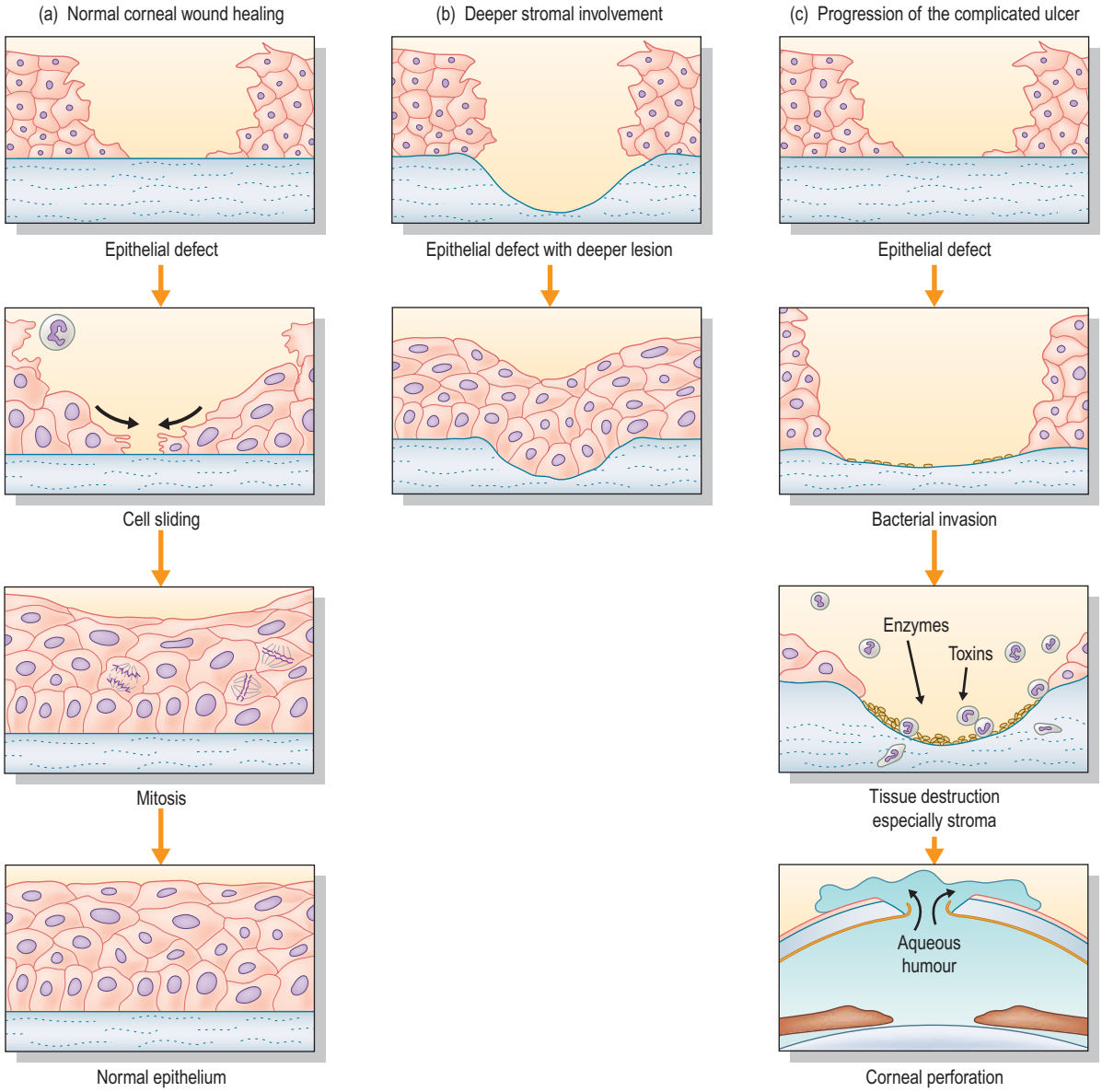


Figure 20.2 Steps in corneal healing:
(a) simple epithelial defect heals by sliding and mitosis
(b) deeper stromal involvement can heal by the above and slow stromal division
(c) infection and toxins can result in complication and even rupture.

corneal disease and the specific pathology of this will be mentioned if relevant for the cases discussed later.

Deep injuries, or ulcers which are progressing (perhaps due to infection), can result in rupture of Descemet's membrane. This elastic, non-cellular layer is normally under some tension, and if ruptured will coil back on itself and rarely re-forms a complete layer afterwards. Healing of the endothelium is limited since these cells have minimal regenerative capabilities in dogs and cats. As such, they heal by hypertrophy and sliding, rather than by mitosis, and this ability is limited. Corneal oedema is common if the endothelium is damaged, and can be permanent.

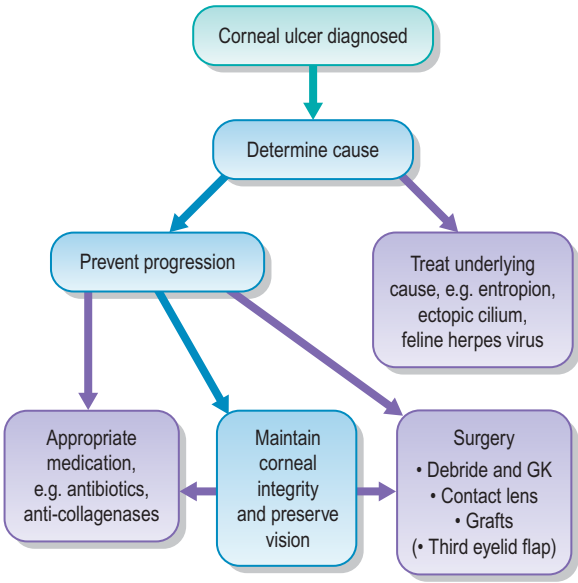


Figure 20.3 Decision-making tree – corneal ulcer. GK – grid keratotomy.

Table 20.3 Common causes of corneal colour change

Colour	Cause
Red	Neovascularization Stromal haemorrhage Symblepharon Neoplasia (rare)
White	Dense oedema Lipid Calcium deposits Leucocytes Stromal scarring Keratic precipitates
Blue–grey	Mild corneal oedema Keratomalacia with infectious agents
Pigmented	Superficial melanin deposition Corneal sequestrum Corneal endothelial pigment (from uveal cysts)

CORNEAL COLOUR CHANGES

As with most ocular pathology, there is only a limited number of ways the cornea can react to insults. Since the cornea is usually transparent, the colour change which occurs following disease is the most striking alteration and can be divided into white, red, blue–grey or pigmented lesions, as listed in Table 20.3. We will encounter these as we discuss the different corneal conditions in the following sections.

21 Recurrent epithelial erosion

INITIAL PRESENTATION

Discharge, abnormal appearance – red

PRESENTING SIGNS

Dogs and cats with recurrent epithelial erosions typically present with mild conjunctival hyperaemia, increased lacrimation and slight blepharospasm in the affected eye. Breeds typically presented include boxers and corgis although any middle-aged dog can be affected, including crossbreeds. Burmese and Persian cats tend to be over-represented compared to the general feline population.

Commonly only one eye is affected but occasionally both eyes are involved at the same time. The degree of discomfort is variable – some patients do not seem sore, while others try to rub the affected eye from time to time. A few patients are particularly uncomfortable although this is quite uncommon. There may be a change in appearance of the eye – the cornea is sometimes slightly oedematous, giving a grey–blue cloudiness to the ulcerated area while some conjunctival hyperaemia is inevitable. Sometimes a secondary bacterial infection can develop and in these cases the ocular discharge will change from serous to mucopurulent in nature and the owner might present the patient because of the discharge.

CASE HISTORY

Owners usually notice the slight redness and watering for several days before bringing the patient to the surgery. Unfortunately, they sometimes wait until the disease has been complicated by a bacterial infection – they do not worry about the increased lacrimation or redness until the discharge becomes purulent! Sometimes there is a history of possible trauma – running through the bushes for example – although this is by no means necessary for the ulcer to develop. If the patient has suffered from similar problems in the past, the owners are more likely to present their pet sooner for evaluation. Often the disease is recurrent in one or both eyes.

CLINICAL EXAMINATION

A full ophthalmic examination should be performed on both eyes. Conjunctival hyperaemia and increased lacrimation are the typical abnormalities present. The degree of blepharospasm is variable. As with all patients presenting with ocular surface disease, Schirmer tear test readings should be taken from both eyes. It is usual for the affected eye to have a higher reading. The patch of ulceration might be visible with the naked eye but it is important to test both eyes with fluorescein – and to flush the excess away properly with sterile saline. Typically the ulcer has an irregular outline with under-run edges – the fluorescein leaches under the loose epithelium (Figure 21.1). This lip of non-adherent epithelium is pathognomonic for the non-healing ulcer. Also by their nature these ulcers are extremely shallow – it is only the epithelium that is denuded, the stroma remains intact.

There may be evidence of corneal vascularization – fine branching vessels from the limbus nearest to the ulcer (Figure 21.2). If the case is presented late in the course of the disease this vascularization might be abundant, producing an obvious granulation reaction and the owner's complaint of a red eye. If the case is complicated by secondary bacterial infection, the discharge will be

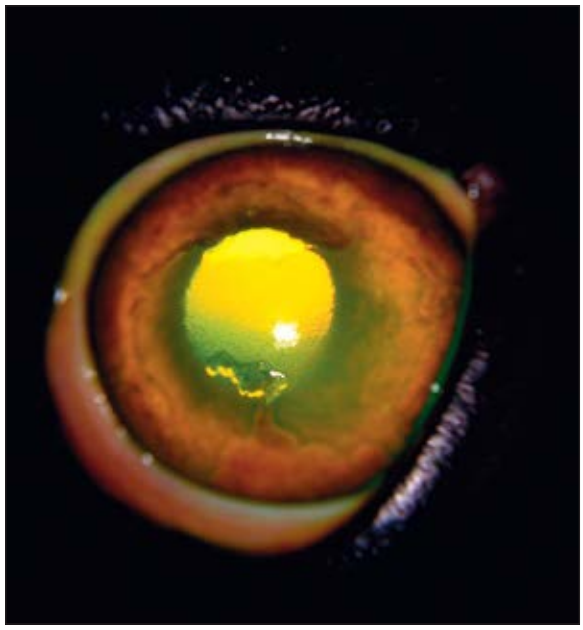


Figure 21.1 Typical epithelial erosion in a 10-year-old boxer. Note the irregular outline and very superficial nature of the ulcer.

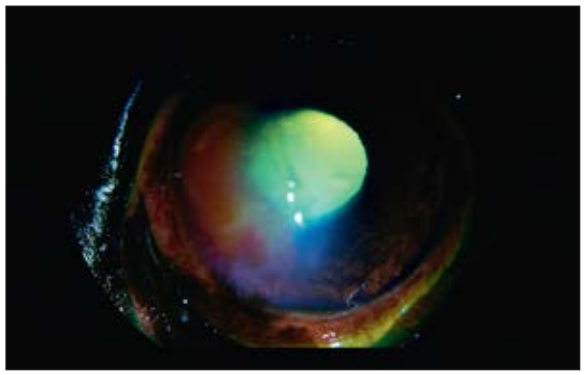


Figure 21.2 Another epithelial erosion, also in a boxer, with moderate corneal vascularization and some corneal oedema.

mucopurulent in nature – although usually it is purely serous. Occasionally corneal oedema is present in the ulcerated area and adjacent tissue but this is not usually a particularly obvious clinical sign. Normally there is no intraocular involvement, although mild miosis might be apparent due to a reflex uveitis. This is more likely in long-standing cases, or when the patient seems particularly uncomfortable – the spasm of the iris constrictor muscle will cause ocular pain.

DIFFERENTIAL DIAGNOSES

Basically any cause for corneal ulceration should be considered, such as:

- trauma (cat scratch, foreign body)
- entropion
- ectopic cilia
- trichiasis
- distichiasis
- keratoconjunctivitis sicca
- topical irritants (e.g. shampoo)
- bullous keratopathy (secondary to corneal endothelial degeneration).

CASE WORK-UP

Minimal work-up is required since the diagnosis should be obvious from the presenting signs and the findings of the ophthalmic examination. As mentioned in the above paragraph on ophthalmic findings it is important to test both eyes with fluorescein. It is also sensible to check for foreign bodies, particularly behind the third eyelid or in the conjunctival fornix, and especially if the ocular discharge is purulent in nature. It is not usually necessary to perform culture and sensitivity testing on the ocular discharge unless it is mucopurulent or purulent in nature.

NURSING ASPECTS

Nursing the patient with a non-healing ulcer usually involves topical medication. Hospitalization is not usually required and, as such, the nurse should ensure that the owner is competent at putting in the drops or ointment, and is able to medicate frequently enough. Patients should not be allowed to rub the eye(s) – they are most likely to do this immediately after the application of topical medication and should therefore be distracted for a few minutes afterwards. Elizabethan collars are not routinely required. If the patient persistently tries to rub they might be necessary but most animals are fine without – providing that they have adequate analgesia and are closely monitored after the application of topical medication.

EPIDEMIOLOGY

Recurrent epithelial erosions have a variety of synonyms – non-healing ulcer, indolent ulcer, boxer ulcer, refractory corneal ulcer, spontaneous chronic corneal epithelial defect (SCCED) and so on. As the names imply, they are shallow, slow to heal and have a tendency to recur. The condition was originally described in the boxer, and although it remains most commonly seen in this breed it can occur in any breed of dog, particularly middle-aged and older patients. It also occurs in cats – Persians and Burmese breeds are presented more commonly than domestic types.

The condition is thought to represent a corneal epithelial dystrophy characterized by epithelial basal cells which produce an abnormal basement membrane and have reduced numbers of hemidesmosomes for attachment. These two abnormalities have been demonstrated in boxers affected with epithelial erosions. The normal attachment of the epithelium to the underlying stroma is dependent both on the basement membrane (augmented with anchoring fibrils) and on hemidesmosomes, and as such it is easy to understand how abnormalities with both of these structures could lead to epithelial non-attachment. It also explains why the ulcers often develop in the absence of trauma or any other inciting cause, and contributes to the problems sometimes encountered in achieving proper healing.

Histologically, affected dogs have separation of their basal epithelial cells from the underlying basement membrane and there are multiple layers of cells in the adjacent lip of non-adherent epithelium. Both hyperplastic and degenerate cells can be present in this lip of abnormal epithelium, together with inter- and intracellular oedema. The abnormal basement membrane can have splits within it and the adjacent stroma can also contain abnormal hyalinized acellular components.

CLINICAL TIPS

On initial diagnosis of a recurrent epithelial erosion or refractory corneal ulcer (or whichever name you choose!) it is important to explain the condition fully to the owners. If they are aware of the nature of the disease, including the expected healing time, potential complications and various treatment options available, they are more likely to cooperate with treatment plans and improved results can be expected. If not fully appraised, owners become dissatisfied as the condition can drag on for weeks, or even months, and in this situation owner compliance is likely to be poor.

Draw up a treatment plan initially, with a cut-off point for referral if the condition has not resolved after a certain period of time – a month for example.

Show the owner the ulcer and its progress. It is easy for them to see the fluorescein-stained lesion and it also helps with their understanding. When they feel involved in the monitoring of the case, compliance with treatment will be improved.

In cats with recurrent epithelial erosions it is important to warn the owner of the risk of sequestrum formation. This can occur following any chronic feline ulcer and usually requires referral for surgical removal via superficial keratectomy together with conjunctival grafting or corneoscleral transposition.

TREATMENT OPTIONS – MEDICAL/SURGICAL

There are various treatment options available for recurrent epithelial erosions. Normally these will consist of initial medical management but can progress to surgical procedures of varying complexity depending on the response to treatment.

Debridement of any loose epithelium is fundamental to achieving healing. This is normally performed under topical anaesthesia and should be performed on initial presentation – the characteristic lip of non-adherent epithelium needs to be removed before normal adhesion can occur. If the patient is fractious or the eye particularly painful, then sedation might be required; however, this is rarely necessary. One drop of topical anaesthetic is applied (e.g. proxymetacaine) and repeated 5 minutes later. The patient's head is held still and a dry, preferably sterile, cotton bud is used to remove all loose epithelium. The cotton bud can be gently rubbed over the ulcer and rolled at the edges to pull off the thin, loose tissue (Figure 21.4). Each time the tip becomes moist it should be replaced with a new cotton bud. Normally three to five are required to fully debride the ulcer. This will enlarge the ulcer significantly and it is important to explain this to the owners – it is quite usual to double

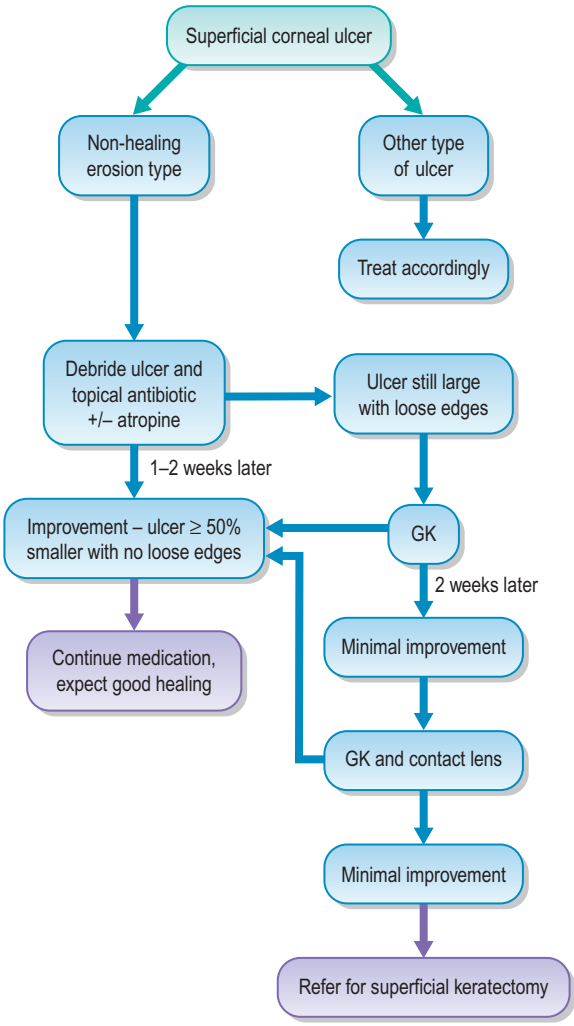


Figure 21.3 Decision-making tree – recurrent epithelial erosion. Gk – grid keratotomy.

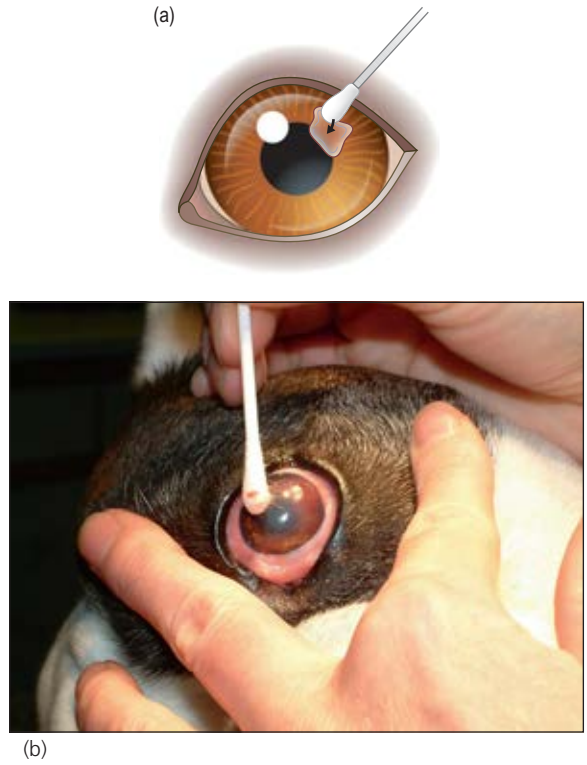


Figure 21.4
(a) Debridement of superficial corneal ulcer using a dry, sterile cotton bud after the application of topical anaesthetic.
(b) Debridement procedure.

or even triple the size of the erosion just with simple debridement.

Topical antibiotics are prescribed (broad spectrum against the normal conjunctival contaminants, e.g. fusidic acid (Fucithalmic Vet, VetXX)), together with systemic NSAIDs (in dogs) to prevent ocular pain and thus reduce the risk of self-trauma. Under no circumstances should topical anaesthetics be used to promote patient comfort: although they do provide good analgesia, they damage the epithelium and slow healing further – they are for diagnostic and minor surgical procedures only and their use for treatment of any ocular problem is contraindicated. Elizabethan collars are not normally required unless the patient is determined to self-traumatize – they should not be supplied routinely.

The patient should be checked a week later. If there is significant improvement – demonstrated by a 50% reduction in ulcer size plus no return of the lip of non-adherent epithelium – then the treatment continues and the ulcer should heal in another week. Unfortunately, simple debridement is often not sufficient. Thus, if the ulcer is not much smaller, or if some non-adherent epithelium has returned, then the next stage of management includes further debridement plus a grid keratotomy (GK). This procedure is not recommended in cats since the development of a sequestrum can be triggered following a GK – further debridement and contact lens placement is the preferred next stage in



Figure 21.5 Performing a grid keratotomy (GK).



Figure 21.6 Appearance immediately following GK. Since this ulcer extended over almost all of the cornea post-debridement the GK has done likewise. However, in a smaller ulcer, only the area denuded of epithelium and 2 mm beyond it into normal epithelium needs the GK – not the entire cornea.

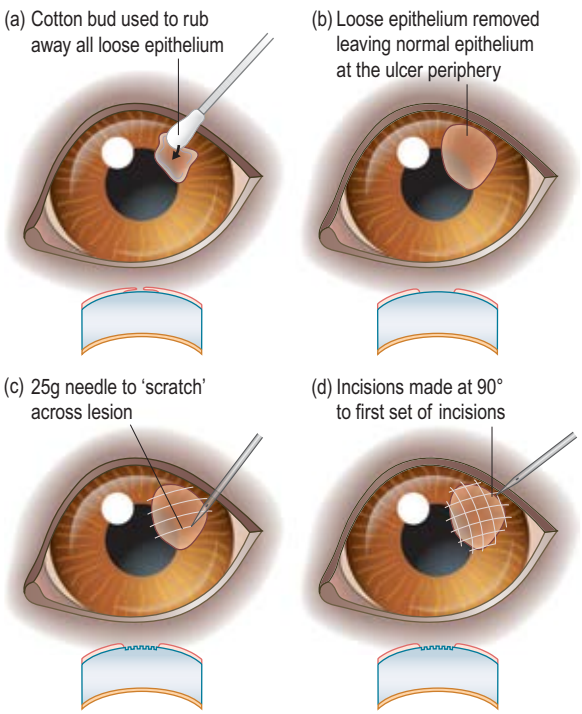


Figure 21.7 Diagram illustrating how to perform a grid keratotomy (GK).

cats. Chemical cautery is no longer routinely advised but will be mentioned later since in experienced hands it can provide a good alternative to GK. Remember that the non-healing superficial ulcer is the only indication for performing a GK – it should not be used for any other type of ulcer.

If the patient is compliant, topical anaesthesia is all that is required to perform the GK; however, if the animal is wriggly, or the veterinary surgeon inexperienced, it is safer to perform the procedure under sedation. The cornea is debrided again as described above. A 23–25 gauge needle is used to perform the GK. It is used to scratch across the cornea from 1 to 2 mm either side of the ulcer, i.e. from normal cornea, through the ulcer and to normal cornea the other side, in a grid pattern with the lines approximately 1 mm apart (Figures 21.5 and 21.6). Figure 21.7 shows in diagrammatic form how to perform the GK. The needle should be pressed hard enough to indent the stroma without actually cutting too deeply through it. Some people find that bending the tip of the needle assists in assessing the depth more accurately. The GK is easier to perform than the multiple punctate keratotomy. With the latter a needle is used to make multiple holes in the anterior stroma of the cornea. However, it is more difficult to gauge the depth of the punctures – especially if the patient is awake – and so the GK is preferred, since there is more control in dragging the needle across the cornea rather than stabbing into it. Inadvertent corneal perforation can occur with the punctate keratotomy and clearly should be avoided! A useful tip, in the awake patient, is to keep the hand holding the needle slightly below the patient while performing the GK. Thus, should the patient suddenly jerk its head, it is unlikely that the needle will accidentally penetrate too deeply – most dogs jerk their heads up and slightly backwards. The aim with both grid and punctate techniques is to expose normal corneal stroma for the new epithelium to adhere

to and to allow normal hemidesmosomes to form. As such, the sliding epithelium has something to attach to, and does not curl over on itself in the characteristic loose lip at the ulcer edge.

Prophylactic topical antibiotic cover and systemic NSAIDs continue. There is no need to change the antibiotic agent unless there is evidence of a secondary infection and suspected antibiotic resistance. If this is the case, then the choice of an alternative should rest on the results of culture and sensitivity testing. It should be remembered that antibiotic agents do not affect the rate of healing and swapping from one to another each time the patient re-presents to the surgery is bad practice. If the eye is painful due to a reflex uveitis, then a few days of topical atropine will greatly improve patient comfort. Cats tend to have an intense dislike of the taste of atropine as the drops drain down the nasolacrimal ducts – in this species ointment is preferred to prevent excessive salivation and even frothing at the mouth. Dogs tend to have less dramatic reactions to the taste but owners should always be warned that the patient might salivate following the topical administration of atropine drops. Topical NSAIDs are not normally required – atropine will be a more effective analgesic in the presence of reflex uveitis by paralysing the pupil constrictor muscle.

The patient should continue to be checked on a weekly basis. If the ulcer has not healed 2 weeks after performing a GK then the procedure can be repeated. The use of a contact lens should be considered at this stage. A bandage soft contact lens, placed after debridement ± GK, will promote patient comfort while protecting the delicate epithelial cells as they migrate and attempt to adhere. The corneal diameter and curvature should be measured to ensure a good fit of the contact lens. The majority of manufacturers supply a measuring ruler. The most common size for dogs is 18 mm diameter × 9.8 mm curvature and for cats 16 × 9.2 mm. However, there can be marked individual variation and certainly one size does not fit all! A poorly fitting contact lens is the major reason for early loss of the lens (Figure 21.8).

The lens is placed under topical anaesthesia, with sedation if necessary. The cornea is gently flushed with sterile saline to remove any debris such as remaining loose epithelium post-GK. The lens is also flushed prior to placement. It should be placed under the third eyelid and should reach to the limbus. No air bubbles should remain between the corneal tear film and the contact lens – gently retropulsing the globe should eliminate these providing the lens is the correct size. No wrinkles should be present either – particularly at the periphery of the lens – and it should fit snugly against the cornea in all areas. Lens retention can be promoted by placing a temporary tarsorrhaphy (single mattress) suture at the lateral canthus – for example, using 4/0 silk or polyglactin 910 (Vicryl, Ethicon). This helps particularly in brachycephalics such as the boxer, where a relative lagophthalmos can predispose to early loss of the contact lens. Good visualization of the cornea is still possible – unlike if a third eyelid flap is placed.

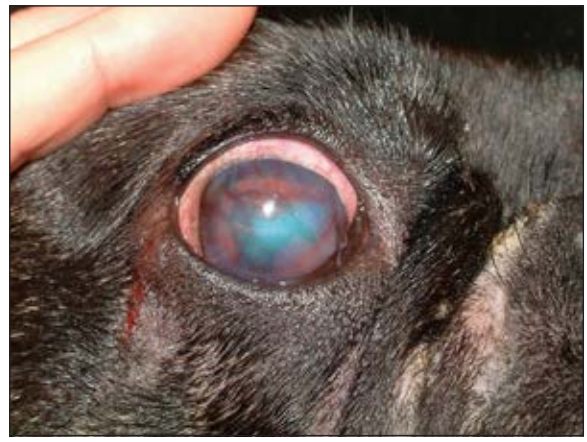


Figure 21.8 Poorly fitting contact lens – the lens is too big and is puckered close to the nictitans membrane. As soon as the third eyelid sweeps across the cornea the contact lens will be pushed out (usually in the waiting room while the owner is on their way out!).

When a contact lens is in situ it is important that drops rather than gels or ointments are used topically. Thus a broad spectrum agent such as chloramphenicol, together with topical atropine if required, is a good choice. An Elizabethan collar might be prudent at this stage.

The contact lens should be retained for 2–4 weeks and the patient checked on a weekly basis. Fluorescein testing should not be performed when a contact lens is present – the dye will pool under the lens resulting in a bright green eye and also can be absorbed into the lens such that it has to be removed anyway. Thus it is better to leave the lens for 2 weeks before removing it, flushing the eye, testing with fluorescein, and replacing with a fresh lens if required.

Other treatment options, if a contact lens is not available, include the third eyelid flap, or chemical cautery. The former has the advantage of being easy and quick to perform but does not allow regular checking of the

cornea and provides less epithelial protection than a good fitting contact lens. If used, the third eyelid flap should be sutured to the bulbar conjunctiva with fine suture material (4/0 or smaller) rather than through the upper lid. Although the latter procedure does, in theory, allow the sutures to be untied, the flap taken down, the cornea checked and the flap replaced without further anaesthesia, in practice this is rarely possible. A main disadvantage of the upper lid suturing is that the globe continues to move independently below the flap, such that epithelial rubbing still occurs. If the third eyelid is sutured to the bulbar conjunctiva it moves with the eye and thus provides more epithelial protection. The third eyelid flap is left in place for 7–14 days.

Chemical cautery is rarely performed today and should never be used in cats. It causes severe corneal irritation and more scarring than modern techniques. When used, only a tiny amount needs to be dabbed onto the ulcer edges. A cotton bud dipped in the agent (e.g. phenol) is an excessive amount. A tiny piece of cotton wool wrapped around the tips of the smallest haemostats in the practice is all that is required! This is lightly touched all around the ulcer edges (not in the centre of the ulcer) after debridement, and copious sterile saline is used to flush the eye afterwards. A marked vascular and granulation reaction is common during the healing process.

If debridement, GK and contact lens placement have not resulted in full healing, then the next step is a superficial keratectomy. This is commonly performed in cats earlier in the case management to help reduce the risk of sequestrum formation. Surgical removal of all unhealthy epithelium, plus some anterior stroma, can be performed under general anaesthesia. Referral to a suitably qualified ophthalmologist is advised since magnification (using an operating microscope) and delicate instrumentation are required for accurate, and thus successful, surgery.

The healing process for refractory ulcers can be variable. In many instances the ulcer will heal by epithelial sliding as discussed above, and no vascularization will occur. This provides minimal scarring and is ideal. However, in some cases, a marked vascular reaction can develop, even with overt granulation tissue at the edges of the shrinking ulcer (Figure 21.9). This can be alarming to owners – the eye actually looks far worse as the healing takes place, and they should be warned that this can occur. On the whole it is nothing to worry about, and will subside of its own accord once full epithelialization has occurred. Judicious use of topical NSAIDs can reduce an excessive reaction. Topical steroids can also be used only once full epithelialization has occurred. However, it is often better to let nature take its course at this stage – the patient has often been on drops for several weeks and both patient and the owners will appreciate the cessation of these! If, after 2 weeks, there is still abundant granulation tissue, then further treatment can be considered, but often this is not required.

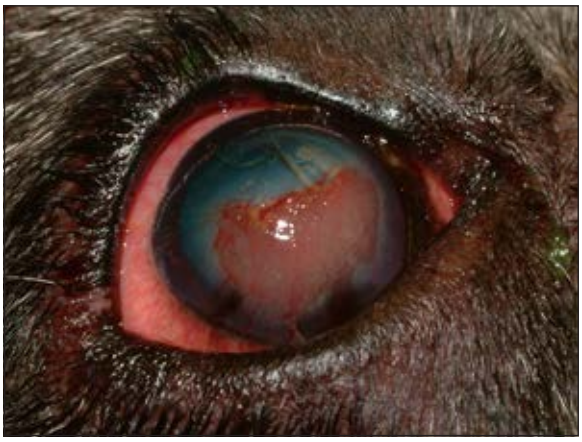


Figure 21.9 Healing recurrent epithelial erosion – note the marked corneal vascularization. A small ulcerated area remains dorsal to this (a mucus thread is also present on the cornea). This eye does not require further debridement and will heal in another week or so.

CLINICAL TIPS

(See also Table 21.1.)

- Debride with a dry cotton bud
- Never dispense topical anaesthetic as a treatment option – it will cause epithelial damage and slow healing
- A superficial recurrent epithelial erosion is the *only* indication for GK – do not perform a GK on any other type of ulcer
- Sedate the patient if necessary – but it still requires topical anaesthesia
- If sedated, do not be tempted to scratch too deeply – just indent the stroma, do not incise it!
- If unsedated, always have your hand holding the GK needle below its face so if the patient jerks its head back inadvertent corneal perforation will not occur

- Remember that antibiotics do not make the ulcer heal any quicker
- Contact lenses should be a correct fit to avoid early loss
- Do not test with fluorescein while a contact lens is in place
- If healing is not satisfactory, refer!

Table 21.1 Management tips for superficial corneal erosions

Good management	Bad management
• Initial client education • Careful debridement • Planned treatment regime • Regular reassessment • Cut-off point for referral	• Failure to recognize condition • Use of topical anaesthetics as treatment • Change of antibiotic without good reason • Incomplete debridement • Failure to offer referral if not responding

PROGNOSIS

The prognosis for refractory superficial corneal ulceration is good. Healing will occur eventually and this can be expedited by careful management. Owners must be aware that the disease can be bilateral, and that recurrences can occur in either eye. However, if there is sufficient scarring to strengthen the cornea, without it being excessive such that vision is affected, this can reduce the risk of further ulcers in the affected area.

CASE EXAMPLE 21.1

Patient details: Boston terrier, 7 years old, male (entire)

PRESENTATION AND HISTORY

The dog had had previous ulcers in both eyes in the past and the owner was attuned to the problem. As soon as the dog started to have slightly increased tearing and blepharospasm she presented the dog to the surgery.

CLINICAL SIGNS

The dog showed mild discomfort with the right eye – increased blink rate and slight lacrimation. Moderate conjunctival hyperaemia was noted. A 4 mm diameter irregular area of superficial ulceration was visible centrally. This was confirmed with fluorescein. The left eye was comfortable but some subcentral corneal scarring and medial corneal pigment were noted. All ocular reflexes were normal and vision was unaffected. Schirmer tear test readings were 18 mm in the left eye and 21 mm in the right eye.

WORK-UP

No work-up was deemed necessary.

TREATMENT AND OUTCOME

- Ulcer debrided under topical anaesthesia, enlarging it to three-quarters of the corneal surface. Topical fusidic acid twice daily and one drop of atropine applied.
- One week later – ulcer slightly smaller but with underrun edges again. Debridement repeated.
- One week later – minimal change. Debridement and grid keratotomy; medical treatment continued.
- Two weeks later – owner very concerned. Eye extremely red and 'angry looking' (Figure 21.10). Marked vascularization peripherally but central area still fluorescein positive. Dog sedated, further debridement and contact lens placed. Initial contact lens fitted was too big (see Figure 21.8) and so a smaller lens was fitted. Lateral tarsorrhaphy suture to reduce lagophthalmos and promote lens retention (Figure 21.11). Chloramphenicol drops four times daily and

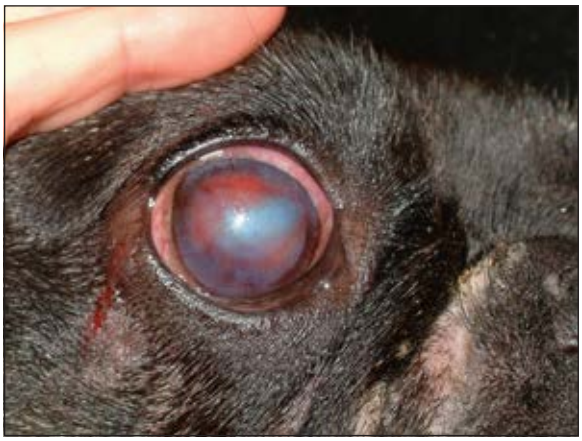


Figure 21.10 Boston terrier with marked superficial vascularization as healing response to superficial corneal erosion.

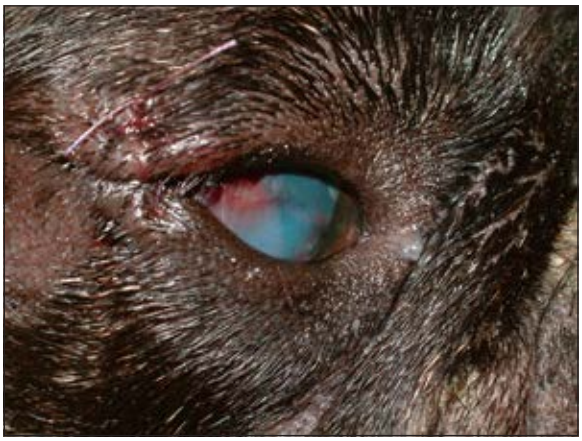


Figure 21.11 Same Boston terrier as Figures 21.8 and 21.10 with a better fit of contact lens and temporary lateral tarsorrhaphy suture. The contact lens should really be under the third eyelid but has moved slightly – this could allow it to be lost sooner than would be ideal.

carprofen 2 mg/kg/day dispensed. Elizabethan collar supplied.

- One week later – lens in place and eye comfortable.
- Two weeks later – lens removed and ulcer fully epithelialized. Chloramphenicol continued for a further 5 days.
- One week later – vascularization subsiding and minimal scarring. Dog and owner both happy.

This case illustrates the protracted nature of superficial corneal erosions – from presentation to healing took 7–8 weeks – but the owner was aware that this was likely to be the case and was keen to avoid general anaesthesia. Earlier intervention with a superficial keratectomy would probably have resulted in faster healing but since the dog was gradually improving the management plan was certainly acceptable and the outcome satisfactory.

22 Deep corneal ulcer

INITIAL PRESENTATION

Pain, discharge, abnormal appearance – red

PRESENTING SIGNS

Both dogs and cats can present with deep corneal ulcers. Usually the owner will notice a painful eye – blepharospasm and conjunctival hyperaemia (the 'red' eye). Some form of ocular discharge is inevitable. This might be purely serous and present as a very wet eye, but is often mucopurulent or purely purulent in nature. The latter is particularly a feature if the owner has failed to bring the pet into the surgery immediately – deep ulcers can be complicated by bacterial infection quite commonly. The pet might have been seen to rub the eye frequently as well.

CASE HISTORY

In most cases the presentation of a deep corneal ulcer is acute in nature. The owner might have been out walking the dog in the woods and it came back with one eye closed and weeping for example. Or the cat was heard to be fighting with a neighbouring tomcat, resulting in a deep corneal scratch. Thus a history of a possible traumatic episode is very common. However, on occasion, there is no such trigger factor in the history and in these cases it is important to check the patient very carefully for other possible causes – an ectopic cilium for example.

CLINICAL EXAMINATION

Obviously a full clinical examination should be performed as well as concentrating on the eyes. This is particularly important if a traumatic injury is suspected. The affected eye is likely to be partially closed with the third eyelid protruding. This can make detailed examination difficult, especially if chemosis is also present (more common in cats – the conjunctival swelling can be so dramatic as to render corneal examination almost impossible). Sedation is sometimes necessary to fully evaluate the cornea, but before rushing to do this a few things can be done to improve your examination:

- If the eye is painful, then a drop of topical anaesthetic can be placed and repeated 2–3 minutes later.
- If any ocular discharge is present and a swab is considered for bacterial culture and sensitivity testing, then this should be taken before the application of topical anaesthesia.
- The third eyelid can still be elevated and present a barrier to corneal examination despite numbing the cornea. By placing the patient at the end of the examination table and making them look down, the third eyelid is retracted back to its normal position. Thus if you bob down and look up to the patient, your examination is likely to be more rewarding.

The ophthalmic examination should include measuring aqueous tear production. If the eye is very painful, or the ulcer is very deep and you are concerned about corneal rupture, then perform the Schirmer tear test on the fellow eye only. Acute keratoconjunctivitis sicca is an under-recognized cause of sudden onset, deep corneal ulceration in the dog (see Case example 16.2). The eye is assessed for vision, and the pupil should be checked – often some miosis is present representing a reflex uveitis and this should be addressed when formulating a treatment plan. A frank uveitis with aqueous flare or even hypopyon can be present in conjunction with deep

corneal ulceration, especially if bacterial infection is present. Careful evaluation of the conjunctival sac, including behind the third eyelid, should be undertaken since foreign bodies can be retained here. Intrinsic factors causing the ulcer, such as ectopic cilia or entropion, should also be looked for (Figure 22.1).

The ulcer is usually obvious as a defect or crater in the cornea (Figure 22.2). It can be assessed for depth both before and after staining with fluorescein. It is important to flush out excess dye with sterile saline to prevent pooling in the defect which can confuse the interpretation of the test. The exposed stroma will retain fluorescein, while Descemet's membrane does not. Thus a deep ulcer which does not stain is likely to suggest a descemetocoele which should be treated as an ocular emergency. If the ulcer is actually healing, then the bottom of the lesion might not take up dye if the epithelium has fully covered all the stroma. The stromal edges are likely to be more gently sloped toward the depth of the lesion rather than being almost vertical when recently ulcerated.

The depth of the ulcer is important when formulating a treatment plan. Also of importance is the presence or absence of blood vessels. These grow from the limbus towards the damaged cornea at the rate of about 1 mm per day. This can be used to determine the length of time the lesion has been present. If the blood vessels are getting close to the ulcer, then medical management might be a feasible option. Some corneal oedema is often present around deep corneal ulcers so a grey ring to the lesion is commonly seen. This should be differentiated from the keratomalacia seen in melting ulcers (see Case example 23.2). In this instance the cornea will be grey and gelatinous in appearance, and when examined from the side the outline of the cornea will be distorted (keratoconus). Blinking and tear film distribution should be evaluated in all cases of deep corneal ulcer. Brachycephalic breeds often have some degree of lagophthalmos, and corneal exposure combined with poor or incomplete blinking can compromise healing.

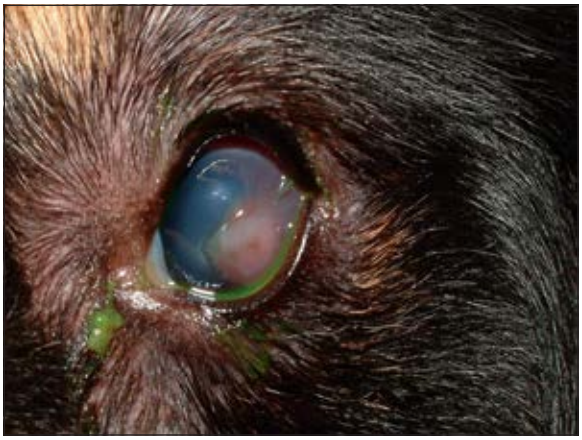


Figure 22.1 Nasty granulating corneal ulcer associated with a large distichium on the upper eyelid. Although it is uncommon for distichia to cause ulceration, if the lash is directed straight onto the cornea and rubbing, then ulcers do sometimes arise.



Figure 22.2 Acute ulceration in an English bulldog with keratoconjunctivitis sicca. Note the two-step crater – the deeper portion looks dark and represents a descemetocoele.

DIFFERENTIAL DIAGNOSES

- Traumatic injury
- Foreign body
- Acute keratoconjunctivitis sicca
- Chronic keratoconjunctivitis sicca
- Ectopic cilium (occasionally distichiasis)
- Entropion
- Complication from superficial ulcer
- Chemical burn (acid or alkali – use Litmus strip to check which)
- Associated with corneal sequestrum in cats

CASE WORK-UP

Only limited case work-up is usually required for deep corneal ulcers since the diagnosis is easily made on ophthalmic examination, and unless multiple trauma has occurred (e.g. dog fights or road traffic accidents), further systemic evaluation is not normally required. As mentioned in the section above on the clinical examination, Schirmer tear test readings should always be taken. This is particularly important if a central descemetocoele is present since acute keratoconjunctivitis sicca is an important, and often forgotten, cause for these (see Chapter 16). If there is a mucopurulent or purulent ocular discharge, a swab should be taken for bacterial culture and sensitivity. Samples for cytology should also be considered. Treatment plans can be drawn up once the specific cause of the ulcer is identified.

NURSING ASPECTS

The treatment for corneal ulceration can be medical, surgical or a combination of both. Nursing care will be required. Frequent topical medication is usually necessary, using drops, ointments or sometimes both. Drops should always be applied before ointments if both are employed, and a minimum of 5 minutes (but ideally 10–15) should be left between different topical applications. Nurses should gently bathe any discharge from the eye using soft woven gauze swabs (not cotton wool which can shed fibres) and sterile saline. Patients should be evaluated for signs of discomfort and the veterinary surgeon in charge of the case should be informed of any change in patient comfort such that appropriate analgesia can be prescribed. Self-trauma and rubbing (either with the paws or against the bedding for example) must be avoided. By making the patient comfortable this can usually be prevented and Elizabethan collars are not always required routinely – providing proper monitoring is undertaken.

If surgical treatment is planned, the nurse will be involved in preparing the patient, including anaesthesia and surgical preparation of the eye along with liaison with the surgeon regarding the instruments and other surgical equipment necessary for the procedure. Postoperative nursing instructions, both for the time the patient is hospitalized and afterwards for the owners, should be considered.

EPIDEMIOLOGY

The cause for deep corneal ulcers is usually evident from the clinical examination. Traumatic incidents are common but progression from a superficial ulcer is also frequent – especially if bacterial contamination has occurred and if the owner has delayed seeking veterinary attention. Ulcers of this sort tend to occur on an individual basis – pathogens are usually opportunistic and contaminate the injury rather than being primary corneal pathogens with infectious spread between animals. Thus Gram-positive bacteria are usually present initially but Gram-negative ones such as *Pseudomonas* can become involved. Fungal infectious are rare in the UK but should be considered if the ulcer is not responding to conventional therapy.

CLINICAL TIPS

- Thoroughly flush out fluorescein with sterile saline to prevent false positives
- Make the animal look down to retract the third eyelid and facilitate corneal examination
- Consider topical anaesthesia if blepharospasm excessive
- Sedation occasionally necessary for thorough examination
- Remember to check behind the nictitans membrane for foreign bodies
- Do not forget Schirmer tear test (both eyes if possible)

TREATMENT OPTIONS – MEDICAL

If the ulcer is deep, but not down to Descemet's membrane, and there is evidence of corneal vascularization, then medical management is an option (Figure 22.3). However, if a descemetocoele is present, then the eye is in danger of imminent rupture and surgery must be recommended. Similarly, if the patient is suffering from acute keratoconjunctivitis sicca as the underlying cause for the ulcer, then surgical procedures, in addition to addressing the lack of tear production, are usually necessary.

For medical management a combination of both topical and systemic medication is normally necessary (Table 22.1). The frequent application of topical antibiotics is required. These should be chosen according to the

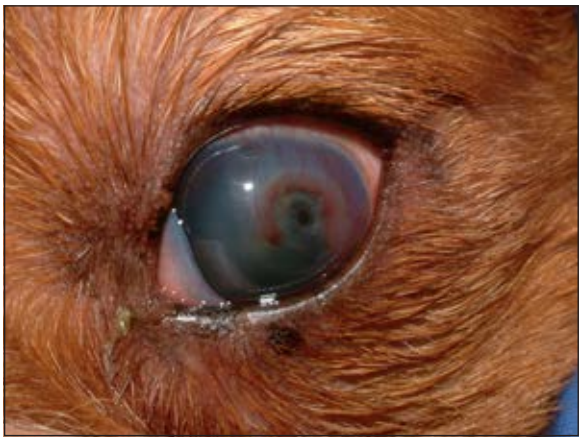


Figure 22.3 Deep corneal ulcer (descemetocoele) in a 4-year-old terrier. Although this is a deep ulcer, there is good corneal vascularization and this can be treated medically to achieve healing.

Table 22.1 Medical management of uncomplicated deep corneal ulcers

Agent	Frequency
Topical antibiotic, e.g. chloramphenicol, gentamicin (or chosen according to culture and sensitivity testing)	Four to eight times daily
Topical atropine	Twice daily (or less – to effect)
Systemic NSAID	Once or twice daily
Topical NSAID	Two to three times daily – only necessary if overt uveitis is present
Autologous serum	Hourly if corneal melting occurs – consider referral for surgery

likely pathogens involved. The conjunctival sac normally contains mainly Gram-positive bacteria (such as staphylococci and streptococci) and these tend to be the most frequent opportunistic pathogens. Antibiotics such as fusidic acid and chloramphenicol are good choices, and are licensed for use in dogs and cats. Gentamicin can be used, particularly if the cornea has any grey, melting appearance, which could suggest a *Pseudomonas* infection. Swabs should always be considered, and if the ulcer has not responded favourably after a few days, then culture and sensitivity testing is a very good idea, particularly if one is considering changing the antibiotics. The frequency of application will depend on the degree of infection suspected and the formulation of the drug. Sometimes as often as hourly is required. Fortified antibiotic solutions are occasionally used – for example, an intravenous formulation of a cephalosporin can be placed into artificial tears and used at a much higher concentration than is available commercially. Potent topical antibiotics such as ciprofloxacin and enrofloxacin should not be used as first-line antibiotics but are useful in infected cases while awaiting culture and sensitivity results.

In addition to topical antibiotics, topical atropine is usually advised to relieve the ciliary spasm associated with reflex uveitis and to help stabilize the blood–aqueous barrier, thus reducing further uveitis. It should not be used in keratoconjunctivitis sicca (since it lowers tear production even further – tropicamide can be used instead although more frequent application will be required). The atropine should be used to effect, i.e. to keep the pupil moderately dilated. Initially this might be twice daily, reducing to once, or even every other day. Remember that the drops drain down the nasolacrimal duct and taste bitter so might make patients dribble and salivate excessively – especially cats where ointment formulations are preferred.

Topical non-steroidal anti-inflammatory agents are sometimes used for deep ulcers. They should only be chosen if an active uveitis is present (but not with frank hyphaema). If the pupil dilates readily with atropine then they are probably not required. They will slow corneal vascularization which can lead to a delay in healing. The use of systemic NSAIDS is probably preferred in many instances.

Autologous serum is used topically for deep corneal ulcers which show a tendency to melt. It contains anti-collagenases which reduce the keratomalacia that can result from the excessive collagenase activity of neutrophils, bacteria or degenerating keratocytes. Blood is collected from the patient, spun down, and the serum dropped into the eye on a 1–2 hourly basis. The serum should be handled in an aseptic manner and kept in the fridge. Fresh supplies should be collected every 24–48 hours. If the ulcer is so severe as to warrant its use, it is probably wise to hospitalize the patient – progression rather than improvement might require emergency surgery to prevent corneal rupture. Serum is not beneficial in uncomplicated deep ulcers.

Systemic medication takes the form of NSAIDs and sometimes a broad spectrum antibiotic such as a cepha-

losporin or amoxicillin/clavulanic acid (particularly if frequent topical medication is not feasible although systemic medication is less effective).

Improvement in the ulcer will manifest as a shallowing of the crater – the edges will become more sloping, rather than vertical, as the epithelium slides into the defect. The blood vessels will grow towards the edge of the lesion, and once they reach it the defect will epithelialize quickly. At this stage the frequency of topical application can be reduced. Once fluorescein is not retained the ulcer can be said to have healed, but a stromal facet will remain and it can take weeks for this to infill. As such, the cornea will be weakened and vulnerable during this time. Topical antibiotics and atropine can be discontinued, together with systemic medication. The use of topical lubricants such as carbomer gel should be considered to help tear film dynamics and perhaps provide a little protection to the weakened area. The use of topical NSAIDs is helpful if vascularization is excessive, but topical steroids should probably be avoided if any corneal thinning remains. Normally the vascularization will reduce naturally in 2–4 weeks following epithelialization.

TREATMENT OPTIONS – SURGICAL

If the ulcer is not improving after 48 hours of intensive medical therapy, or if a descemetocoele is present, then surgery is advised. Contact lenses and third eyelid flaps are not usually sufficient to provide adequate corneal support and should not be used if active infection is present. Although corneal glue can be employed, its use is probably best left to specialists. Thus, in practice, the use of conjunctival grafts is probably the best choice. It is important that the surgeon has been taught how to perform these, and has adequate magnification and the correct instrumentation to do so. If not, then referral is recommended.

In general a pedicle conjunctival graft is the most versatile type to use (see Figure 22.4). Details of exactly how to perform this surgery are beyond the scope of this book but the basic technique is as follows:

- After gentle debridement of the ulcer the harvested conjunctiva is rotated into the defect and sutured to the walls of the ulcer. An absorbable suture material such as 8/0 polyglactin 910 (Vicryl, Ethicon) is normally chosen.
- The graft should be thin, and rotated perpendicular to the eyelids so that blinking is unlikely to drag on it and result in early dehiscence.



Figure 22.4 Conjunctival pedicle graft in a Cavalier King Charles spaniel with acute deep corneal ulceration following a scratch by a cat.

- Medical treatment is continued and the 'stalk' of the graft is normally trimmed 3–6 weeks later, leaving a small pale button of conjunctiva which gradually scars down such that vision is usually reasonable.
- It should be remembered that if the ulcer is melting, this keratomalacia can continue if the cornea is not properly debrided prior to graft placement, such that the graft also melts out of the unstable cornea (see Case example 23.2 and Figure 23.3).
- This situation can be disastrous for the eye and enucleation may prove the only treatment option at this stage.

Further complications of ulcers are discussed in the next case.

Corneoscleral transposition can be used instead of conjunctival grafts but is best left to specialists since magnification with the use of an operating microscope is essential for accurate surgery.

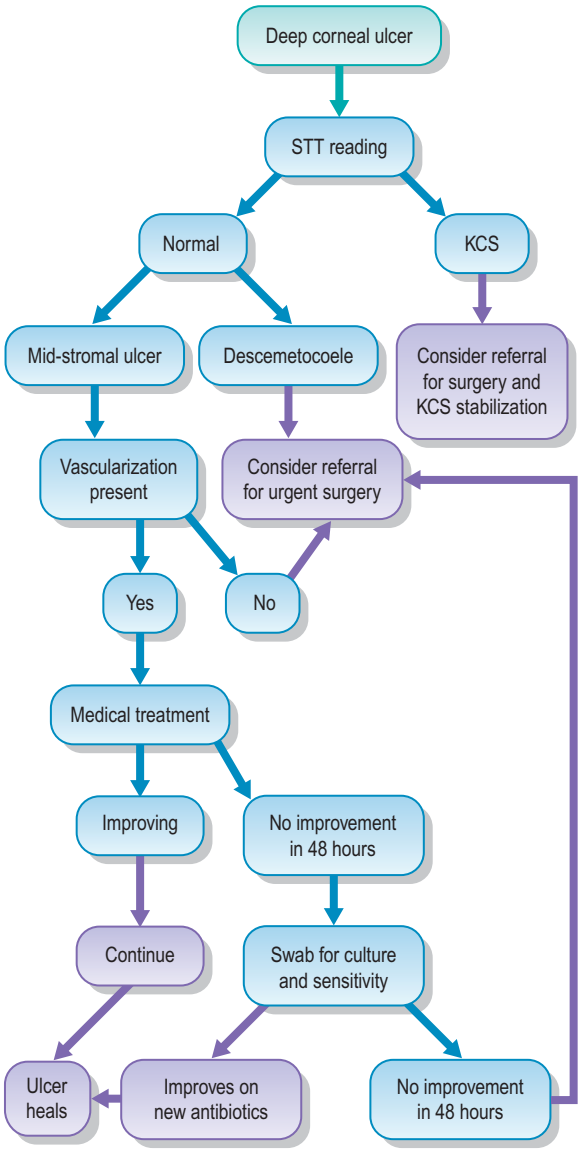


Figure 22.5 Decision-making tree – deep corneal ulcer.

PROGNOSIS

The prognosis for deep corneal ulcers must always be somewhat guarded. The risk of corneal rupture is present and this can sometimes lead to iris prolapse. There is the risk of bacterial spread into the anterior chamber, resulting in a globe-threatening panophthalmitis. However, with careful management, many cases do very well. Some corneal scarring is inevitable – with both medical and surgical management – but a comfortable, functioning eye can often be the end result. Referral should always be considered for deep ulcers.

CASE EXAMPLE 22.1

Patient details: Staffordshire bull terrier, 3 years old, male

PRESENTATION AND HISTORY

The dog was presented with a watery right eye which was held closed. The owners had noticed this a week previously, but had failed to bring the dog to the surgery. Apparently he had been out on a walk and came back holding the eye closed. They assumed that he had banged it but thought that it would get better. No treatment had been given and the dog was reportedly well in himself.

CLINICAL SIGNS

The right eye was unremarkable. The left showed marked blepharospasm and increased lacrimation – the discharge was purely serous. Conjunctival hyperaemia was present, together with marked corneal vascularization extending several millimetres from the limbus towards the central cornea where a deep linear ulcer was noted (Figure 22.6). Slight anisocoria was present (left pupil smaller) but there was no overt uveitis. Schirmer tear test readings were 25 mm in the left eye and 18 mm in the right (in 1 minute). Fluorescein dye stained the walls and base of the ulcer.



Figure 22.6 Three-year-old Staffordshire bull terrier with a deep linear corneal ulcer of the left eye. Marked corneal vascularization shows that this has been present for several days.

WORK-UP

It was explained to the owners that this was a very deep ulcer (probably caused by a foreign body which had fallen out, or a scratch from a bush or similar). Referral for a conjunctival graft was recommended but the owners did not want to pursue this option. No further work-up was undertaken.

TREATMENT

The dog was sedated and a bandage contact lens applied (Figure 22.7). A temporary lateral tarsorrhaphy suture was placed to promote lens retention. Chloramphenicol drops four times daily and once daily atropine drops were dispensed, together with 5 days of carprofen (4 mg/kg/day).

OUTCOME

The contact lens was well tolerated and remained in situ. The ulcer could be seen to be healing beneath it. Three weeks after inserting the lens it was removed, along with the tarsorrhaphy suture (which had loosened significantly). The ulcer had fully epithelialized and only a minimal stromal facet remained. The vascularization had



Figure 22.7 The same patient as Figure 22.6 with a bandage contact lens and temporary tarsorrhaphy suture.

reduced. Treatment was stopped. Two weeks later the eye looked fine, with normal vision and minimal scarring.

This case shows that if deep ulcers are sufficiently small and do not become infected, minimal intervention can result in good healing. The dog was young and healthy with no systemic disease which could compromise healing (hypothyroidism for example). Thankfully the delay in presenting the dog did not result in opportunistic infection and the presence of the corneal vascularization suggested that surgery might not be required since healing had already started.

23 Complicated corneal ulcer

INITIAL PRESENTATION

Discharge, pain, abnormal appearance – red

The previous two case descriptions illustrate very common presentations in general practice – the superficial non-healing ulcer and the deep ulcer. The treatment plan outlines ways to treat these conditions but assumes that everything goes well. In this chapter we will discuss the ulcer that does not heal according to plan.

PRESENTING SIGNS

It is likely that the animal has already been presented for treatment, although some cases might have been present for a while before the owner brings the pet along to the surgery – the cat might have been missing for a few days for example. The animal may have been taken to another surgery, the owners could have failed to return when asked, or – in spite of everything being followed correctly – things could have taken a turn for the worse. As such, the presentation will be of a painful eye. The animal is likely to be holding the affected eye closed and it is likely that significant ocular discharge will be noted – most probably mucopurulent in nature.

CASE HISTORY

It is usual for the history to include the suggestion of trauma – the cat comes back early one morning with one eye closed for example, although this is not true in 100% of cases. On initial presentation a corneal ulcer will have been diagnosed which was treated symptomatically with a broad spectrum topical antibiotic. A few days' improvement may be reported before the eye deteriorated – it became more painful, changed in appearance to the owner or the discharge altered and became more copious. The patient may have had surgery already – for example, a third eyelid flap which on removal revealed a nasty corneal lesion.

CLINICAL EXAMINATION

The ulcer should be obvious on clinical examination. However, the conjunctiva is likely to be hyperaemic, the nictitans membrane elevated and chemosis might also be present which could make visualization of the cornea difficult (Figure 23.1). Sometimes the ocular discharge will be extensive such that the eye is 'glued' shut. Gently bathing this away (and taking swabs for culture and sensitivity at the same time – but see below) will assist in examination. Overt ulceration might not initially be visible – there is likely to be significant vascularization (even granulation tissue) on the cornea, along with oedema, both of which obscure the actual ulcer. If areas of cornea appear gelatinous, and creamy or grey in colour, this could suggest keratomalacia or corneal melting (Figure 23.2).

To make it easier to examine a painful cornea, the patient should be placed at the end of the consulting table and made to look down, while you look up from below. This usually makes it possible to view most of the cornea as the nictitans membrane moves into the ventromedial fornix. The use of topical anaesthesia, and even sedation or general anaesthesia, might be necessary to fully evaluate the eye in some patients.

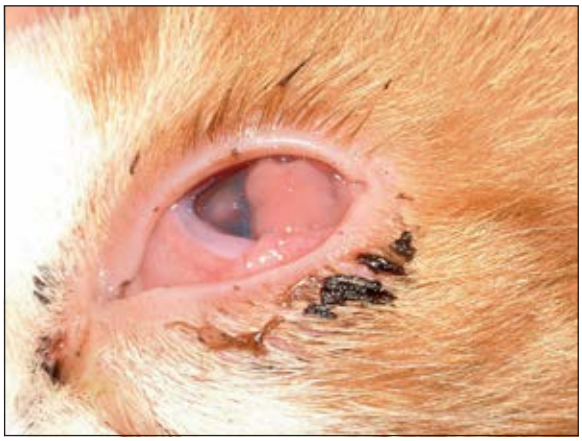


Figure 23.1 Nasty corneal ulcer in a cat – notice the abundant granulation tissue, more medial ulcer and vascularization, chemosis, nictitans elevation and copious discharge.

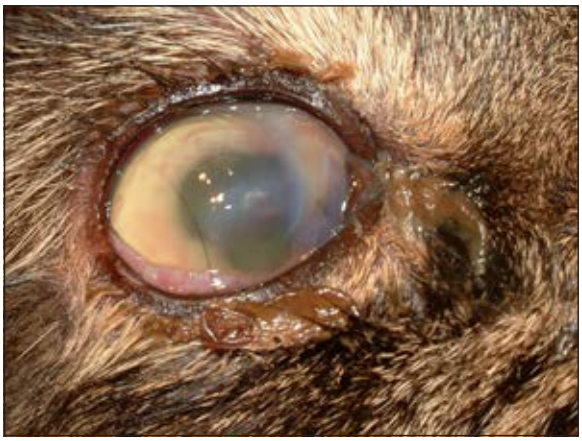


Figure 23.2 Melting ulcer in a cat – note the overall gelatinous appearance to the cornea and general opacity. The darker central area is the site of the original ulcer. Atropine has partially dilated the pupil, which looks distorted due to the abnormal corneal curvature. A copious discharge is also present and a hair is adhered to the cornea.

DIFFERENTIAL DIAGNOSES

- Progression from simple ulcer
- Infected corneal ulcer
- Melting corneal ulcer
- Undetected foreign body
- Undetected intrinsic factors – distichia, entropion, ectopic cilium
- Undetected keratoconjunctivitis sicca (check Schirmer tear test reading in fellow eye)
- Progression from intraocular disease (e.g. following topical steroids to treat uveitis)

CASE WORK-UP

It is essential that swabs are taken for culture and sensitivity testing from all potentially infected corneal ulcers. These can be taken before topical anaesthesia. Samples can be collected from the ocular discharge but more accurate isolation will be obtained from the ulcer edge itself – a bacterial swab moistened with sterile saline can be gently rolled onto the edge of the ulcer. This can also be used to perform Gram staining (by then rolling the swab onto a microscope slide and staining it appropriately). Cytology should also be considered – samples collected by swabs do not yield high numbers of cells and so the use of a cytobrush or Kimura spatula (or the blunt end of a scalpel blade) is preferred. Topical anaesthesia is necessary before such sampling. Samples should be spread onto a microscope slide and air dried. Staining with Diff-Quick or similar stains can be useful to look for the presence of bacteria, the type and number of cells (usually a combination of neutrophils and degenerate keratocytes in a bacterial infection) and the presence of inclusion bodies (for example, with *Chlamydophila*). The presence of fungal elements should be considered – although relatively rare in the UK, these can have devastating consequences if not identified and treated accordingly.

The eye should be fully examined for the evidence of anything that could be contributing to the poor healing, or exacerbating the ulcer. Retained foreign bodies and abnormal cilia are probably the most common culprits after bacterial infection.

NURSING ASPECTS

The specific nursing for complicated corneal ulcers is similar to those for deep ulcers (see Chapter 22). Thus regular bathing of any discharge and the frequent application of topical medication are necessary. It is also important to alert the attending veterinary surgeon to any change in the condition of the patient, both of the eye itself and of the animal's general demeanour – a sudden deterioration could require urgent surgical intervention.

EPIDEMIOLOGY

The complication of bacterial infection is the most common cause of ulcer progression. The conjunctival sac normally carries a mixed population of mainly Gram-positive bacteria (e.g. staphylococci, streptococci and corynebacteria). These can act as opportunist pathogens once the corneal epithelium is breached. However, infection with more pathogenic organisms, mainly Gram-negative bacteria (e.g. *Escherichia coli*, *Pseudomonas*), is likely in more severe ulcers. Mycotic infections do occur occasionally in the UK but are common in some parts of the world. Cytology is important for their diagnosis initially, followed by culture to identify the exact fungus. *Candida albicans* and *Aspergillus* spp. are reportedly the most frequent isolates in the UK.

Not all complicated ulcers are infected. Sometimes degenerating keratocytes or neutrophils can release collagenases which cause further corneal damage. Clearly if other pathological processes are occurring in the eye simultaneously – uveitis or glaucoma for example – the risk of complications with an ulcer is higher.

CLINICAL TIPS

- Perform a thorough ocular examination each time the patient is examined – the situation can change very rapidly
- If the patient is hospitalized it should be carefully examined (i.e. in the consulting room in the dark) at least twice daily
- The treatment plan should be altered depending on the case progression
- Repeating the swabs for culture/sensitivity or cytology is useful, especially if there is a sudden change in the appearance of the eye or the discharge
- Consider referral for melting ulcers
- If the patient is taken to surgery, samples should be taken from the surgical field

TREATMENT OPTIONS – MANAGEMENT

The owner should be fully apprised of the severity of the condition from the outset. A treatment plan should be drawn up and followed. Obviously this must be tailored to the individual and will include factors such as patient compliance, owner finances and commitment, as well as the condition of the eye itself.

The first decision to be made should be: Is the eye salvageable? In some cases the cornea will be so severely compromised that the ulcer has ruptured, the iris has prolapsed and a panophthalmitis has resulted (Figure 23.3). If this is the case, then enucleation might be the best option for the patient. However, this is an irreversible decision and so one should be convinced that there is no alternative. If the eye has the potential for vision, enucleation is not advised. A positive dazzle response, together with a consensual pupillary light reflex (i.e. constriction of the fellow eye when a bright light is shone into the affected eye), are encouraging signs. If neither is present, then removing the eye might be indicated. If you are not sure whether the eye can be saved, referral should be considered. The welfare of the patient is paramount.

In order to achieve healing some patients might require very frequent topical medication (every 1–2 hours) over several days, followed by ongoing medication for several weeks, often in conjunction with one or more surgical procedures. If the patient is very nervous, or aggressive, then such intensive medication might be difficult for owners and professionals alike, as well as being traumatic for the patient.

If the final outcome is a very scarred cornea, such that vision is severely limited in the affected eye, one needs to evaluate the case very carefully before deciding



Figure 23.3 Six-month-old Boston terrier. A deep corneal ulcer has received a conjunctival graft. Unfortunately, this did not hold and the ulcer continued to deepen, resulting in keratomalacia and iris prolapse as the cornea ruptured. A profuse growth of *Pseudomonas*, resistant to gentamicin (which the dog had been treated with), was isolated but by this time enucleation was the only option.

whether to try to save the eye. Having said that, it is amazing how well some cases can do (see Case example 23.1 below). Thus it is often sensible to attempt treatment initially, ensuring that the owner is fully informed of the situation, and is aware that the eye might require removal if an improvement is not seen within a few days.

TREATMENT OPTIONS – MEDICAL

The medical management of infected ulcers is intensive (see Table 23.1). It will revolve around appropriate antimicrobial drugs. Gram staining is important to assist the choice of medication while awaiting the results of culture and sensitivity testing. Since Gram-negative pathogens are commonly isolated, a suitable choice of topical antibiotic could be gentamicin or tobramycin. Both have good activity against agents such as *Pseudomonas* although resistant strains are encountered. If Gram-positive organisms are identified, then a fluoroquinolone antibiotic such as ciprofloxacin or ofloxacin might be more appropriate. Both gentamicin and ciprofloxacin can be used alternately pending sensitivity results. Initial topical application should be every 2–3 hours. Subconjunctival injections of gentamicin or a cephalosporin can also be considered.

In addition to antibiotic cover, topical atropine is usually recommended to reduce the ciliary spasm encountered with severe corneal disease. It will also help to stabilize the blood–aqueous barrier and reduce the severity of a concomitant uveitis. Atropine should be used to effect, i.e. frequently enough to keep the pupil dilated. This is usually two or three times daily. Atropine should not be used in cases of keratoconjunctivitis sicca since it lowers tear production even further. Cats do not tolerate the drops well, and so an ointment formulation might be more appropriate.

Medical management can also consider the use of autogenous serum. This is particularly useful in the case of keratomalacia, i.e. melting ulcers. Here a combination of collagenase activity – both exogenous (from the bacteria) and endogenous (from degenerating keratocytes and neutrophils) – contributes to liquefaction of the corneal cells. Untreated this will lead to complete corneal dissolution and loss of the eye. Hourly serum can arrest such melting by providing protease inhibitors to the cornea. Blood is taken from the patient and spun down. The serum is placed in a sterile container and kept in the fridge. It should not be used for more than 48 hours and is not suitable for home medication (owners are unlikely to follow a strict aseptic technique and serum is the perfect culture medium for bacteria!). Alternatives to autogenous serum include acetylcysteine and EDTA.

Topical NSAIDs are sometimes used if a severe uveitis is also present. However, systemic agents are normally given, and often will be sufficient. A broad spectrum antibiotic such as a cephalosporin should also be administered.

If the ulcer has not stabilized after 24–48 hours then surgical intervention should be considered. It might be necessary to refer the patient at this stage. Encouraging signs will be a reasonable vascular reaction approaching the ulcer edge, a less gelatinous appearance to the cornea (especially the area immediately adjacent to the actual ulcer) and a shallowing of the ulcer itself. Less discharge should be present and the animal should be more comfortable (Table 23.2). However, if the ulcer seems to be getting deeper despite the intensive treatment regimen, or the cornea is becoming more jelly-like, then surgery is indicated.

Figures 23.4 and 23.5 show a *Pseudomonas*-positive melting ulcer in a domestic short haired cat on presentation and 4 weeks later after 2 weeks of hospitalization and intensive medication, followed by a fairly intensive

Table 23.1 Medial management of complicated ulcers

Agent	Frequency
Topical antibiotic chosen on Gram stain: • Gram negative – gentamicin • Gram positive – ciprofloxacin or ofloxacin	Every 1–2 hours (over 24-hour clock – do not stop at night!)
Topical atropine	Two to four times daily (or less – to effect) (not if keratoconjunctivitis sicca)
Autogenous serum	Hourly if corneal melting occurs – consider referral for surgery
Topical NSAID, e.g. ketorolac	Up to four times daily – necessary if overt uveitis is present
Systemic antibiotic, e.g. cefalexin	Every 12 hours
Systemic analgesia – opiates plus NSAID (normally both required initially)	Every 8–12 hours depending on agent chosen

Table 23.2 Good and bad signs in ulcer appearance

Good signs	Bad signs
• Cornea less gelatinous in appearance • Ulcer edges less irregular • Corneal vascularization • Pupil well dilated • Less ocular discharge • Patient more comfortable	• Continued keratomalacia • Ulcer enlarging • No corneal vascularization • Pupil miotic • Copious discharge – especially if suddenly watery (could be aqueous) • Patient in significant pain • Hyphaema • Hypopyon • Corneal rupture and iris prolapse

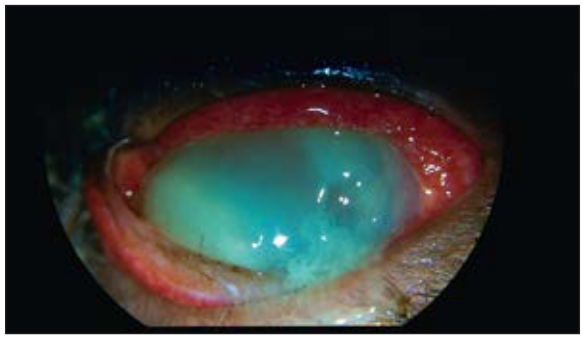


Figure 23.4 Domestic short haired cat with infected (*Pseudomonas*) melting ulcer in the left eye.

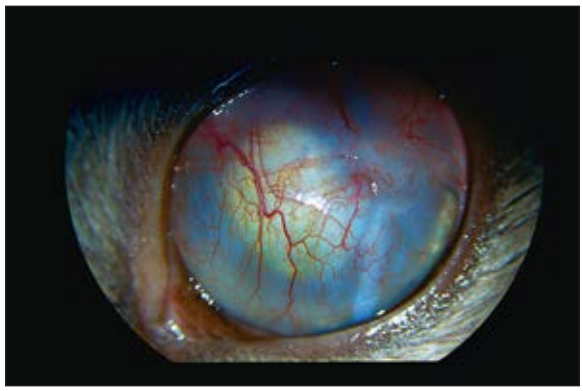


Figure 23.5 Same cat as in Figure 23.4 a month later after intensive medical treatment.

home regimen. Surgery was not required and the eye was salvaged with reasonable vision. However, the treatment was very intensive which was only possible due to the cat's good temperament and the owner's determination not to give up!

TREATMENT OPTIONS – SURGICAL

The surgical management of complicated ulcers rests with the removal of infected or unstable corneal tissue followed by support of the weakened structure. Thus keratectomy with grafting (usually conjunctival rather than corneoscleral transposition) is required, and suitable experience and equipment are necessary for this. Referral might be appropriate. The keratectomy will result in small pieces of cornea which can be submitted for histopathology and culture. This is important even if cytology and bacterial culture have already been performed – there could have been a change in pathology at the ulcer site (which could explain why medical treatment was not successful) or the isolates might have been from the conjunctival fornix and not the ulcer at all.

Conjunctival grafts will support the weakened corneal stroma while bringing a blood supply directly to the damaged tissue. They are preferred over corneoscleral transposition if keratomalacia is present since the corneal melting can continue into the newly transplanted tissue and result in surgical failure. Grafts using porcine submucosa material (BIOSIS) also run the risk of rejection. Sutures are usually 8/0 polyglactin 910 (Vicryl, Ethicon) but it must be remembered that as multifilament material the risk of infection tracking into the suture sites is slightly greater than if a monofilament material is chosen.

Postoperative medication will be similar as for the medical treatment options above. Frequent topical medication will be required still, as well as atropine and systemic antibiotics and NSAIDs. Topical serum should not be necessary as the graft should be providing nutrients including anticollagenases.

Once the cornea is fully healed, usually in 3–4 weeks, the conjunctival graft can be trimmed. This will leave a small area adhered to the cornea while the surrounding tissue should clear well, such that good vision is restored.

The use of third eyelid flaps is not recommended for infected or progressing corneal ulcers. They do not provide any direct support to the ulcerated cornea, they reduce the penetration of topical medication and totally obscure the cornea such that frequent monitoring of the underlying lesion is not possible. It is not uncommon for ulcers to progress under the flap: it is warm and moist under a flap – ideal conditions for continued bacterial infection – with the added disadvantage that discharges are not removed but left to accumulate between the cornea and third eyelid, thus contributing to further

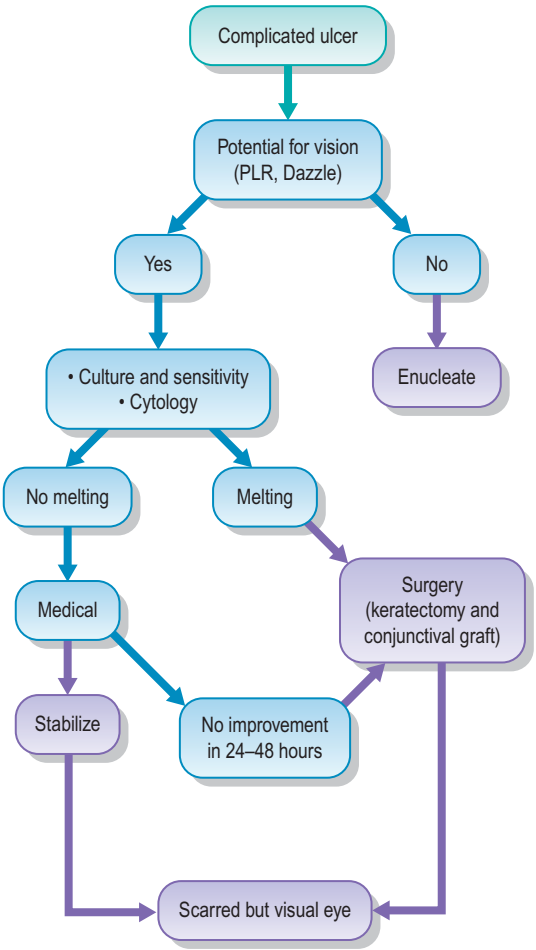


Figure 23.6 Decision-making tree – complicated corneal ulcer.

corneal damage. Undoing a third eyelid flap to discover that the ulcer has ruptured occurs on occasion such that enucleation is then the only treatment option.

PROGNOSIS

Owners should always be given a guarded prognosis for complicated ulcers. However, if an underlying reason for the complication – such as a previously undetected ectopic cilium or fragment of grass seed – is identified, the prognosis obviously improves once this has been removed. If the ulcer is stabilized and responds to treatment, the owners should be aware of the realistic expected outcome. This is a scarred, but visual eye. They should not expect the eye to return to complete normalcy.

CASE EXAMPLE 23.1

Patient details: Domestic short haired cat, 10 years old, male (neutered)

PRESENTATION AND HISTORY

The cat was presented as a second opinion because the owners were very concerned about the lack of improvement in the cat's eye. It had been diagnosed with a corneal ulcer a month previously, after having been absent from home for 3 days. It was initially treated with chloramphenicol drops three times daily but a week later the eye was still red, sore and discharging and a third eyelid flap was placed. On removal 2 weeks later the eye looked dreadful and enucleation was advised. The owners declined and sought specialist advice.

CLINICAL SIGNS

The right eye was totally red on presentation and blind with no menace response although a dazzle reflex was present but no consensual light reflex in the left when light was shone in the right. Closer examination (Figure 23.7) revealed severe central granulation tissue with peripheral corneal ulceration and melting dorsomedially. The bulbar surface of the nictitans membrane was partially adhered to the granulated cornea. Intraocular examination was impossible.

WORK-UP

Samples were taken for Gram staining (revealing mostly Gram-positive organisms) and culture and sensitivity testing (mixed staphylococci and streptococci with good sensitivity). The owner was adamant that the eye should not be removed. Ultrasonography was performed under sedation and the anterior chamber appeared formed with no corneal rupture. Some debris was present in the anterior chamber (blood or hypopyon). Since the eye appeared viable surgery was scheduled.

TREATMENT

Under general anaesthesia the adhesions between nictitans membrane and cornea were broken down. The cornea was debrided and an island conjunctival graft sutured into place covering the ulcerated cornea. The graft had been harvested from the fellow eye since the right eye contained no normal conjunctiva. Topical ciprofloxacin every 2 hours and three times daily atropine ointment were dispensed, along with systemic cefalexin and 48 hours of buprenorphine injections.

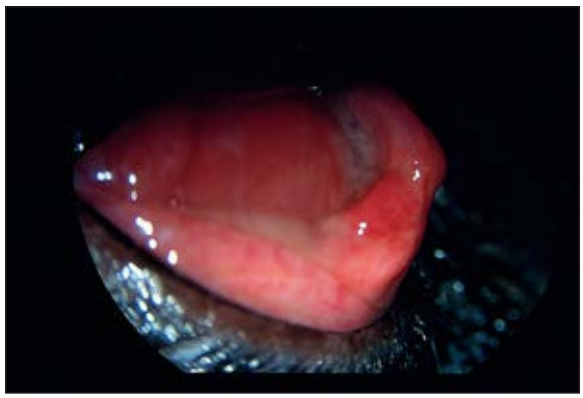


Figure 23.7 Right eye showing granulation reaction, peripheral corneal oedema and ulcer (medially), and severe conjunctival hyperaemia.

OUTCOME

A week later the graft was vascularized and viable. Ciprofloxacin was reduced to four times daily and atropine once daily.

Three weeks later the graft was still healthy. No ulceration was present and treatment was changed to neomycin, polymyxin B and dexamethasone drops (Maxitrol, Alcon) four times daily (Figure 23.8).

Ten weeks after surgery the eye was comfortable and visual. The corneal vascularization continued to clear but the cat needed to stay on once-daily steroid drops to keep the inflammatory reaction at bay (Figure 23.9).

This case illustrates the amazing healing capacity some cats possess! Enucleation appeared to be a sensible option at initial presentation but with complicated surgery and intensive after-care, together with very dedicated owners, the cat not only retained its eye but also regained good vision.

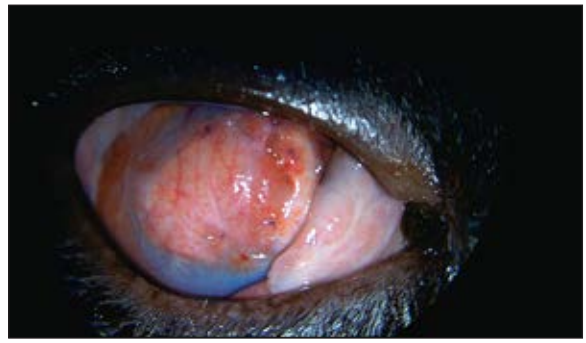


Figure 23.8 Same cat as in Figure 23.7, 3 weeks following surgery.



Figure 23.9 Same cat as in Figure 23.7, 10 weeks following surgery. The eye is visual.

CASE EXAMPLE 23.2

Patient details: Tibetan terrier, 13 years old, female (neutered)

PRESENTATION AND HISTORY

The patient suffered from diabetes mellitus and developed sudden onset cataracts secondary to this. She did not cope with the blindness and underwent cataract surgery on the right eye. Initially this did very well, and vision was good. However, 6 weeks after surgery the owner noticed that the eye became cloudy and started to discharge. She had an appointment with the ophthalmologist the following week so continued with the eye drops that the dog was on at the time (prednisolone acetate and tropicamide both twice daily). The eye continued to deteriorate.

CLINICAL SIGNS

On presentation vision was very limited. The conjunctiva was inflamed and the eye was painful. The corneal wound was grey with an irregular cornea while a large area of corneal melting was present ventrally, resulting in keratoconus (Figure 23.10).

WORK-UP

Swabs were taken from the ocular discharge and the gelatinous part of the cornea (which appeared very unstable). No bacterial growth was isolated, nor were any bacteria observed on an initial Gram stain. Blood glucose samples were taken regularly to monitor diabetic control.

TREATMENT AND OUTCOME

Intensive medical treatment was started – ciprofloxacin every 2 hours, hourly autogenous serum, atropine drops and ketorolac drops every 6 hours, plus systemic methadone, carprofen and cefalexin.

Twenty-four hours later the cornea appeared to be stabilizing – the ventrally melting cornea was more 'firm' and no further keratomalacia was noted. Medication was continued. Five days later the corneal 'melt' came off as the eye was bathed. Ulceration was still present but this seemed superficial. Marked hypopyon was noted (Figure 23.11). The eye was blind. Ultrasonography under sedation revealed a total bullous retinal detachment and significant vitreal debris. The owner was warned that vision would not return and was given the option of continued intensive medical management to salvage the eye, or enucleation. Since the dog was still in discomfort, and did not tolerate hospitalization well, enucleation was performed. Histopathology was unfortunately not performed.



Figure 23.10 Thirteen-year-old diabetic Tibetan terrier with melting corneal ulcer 6 weeks following cataract surgery.



Figure 23.11 Same patient as in Figure 23.10, 5 days later. Although the cornea is stabilizing, a severe uveitis is present with marked hypopyon.

This case illustrates how although the cornea might be starting to improve other serious consequences can occur in the eye. The owner should have presented the dog immediately on noticing a change in the eye (she did have written instructions to this effect!) but instead continued using topical steroids. The cornea was probably ulcerated for several days (ulceration following cataract surgery is always a risk, but occurs more commonly in diabetic patients). The keratomalacia will have been enhanced by the steroids. The previous intraocular surgery and severe corneal disease both resulted in destabilization of the blood–aqueous barrier and a severe panuveitis developed, resulting in retinal detachment. Unfortunately, enucleation was the only sensible option left.

24 Puppy scratched by cat

INITIAL PRESENTATION

Pain, discharge

PRESENTING SIGNS

The young puppy presented due to an encounter with the local cat – sometimes the family cat, sometimes belonging to a neighbour – is a familiar problem in general practice and, as such, it deserves some detailed consideration. The presenting signs are usually textbook classic. The puppy suddenly yelps and holds one eye tightly closed. Meanwhile the cat saunters away unconcerned! The owners present the patient with minimal delay.

CASE HISTORY

As with the case presentation the history is normally very straightforward. The puppy is normally very young – less than 5 months and often only 12–16 weeks. Frequently it has only been in the house for a few days. The puppy was playing and suddenly yelped or screamed and came running away from a cat. It might have encountered the neighbour's cat in the garden, or cornered the family cat in the kitchen, but the outcome is the same. The cat lashed out at the boisterous puppy and caught it in the eye.

CLINICAL EXAMINATION

The most difficult part of the clinical examination is to keep the puppy still for long enough to evaluate it! A general clinical examination should be performed. Occasionally the pup might have other scratch injuries (usually to the face) or it might even become shocked and need immediate attention for this. However, we will assume that it just has an injury to one eye. Full evaluation is required since the trauma could result in simply a very superficial corneal scratch or the far more serious ocular penetration including lens damage.

It is common for the conjunctiva to swell (chemosis) and become markedly hyperaemic, making direct examination of the cornea difficult. The application of topical anaesthesia can help visualization but if the puppy is struggling it is often more sensible to sedate it, or even administer general anaesthesia for a more thorough examination. This is particularly important if a full thickness laceration is suspected – resistance to examination could result in a catastrophic iris prolapse for example. Examination under sedation or anaesthesia obviously has its limitations but it is preferable to exacerbation of the ocular damage. One advantage, however, is that it is possible to fully check the conjunctival sac for remnants of cat claw which could have broken off and if not removed could act as a foreign body later on.

The globe should be evaluated to see if it has been perforated by the cat scratch. The report of a very wet eye, often with a bloodstained discharge, might suggest this (Figure 24.1). Fluorescein can be safely used, even if the eye has been perforated, providing moistened sterile paper strips or single-use vials are used. If the corneal integrity has been broken, a small stream of aqueous might be seen leaking out through the retained dye in the surrounding damaged cornea. Checking the depth of the anterior chamber is useful – it will be shallower than the fellow eye if perforation has occurred. Also the shape of the pupil might alert you to a full thickness injury – normally it would be miotic following the injury but should remain spherical if the cornea is intact. If it is markedly distorted, this suggests corneal rupture with the iris moving forwards to plug the hole



Figure 24.1 Full thickness laceration, resulting in iris prolapse in a 4-month-old Labrador puppy. Note the sticky bloodstained discharge (clotted aqueous) and bulging brown iris with surrounding grey corneal oedema.

(anterior synechia or even iris prolapse) and resultant misshaping of the pupil (see Figure 24.9).

Assuming that the cornea has not been ruptured, the injury can be treated as a superficial or simple deep ulcer. However, cats' claws often rip through the full thickness of the cornea. If this has occurred, in addition to checking the position of the iris it is essential with any such injury to check the integrity of the lens. Tears in the anterior lens capsule can be catastrophic. If not diagnosed on initial presentation they can result in a serious phacoclastic uveitis, secondary glaucoma and loss of the eye. If the anterior lens capsule cannot be directly visualized, then careful ultrasonography can be considered. However, the ultrasound gel should not be allowed to enter the anterior chamber. If it is not clear whether the lens is involved, then early referral should be considered and offered to the client.

DIFFERENTIAL DIAGNOSES

The list of differential diagnoses is minimal since the history and presentation are classic for a cat scratch injury. Other traumatic incidents such as a thorn or even blunt trauma from a football can be considered but the history will usually rule out such events. Progression of an undiagnosed ulcer is possible, but unlikely. Ocular congenital conditions are rarely likely to cause such a presentation with the exception of congenital keratoconjunctivitis sicca which could present with a suddenly ruptured corneal ulcer.

CASE WORK-UP

The work-up for a puppy scratched by a cat is minimal. Once the degree of ocular damage has been ascertained, further diagnostics can be undertaken. These might include ultrasonography, as outlined briefly above, to evaluate lens integrity and ensure that the posterior segment is not involved. If the anterior lens capsule has been breached, then early referral should be considered.

Phacoemulsification of the damaged lens offers the best long-term prognosis for the puppy. If the lens appears intact, and immediate referral is not being offered, it might be appropriate to take a swab from the edge of the laceration for bacterial culture and isolation – cats' claws can harbour a wide range of potentially pathogenic organisms and early detection can allow the most appropriate medication to be chosen. Additional work-up is not usually required.

NURSING ASPECTS

Some specific nursing factors need considering when dealing with ocular injuries in young puppies. To start with, they will not be fully trained, and so are unlikely to sit on command and keep still for the application of topical medication. As such, it is usually easier if two people are available at treatment times. Self-trauma is more likely in puppies than in older patients and so the use of Elizabethan collars is common. However, many young animals will react quite violently to the collar, and care must be taken to ensure that they do not exacerbate the ocular damage further by thrashing around due to panic and fear of the collar. They should also be kept quiet while the cornea heals – which again is difficult in young playful animals. Nurses can spend time with the owners explaining ways to keep the pet calm without resorting to sedatives!

General aspects of nursing are similar to previous ulcer cases – gently cleaning away any discharge with sterile saline on soft swabs or moist cotton wool prior to the topical application of various medications. The patient should be stroked and fussed after treatment so that they learn that it is not an unpleasant experience and also to distract them such that they are less likely to rub the eye.

EPIDEMIOLOGY

The epidemiology of cat scratch injuries is easy to understand when remembering that the menace response is a learned response. It is not innate. The response is not present immediately the eyes open (at about 10–14 days in puppies), but develops gradually by about 4 months of age. Thus, when the playful puppy bounds up to the less than enthusiastic cat, it does not realize that the cat's paw rapidly reaching out to it is a threatening gesture which is likely to hurt. And in the matter of a few seconds the poor puppy has come off worse from the encounter.

CLINICAL TIPS

- When any puppy is presented – at first vaccination or for a post-purchase check-up for example – ask if there are any cats in the house and ensure that they have their claws cut!
- Apply topical anaesthesia and light sedation to examine the injured puppy properly
- Consider referral if the cornea has ruptured, especially if lens damage might have occurred
- Lacerations are more commonly in the dorsolateral quadrant where protection from the nictitans membrane is minimal
- Look from the side at the profile of the cornea, iris and lens to see if there is disruption of the normally parallel curves of these structures
- Treat aggressively for uveitis
- Do not use ointments if the eye has been perforated

ANATOMY AND PHYSIOLOGY REFRESHER

The anatomy of the anterior chamber should be remembered when considering corneal lacerations. Corneal healing has been previously described. If an injury penetrates full corneal thickness and continues into the eye, the damage which follows will depend on whereabouts the injury has occurred. A peripheral laceration will result in the claw catching the iris. This will result in immediate haemorrhage – the anterior face of the iris is highly vascular – but the lens will probably be undamaged (Figure 24.2a). Thus, even if an iris prolapse occurs, the prognosis is probably still reasonable – the uveitis is likely to be moderate. This is assuming that bacterial infection does not complicate the picture.

If the injury has occurred in the central cornea, then the lens is more likely to be involved since anatomically it is the next solid structure for the claw to catch (Figure 24.2b). Once the anterior lens capsule is torn, cortical material leaks out into the anterior chamber. This causes a phacoclastic uveitis – basically the released lens proteins are highly antigenic and cause a tremendous inflammatory reaction which is often poorly responsive to traditional therapy with both steroid and NSAID agents. Removal of the antigenic lens material by phacoemulsification provides the best prognosis if a tear of more than 1 mm has occurred. Pinpoint tears can self-seal, limiting the amount of lens loss and causing a less catastrophic uveitis. In these less serious cases lens removal might not prove necessary.

TREATMENT OPTIONS – MEDICAL

No matter how severe the injury to the puppy's eye, medical treatment will be required, sometimes alone but sometimes in conjunction with surgery. If the cat scratch is superficial, i.e. no deeper than one-third of the corneal stromal thickness, medical management should suffice. The injury can be treated in the same way as an uncomplicated ulcer. Thus a combination of broad spectrum antibiotic drops such as chloramphenicol four to six times daily in conjunction with topical atropine if any reflex uveitis is present should suffice.

The eye should be checked regularly – every 1–2 days – since although it should heal quickly, it could also deteriorate rapidly. For example, the owner might not be able to medicate effectively resulting in bacterial contamination, or the puppy might not be on restricted play and in its exuberance might knock the already injured eye again such that further damage ensues.

Deeper injuries require immediate systemic antibiotics (e.g. a cephalosporin) and systemic NSAIDs in addition to the topical therapy. Systemic steroids are sometimes used instead of NSAIDs but never without antibiotic cover. The risk of endophthalmitis is present and, as such, aggressive medical management is important.

TREATMENT OPTIONS – SURGICAL

Surgical intervention is often required and it must be decided if referral is in the best interests of the puppy. Certainly if the cornea has been ruptured this should be considered for the best outcome but obviously owner finances, owner commitments and specialist availability will all factor.

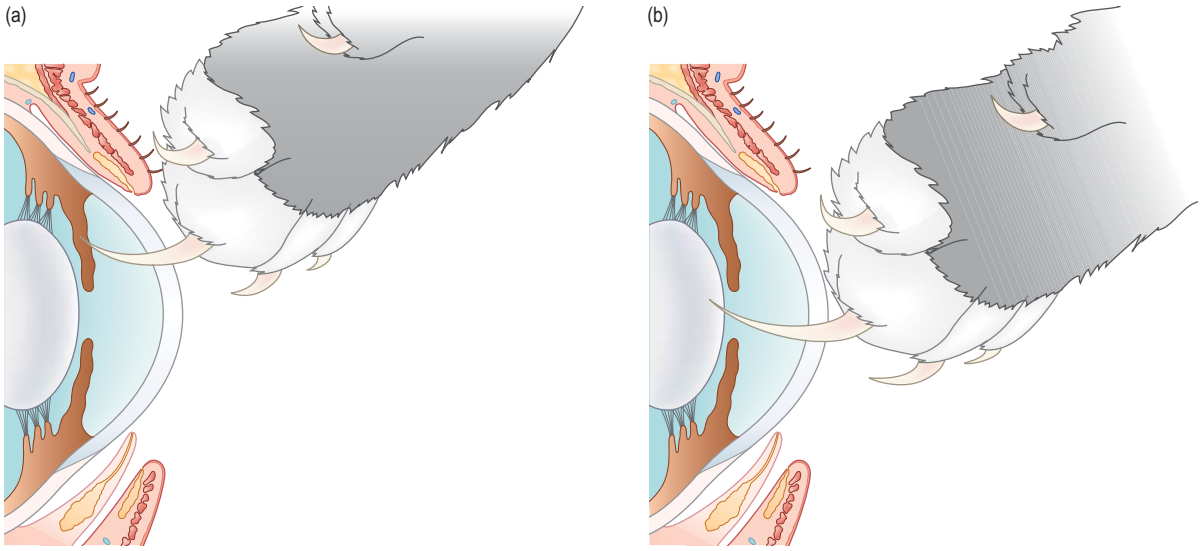


Figure 24.2

(a) A peripheral cat scratch will result in iris prolapse.

(b) A central laceration will result in damage to the lens, more severe uveitis and possibly require emergency phacoemulsification of the lens.

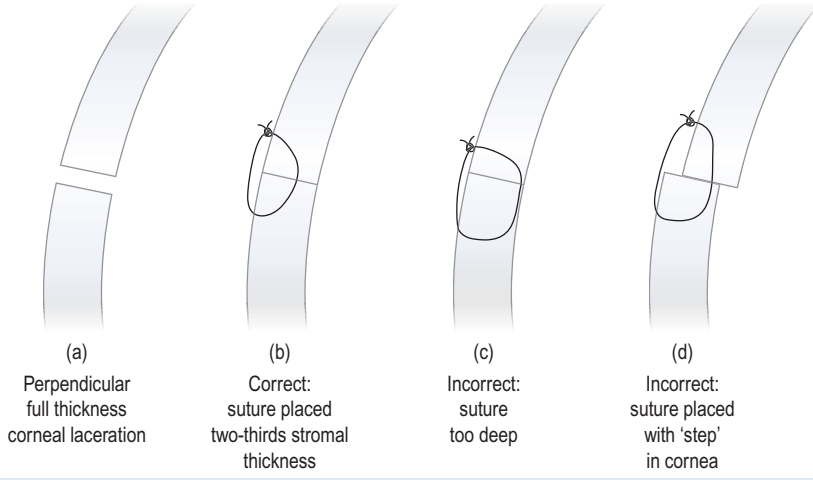


Figure 24.3 Corneal suture placement for perpendicular laceration.

Cat scratch lacerations can be perpendicular to the cornea (these tend to heal more readily) or can be oblique through the stroma. If the former, it is possible to place direct sutures (e.g. of 8/0 polyglactin 910, Vicryl, Ethicon) in a simple interrupted or continuous pattern. This will provide immediate support to the weakened area and promote rapid healing. Prior to any surgery the application of several doses of topical antibiotic, plus atropine and possibly a topical NSAID (or certainly a systemic one), should be undertaken. Careful surgical preparation is required and while under anaesthesia it is important to check that no fragments of claw have become embedded in the conjunctiva or fornix. The placement of the sutures is important: they should extend from one-third to halfway through the stroma – never full thickness – and should be tightened to allow the edges of the cornea to meet smoothly with no stepping at the surface of the wound (Figure 24.3). Postop-



Figure 24.4 Oblique corneal laceration resulting in flap of cornea seen as grey 'blob' on the front of the eye. This must be differentiated from clotted aqueous. The dog is an older Jack Russell terrier which met an unfriendly cat (rather than a puppy)!

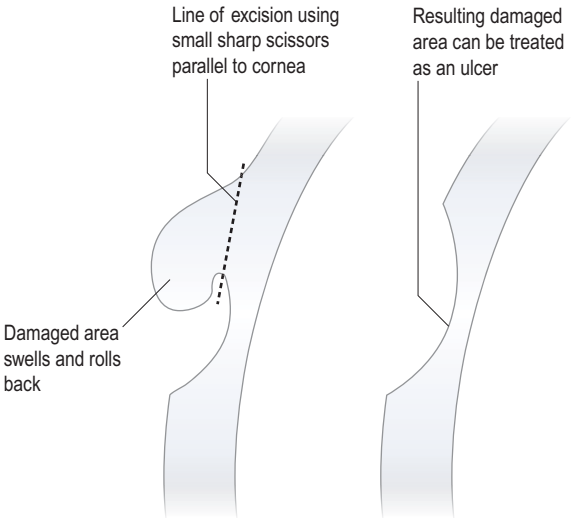


Figure 24.5 Surgical removal of flap of cornea.

erative care is as mentioned above under medical management. Once the wound has healed, i.e. no fluorescein is retained between the sutures and the cornea lies flat around them, it might be necessary to introduce a topical anti-inflammatory agent (such as dexamethasone alcohol or prednisolone acetate) to reduce the likely vascular reaction associated with the sutures starting to dissolve. This reaction can be quite florid in young, growing animals and is often a cause of concern for the owner.

For a laceration which is not full thickness but is an oblique cut, it is possible to trim the oedematous loose flap of cornea under general anaesthesia with small, sharp scissors (Figures 24.4 and 24.5). This will prevent the devitalized flap from acting as a site for collagenase release which could then lead to corneal melting (discussed in Chapter 23). Removing the loose flap will result in a larger stromal defect such that direct suturing is not possible to close this gap. The injury can be treated as a simple but fairly deep ulcer and allowed to heal without further surgery. However, the cornea will be weakened and, as previously alluded to, the patient might not be quite as cooperative as an older animal with respect to keeping calm and not leaping about and getting over-excited. As such, further protection with the use of a conjunctival graft might be prudent. Not only would this provide a blood supply with nutrients, anticollagenases and so on, it would also provide the all-important structural support. Additionally, it is possible to carefully use topical steroids immediately following graft placement should a significant uveitis be present.

Serious injuries with iris prolapse should be referred unless magnification and experience are available! It is necessary to evaluate the iris and decide if it can be replaced or if it requires amputation. The anterior chamber is usually inflated with viscoelastic agents to assist the surgery and help to evaluate the lens. If the lens capsule has been damaged, the surgeon must decide if the lens needs removal. Phacoemulsification at the same time as corneal repair is often required in these situations. If the lens damage has not been appreciated initially and the cornea has been repaired, only to find a few days later that the eye is becoming very inflamed with white matter in the anterior chamber (a combination of released lens material which has become cataractous together with hypopyon), then referral at this stage is important if the eye is to have any chance of being salvaged. The specialist is likely to need to operate quickly in order to remove all the damaged lens and carefully flush the anterior chamber. However, once a phacoclastic uveitis has developed, the prognosis is guarded. There is often an overwhelming inflammatory reaction, which, even with surgery, and high dose topical and systemic steroids, cannot be fully controlled and leads rapidly to a secondary glaucoma. In these cases enucleation is then the only option. In addition to the inflammatory reaction which develops following ocular

penetration, we must remember that the cat claw could have inoculated bacteria into the anterior segment. These can rapidly multiply resulting in a panophthalmitis and loss of the globe though infection.

PROGNOSIS

The prognosis for an eye that has been lacerated by a cat will clearly depend on the degree of damage present (Figure 24.6). However, the owners should be fully aware of the serious nature of the injury from the start. Superficial damage does carry a good prognosis and lesions heal with minimal scarring. Although those which require surgery will result in more scarring, providing the injury was not full thickness the prognosis is still reasonable. If a full thickness injury has occurred, but with minimal lens damage, some animals can still do very well and retain functional vision (Figure 24.8). It is those cases in which corneal rupture has occurred that carry a poor outlook. The presence of an iris prolapse, severe hyphaema and collapse of the anterior chamber require specialist surgery and even then the inflammation which ensues can be difficult to control. It is not usually the corneal injury which provides the problems, but the reaction of the uveal tract. The blood–aqueous barrier in young animals is unstable and more labile than in adults, resulting in a more rapid and severe inflammatory reaction. A tear in the anterior lens capsule, especially if more than 1–2 mm in size, will result in the release of lens cortex into the anterior chamber. This material is highly antigenic and a tremendous uveitis can follow which triggers a painful, blinding secondary glaucoma. Removing the eye is then the only option.

Owners should also be aware that even if the puppy does well in the short term, the longer term prognosis is still guarded. It is not uncommon for the wound to heal well, the inflammation to settle and the puppy come off all treatment, only to suffer from glaucoma several months later.

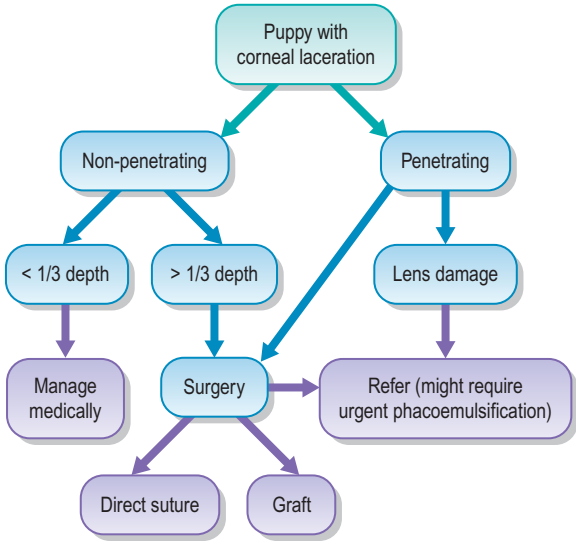


Figure 24.6 Decision-making tree – puppy scratched by cat.

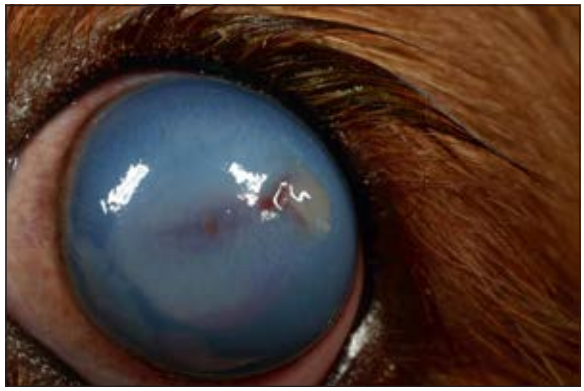


Figure 24.7 Cavalier King Charles spaniel puppy which was scratched by a cat 12 hours previously. Note the corneal laceration and bleb of clotted aqueous sitting on the cornea, marked corneal oedema and hyphaema. The lens was ruptured and emergency phacoemulsification was required. Image courtesy of Christine Heinrich, Willows Referral Centre, Solihull, West Midlands.



Figure 24.8 Young Labrador which was scratched by a cat 6 months previously. The corneal laceration was small and healed without surgery. Note the anterior capsular and cortical cataract and wrinkling of the anterior lens capsule, together with mild posterior synechia formation. However, the iris looks fine with no ongoing uveitis and the eye is visual. A large tear in the lens capsule could have been catastrophic.

CASE EXAMPLE 24.1

Patient details: Lurcher, 4 months old, male

PRESENTATION AND HISTORY

The puppy had been playing in the garden and put its head under the fence. The neighbour's cat then attacked the intruder. The puppy screamed and ran inside holding the right eye closed. The owners brought it straight along to the surgery.

CLINICAL SIGNS

The puppy was frightened but otherwise well. Only the right eye was affected. A watery discharge was present. A large bleb was noted laterally close to the limbus. It was the same colour as the iris but appeared slightly gelatinous. This was an iris prolapse. Minimal conjunctival hyperaemia was noted. Dyscoria was present with the pupil drawn out laterally towards the corneal injury (Figure 24.9). Vision was present and the lens appeared undamaged.

WORK-UP

No further work-up was undertaken.

TREATMENT

General anaesthesia was induced and the injury was examined and found to be adjacent to the limbus, but not crossing it. The iris was flushed with sterile saline, the fibrin removed from it and it was gently replaced through the corneal wound. This was then directly repaired, and a small advancement conjunctival graft was sutured over the top for added security (given the lively nature of the puppy!). All sutures were 8/0 polyglactin 910 (Vicryl, ethicon). Postoperatively he was treated with a week of amoxicillin/clavulanic acid tablets, chloramphenicol drops six times daily, atropine drops twice daily and prednisolone acetate four times daily, all for 2 weeks, and then continued with just prednisolone acetate at reducing levels for the following 6 weeks.

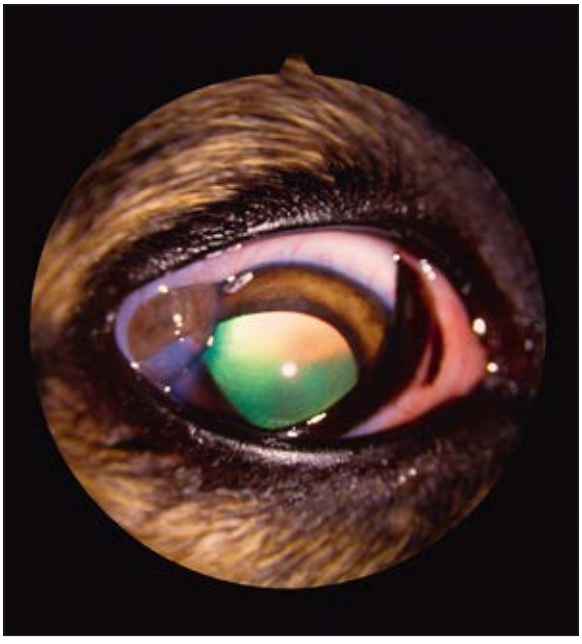


Figure 24.9 Iris prolapse in a 4-month-old lurcher which met the neighbouring cat through the fence. Note the dyscoria, bleb of grey–brown tissue on the cornea but lack of haemorrhage or lens damage.

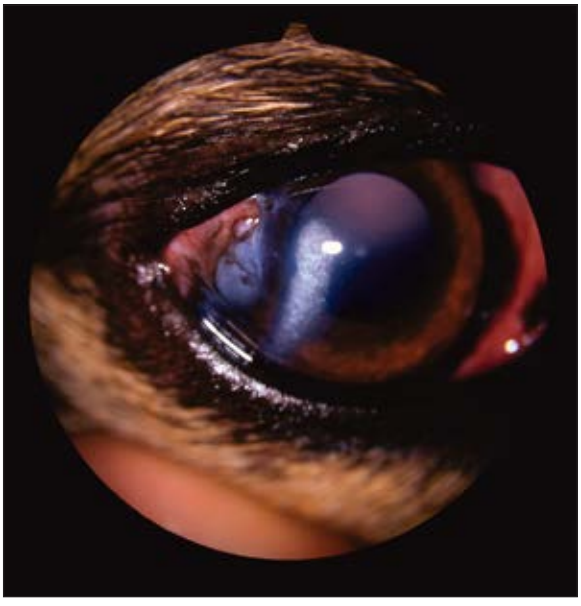


Figure 24.10 Same lurcher as in Figure 24.9, 3 months later. Note the lateral corneal scar and slightly pulled corneal graft, plus the arc of corneal lipidosis. The visual axis is clear and vision is normal.

OUTCOME

Healing of the corneal wound and graft was uneventful and minimal uveitis developed. The graft pulled slightly a month following surgery but by this stage the cornea was fully healed. A lipid keratopathy developed adjacent to the injury – probably as a combination of altered blood supply to the area exacerbated by the topical steroids (Figure 24.10). However, this was not in the visual axis and remained of no concern to the dog.

This case illustrates that with rapid presentation and immediate surgery such injuries can do very well. The lack of haemorrhage and lens damage certainly contributed to the successful outcome.

25 Corneal foreign body

INITIAL PRESENTATION

Pain, discharge

PRESENTING SIGNS

Typically patients present with an acutely painful eye. For dogs they might have been running in the park, through the bushes, and come back holding one eye closed, with increased lacrimation and possibly attempts to rub the affected eye. Cats are more likely to return home with one eye closed or exhibiting severe blepharospasm together with an ocular discharge, normally serous in nature initially but which rapidly becomes purulent. Working dogs will commonly present with a corneal foreign body. Occasionally the patient will only be in mild discomfort and the owners then may notice something abnormal sitting on the surface of the eye.

CASE HISTORY

Normally there is very little history – just the information that the pet had been perfectly OK when previously seen but suddenly the eye was painful, half closed and watering.

Usually only one eye is affected. No previous ocular problems have been reported in most cases (although some dogs will get repeated foreign bodies, especially working breeds!).

CLINICAL EXAMINATION

General clinical examination is unremarkable. Mild to marked blepharospasm will be present and increased lacrimation will be noted. This can be very dramatic if the foreign body has actually perforated the cornea, since aqueous leakage will also have occurred. If there is any delay between the owners noticing the problem and presenting to the surgery, bacterial contamination could have developed such that the discharge becomes purulent in nature. Conjunctival hyperaemia and chemosis (the latter especially in cats) are also likely to be present. Gently opening the eyelids might reveal a foreign body on or in the cornea. If the patient is unwilling to open the eye then topical anaesthesia might assist examination. Alternatively, sedation should be considered to allow full evaluation, particularly if a corneal perforation is suspected.

In addition to identifying the presence of the foreign body, it is important to check the rest of the eye. This will include examining beneath the nictitans membrane – fragments of plant matter can easily lodge here as well as in the cornea and should not be overlooked. A reflex uveitis is likely to be present such that the pupil is miotic. If the foreign body has perforated the cornea, then hyphaema and aqueous flare might be present (Figure 25.1). Viewing the patient's eye from the side, and comparing it to the normal eye, will help to tell if the anterior chamber is of normal depth – it can be more shallow than usual if perforation has occurred and aqueous leakage has followed.

It will be clear from this description that it is important to assess the location of the foreign body within the cornea. This can be divided into surface, penetrating and perforating, in increasing order of seriousness (Figure 25.2). As the name suggests, a surface foreign body will sit on or in the epithelium. The eye is likely to be uncomfortable, rather than very painful, and only mild intraocular signs (such as slight miosis from a reflex uveitis) will be present. Penetrating foreign matter will be embedded



Figure 25.1 Perforating corneal foreign body in a cat. Note the miosis and hyphaema – the latter extends to the entry wound which is the grey lesion dorsally (the actual foreign body cannot be seen in this photograph but was a piece of cat claw). The corneal vascularization seen at 12 o'clock indicates that the injury occurred a few days previously.

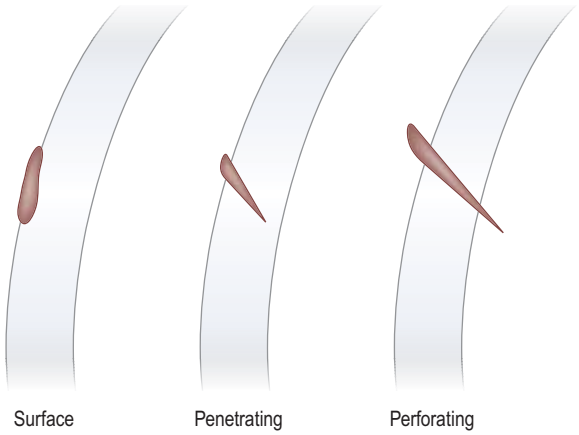


Figure 25.2 Classification of corneal foreign bodies.

within the stroma of the cornea. Some ulceration is likely around the entry site (check with fluorescein dye). If the foreign matter has broken off flush with the corneal surface, or is small and smooth in outline, the patient might be remarkably comfortable. Intraocular involvement will again be minimal.

The most serious foreign body penetrates the full thickness of the cornea. In these cases the eye is likely to be very wet due to aqueous leakage as well as increased lacrimation due to trigeminal pain. The use of fluorescein dye can help to determine if leakage is still occurring around the foreign body. Staining the cornea will result in green dye over the cornea. Instead of flushing away the excess dye as usual, the area immediately surrounding the foreign body is carefully evaluated. If leakage is still occurring, then a small stream of clear, transparent aqueous will be seen dribbling through the green dye. This is called the Seidel test and is pathognomonic for corneal perforation. The anterior chamber might be shallower than the fellow eye. Fibrin will be released and might be free in the anterior chamber, or more likely adhered to the foreign body. If the iris has been touched on entry, then hyphaema is normally present. The pupil will be miotic.

Extreme care should be exercised in examining such patients: if they struggle or squeeze their eyelids tightly closed this could encourage the foreign body to penetrate further into the eye – even becoming loose in the anterior chamber, with sudden loss of globe tension as aqueous flows through the corneal hole which had been plugged by the foreign body. Iris prolapse can also occur and without appropriate emergency management these eyes can be irreparably blinded.

DIFFERENTIAL DIAGNOSES

- Corneal laceration
- Iris prolapse
- Feline corneal sequestrum

CASE WORK-UP

Thorough general clinical examination is advised – there might be other foreign bodies present (down the ears, between the pads of the feet and so on). As mentioned above, it is important to check under the nictitans membrane (either just with topical anaesthesia or under sedation/general anaesthesia depending on the individual). It would be most distressing to successfully remove a corneal foreign body, only to have the patient return a few days later with a purulent bacterial discharge, marked conjunctival hyperaemia and an infected corneal ulcer ventromedially beneath the third eyelid where a retained conjunctival foreign body has rubbed.

Most foreign bodies are of plant matter – other items include paint flecks, metal filings and so on. Since plant matter can be a site of pathological bacterial (or even

fungal) contamination, it is sensible to consider sampling for bacterial (± fungal) isolation and sensitivity testing. Doing this at the outset might result in a quicker resolution for the patient.

NURSING ASPECTS

As it is likely that the patient will require general anaesthesia to remove the foreign body, it is important to check when they last ate. Any concurrent illnesses and medication should be discussed. Topical medication will be required after the surgery (and sometimes before – mydriatics and NSAIDs for example) and care should be taken to ensure that the patient does not rub the eye.

CLINICAL TIPS

- Use topical anaesthesia to assist examination
- Check under the nictitans membrane and the conjunctival fornix for other pieces of foreign body
- Fluorescein is recommended to check the degree of ulceration
- Fluorescein can also be used to see if corneal leakage is occurring (Seidel test)
- Compare the depth of the anterior chambers to see if perforation and aqueous loss have occurred
- Look for fibrin or haemorrhage in anterior chamber which could suggest perforation

ANATOMY AND PHYSIOLOGY REFRESHER

Due to the laminar arrangement of collagen fibrils within the corneal stroma, the angle of entry of the foreign body is likely to have a significant effect on its final resting location. Thus an oblique entry is likely to penetrate through the lamellae while a perpendicular entry is more likely to result in perforation. Clearly blunt-ended objects are more likely to remain on the epithelium and sharper objects such as thorns are more likely to penetrate the stroma. An area of ulceration will be present just around the entry site.

TREATMENT OPTIONS – MEDICAL

All foreign material can potentially contaminate with bacterial or occasionally fungal matter and this is one reason why removal should be performed. Only with inert material, such as a small fragment of non-ferrous metal that has broken off within the stroma and without penetrating, can leaving it be considered. However, frequent monitoring should be undertaken and should there be any change in appearance – a vascular reaction for example – then removal should be performed. Thus medical management is really only an option in a very, very limited number of cases. Topical treatment will be required following removal and this is discussed below.

TREATMENT OPTIONS – SURGICAL

The surgical approach to removal of corneal foreign bodies will vary according to the location of the material. Thus we will discuss superficial or epithelial, penetrating and perforating in turn. In general, superficial foreign bodies can be easily removed in general practice. Whether one attempts the removal of penetrating or even perforating ones will depend upon the level of experience of the surgeon and the facilities available. One must be able to deal with the potential sequelae of attempted removal before starting out. Thus, one should be aware that there could be aqueous leakage on removal of the foreign object. There could be anterior synechiae which need breaking down or an iris prolapse which requires attention. The foreign body could slip out of your grasp and become lost in the anterior chamber. If you cannot deal with these potential complications then it is wise to consider referral to someone who can. There is nothing more disheartening for the referral ophthalmologist than receiving that emergency phone call saying 'I've got this dog anaesthetized at the moment and I went to grab a piece of thorn which was lodged in the cornea but it's slipped into the eye, lots of aqueous has escaped and I can't see the thorn any more. What should I do?' By being aware that this could happen, even with what appears to be a simple removal, and referring to a suitably qualified and equipped colleague, the patient can be given the best chance of a successful outcome should such events occur!

Superficial foreign bodies

The superficial foreign body is usually embedded just in the epithelium or anterior stroma. Often it can be simply removed using a sterile cotton bud or cellulose sponge

after the application of two or three doses of topical anaesthesia with light sedation if necessary. Using a dry cotton bud the foreign body can be gently dislodged – rolling the cotton bud as you lift across the foreign body sometimes helps. Alternatively, it might be possible to flush it off the cornea using a 5 or 10 ml syringe filled with sterile saline. Once the object is removed, the conjunctival fornix and beneath the nictitans membrane should be checked for further remnants of foreign material.

If gently touching and flushing are not successful, then the patient should be anaesthetized. Using good magnification and lighting the object should be gently touched with a sterile cotton bud again. If this does not loosen it then a 25 gauge needle can be used to lift the material from the epithelium – the needle should be introduced lateral to the object and parallel to the plane of the cornea. Once removed, a small ulcer will be present. If the edges of this are not clean, or if the foreign body has been present for more than a few hours, it is sensible to take a swab from the ulcer site for bacterial culture and sensitivity testing. While awaiting results the patient should be treated with a broad spectrum antibiotic topically, such as chloramphenicol or fusidic acid. One drop of topical atropine can be applied to relieve ciliary spasm and promote ocular comfort – any reflex uveitis is usually very mild and, as such, this single application is normally sufficient. Systemic antibiosis is not usually required. The resultant ulcer should heal in a matter of days providing that bacterial infection does not complicate things.

Penetrating foreign bodies

The penetrating foreign body will always require the patient to undergo general anaesthesia prior to attempting removal. Careful pre- and post-anaesthetic examination will determine the depth of the material and the angle of entry. The former is obviously important regarding whether anterior chamber penetration could occur if the object slips further away as you attempt to remove it. The latter is significant since removal is best achieved by directing your movements perpendicular to the line of entry.

Once the patient is positioned – with the cornea in the horizontal plane (the use of vacuum positioning cushions is invaluable) – the eye is stabilized with stay sutures or small mosquito forceps grasping the conjunctiva close to the limbus. Removal is best achieved using one or two 25 gauge needles rather than a pair of forceps (Figure 25.3). It is tempting to use a pair of fine rat-toothed forceps to grasp the end of the foreign body but unfortunately this tends to force the object further into the cornea and the end can fragment, rather than it being gently pulled out as one would hope! So instead needles are advocated. Special sharp-pointed foreign-body spuds can be purchased, but needles do just as well! These should be used to impale the object close to its entry, at right angles to its entry plane. Once impaled with one or two needles (opposite each other) the object can be gently withdrawn through its line of entry. Sometimes it is necessary to undermine the epithelium at the entry site in order to get a good purchase on the material. Once removed the corneal surface should be thoroughly flushed with sterile saline solution.

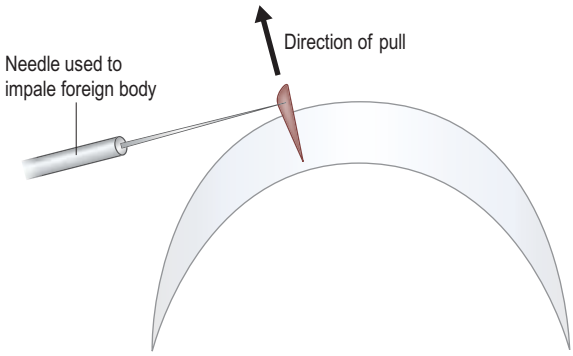


Figure 25.3 Removal of a penetrating corneal foreign body.

It is not normally necessary to suture the cornea, since the foreign material was only partial thickness, and the lamellae should fill the defect quickly. However, the surrounding stroma should be assessed for viability – any necrotic areas should be removed as they could act as a focus for stromal liquefaction, especially in the presence of bacterial infection. Thus the penetrating foreign body should be treated as a complicated ulcer once removed. Frequent broad spectrum topical antibiotics, with culture and sensitivity testing if appropriate, together with topical atropine and occasionally NSAIDs should a significant uveitis be present, are all advised. All being well, the cornea should heal in a few days and minimal scarring should be expected.

The main complications with penetrating foreign bodies are associated with problems encountered during removal and infections afterwards. Thus the foreign body could fragment, making it impossible to remove in one piece. In this case the entry tract might need opening with a blade (e.g. Beaver blade No. 65) to gain access

to the material. If the material is deep within the stroma, then corneal suturing or a conjunctival graft might be required. The foreign body could have perforated the cornea fully, and be plugging the defect, such that removal resulted in a loss of aqueous. Normally a single suture and re-inflation of the anterior chamber (with a balanced salt solution, sterile saline or even air) will suffice. If the foreign body does not come out as expected, but enters the anterior chamber completely, its removal is discussed briefly below.

Bacterial (or even fungal) infections can occur following foreign body removal. The rapid removal and concomitant cover with a topical broad spectrum antibiotic should prevent this, but if the cornea does not start to heal in a couple of days, then aggressive medical management is required. This is discussed in the section on complicated ulcers.

Perforating foreign bodies

Removal of the perforating corneal foreign body can be more challenging. Referral should be considered. If the foreign body has penetrated the iris or lens, then referral is definitely advised. Some cases require urgent lens removal via phacoemulsification if the lens has been ruptured (see Chapter 24). However, sometimes referral is not possible, and so the approach to removal will be discussed here.

Ideally a full intraocular ophthalmic kit, magnification with surgical loupes (3–4.5× magnification) and viscoelastic material should be available. Initially removal is attempted by impaling the foreign body using needles perpendicular to it, as described above. Some surgeons advocate pre-placing a corneal suture (such as 8/0 polyglactin 910, Vicryl, Ethicon) but personally I find that these just get in the way. Once removed, the anterior chamber can be flushed and any anterior synechiae gently broken down using an iris repositor. The corneal perforation is closed with a single suture. If the surrounding cornea is devitalized, then a small conjunctival graft might also be advised.

If it is not possible to remove the foreign body via its entry site, then it must be 'pushed out' from inside. A small incision (3 mm) is made in the clear cornea close to the limbus and, if available, viscoelastic material is injected into the anterior chamber to keep it formed and to protect the corneal endothelium. A small pair of rat-toothed forceps (e.g. St Martin's) is introduced parallel to the plane of the iris and grasps the foreign body which is then pushed out from its entry wound. This is less traumatic to the eye and less likely to cause fragmentation and severe uveitis than by pulling it into the eye and removing it though the limbal incision. Once the foreign body is removed, the entry site is cleaned and sutured and the viscoelastic fully removed. Hyphaema and fibrin can be removed at the same time. The incision is closed and the anterior chamber re-inflated using a balanced salt solution or sterile saline should the former not be available.

In addition to the risk of bacterial contamination – not only of the cornea but also in the anterior chamber this time – some degree of uveitis is inevitable following a penetrating corneal body. If unrecognized and untreated, the uveitis can be so severe as to blind the patient, and secondary glaucoma is not uncommon. Thus, in addition to antibiotic cover (e.g. topical enrofloxacin or ciprofloxacin and systemic cefalexin), and topical atropine to ensure mydriasis (thus preventing synechia formation), topical and systemic anti-inflammatory agents should be employed. Providing the cornea has been carefully sutured, and appropriate antibiotic cover is supplied, then topical steroids such as prednisolone acetate or dexamethasone can be used. If ulceration is present or you are not confident with the sutures, then topical NSAIDs can be used initially, swapping to steroids once full epithelialization has occurred. Anti-inflammatory medication is normally required for several weeks following surgery and careful reassessment is required to check case progression. Deterioration with endophthalmitis or glaucoma does occur, thankfully not commonly, but should either of these severe complications develop, enucleation is the only alternative for patient wellbeing.

PROGNOSIS

The prognosis for a corneal foreign body will depend on the location within the cornea, the length of time it has been present, the nature of the material and the surgeon's skill in removal.

- A small plant seed lodged in the epithelium which is removed within hours of injury and treated as a simple ulcer would be expected to heal in a matter of days, with no significant corneal scarring and thus an excellent prognosis.
- The perforating foreign body which had been present for several days before presentation and thus involved both infection and severe uveitis could carry a guarded prognosis for restoration of normal vision on removal. The risks of endophthalmitis/panophthalmitis and secondary glaucoma are unfortunately quite high if

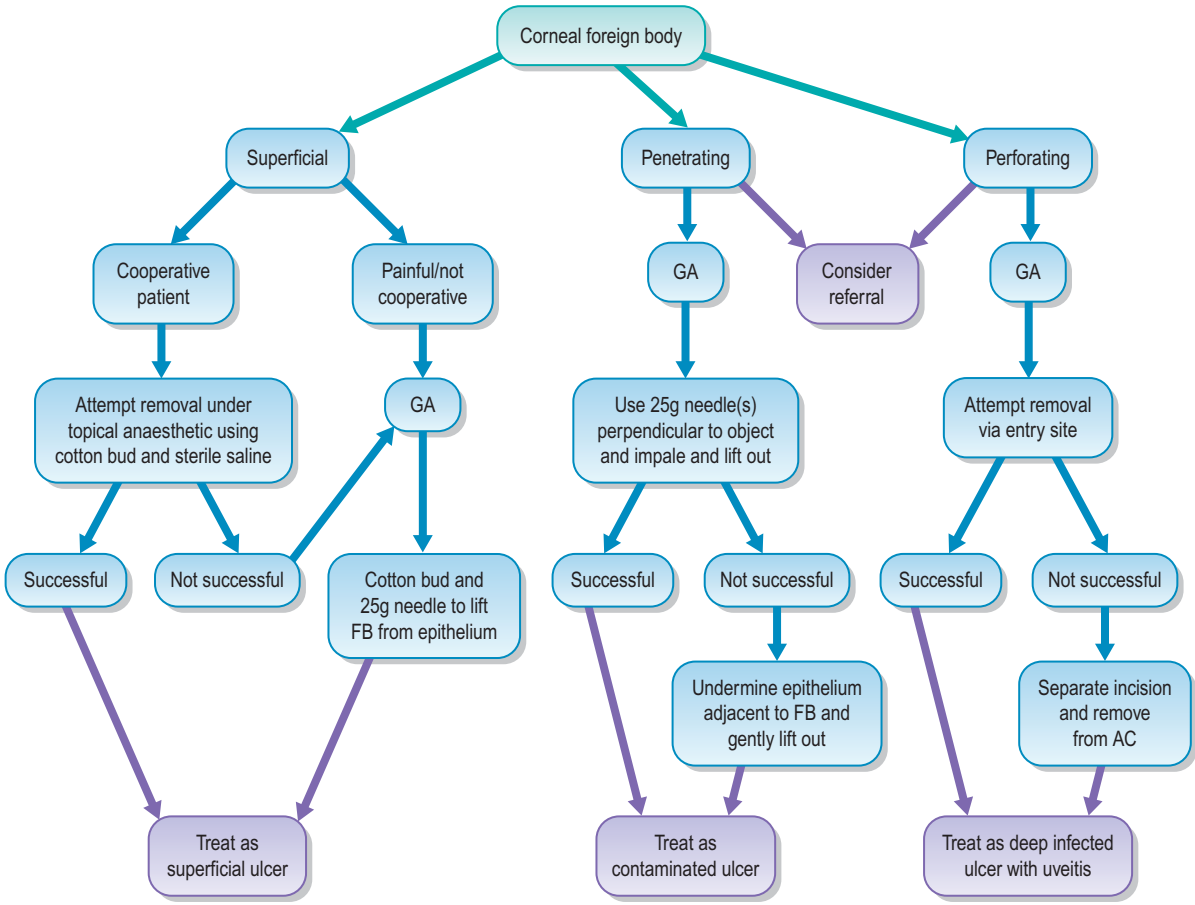


Figure 25.4 Decision-making tree – foreign body. GA – general anaesthesia.

rapid removal of the foreign body and aggressive after-care are not undertaken.

- For penetrating or perforating foreign bodies referral to a suitably qualified colleague should be considered. If the iris has been damaged by the foreign body then either anterior or posterior synechiae might remain following removal but a comfortable visual eye can still result (Figure 25.5).



Figure 25.5 Young German short haired pointer (GSHP) following removal of a perforating foreign body. Although posterior synechiae resulting in dyscoria are present, and some lens damage with cataract formation has occurred, the eye is quiet and visual.

CASE EXAMPLE 25.1

Patient details: Boston terrier, 2 years old, male (entire)

PRESENTATION AND HISTORY

The dog had been out in the garden and about an hour later the owner noticed that the right eye was causing him problems – he was holding it partly closed, attempting to rub and it was weeping.

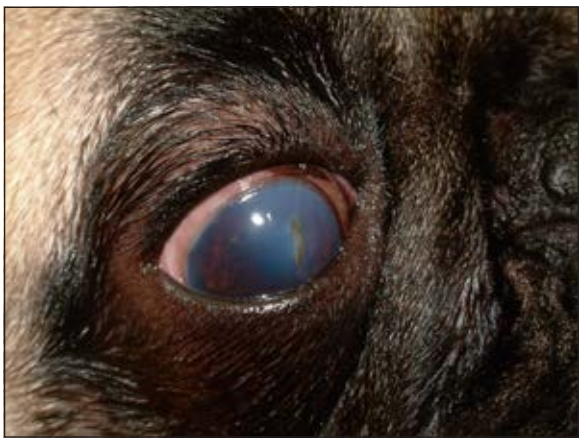


Figure 25.6 Superficial corneal foreign body in a Boston terrier. Note the oedema surrounding the plant material and mild miosis.

CLINICAL SIGNS

The dog was in good general health. The left eye was unremarkable. The right showed blepharospasm and increased lacrimation. The conjunctiva was slightly swollen but no marked hyperaemia was present. Corneal oedema was noted surrounding a piece of plant matter which was embedded in the anterior cornea (Figure 25.6). The pupil was mildly miotic. On examining the eye from the lateral aspect it was apparent that the foreign body was lodged superficially.

WORK-UP

The clinical examination revealed an obvious corneal foreign body and no further work-up was required.



Figure 25.7 Retrieved foreign body from Boston terrier in Figure 25.6.

TREATMENT

Topical anaesthesia (proxymetacaine) was placed in the eye and the foreign body was gently touched using a sterile cellulose sponge (although a cotton bud would also be appropriate). The plant material was easily dislodged (Figure 25.7). The cornea was flushed with 10 ml sterile saline. A pair of non-traumatic fixation forceps (Von Graefe forceps, see Figure 13.1) was used to grasp the nictitans membrane and the ventral conjunctival fornix was checked for further foreign body fragments – none was present. One drop of atropine was placed in the eye and fusidic acid was dispensed twice daily for 5 days.

OUTCOME

The dog was checked 5 days later and the cornea was fully epithelialized. The corneal oedema had reduced and the pupil remained dilated. A further week later minimal corneal scarring remained, the eye was visual and no complications arose.

This case illustrates that some foreign bodies can be very easy to remove and do not even require sedation or anaesthesia if the patient is cooperative. However, if there is any suspicion that the material has perforated the cornea, this approach should not be attempted!

26 Corneal endothelial dystrophy

INITIAL PRESENTATION

Abnormal appearance – cloudy

PRESENTING SIGNS

This condition is usually encountered in dogs and is very rare indeed in cats. The dog is presented with the complaint of cloudy eyes. The owner often thinks that it is developing cataracts. Normally both eyes are affected but to differing degrees – the owner might only be concerned about one eye. The dog is likely to be comfortable with no or minimal conjunctival hyperaemia – the owner is probably not worried about the eyes looking red. However, sometimes the owner feels that the eyes are bulging (see Clinical examination). Vision is usually normal or only minimally impaired – it is the change in appearance of the eyes which concerns the owner. The condition is normally insidious in onset and is most common in older patients.

CASE HISTORY

The history is usually relatively straightforward. The owner has probably been aware of a cloudy appearance to the eyes for weeks or even months, which is getting progressively more noticeable. They initially assume that the dog is just getting old, or developing cataracts, and it is only when the clouding becomes quite severe that they bring the pet along. It is normally otherwise well, with no relevant history of previous ocular problems. Thus it is not uncommon for the owner to comment on the appearance of the eyes in passing during an annual vaccination consultation for example.

CLINICAL EXAMINATION

General clinical examination is usually unremarkable. On ocular examination the eye(s) appear slightly blue–grey in colour and the cornea appears slightly dull. Menace responses are usually normal as are pupillary light reflexes. The view of the iris and anterior chamber is hazy due to the presence of corneal oedema which manifests as a blue–grey mottled appearance. No ulceration is usually present (unless the disease has progressed to bullous keratopathy with ulceration – see the later section on prognosis).

The oedema can affect just one quadrant (e.g. dorsolaterally) or the entire cornea, and can be uni- or bilateral (but is not usually symmetrical – one eye will probably be more severely affected initially). The corneal oedema outlines the shape of the cornea and this gives the false impression that the eye is 'bulging'. Normally the cornea is completely transparent and its convex shape is not apparent – one sees the flat iris instead – both from in front and laterally. Once the outline is visible due to the oedema then the shape is more obvious and the appearance is of the eye bulging. This must be differentiated from globe enlargement due to glaucoma or exophthalmos causing the eye to be placed more anterior than usual. The corneal oedema gives a mottled appearance and by gently pressing the globe over the sclera (through the upper lids for example) the oedema becomes denser. Palpation in this way is unlikely to be painful – whereas with a case of glaucoma or severe uveitis causing the oedema the patient would resent such examination due to the discomfort this would cause. Conjunctival hyperaemia can be present but is normally only very mild, providing ulceration is not a feature. Similarly, no significant ocular discharge is normally present.

Intraocular examination is unremarkable – details might be hazy but the use of distant direct ophthalmos-

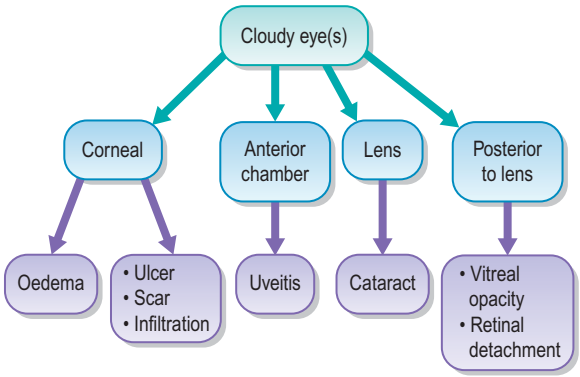


Figure 26.1 Differential diagnosis for cloudy eyes.

copy will help to determine whether the lens is clear or is cataractous, while indirect ophthalmoscopy will assist in evaluating the fundus.

DIFFERENTIAL DIAGNOSES

(See Figure 26.1.)

- Glaucoma
- Keratitis
- Exophthalmos
- Corneal ulceration with oedema
- Corneal lipidosis
- Lipid keratopathy
- Cataract
- Anterior lens luxation with lens pressing on corneal endothelium causing oedema
- Uveitis causing oedema (e.g. canine adenovirus 1 associated)
- Retinal detachment resulting in leukoria
- Lipid-laden aqueous

CASE WORK-UP

Ophthalmic work-up for the case will include fluorescein testing and measurement of intraocular pressure. The latter will rule out both glaucoma and uveitis, although for both conditions other ocular abnormalities will be present along with the oedema. Thus the diagnosis of corneal endothelial dystrophy or degeneration is often one of exclusion. The patient is normally clinically well and, as such, further diagnostic work-up is not normally required.

NURSING ASPECTS

Many cases of corneal endothelial dystrophy do not require treatment. However, if the oedema is so severe that the animal is having visual difficulties, or if bullous keratopathy has developed, then treatment might be necessary. If this is with hypertonic sodium chloride it should be remembered that this often stings. Preventing rubbing immediately post-application is required and distracting the patient (e.g. by feeding or taking for a short walk) is helpful.

EPIDEMIOLOGY

Corneal endothelial dystrophy or degeneration is not particularly common. The dystrophy affects certain breeds, such as the Chihuahua, Boston terrier and dachshund varieties. Dogs with corneal endothelial dystrophy tend to be affected at a younger age (5–9 years) than those with the degeneration (usually 10 years or older). The latter can occur in any breed, although springer spaniels, Dalmatians, Basset hounds, wire-haired fox terriers and retrievers are perhaps over-represented. With the dystrophy females are affected more frequently, but no sex predisposition is present with the degeneration.

The corneal endothelium is the inner layer of the cornea and is only a single layer thick. It has poor regenerative capabilities in dogs and cats and cell density reduces with age. In addition to acting as a physical barrier, the other main function of the endothelium is to pump excess fluid from the corneal stroma such that it remains in a relatively dehydrated state (which promotes transparency). An energy-dependent sodium–potassium transport pump removes this fluid to the aqueous. Failure to do this results in swelling of the collagen fibres as well as accumulations of fluid between the cells – both of which reduce transparency and result in the clinical appearance of oedema.

An endothelial dystrophy occurs in the Chihuahua, Boston terrier and dachshund breeds as mentioned above. By definition the dystrophy is a primary, bilateral, inherited disorder with no inflammation or systemic disease occurring. It manifests with stromal oedema, thickening of Descemet's membrane and fibrous tissue proliferation. The endothelial cells themselves are reduced in number, enlarged and often take on abnormal shapes. Age at onset varies from 5 to 9 years.

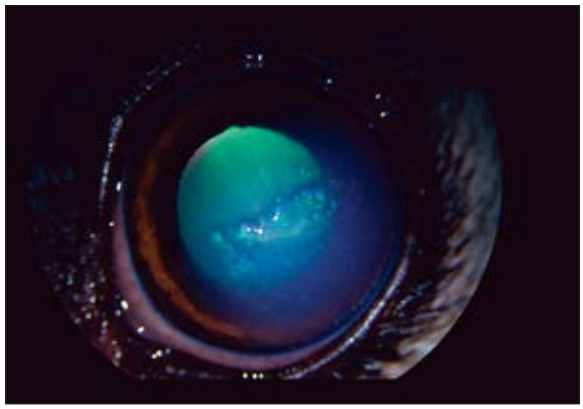


Figure 26.2 Corneal oedema in a 10-year-old Jack Russell terrier that has developed bullae. These are likely to ulcerate.

Degeneration of the endothelium is perhaps more common and is seen in older dogs. With age the endothelial cells become less efficient and reduce in number such that it is easy to understand how oedema can result. The clinical appearance of endothelial dystrophy and degeneration are the same. An inherited corneal dystrophy has been described in the Manx cat but is very unusual.

Once the fluid starts to build up within the stroma it can form small pockets or bullae. These eventually coalesce and can blister to the surface of the epithelium (Figure 26.2). Seen as small bullae on the surface, they can result in small ulcers which can be difficult to get to heal (see below). The term bullous keratopathy applies to this more severe form of the disease.

CLINICAL TIPS

- View the patient from the side as well as in front to appreciate the corneal clouding
- The view of the iris is hazy (unlike with cataract)
- Gentle pressure on the globe through the upper lid will result in increased density of oedema
- The eyes are visual and non-painful, ruling out things such as cataract, glaucoma and severe uveitis

TREATMENT OPTIONS – MEDICAL

Many cases of corneal endothelial dystrophy/degeneration do not require treatment. Providing there is no ulceration and vision is reasonable, the patient can be monitored. Owners should be alerted to the fact that ulcers could develop and should return their pet for examination if there are any signs of pain, blepharospasm and increased lacrimation.

If the oedema is so severe as to reduce vision – like looking through a very steamed-up window – then medical treatment can be considered. This usually consists of hypertonic (5%) sodium chloride ointment. This is not available commercially in the UK but can be obtained from the pharmacy at Moorfields Eye Hospital. The aim of this is to draw the excess fluid out to the epithelium and ocular surface from where it evaporates. The treatment is successful in some, but not all, patients. The oedema will reduce slightly but some clouding will remain and owners must be aware that the eyes will not return to a normal appearance. The ointment needs frequent application – three to four times daily. Owners need to be made aware that it can sting and should ensure that the patient does not rub their eyes. If the cornea does develop ulcers then the hypertonic sodium chloride ointment might sting even more. Should this be the case, it can be reserved for periods between ulcers.

Bullous keratopathy is the more severe form of corneal endothelial dystrophy/degeneration. Small blister-like accumulations of fluid build up within the stroma and burst through the epithelium, causing small ulcers. These can be refractory to medical management in some cases. In general, they are treated in a straightforward manner initially with topical antibiotic agents, atropine to dilate the pupil should any reflex uveitis be present, plus topical lubricants to promote comfort. Vascular infiltration of the cornea is to be encouraged as this can help expedite epithelialization. If the ulcers do not heal after 2–3 weeks, they can be approached in the same way as recurrent epithelial erosions, often with the use of bandage soft contact lenses (see Chapter 21).

Long-term medical management of cases with bullous keratopathy will aim to reduce the build up of bullae using hypertonic sodium chloride ointment, together with gel-based topical lubricants to help smooth the irregular corneal epithelium and distribute the tear film more evenly.

TREATMENT OPTIONS – SURGICAL

Simple cases of corneal endothelial dystrophy/degeneration do not require surgery. In humans, severe cases (Fuchs' dystrophy) require corneal transplants to pre-

serve vision, but these are not widely used in veterinary ophthalmology – rejection and graft opacification are both frequent and severe complications and transplants rarely result in a clear cornea in our patients. As such, surgery is used only in cases where severe, repeated ulceration is a problem, and the aim is to relieve patient discomfort rather than restore vision.

The two main treatment options available in such cases are the use of large conjunctival grafts over the ulcerated areas, or thermal keratoplasty, where small burns are made throughout the cornea to encourage fibrosis and scarring within the stroma such that bullous formation is prevented. Such eyes are often blind. This surgery is usually reserved for such end-stage eyes and it should only be undertaken by experienced veterinary ophthalmologists.

PROGNOSIS

The prognosis for corneal endothelial dystrophy/degeneration is normally fair depending on the age of the patient, the rate of progression of the oedema and the presence of ulceration. Obviously when younger patients are affected with the dystrophy one must offer a slightly more guarded prognosis since the disease is inevitably progressive and at present we have no treatment options available to us to prevent this progression. However, medication with hypertonic sodium chloride ointment, as outlined above, can delay the rate of progression.

Owners can be reassured that in the early stages of the disease patients retain vision and are comfortable. Even when the corneas appear very cloudy, vision is usually sufficient to retain a reasonable quality of life. However, if bullous keratopathy develops, then the patient could well experience repeated periods of discomfort and ulceration, and the resultant scarring (vascularization, pigmentation and stromal opacification) can reduce vision further. Thus more severely affected patients can go blind from the disease, although thankfully this is not particularly common.

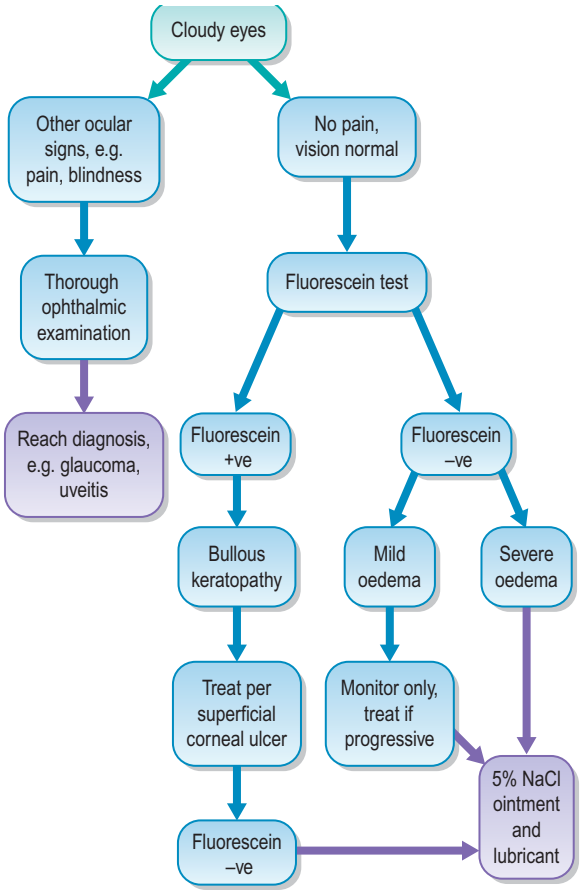


Figure 26.3 Decision-making tree – corneal oedema.

CASE EXAMPLE 26.1

Patient details: Labrador retriever, 10 years old, male

PRESENTATION AND HISTORY

The dog was presented for routine vaccination and the owners asked for his eyes to be checked as they felt that he was developing cataracts. Apparently the eyes had been gradually becoming cloudy over the previous few months. No other problems were reported and vision was not noticeably reduced.

CLINICAL SIGNS

General clinical examination revealed the dog to be slightly overweight with mild hip arthritis. Vision was good but both eyes were abnormal. Mild corneal oedema was noted in both eyes, worse in the right (Figure 26.4). The corneal surfaces were smooth and no ulceration was present in either eye, but a mottled blue–grey appearance was present. No significant intraocular changes were noted – just age-related nuclear sclerosis. Intraocu-

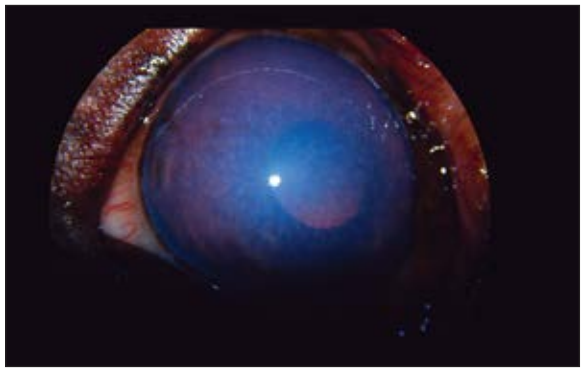


Figure 26.4 Right eye of 10-year-old Labrador with corneal endothelial degeneration.

lar pressures were not measured (but would be expected to be normal).

WORK-UP

The clinical examination allowed the diagnosis of corneal oedema due to endothelial degeneration to be reached, such that further work-up was not required.

TREATMENT

No treatment was deemed necessary since the eyes were comfortable and visual. However, the owners were warned that the dog could develop corneal ulcers in future and were advised to have him checked every 3–4 months.

OUTCOME

The oedema did gradually progress over the following 18 months and one episode of ulceration occurred in the right eye. This was debrided on three occasions and then a contact lens was placed for 3 weeks before healing was achieved. Following this the dog was placed on lubricating gel (carbomer gel, Viscotears, CIBA Vision) twice daily. The use of hypertonic sodium chloride was discussed but the owners were happy that the dog was comfortable with reasonable vision and did not want to have to medicate him as frequently as four times daily, given that it would not fully resolve the oedema.

This case shows that once the diagnosis has been reached there will be inevitable progression but frequently this does not cause the patient too many problems – the appearance of the eyes can be alarming but once owners are reassured that the cosmetic appearance is of little consequence to the dog they are generally happy to accept the situation.

27 Proliferative (eosinophilic) keratoconjunctivitis

INITIAL PRESENTATION

Abnormal appearance – red/cloudy

PRESENTING SIGNS

Proliferative (eosinophilic) keratoconjunctivitis occurs in cats (and horses) but not dogs.

Typically a young to middle-aged (4–9 years old) domestic short haired cat will be presented due to a change in appearance of one eye. The owners report a pink 'film' growing over the eye. Normally the cat is not uncomfortable, or shows only mild blepharospasm. The lesion has often been present for a few weeks but appears to be getting larger. A pale coloured ocular discharge might be reported.

CASE HISTORY

There is normally no history of a predisposing factor. However, it is not uncommon in cats which go out, and owners might think that the cat has possibly been involved in a fight and thus assume that a traumatic incident triggered the development of ocular signs. This is actually rarely the case. The cat might have had a history of previous cat 'flu but again this is not necessary for the condition to develop. Usually the lesion has been gradually worsening for a few weeks before the cat is brought to the surgery and it is the change in appearance of the eye which alerts the owner to a problem. The lack of apparent pain will frequently explain why there is a delay between the owner noticing the abnormality and presenting the cat. Sometimes a scant, pale, mucoid-type discharge is present, of which the owners have been aware. In some patients both eyes can be affected, although not normally in a symmetrical manner.

CLINICAL EXAMINATION

General clinical examination is usually normal. On ophthalmic examination menace responses are unaffected and pupillary light reflexes normal. Schirmer tear test readings can be normal, elevated or slightly reduced. Abnormalities are confined to the cornea and conjunctiva with intraocular contents being normal.

A pale pink mass, similar to granulation tissue, is seen extending across the cornea from the limbus. This is usually either at the temporolateral or ventronasal quadrant (Figure 27.1). The conjunctiva adjacent to the lesion is hyperaemic but no chemosis is usually present. A collection of white coloured tissue – almost globules or blobs – can be present on the surface of the pink mass, or adjacent to it. Some whitish discharge can accompany the signs. This looks similar to cottage cheese in consistency. There is normally corneal vascularization. Sometimes the nictitans membrane can be affected – particularly the bulbar side – so this should be examined. Ulceration is not usually a feature of the condition but care must be taken in interpreting fluorescein tests – the excess dye must be thoroughly flushed with sterile saline solution. Uptake of the green dye is quite possible on the pink fleshy tissue itself, often in tiny pinpoint patches, but not actually on the cornea – the epithelium usually remains intact.

If the condition is quite advanced, the pink tissue can extend all the way across the cornea and in these cases vision will be affected. The palpebral surface of the eyelids can be involved. Although normally initially unilateral, both eyes can be affected (especially in advanced or untreated cases) but usually to different degrees – the

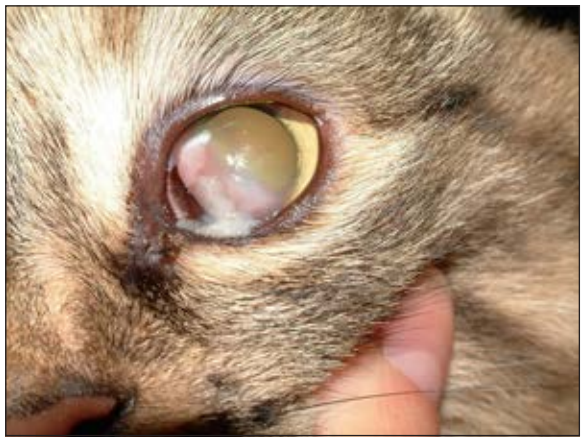


Figure 27.1 Proliferative keratoconjunctivitis in the left eye of a 6-year-old domestic short haired cat. Note the pink fleshy matter growing across from the ventromedial canthus and corneal vascularization. A white mucoid discharge is present but no increased lacrimation and minimal ocular discomfort is present.



Figure 27.2 Bilateral proliferative keratoconjunctivitis in a domestic short haired cat. The cat's brother was also affected but only in one eye. No studies have considered inherited tendencies to this condition.

owner might have only been aware of one eye being abnormal but on careful ophthalmic examination it is discovered that the other eye has more subtle lesions (Figure 27.2).

DIFFERENTIAL DIAGNOSES

- Corneal granulation tissue – healing ulcer
- Granulation reaction to corneal foreign body
- Chronic superficial keratitis
- Chronic herpetic keratitis
- Conjunctival lymphoma
- Keratoconjunctivitis sicca
- Corneal mass – squamous cell carcinoma (rare)

CASE WORK-UP

A full ophthalmic examination is obviously required to rule out other causes for the corneal infiltration – an infected ulcer or corneal foreign body for example. The white 'cottage cheese' discharge adherent to the granulation-type lesion is often considered pathognomonic but nonetheless further diagnostic tests are recommended to reach a definitive diagnosis. Cytology is the most appropriate test.

Under topical anaesthesia samples are collected from the lesion. This can be done either using a Kimura spatula (or more commonly the blunt end of a scalpel blade) or with a cytobrush. The collected material is gently pressed or rolled onto a microscope slide and allowed to air dry. Staining with Diff-Quik will allow rapid in-house examination of the sample although professional interpretation should also be considered. It is usual to see a collection of epithelial cells, mast cells, neutrophils, lymphocytes and, of course, eosinophils. However, the last are not necessarily abundant, and for this reason the condition can be called proliferative keratoconjunctivitis instead of eosinophilic! If eosinophils are present, it is usual to also find eosinophilic granules within the debris of the cytology samples as well as disrupted cells. Bacterial components are not normally a feature. However, if they are noted, a swab for bacterial culture and sensitivity should be taken.

In addition to cytology, a full haematology screen can be considered – a few cases do show a circulating eosinophilia.

Swabs can also be submitted for feline herpes virus (FHV-1) polymerase chain reaction (PCR). This is particularly important in cases where a history of upper respiratory infection or previous corneal disease (including ulceration) is present. Since the treatment of eosinophilic keratoconjunctivitis usually requires topical steroids, which can allow FHV-1 recrudescence, identification of this pathogen is particularly important.

NURSING ASPECTS

Nursing patients with eosinophilic keratoconjunctivitis is minimal and entails topical, and occasionally systemic, medication. Thus owners should be instructed how to apply the medication and with what frequency.

EPIDEMIOLOGY

The cause for eosinophilic keratoconjunctivitis is unknown. There is no sex or breed predisposition and affected cats are normally young to middle aged. The condition is seen in cats and horses, but has not been reported in dogs. The condition does not appear to be associated with the feline eosinophilic granuloma complex – only a few cats show a circulating eosinophilia and skin lesions are not present. A percentage of cats (up to 76% in one study) are positive for FHV-1 on PCR testing of conjunctival swabs or corneal scrapes. However, identification of the virus does not necessarily mean that it is involved in the disease – other clinical signs of recurrent herpetic lesions should be present. If they are, then treatment should be directed at both the FHV-1 and the eosinophilic keratoconjunctivitis.

The condition can be considered a chronic immune-mediated disease. This could be due to a type I hypersensitivity reaction mediated via IgE or mast cell and eosinophil degranulation, or could represent a type IV reaction mediated via sensitized T lymphocytes and stimulation of local eosinophilic reactions.

CLINICAL TIPS

- Careful examination following fluorescein dye is required (ideally using blue light) to determine whether corneal ulceration is truly present or whether the fluorescein merely stains the granulation-type tissue
- Check beneath the nictitans membrane for further deposits
- Check both eyes thoroughly – the condition can be bilateral
- Consider cytology and FHV-1 PCR as part of diagnostic work-up

ANATOMY AND PHYSIOLOGY REFRESHER

The cornea is normally totally avascular. Blood vessels can grow from the limbus bringing with them inflammatory mediators which are usually present in abundance in the conjunctiva. Leucocytes are often the source of angioblast and fibroblast growth factors. Once an inflammatory reaction has been triggered it can self-perpetuate, leading to the clinical appearance described above.

TREATMENT OPTIONS – MEDICAL

The treatment of eosinophilic/proliferative keratoconjunctivitis is medical rather than surgical. Most cases respond well to topical steroids. Thus it is important to establish whether true corneal ulceration is present before prescribing these. Prednisolone acetate or dexamethasone is normally chosen, either in drop or ointment formulation. The frequency of application will clearly depend on the severity of signs – usually beginning with three or four times daily application and gradually reducing over time depending on the response to treatment.

Since the disease is immune mediated, long-term medication is frequently required. As such, it is good practice to warn the owners of this at the outset. Once the condition has reached remission – noted by clearing of the cornea and disappearance of the white deposits – the frequency of medication can be reduced to once daily, then every other day. Careful ophthalmic examination will often reveal the presence of 'ghost vessels', i.e. the empty tracts where the vascularization had been present. Withdrawal of anti-inflammatory medication too soon will allow these to rapidly re-cannulate with a flare-up of the condition.

In some cats it is possible to stop treatment completely after several months, but again owners need to be aware of the possibility of further episodes of disease. These might not develop for months to years. As such, episodic treatment is acceptable in some cases. Others require life-long low dose topical treatment to keep the condition in remission. The risks of corneal ulceration should be explained to the owners such that the medication is stopped should any of these signs develop and veterinary attention is sought.

An alternative to topical steroid use is ciclosporin. This does not seem to be as effective as steroids in the early stages of the disease; however, once the clinical signs

have subsided it can be effective for long-term control. Topical NSAIDs do not seem to be effective.

Some cases do not respond well to topical steroids alone, or the owners might find frequent topical medication difficult. In this situation systemic steroids can be used. Prednisolone at a dose of 5 mg twice daily for 3–5 days then once daily for a couple of weeks before reducing to the lowest alternate day medication necessary to keep the condition at bay is normally sufficient for most cats. Alternatively, the systemic medication can be used until the condition has clinically resolved, before switching to a low dose topical medication for maintenance. Megestrol acetate has been used effectively historically but due to the more severe potential side effects of this drug it is rarely recommended today.

As mentioned previously, FHV-1 infection can sometimes be present along with the eosinophilic keratoconjunctivitis. If this is the case, topical steroids can bring on a recrudescence of the herpetic keratitis with dendritic or superficial corneal ulcers, and occasionally respiratory signs as well (particularly if systemic steroids are used). In cats with a known history of herpetic keratitis it is sensible to medicate with topical antiviral agents (such as trifluorothymidine) at the same time as the topical steroids. This should reduce the risk of further acute herpetic outbreaks. Management of these cases can be complicated and it is sensible to discuss this with your referral ophthalmologist.

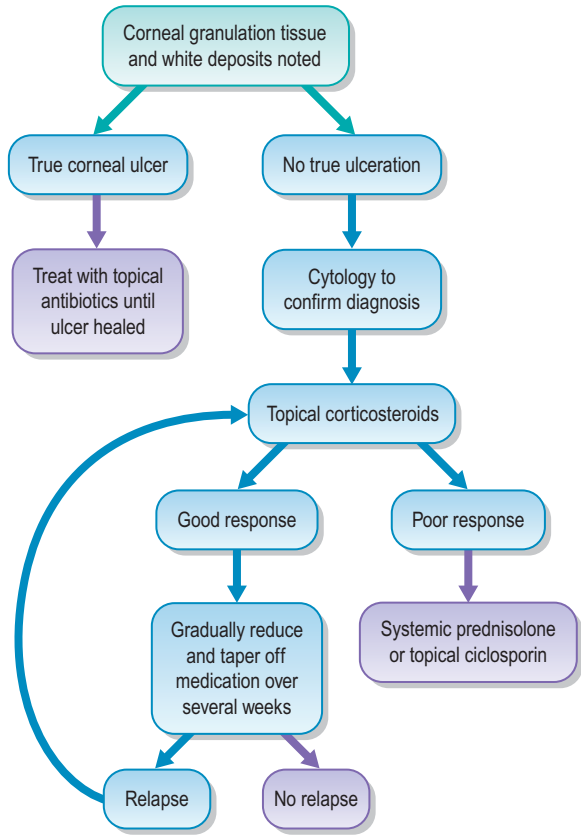


Figure 27.3 Decision-making tree – proliferative keratoconjunctivitis.

TREATMENT OPTIONS – SURGICAL

Surgery is not usually required for this condition. The only exception would be the case that did not respond as expected. In this situation a superficial keratectomy could be performed to remove the tissue, and also to submit it for histopathology in order to confirm the diagnosis.

PROGNOSIS

The prognosis for eosinophilic/proliferative keratoconjunctivitis is generally good, providing the owners are informed at the outset that the condition might require long-term treatment to prevent relapses. Most cases respond very well to topical medication, with a clinical resolution of signs within 4–6 weeks. Some cases can have the medication completely withdrawn after further weeks to months at reducing doses. Complete resolution with no recurrences is possible but less common. More frequent are the cases which respond very well but suffer from periodic episodes of clinical disease. Usually when a flare-up occurs the owners will notice this more quickly such that the condition is less severe than when the cat was initially presented and, as such, responds well to lower levels of medication. If the relapses are frequent, then long-term, low dose medication is advised.

CASE EXAMPLE 27.1

Patient details: Domestic short haired cat, 7 years old, male (neutered)

PRESENTATION AND HISTORY

The owner presented the cat because she noticed that his right eye was cloudy and pink in appearance. This had been present for about 3 weeks and was worsening. No discomfort was reported and the cat was clinically well. The cat did go out and had been a fighter in his younger days – the owner thought that perhaps he had been scratched in the eye. No treatment had been given.

CLINICAL SIGNS

Abnormalities were confined to the right eye. The cornea was opaque with a pinkish discolouration and multiple branching corneal vessels. Small white globular deposits were adherent to the corneal surface. Some conjunctival hyperaemia was noted and the lateral lower lid margin was slightly inflamed. No ulceration was present and only a scant mucoid discharge was noted (Figure 27.4).

WORK-UP

Topical anaesthesia was administered and cytology samples were collected using the blunt end of a scalpel blade scraped gently across the lesion laterally. These were spread onto a microscope slide, dried and stained with Diff-Quik. Multiple damaged epithelial cells, neutrophils and lymphocytes were noted, together with scant mast cells and the occasional eosinophil. The diagnosis was proliferative/eosinophilic keratoconjunctivitis.



Figure 27.4 Right eye of 7-year-old domestic short haired cat showing corneal opacity, vascularization and multiple small white corneal deposits. Mild eyelid inflammation and conjunctival hyperaemia are also present.

TREATMENT

The cat was placed on topical dexamethasone (Maxidex, Alcon) four times daily and reassessed in 7 days. The owner was warned to stop the drops and return immediately should any signs of ulceration develop – blepharospasm, increased lacrimation and attempts at rubbing, especially after application of the drops. On the second check the eye had improved well and the drops were reduced to three times daily for 2 weeks and then by a drop every further 2 weeks and finally stopped.

OUTCOME

After the first course of treatment the cat was fine for 3 months. However, the owner noticed that the white lesions returned and brought him back. Several white lesions were present along with fine corneal vascularization. The drops were restarted at three times daily but the cat became difficult to treat – he would run away from the owner whenever she approached and took to staying out most of the time. The eye deteriorated as a result and so treatment was changed to prednisolone tablets in his food, 5 mg daily for a week then 2.5 mg daily for another 3 weeks. Re-examination confirmed complete resolution with just faint ghost corneal vessels remaining. Prednisolone was continued at 2.5 mg every other day for another 2 weeks and then stopped, but replaced with dexamethasone drops every other day instead. The owner could manage this and the cat was maintained on this for 3 months before stopping treatment completely. The owner was aware that further flare-ups could occur.

This case shows that although a good initial response to treatment is frequently seen, cats can suffer from more than one episode of inflammation and longer treatment courses are sometimes necessary.

28 Brachycephalic pigmentary keratitis

INITIAL PRESENTATION

Abnormal appearance – cloudy, blind

PRESENTING SIGNS

Brachycephalic pigmentary keratitis can present in a variety of ways. The most straightforward is where the owner has noticed a film appearing over the dog's eyes with an apparent reduction in vision – particularly for things immediately in front or beneath them. Sometimes there has been recurrent ulceration and thus the dog could have a sore, watery red eye as the reason for seeking veterinary advice. Alternatively, the dog could be presented with a sticky ocular discharge. Normally dogs are young to middle aged and in good general health.

CASE HISTORY

It is not uncommon for a history of previous ulceration to have occurred in one or both eyes. The owners might have been aware of the change in appearance of the eyes associated with a dark film across them. Some increased lacrimation with tear overflow might have been noted; alternatively, the eyes could be slightly dry with a mucoid or mucopurulent discharge. The condition is chronic in nature, and it is possible that the owners were unaware of current problems – perhaps the dog was brought in for routine vaccination and the ocular abnormalities were found incidentally.

CLINICAL EXAMINATION

General clinical examination is usually normal. However, multiple, often quite subtle, abnormalities are detected on ophthalmic examination. The most striking feature is a film of dark pigment extending over the corneas. This is usually bilateral, but not necessarily symmetrical. It is normally most dense ventromedially and extends in a triangle with the apex centrally (Figure 28.1).

However, other less obvious changes are likely to be present. The dog can probably see, so menace responses will be present, but these might be reduced, especially if directing the threatening gesture from a ventral or ventromedial direction. Palpebral reflexes should be checked – they are likely to be normal in that the animal will blink, but the eyelids might not fully meet on blinking due to some degree of lagophthalmos. Blink rates might be reduced and incomplete or partial blinks are not uncommon. It is sensible to ask the owners whether the dog sleeps with his eyes fully closed – sometimes they do not. Schirmer tear test readings should be evaluated. These could be normal, increased (especially if corneal ulceration is present) or reduced. Low grade keratoconjunctivitis sicca is not infrequent in brachycephalic dogs.

Careful evaluation of the lid margins should be undertaken. A ventromedial entropion is often present. The eyelid margins will appear tight against the cornea, with hairs from the lid touching the corneal surface. The third eyelid is not normally elevated or providing any corneal protection. Hairs are often also present on the caruncles – the small fleshy protuberances at the medial canthi – and these can both irritate the cornea and act as a wick to draw tears down the face rather than down the nasolacrimal punctae.

Nasal fold trichiasis can be present. Here the hairs from the skin over the nasal folds are in contact with the corneal surface. Viewing the patient from the side and above as well as directly in front will help to determine whether significant trichiasis is present. Close evaluation



Figure 28.1 Bilateral pigmentary keratitis in a young Pug. Note the ventromedial pigment in both corneas.



Figure 28.2 Ten-year-old Shih Tsu with pigmentary keratitis. Marked corneal scarring is present from previous ulceration and trichiasis from hair at the medial canthus is present. Subclinical distichia are visible on the upper lid. Tear production was normal. This patient was treated with just topical lubrication, including soft white paraffin ointment onto the medial canthal hairs to smooth them away from the cornea and reduce the irritation.

of the nasolacrimal punctae should be performed. These are normally present, and of normal size, but the medial entropion causes the openings to be physically closed (like a straw flattening on itself). This will contribute to any epiphora. The skin ventromedial to the eye should be checked – the epiphora could allow a low grade moist dermatitis to develop here. Distichia can be present – commonly in English bulldogs for example – but as these are often subclinical they must be carefully examined to see if they are actually in contact with the cornea or the tear film and are truly contributing to the corneal disease, or whether they are purely an incidental finding (Figure 28.2).

Conjunctival hyperaemia is not a consistent finding – it will be present if there is concurrent corneal ulceration but otherwise the conjunctiva is often normal. Detailed corneal examination is required. The pigment is normally superficial but can be quite thick in places. It will almost always be associated with some degree of corneal vascularization – this is best appreciated at the leading edge of the pigment since the dark colouration will obscure any blood vessels beneath it. If ulceration has occurred previously it is most common centrally in brachycephalics and so some white stromal scarring and/or irregular corneal outline might be present in this area. Active ulceration can be shallow or quite deep with a marked vascular reaction and all the signs typically present with any ulceration. It goes without saying that fluorescein testing is mandatory. As usual, the excess dye should be carefully flushed away using sterile saline solution – the surface of the cornea is likely to be irregular due to the pigment and previous scarring, and pooling of dye in these patches could lead to false positives.

Although intraocular contents are usually normal, evaluation of them can be difficult. Distant direct ophthalmoscopy can be used to assess pupil size and gain a tapetal reflex (albeit reduced). Indirect ophthalmoscopy offers the best way of evaluating the fundus since the view through semi-opaque ocular media is superior with this method over direct ophthalmoscopy.

DIFFERENTIAL DIAGNOSES

- Corneal ulceration
- Keratoconjunctivitis sicca
- Other corneal pigment – endothelial from iris cysts, persistent pupillary membrane remnants or chronic hyphaema
- Distichiasis
- Nasal fold trichiasis
- Medial entropion
- Chronic superficial keratitis
- Limbal melanoma
- Pigmentary glaucoma (pigment dispersal syndrome)

CASE WORK-UP

Once a thorough ocular examination has been performed to determine the underlying reason for the pigment deposition, then a management plan can be

drawn up. This will probably include both surgical and medical options and if the former is indicated a full general examination should be undertaken. Particular attention should be paid to the presence of other brachycephalic problems – notably upper airway conditions – which might require surgery as well as the eyes, or at least should be borne in mind when planning general anaesthesia. Apart from this, there is limited work-up necessary since the ophthalmic examination should be sufficient to reach a definitive diagnosis.

NURSING ASPECTS

The patient is likely to require both surgical and medical management. Thus the nurse will be involved perioperatively in patient preparation, anaesthesia and surgical recovery. Extensive treatment prior to surgery is not usually necessary unless some bacterial skin infection is present associated with the nasal folds for example, in which case a course of systemic antibiotics might be required as well as appropriate topical medication. Once anaesthetized, the nurse will be required to assist surgical preparation and positioning in theatre, along with monitoring the anaesthetic.

Careful monitoring is required during recovery with brachycephalics in particular due to their compromised upper airway anatomy. Prevention of self-trauma is required and sometimes an Elizabethan collar might be necessary. Postoperatively, topical and systemic medication is usually necessary, the former sometimes long term, and nurses are invaluable in instructing clients about this and checking their proficiency at medicating their pet.

EPIDEMIOLOGY

The epidemiology of chronic corneal pigmentation in brachycephalics is complicated. The pigment itself is derived from limbal or perilimbal tissues and the melanocytic cells migrate into the cornea with the vascularization. In addition, some melanin pigment can be deposited from fibroblasts and macrophages. The migration of pigment cells is seen alongside the vascularization but other signs of active inflammation such as stromal inflammatory cell infiltration and granulation tissue deposition can also be present.

Thus a chronic low grade inflammation is necessary to allow the pigment to migrate and be deposited. This is normally due to more than one factor and is associated with the brachycephalic conformation. The shape of the orbit, with its flattened, shallow profile (I describe it to owners as being shaped more like a teaspoon than the ice-cream scoop of mesaticephalic breeds) leads to lagophthalmos. The large palpebral fissure also results in chronic exposure. Tear film dynamics can be poor – even if Schirmer tear test readings are normal it is possible for the tears not to be spread across the corneal surface properly due to the exposure of the globe and ineffective blinking. This can lead to corneal drying in the central region and associated ulceration. Dogs which do not fully close their eyes when sleeping are particularly susceptible.

Eyelid anatomy in the medial canthal region contributes as well, with low grade entropion and trichiasis causing chronic irritation to the ventromedial cornea. Hairs on the caruncles also cause irritation and wick the important tears away from the tear film and down the face, further reducing the efficacy of lubrication. Distichiasis, aberrant cilia from the meibomian orifices along the lid margins, can irritate and trigger an inflammatory reaction on the cornea. Further incriminating hairs are those on the nasal folds. Thus trichiasis – hairs in a normal position which are causing problems – can exacerbate as well.

In addition to poor tear film dynamics, keratoconjunctivitis sicca might be present. Chronic keratitis, with or without ulceration, is common and the history of recurrent ulceration or repeated infections should alert one to this possibility.

Consequently, the development of pigmentary keratitis is usually multifactorial and evaluation of the various contributory factors is required to provide the best treatment for the patient.

CLINICAL TIPS

- Assess the efficacy of blinking – watch the dog for a few minutes to determine whether the blink rate is normal and whether each blink is complete or if partial ones occur
- The presence of distichia does not necessarily mean that they are clinically significant – see Chapter 7
- View the dog from above and the side as well as in front to determine globe position and whether nasal fold trichiasis is relevant
- Flush fluorescein dye to prevent false positives on the irregular cornea
- Use indirect ophthalmoscopy to evaluate intraocular contents (especially the fundus)

ANATOMY AND PHYSIOLOGY REFRESHER

Since brachycephalic corneal pigmentation is due to a combination of factors, a full review of all aspects is beyond the scope of this section. However, the typical anatomy of these breeds does play a crucial role in the disease. The shallow orbit and large palpebral fissure contribute to globe exposure (lagophthalmos) and poor spread of tears across the prominent globe is exacerbated by reduced blinking – both decreased rates and incomplete blinks. Aqueous tear production can also be reduced. Tears are spread across the cornea during blinking and also propelled towards the nasolacrimal punctae. If the latter are physically closed due to entropion, then the tears will escape over the face. The presence of aberrant hairs on the caruncles will encourage this further.

The cornea of brachycephalics has been shown to have reduced numbers of trigeminal nerve endings compared to mesaticephalic breeds. As such, they can develop dry patches or even overt ulceration without 'feeling' them and showing signs of discomfort. As a result of this, any ulceration can progress quite rapidly before the dog demonstrates any signs to the owner. Eyelid and cilia defects can cause low grade irritation which allows for a low level of inflammation to develop, leading to further pigment deposition.

TREATMENT OPTIONS – MEDICAL

If corneal ulceration is present this must be treated accordingly – often with aggressive topical medication. However, the long-term approach to the problem of pigment deposition requires both medical and often surgical approaches. Surgery aims to correct predisposing factors; medication aims to reduce the inflammation and improve tear dynamics to limit the tendency for further pigment deposition. Without improving any anatomical defects, medical management alone is unlikely to be effective long term.

Since inflammation is always present and vascularization is the source of the pigment, anti-inflammatory agents applied topically are effective. Providing no ulceration is present, topical corticosteroids can be used. Dexamethasone once or twice daily for several weeks can cause the superficial blood vessels to shrink and prevent further pigmentation but will not reverse that which is already present. However, as the underlying inflammation is reduced an impression of pigment thinning is sometimes seen. Long-term use of steroid is not recommended but intermittent courses might be required.

Even if tear production seems adequate, topical lubricants with carbomer gels or liquid paraffin ointments are beneficial. They will help to keep the central cornea moist such that the stimulus for further keratitis is reduced. If tear production is low, then topical ciclosporin is required, usually twice daily. Early treatment with this when Schirmer tear test readings are 12–15 mm/min will reduce the severity of the keratoconjunctivitis sicca. In addition, long-term ciclosporin has been shown to slowly reduce the amount of pigment present (more so than can be explained by the anti-inflammatory action alone). For this reason it can be used even in cases where aqueous tear production appears adequate and no overt lagophthalmos is present. However, the high ongoing financial cost of this must be weighed against any low grade clinical improvement.

TREATMENT OPTIONS – SURGICAL

Several different surgical procedures can be performed to improve the anatomy of these patients and thus reduce the pigment deposition. Aiming to prevent further pigment is preferable to removing that which has already built up as will be discussed later. The most common and effective procedure is the medial canthoplasty. This has the effect of correcting any medial entropion, removing the caruncular trichiasis and reducing the palpebral fissure, thus minimizing the lagophthalmos all in one operation. Owners must be warned that the

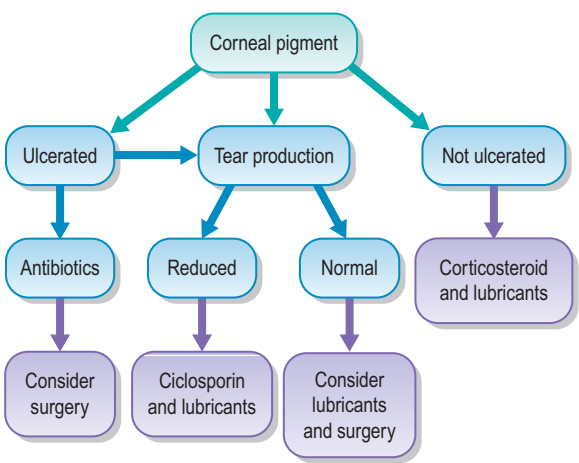


Figure 28.3 Decision-making tree 1 – brachycephalic pigmentary keratitis.

surgery will alter the appearance of their pet – the eyes will look smaller and further apart – but by emphasizing the benefits of reduced ulceration and maintaining vision, most owners are happy to proceed.

It is usual to perform the procedure bilaterally. Magnification makes the surgery much easier and more accurate. If this is not available, or the surgeon is not experienced with ocular surgery, then referral should be considered. If this is not possible for various reasons then it might be safer for the general practitioner to perform a lateral canthoplasty instead, although this procedure only addresses the lagophthalmos and not the medial canthal abnormalities.

The most straightforward method for medial canthoplasty is as follows:

- Under general anaesthesia a strip of skin is removed from the upper and lower lids at the medial canthus.
- A No. 15 scalpel blade is used to make the incision, ensuring that the lid margin is incised at right angles, and small sharp scissors are used to cut away the entire lid margin.
- The caruncle is fully excised along with the abnormal hair follicles it contains. Care should be exercised to avoid the nasolacrimal punctae – cannulation with coloured nylon will alert the surgeon to their location.
- The canaliculi must also be avoided – cutting too deeply around the caruncle could damage them and so this is why coloured nylon is advised to render the location of the ductules more obvious.
- The wound is closed in a two-layered manner using a figure-of-eight suture at the newly created medial canthus – both to attain good lid alignment and to avoid a suture knot close to the cornea.
- Suitable material is 6/0 polyglactin 910 (Vicryl, Ethicon) which, although braided, is soft and dissolvable such that sedation for suture removal is rarely required (Figures 28.4 and 28.5).

A lateral canthotomy is less frequently performed since it does not address the problems at the medial canthus. However, the surgery is less difficult since access to the site is easier and there is no need to worry about punctae! The surgery can be performed in general practice without the necessity for magnification. Sharp incision delineates the area of lid to be removed which is then cut out using scissors. A two-layered closure with a figure-of-eight suture at the new lateral canthus is recommended as for the medial canthoplasty. The length

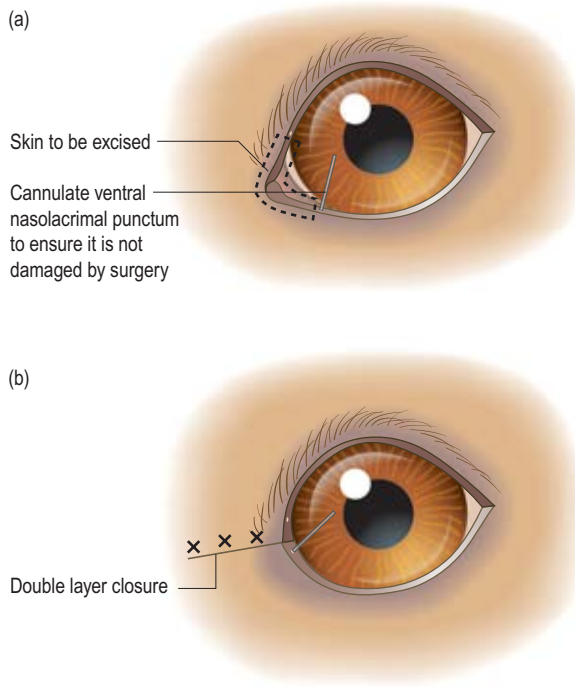


Figure 28.4 Procedure for medial canthoplasty.



Figure 28.5 Same dog as Figure 28.1 immediately after surgery – the palpebral fissure is reduced and the medial entropion has been corrected via a medial canthoplasty.

by which the lid can be shortened is greater laterally than medially (due to the lack of punctae at this location) and sometimes it can be combined with a medial canthoplasty to reduce the palpebral fissure further.

If nasal fold trichiasis is present, a nasal fold resection is advised. Mild cases can be treated just with lubrication

of the nasal hairs (using ophthalmic liquid paraffin, not Vaseline). However, surgery is a better option long term. Under general anaesthesia the offending ridge of skin is simply excised and a double layer closure performed. Skin sutures should be soft and their ends must not contact the cornea. Since low grade dermatitis can be present in the nasal fold it is sensible to use perioperative systemic antibiotics. Owners should be fully aware that the appearance of the dog will be altered by the surgery.

Owners often ask if the pigment can be simply removed. The answer to this is yes, but it is not a procedure to be undertaken lightly. A superficial keratectomy can be performed to strip away all affected tissue. This will create a large, hopefully fairly superficial, corneal defect which is treated like an ulcer. However, healing is likely to involve vascularization – not just epithelial sliding and mitosis – and as such the likelihood of more pigment being deposited is high. The cornea can repigment in a matter of weeks such that the outcome is often disappointing. In addition, corneal healing in brachycephalics can be delayed and the risk of infection complicating things further is quite high. This surgery is not recommended unless other remedial surgery is also performed, and is generally only advocated for advanced cases where the dog is blinded by the pigment. It is not advised if tear production is reduced.

Whichever surgical procedure is undertaken, frequent monitoring and long-term medical management are required.

PROGNOSIS

The prognosis for corneal pigmentation in brachycephalics is slightly guarded. Since the condition is insidious in onset, it can often be quite advanced before dogs are presented. Owners can be reluctant for extensive surgery if the patient is not showing overt clinical problems. Even by explaining that prevention is more successful than attempts at treating advanced corneal disease, some owners are unwilling to consent to surgery until the dog has had several nasty ulcers and excessive scarring has developed. If ulceration is not a prominent feature, the pigment can gradually creep up over the corneal surface as a dark 'water line'. Vision deteriorates slowly – likened to wearing progressively darker sunglasses – until functional blindness ensues. By operating on these patients early the prognosis for preventing further pigment and other problems associated with the lagophthalmos such as exposure keratitis and ulceration is much better. Ideally, affected dogs should not be used for breeding and we should encourage breeders of Pugs, Pekingese, Boston terriers, French and English bulldogs and so on to avoid choosing such exaggerated anatomy as a desirable trait.

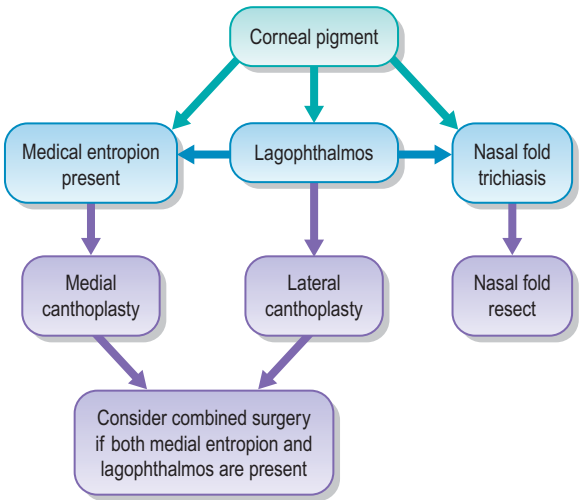


Figure 28.6 Decision-making tree 2 – surgical decision for corneal pigment in brachycephalics.

CASE EXAMPLE 28.1

Patient details: Pekingese, 10 months old, male

PRESENTATION AND HISTORY

The dog was presented as an emergency with a red, painful right eye. The owner had been away for the weekend and the friend who was looking after the dog had said that the eye was a bit red but the dog did not seem worried by it. The dog had already undergone enucleation of the left eye following severe ulceration.



Figure 28.7 Ten-month-old Pekingese with severe lagophthalmos, corneal ulcer, nasal fold trichiasis and ventromedial corneal pigment.



Figure 28.8 Same dog as in Figure 28.7, 1 month following surgery (combined medial and lateral canthoplasty plus nasal fold resection). Blinking is now effective and the corneal ulcer is fully healed although moderate pigment remains.

CLINICAL SIGNS

The left eye was absent. The right eye showed lagophthalmos with a very large palpebral fissure and exposed sclera. Blinking was frequently incomplete with a 3 mm gap left centrally during many blinks. A large central corneal ulcer was noted with dorsal vascularization from the limbus. The prominent nasal fold was in contact with the ulcerated portion of cornea. Significant ventromedial corneal pigment was also present. The pupil was of normal size with no apparent uveitis. Schirmer tear test reading was 18 mm/min (Figure 28.7).

WORK-UP

General clinical examination revealed stenotic nares but no other significant abnormalities.

TREATMENT

Under general anaesthesia the patient underwent both a medial and a lateral canthoplasty and the nasal fold was also resected. It was decided that a medial canthoplasty alone would provide insufficient lid shortening to provide corneal protection and improve tear dynamics, and so the lateral canthus was also shortened. The owner was fully aware that the dog's appearance would be changed but sensibly was most concerned that the dog did not lose its only remaining eye. Postoperative treatment was with topical gentamicin four times daily, carbomer gel and systemic cefalexin.

OUTCOME

The surgery was successful. The ulcer healed well and the vascularization subsided. Blinking was effective and a good tear film could be seen (Figure 28.8). Moderate pigment remained but did not compromise vision and no further episodes of ulceration developed. The pigment actually reduced over the following 6 months.

This case shows that radical surgery is sometimes required to salvage these globes. The owner was upset that this had not been offered when the dog developed the initial ulcer in the first eye and it is impossible to tell whether the globe could have been saved had the attending veterinary surgeon been fully aware of the anatomical defects which contributed to the ulceration.

29 Chronic superficial keratitis

INITIAL PRESENTATION

Abnormal appearance – red

PRESENTING SIGNS

Dogs are presented with a change of appearance – a redness – in one or both eyes. This is normally gradual in onset. German shepherd dogs are most frequently presented, although greyhounds and lurchers, Belgian shepherd dogs and Border collies are also seen together with any other breed on occasion. The dog does not usually show any signs of ocular discomfort although a scant mucoid discharge can be present. More dogs are presented in the summer than winter.

CASE HISTORY

Sometimes there is a history of previous ocular disease, an earlier episode of a similar problem, but usually dogs are presented the first time they develop the condition and as such there is no relevant history. Dogs tend to be clinically well.

CLINICAL EXAMINATION

General clinical examination is normal. On ophthalmic examination one or both eyes can be affected, but the condition is rarely symmetrical. The most striking lesion is a fleshy growth of tissue on the cornea. It normally starts in the ventrolateral or occasionally dorsolateral quadrant, with its base at the limbus growing centrally (Figures 29.1 and 29.2). The lesion is usually pink, but can be darkly pigmented. It appears vascular. The cornea at the leading edge frequently has patchy white deposits within the anterior stroma which can be cellular infiltrates or lipid. No fluorescein uptake occurs (although false positives are common on the raised fleshy tissue and copious flushing with sterile saline is required to show that these areas are not ulcerated). The conjunctiva is hyperaemic in the adjacent bulbar conjunctiva, and sometimes this can extend around the circumference of the globe. The nictitating membrane can be involved with thickening (with a cobblestone appearance) and depigmentation of the leading edge in about 10% of dogs. Vision can be reduced but unless the condition is very severe animals are not normally blinded. Schirmer tear test readings are normal. Intraocular contents are unaffected. Sometimes a mucoid or mucopurulent discharge is present.

DIFFERENTIAL DIAGNOSES

- Healing corneal ulcer with granulation tissue
- Keratoconjunctivitis sicca
- Interstitial keratitis
- Pigmentary keratitis
- Nodular episcleritis

CASE WORK-UP

Since the signalment and clinical signs are usually sufficient to reach a diagnosis of chronic superficial keratitis, or pannus, it is rarely necessary to perform any further work-up. However, in atypical cases cytology is diagnostic. Under topical anaesthesia (assuming the patient's temperament allows!) superficial cell samples are collected using the blunt end of a normal scalpel blade, special Kimura spatula or a cytobrush. These are gently rolled onto a microscope slide, air dried and submitted for evaluation.

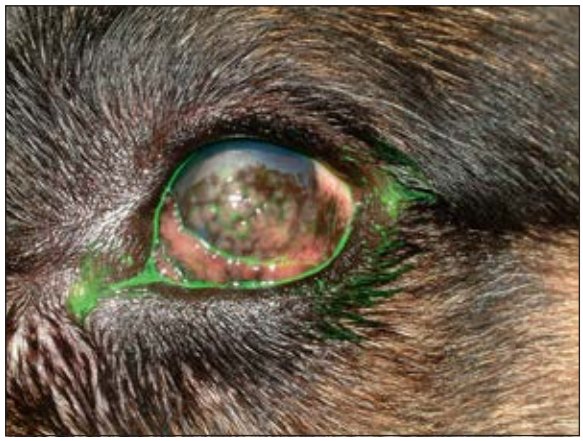


Figure 29.1 Chronic superficial keratitis (pannus) in a 6-year-old German shepherd dog – left eye showing corneal granulation tissue, cobblestone appearance and pigment deposition. Note third eyelid involvement.



Figure 29.2 Right eye of dog in Figure 29.1 with lesions confined to the nictitans membrane (nictitans plasmacytic conjunctivitis).

NURSING ASPECTS

Patients will require frequent topical medication and nurses can be invaluable in teaching owners how to apply the drops or ointment and in encouraging owner compliance.

EPIDEMIOLOGY

Chronic superficial keratitis (CSK), or pannus, is the subepithelial proliferation of blood vessels and connective tissue and the term implies a bilateral superficial keratitis which has an immune-mediated aetiology. Since certain breeds are predisposed to developing the condition (namely German shepherd dogs far more frequently than any other breed), a genetic predisposition is proposed. In addition, the condition is more common, and more severe, in areas of high altitude and long hours of bright sunlight. Thus ultraviolet light is a contributing factor and it is possible that the UV light alters certain corneal antigens in animals with a genetic predisposition and thus triggers the disease. Young adult dogs are affected mostly (3–5 years) although some patients are older before the symptoms develop. The condition is generally more benign in older dogs, and can be controlled with less intensive treatment than in younger animals.

Histologically, the disease is characterized by neovascularization and fibroplasia with a mixed cellular infiltrate consisting of plasma cells, lymphocytes, macrophages and melanocytes. The proportion of these cells varies and is a reflection of the clinical picture – some cases of CSK are more heavily pigmented than others for example. The corneal epithelium remains intact, with no ulceration, but it does alter in structure, becoming thin in some places and hyperplastic in others. Occasionally the pathology is confined to the nictitating membrane and it is then termed nictitans plasmacytic conjunctivitis or plasmoma, rather than chronic superficial keratitis. The underlying pathological mechanisms are identical and it is not understood why some dogs develop one form and not the other.

CLINICAL TIPS

- Carefully check both eyes
- Flush away excess fluorescein to avoid false positives
- Always perform a Schirmer tear test

TREATMENT OPTIONS – MEDICAL

Medical management is the option of choice for patients with CSK. This is usually topical and corticosteroids are the first line of treatment, although ciclosporin is also effective in most dogs. Dexamethasone alcohol or prednisolone acetate are used six to eight times daily in severe cases, four times daily in moderate cases, usually for 2–4 weeks until a good improvement is achieved, when they can be gradually reduced to twice daily (see Table 29.1). Ciclosporin has the advantage of only twice

Table 29.1 Treatment choices for chronic superficial keratitis (CSK)

	Severe CSK	Moderate CSK	Mild CSK
Initial treatment for 2–4 weeks	Dexamethasone six times daily plus ciclosporin twice daily	Dexamethasone four times daily *or* ciclosporin twice daily	Dexamethasone twice daily
Follow-on treatment for 4–8 weeks	Dexamethasone four times daily Ciclosporin once daily	Dexamethasone twice daily *or* ciclosporin once daily	Dexamethasone once daily
Long-term maintenance	Either dexamethasone or ciclosporin once or twice daily	Dexamethasone once or twice daily *or* ciclosporin once daily	Dexamethasone every other day

daily application, but this is countered by its expense. It is also slightly more effective in reducing any pigment deposition which accompanies the pannus. In severe cases both drugs can be used. The choice of drug and frequency of application should be tailored to each patient; life-long low levels of medication are usually necessary to prevent recurrence.

The condition can worsen in conditions of bright light – summer or snowy winters for example – and owners should be aware of this. Keeping the dog out of bright light, walking them at dawn and dusk rather than in full daylight will help. For severely affected patients a form of dog sunglasses (Doggles, from www.doggles.com) is available. These are popular in the USA and Australia but are rarely necessary in the UK under current climate conditions!

If the patient is not amenable to frequent topical medication, or the owner is unable to apply it, then subconjunctival steroid injections offer a reasonable alternative initially. Betamethasone, triamcinolone and methylprednisolone can all be used to 'kick start' the anti-inflammatory process; however, for long-term control, topical treatment is preferable.

TREATMENT OPTIONS – SURGICAL

Occasionally surgery is advocated for the initial treatment of CSK. This entails a superficial keratectomy under general anaesthesia and referral for this should be considered unless the appropriate instrumentation and surgical expertise are present within the practice. It is particularly appropriate as a last resort in cases with severe corneal pigmentation since this is very slow to clear with topical medication. It is not unusual for the entire epithelium and anterior stroma to be removed in such cases. The corneal wound heals rapidly, usually with excessive granulation tissue, and the judicious use of topical steroids is required to reduce this as soon as epithelialization has occurred. Topical ciclosporin and systemic NSAIDs are also required in some patients. Unfortunately, unless the surgery is followed with diligent topical medication, its success will only be short lived.

In addition to surgical keratectomy, cryotherapy and beta irradiation have been used to remove the superficial pigment – but again they cannot replace the topical management.

PROGNOSIS

The prognosis in mild and moderate cases of CSK is good for remission and control of the disease but owners must be made aware that although good control is possible, a complete cure is not. Thus life-long medication

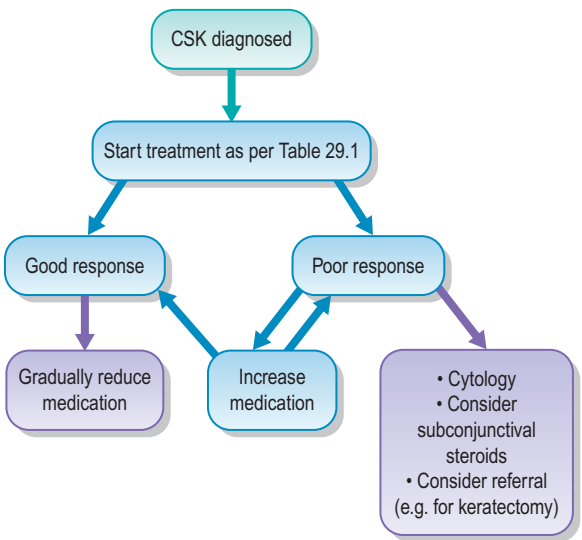


Figure 29.3 Decision-making tree – chronic superficial keratitis (CSK).

is required in most dogs, and the frequency of this might need varying. In some patients the agents used might be changed from time to time as well. The risk of corneal ulceration is present with chronic topical steroid use, but systemic side effects are very rare given the minimal systemic absorption in these large breed dogs. Most lesions will regress well, leaving a small amount of pigment and sometimes lipid or stromal infiltration at the ventrolateral limbus which does not affect vision at all. Unfortunately, owner compliance is sometimes poor and these dogs can have a rollercoaster effect with periods of relative remission and severe flare-ups in between when the owner stops treating. Some dogs can go blind as a result.

CASE EXAMPLE 29.1

Patient details: German shepherd dog, 8 years old, male (entire)

PRESENTATION AND HISTORY

The dog was presented due to severe red eyes and blindness. It had been diagnosed with chronic superficial keratitis 3 years previously and had been prescribed multiple topical agents with variable success. Unfortunately, both owner and patient compliance were lacking. The dog was left for 14 hours a day and no one was able to medicate the dog during this time. The only member of the family who could apply medication was the father (the dog tried to bite his wife and children) and often he would forget to medicate. Ongoing costs of medication were also an issue.

CLINICAL SIGNS

The dog was blind. Severe mucopurulent discharges were noted bilaterally such that the eyelids were stuck together and on gently bathing these away some bleeding occurred from the corneas and eyelid margins. Very severe corneal granulation tissue was noted in both eyes (Figure 29.4) and the nictitans membranes were inflamed and swollen. Mild periorbital hyperaemia was present. Schirmer tear test readings were 16 mm/min in both eyes. Intraocular contents could not be evaluated at all.

WORK-UP

It was advised that swabs should be taken for bacterial culture and sensitivity testing in view of the nature of the ocular discharge but the owner would not agree to this for financial reasons. Cytology was also declined.

TREATMENT

The owner was adamant that it would not be possible for topical medication to be applied more than twice daily. It was explained that control of the disease would be impossible at this dose and that it was not humane to allow the dog to suffer in this way. The owner was aware that the dog was suffering and did not want this to continue (but euthanasia was not something he was considering). Subconjunctival steroids were discussed but these would require general anaesthesia to perform and the owner was reluctant for this, again on cost grounds. As a compromise the dog was prescribed systemic prednisolone. The owner was aware that this could result in systemic side effects and was not the



Figure 29.4 Severe chronic superficial keratitis and nictitans plasmacytic conjunctivitis in an 8-year-old German shepherd dog – left eye.

treatment of choice, but there appeared to be no alternative. Thus prednisolone at 2 mg/kg/day for a week reducing to 1 mg/kg/day for 2 weeks was dispensed together with dexamethasone, neomycin and polymyxin B ointment (Maxitrol, Alcon) twice daily. This topical formulation was chosen since it contains antibiotic as well as the steroid and ointment has a longer contact time than drops.

OUTCOME

The owner failed to return for a follow-up appointment 2 weeks later. A telephone conversation was possible and the owner reported that the eyes were much less red and the dog more comfortable, although he still could not see. The steroids were causing some polydipsia and polyuria but as the dog was kept in the garden most of the time, this was not a problem for the owners. It was made clear that the dog would need to be checked again before further medication could be dispensed, and that without continued treatment the eyes would rapidly deteriorate again. Unfortunately, the dog was never brought back to the surgery.

This case was very frustrating. The lack of owner compliance was the cause for the dog's severe ocular disease and despite the owner being made aware that the condition could be improved dramatically if he would make the effort to treat the dog, this did not occur. If the owner had been dedicated to treating his pet properly from the start the eyes would never have progressed to this end stage and the poor dog would have retained vision.

30 Feline corneal sequestrum

INITIAL PRESENTATION

Pain, discharge, abnormal appearance – red/cloudy

PRESENTING SIGNS

A cat with a corneal sequestrum will be presented with a slightly uncomfortable watery eye. Thus blepharospasm will be present – the cat holds the affected eye partially closed, and the discharge is likely to be serous in nature, although sometimes it can be mucopurulent. It is not uncommon for the discharge to be dark brown in colour – owners may think that the eye has been bleeding due to the colour. The owners might also have noticed a dark discolouration on the cornea, although since this can often be central in position it is not always obvious to them against the dark background of the pupil. The position in the central or subcentral cornea is typical, and the colour can vary from mid amber to very dark brown or black. Any breed of cat can be affected, but Persians of all types and Burmese are over-represented.

CASE HISTORY

The history of previous corneal ulceration is common. Thus the cat could still be under treatment for an ulcer, and on subsequent examination is found to have the typical dark corneal deposition of a sequestrum developing. Slow to heal superficial ulcers are most likely to progress to sequestrum formation. In domestic short haired cats the ulcer is likely to be associated with a traumatic event – a cat scratch for example – while in Persians and Burmese no such history is always present. It is not uncommon for the cat to have had problems with one eye in the past and for similar ulceration and sequestrum formation to recur or to develop in the second eye. Occasionally there will be no history of ulceration and the sequestrum seems to develop spontaneously. In some cats there will be a history of previous upper respiratory disease associated with feline herpes virus (FHV-1). Indeed chronic and recurrent ulcers are frequently associated with FHV-1 infection and secondary sequestrum formation is a well-recognized complication of this.

CLINICAL EXAMINATION

General clinical examination is usually unremarkable, unless an active FHV-1 infection is concurrent. Ophthalmic examination will reveal several abnormalities. The affected eye is usually wet, and Schirmer tear test readings will confirm this with a higher reading than in the normal eye. However, on occasion the opposite can be true – a sticky discharge might suggest an associated keratoconjunctivitis sicca so tear test readings should not be overlooked.

Blepharospasm is normally present. Conjunctival hyperaemia and some chemosis are common, as is superficial corneal vascularization. A darkly pigmented patch of cornea defines the sequestrum. This can be raised above the epithelium or subepithelium (the latter is less uncomfortable). The colour can vary from a mid brown to almost black. The ocular discharge is often also darkly stained; however, this is normal for many cats so should not be over-interpreted. Corneal ulceration is often superficial or mid-stromal and fluorescein dye should always be used for any suspected sequestrum. Variable amounts of corneal vascularization can be encountered – in some patients none is noted, while in others there is profuse superficial vascularization and even granulation tissue deposits at the edge of the sequestrum.

In more severe cases the pupil can be miotic, with rubeosis iridis and some aqueous flare – a reflex uveitis – which is most common if bacterial infection is present with the surface disease. In long-standing cases there might be some degree of secondary lateral lower lid entropion with the hairs rubbing on the cornea and exacerbating the problem – this is more frequent in older domestic short haired breeds and is rarely seen in Persians. However, in this latter breed a medial lower lid entropion can be present which may contribute to the problem. In addition, brachycephalic cats can suffer from a relative lagophthalmos such that the central cornea is not fully protected during blinking and tear film dynamics are abnormal (Figure 30.1). This is a serious problem in some of the 'ultra-type' Persians with the excessively flat face and bulging eyes – some of these poor cats even sleep with their eyes partially open and corneal health can be severely compromised as a result.

Checking the frequency of blinks, and whether these are complete or partial – where the eyelids do not fully meet – is important. In some patients, particularly the predisposed breeds, both eyes can be affected although often not at the same time, and certainly rarely symmetrically if both are involved.

DIFFERENTIAL DIAGNOSES

- Corneal foreign body
- Iris prolapse
- Limbal melanoma



Figure 30.1 Bilateral corneal sequestrum in a Persian. The left eye has the typical central dark discolouration while the right has a superficial corneal ulcer and an amber discolouration to the exposed stroma.

CASE WORK-UP

If there is any mucopurulent discharge then swabs should be taken for bacterial culture and sensitivity testing. These should be taken before the cornea is stained with fluorescein and are more accurate if the swab is gently rolled at the edge of the lesion rather than sampling the discharge on the face. Samples for FHV-1 polymerase chain reaction (PCR) should be taken if there is a suspicion of a viral component.

NURSING ASPECTS

Most affected cats will require topical medication – sometimes for life in predisposed breeds – and owners must be taught how to apply this effectively. Some cats will need surgery, and nursing before, during and after the procedure is important for its successful outcome.

EPIDEMIOLOGY

Corneal sequestrum is traditionally considered to be unique to the cat, although similar symptoms have been described in the horse. Most cats are young adults, but a wide age range from a few months to 17 years old has been reported. The breed predisposition to Persian types is well recognized and this could suggest an inherited component. Indeed, sometimes related cats are affected.

The presence of some form of corneal insult is thought to contribute to the development of the sequestrum. This can be an obvious incident such as a fight with another cat, but more subtle insults, such as inefficient tear dynamics due to poor eyelid conformation and lagophthalmos, or mild entropion or trichiasis can be involved. The pigment has been shown to be melanin, and probably comes from the tear film, although some corneal sources might also be involved.

CLINICAL TIPS

- Check carefully for any predisposing factors – medial entropion, poor blinking, signs of FHV-1
- Carefully examine both eyes
- Perform Schirmer tear test readings
- Consider samples for FHV-1 PCR and bacterial culture and sensitivity
- Use fluorescein dye

TREATMENT OPTIONS – MEDICAL

If the sequestrum is superficial with no obvious ocular discomfort it can be managed medically since spontaneous sloughing can occur. However, regular re-examinations are necessary to ensure that the lesion is not progressing. Medical treatment with topical lubricants such as carbomer gel are advised one to three times daily, with instructions for the owner to bring the cat back to the surgery should any blepharospasm or significant ocular discharge develop. Topical alpha interferon has been used successfully with a reduction in, or even total disappearance of, the pigment in some cases, but many ophthalmologists have been disappointed by its lack of efficacy!

Owners should be warned that it can take many months for the sequestrum to slough but providing the cat is comfortable it is acceptable to follow this management regimen. Some cats can remain comfortable for years with subepithelial pigment, i.e. the discolouration remains in the anterior stroma and does not involve the actual corneal surface. However, the risk of ulceration and exposure of the necrotic pigmented tissue is always present.

Any cat which shows signs of discomfort associated with the sequestrum should be considered for surgery. It is not advisable to just continue with medical management in these cases since the pain could go on for months, the risk of infection is present and the sequestrum could extend deeply through the corneal stroma such that perforation could occur should the lesion start to slough.

TREATMENT OPTIONS – SURGICAL

Surgery for corneal sequestrum involves removing the abnormal tissue via superficial keratectomy. With suitable magnification and instrumentation, and the surgical training to perform such operations, it is possible to undertake this procedure in general practice. However, if any of the above requirements are not present, referral to a suitably qualified and equipped colleague is recommended.

Under general anaesthesia the abnormal cornea, including all pigmented tissue, is excised. Leaving some pigmented material behind can predispose to recurrence. A fixed-depth keratome (surgical knife) of 300 μm is useful, but a rounded Beaver blade (e.g. No. 65) can be used.

If the pigment is quite superficial, less than one-third of the stromal depth for example, then it should heal without requiring a conjunctival graft. Thus it can be left to heal by secondary intention and treated as a superficial ulcer, or a bandage contact lens can be placed to promote ocular comfort during the healing process. Third eyelid flaps can be used for the same purpose, but have the disadvantage of not being able to check on the cornea unless the flap is taken down. Postoperative medication will usually consist of a broad spectrum topical antibiotic drop, with atropine if any reflex uveitis is present. Topical NSAIDs are not usually necessary. Lubricants can also be dispensed.

A conjunctival graft should be used if the pigment extends deeper through the stroma, i.e. more than one-third to half depth. A pedicle graft is most frequently employed, and sutures of 8/0 polyglactin 910 (Vicryl, Ethicon) are normally chosen. Since some sequestra can be quite large before they are presented for surgery, and their location is normally central or paracentral, a conjunctival graft can result in some reduction in vision. As an alternative a corneoconjunctival/corneoscleral transposition can be used. This is best left to a specialist but essentially involves moving a clear, partial thickness section of normal cornea from the periphery to the central keratectomy site, with its base still attached to the conjunctiva or sclera. This results in an opaque periphery where the graft is slid across, but the central visual axis is more clear than if a conjunctival graft was used. Postoperative medication is similar to the superficial keratectomy.

If a conjunctival graft has been used it is normal for the pedicle stalk to be trimmed 3–6 weeks post-surgery. However, reports suggest that it is beneficial to leave some blood supply to the grafted tissue as this seems to help prevent the recurrence of the sequestrum. It does mean that the graft will be more vascular and less transparent than if the stalk was fully transected but this is an acceptable compromise if it does provide protection from further problems (Figure 30.2).

PROGNOSIS

The prognosis for corneal sequestrum is fair – many cases do require surgery and there is a risk of recurrence, especially in predisposed breeds or if FHV-1 associated. Studies have reported a recurrence rate of 20%. This risk is reduced if a conjunctival graft is used following superficial keratectomy but this has the negative effect of



Figure 30.2 The right eye of this Persian has a large central conjunctival graft which was placed several months previously following removal of a very large sequestrum. The left eye has now developed a similar, but smaller, sequestrum and also requires surgery. Vision remains good.

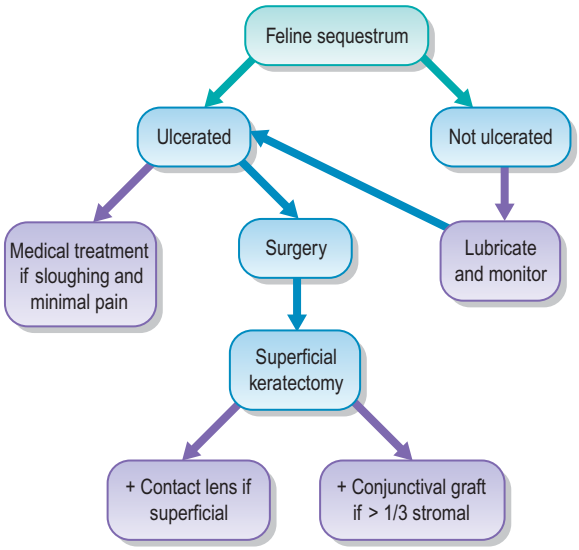


Figure 30.3 Decision-making tree – feline corneal sequestrum.

some reduction in central vision and permanent scarring. However, even with large grafts, which would be seriously compromising to a human, the cats seem to show no significant behavioural signs attributable to a reduction in central vision.

Owners should be warned of the risk to the fellow eye, again more of a concern in predisposed breeds compared to domestic breeds where a known traumatic incident predated the ulceration and subsequent sequestrum formation.

CASE EXAMPLE 30.1

Patient details: Chinchilla cat, 2 years old, female (entire) – breeding queen

PRESENTATION AND HISTORY

The cat was presented with a 3-week history of left ocular pain and discharge, which the owner had treated with chloramphenicol drops. However, this had not helped and she then noticed a dark discolouration on the surface of the eye.

CLINICAL SIGNS

The right eye was unremarkable. The left was uncomfortable and a mucopurulent discharge was present. The cornea showed a dark central lesion with a surrounding gelatinous ring of abnormal cornea. Some corneal vascularization was noted (Figure 30.4). Schirmer tear test readings were 15 mm/min in this eye and 12 mm/min in the right. The pupil was slightly miotic.



Figure 30.4 Left eye of Chinchilla cat with infected corneal sequestrum.

WORK-UP

The clinical examination confirmed a diagnosis of corneal sequestrum. A swab was taken from the discoloured cornea at the edge of the sequestrum and sent for bacterial culture and sensitivity. Attempts at demonstrating FHV infection were not considered necessary. A profuse growth of staphylococcal spp. was isolated, resistant to chloramphenicol and fusidic acid but sensitive to cefalexin and ofloxacin.

TREATMENT

Under general anaesthesia a superficial keratectomy was performed. This removed all the pigment and surrounding abnormal cornea. A pedicle conjunctival graft was sutured into the defect, actually using two grafts – one with a lateral base and one dorsal to ensure a good blood supply to the large damaged area. Topical ofloxacin (Exocin, Alcon) was dispensed four times daily plus once daily atropine ointment and 5 days of oral cefalexin.

OUTCOME

The cat did very well. After 7 days, treatment continued with just ofloxacin four times daily. Two weeks later treatment was stopped. It was decided to leave the graft to meld into the cornea naturally and not to trim it. Two months after surgery the cornea had cleared well and vision was good (Figure 30.5). The owner was advised not to use the cat for breeding and it was neutered. Two years later a similar sequestrum developed in the right eye – the owner presented the cat quickly. Surgery was again successful.



Figure 30.5 Same eye as in Figure 30.4, 2 months later. Vision is good.

This case is typical. The owner was a breeder and treated the cat herself prior to bringing it to the surgery. Unfortunately an infection had developed which was not sensitive to the chloramphenicol (or fusidic acid – both of which could have easily been prescribed initially by the veterinary surgeon!). A large graft was required and thankfully this cleared well such that vision was minimally affected. When the second eye developed similar problems the owner brought the cat along straight away such that a much smaller graft was required in this eye.

31 Chronic feline keratitis

INITIAL PRESENTATION

Discharge, change in appearance – cloudy, pain

PRESENTING SIGNS

Chronic feline keratitis is usually seen in adult cats and in most cases is unilateral in presentation. The owners might have noticed a discharge, a change in appearance of the eye or ocular discomfort, but often all three symptoms are reported to various degrees. Feline keratitis can be ulcerative or non-ulcerative. The former can present as superficial erosions, similar to the recurrent epithelial erosion or indolent ulcer seen in dogs, but usually with a different aetiology – primary feline herpes virus (FHV-1) is the most common cause for recurrent superficial ulcers in cats and we will concentrate on this infection when discussing feline keratitis. Other types of ulcer are usually the result of trauma, such as following a cat fight, and subsequent to other ocular diseases such as entropion, although this is far less common than in dogs. Reactivation of FHV-1 infection following a previous acute episode of ocular and respiratory disease is frequently reported. Occasionally cats can present with keratitis in addition to systemic disease and only when the latter is correctly diagnosed and treated do the ocular signs respond.

CASE HISTORY

As mentioned above, FHV-1 is commonly associated with keratitis in cats and, as such, the history will often include previous ocular or respiratory disease. Rescue or re-homed cats are over-represented so questioning the owner regarding how they acquired the cat is important. Lifestyle is also relevant since outdoor cats, or those which frequently get into fights with other cats, can be at a higher risk of developing recurrent keratitis. It is common for the same eye to be repeatedly affected although in some cats both eyes can be involved, either simultaneously or at different times. Commonly the owner reports that the eye never appears totally normal but it only bothers the cat from time to time. The condition can recur following immunosuppression, for example following an unrelated illness or via the use of systemic steroids for another complaint.

CLINICAL EXAMINATION

In recurrent cases of FHV-1 systemic signs are usually absent but a full clinical examination should still be performed. Ocular signs of mild blepharospasm, increased lacrimation and some conjunctival hyperaemia are usually present. Schirmer tear tests should be performed before any discharge is cleaned away or samples taken. These can be elevated, normal or reduced. Conjunctival hyperaemia is normally present but chemosis is variable. Careful examination for evidence of ocular trauma or other inciting causes should be undertaken. Small corneal opacities, which may or may not stain positive with fluorescein dye, and fine vascularization are common findings. Larger superficial ulcers with irregular outlines and under-run edges are encountered and readily identified on careful examination (Figure 31.1).

However, if FHV-1 is suspected as the cause for the keratitis then staining with rose Bengal should be considered. This is more sensitive than fluorescein at highlighting the small areas of epithelial devitalization and the demonstration of these dendritic ulcers is considered pathognomonic for FHV-1 keratitis (Figure 31.2). Careful evaluation of limbus is advised since acute herpetic infec-



Figure 31.1 Three-year-old British Blue with irregular superficial corneal ulcer. The cat had suffered from upper respiratory tract infection as a kitten and went on to develop recurrent ulcers in both eyes. Viral isolation was positive for FHV-1.

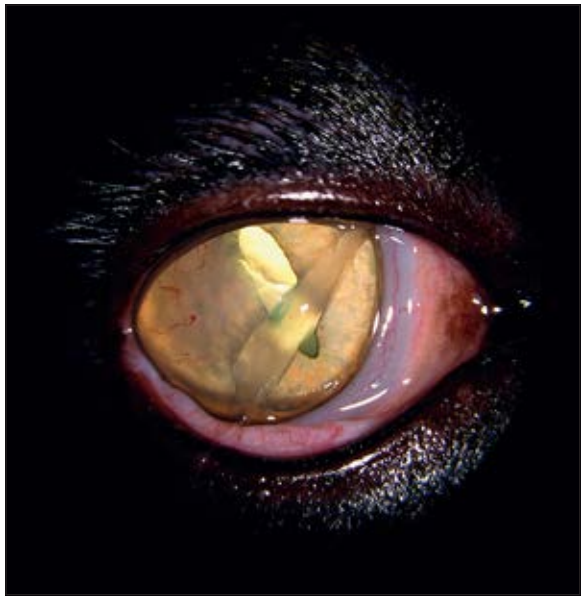


Figure 31.2 Right eye of a domestic short haired cat with FHV-1. Fine 'dendritic' ulcers are present dorsolaterally, highlighted in magenta pink due to rose Bengal staining. A mucoid discharge is adherent to the cornea and Schirmer tear test readings were 2 mm in this eye and 8 mm in the normal left eye (keratoconjunctivitis sicca can develop secondary to FHV-1 and should not be overlooked).

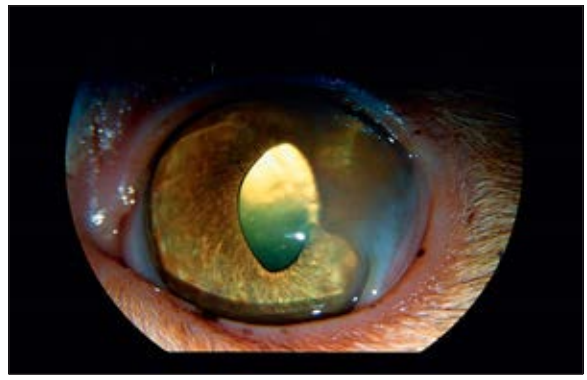


Figure 31.3 Lateral symblepharon in the right eye of a domestic short haired cat. No keratitis is present but the conjunctival adhesions restrict globe movement slightly.

tions in young cats will often result in limbal damage such that the neat dark line present between the clear cornea and white sclera is disrupted. Similarly, examination of the conjunctiva, especially around the nasolacrimal punctae and fornices, might reveal evidence of symblepharon (Figure 31.3), which is another frequent complication of FHV-1 and contributes to both chronic epiphora and corneal ulceration via reduced ocular and third eyelid motility as a result of the adhesions.

Severe corneal scarring with FHV-1 is usually stromal and ulceration can be a typical feature with small punctate or larger coalesced lesions. Extensive vascularization and oedema can occur. A mild reflex uveitis can be present, with miosis and some rubeosis iridis, but intraocular involvement is not an obvious part of the clinical examination.

DIFFERENTIAL DIAGNOSES

- Feline herpes virus (FHV-1) keratitis – with or without ulceration
- Keratoconjunctivitis sicca (can be secondary to FHV-1)
- Corneal ulceration – post-traumatic
- Corneal foreign body
- Proliferative (eosinophilic) keratoconjunctivitis
- Corneal sequestrum
- Lipid keratopathy (rare in cats)

CASE WORK-UP

Thorough clinical examination will lead to the suspicion of FHV-1 and should rule out other causes of keratitis

such as primary keratoconjunctivitis sicca or corneal sequestrum. However, as these are often associated with FHV-1, attempts at viral isolation or polymerase chain reaction (PCR) from both conjunctival and oropharyngeal swabs are advised since the treatment protocol might be changed if positive laboratory results are obtained. Corneal scrapes can be used for PCR and are probably more sensitive than conjunctival swabs since the samples are from the area most likely to be infected with virus. Cytology can be useful to rule out differentials such as proliferative keratoconjunctivitis. Some cats with herpetic keratitis are immunosuppressed and so screening for feline leukaemia virus (FeLV) and feline immunodeficiency virus (FIV) is sensible.

NURSING ASPECTS

General principles of cleaning discharges and careful application of medications apply, with no specific considerations other than barrier nursing since these patients can be infective. Topical medication may be required on a frequent basis which can be difficult for owners to manage, so sometimes cats can be hospitalized during the initial treatment period (although the stress this induces can prolong healing times!).

EPIDEMIOLOGY

Feline herpes virus is ubiquitous and cross-reference to the section on feline conjunctivitis should be made. With keratitis rather than conjunctivitis the condition is usually seen on an individual basis in adult cats rather than as outbreaks in young animals. Instead of the symptoms being derived solely from the direct cytopathic effect of the virus, they are considered to also involve a cell-mediated immune response to the viral particles within the corneal stroma. The clinical signs of keratitis can also develop as a consequence of the destruction of limbal stem cells during the acute phase of the primary infection, such that the corneal epithelium cannot be generated for repair or cellular turnover and conjunctivalization of the cornea results.

Recrudescence can occur with reactivation of virus particles in the trigeminal and other sensory ganglia and probably some latency occurs in the corneal stroma as well. The variety of clinical signs is clearly reflected in the subtle differences in pathogenesis and the exact location, and host response, to the viral particles. Keratoconjunctivitis sicca can develop following viral damage to the lacrimal gland. Conversely, epiphora can result from occlusion of the nasolacrimal punctae by symblepharon formation.

CLINICAL TIPS

- Careful ophthalmic examination with attention to the nasolacrimal punctae and presence of symblepharon
- Differentiate corneal vascularization from conjunctiva/symblepharon
- Consider rose Bengal staining for subtle areas of corneal devitalization
- If using rose Bengal, remember to flush away excess thoroughly – it stings!
- Some degree of ulceration is very common with chronic herpetic keratitis
- Perform Schirmer tear tests since keratoconjunctivitis sicca can result from FHV-1
- Although usually unilateral, check the fellow eye for evidence of previous infection, such as subtle symblepharon or fine corneal scarring
- Remember that repeated sampling might be necessary to obtain positive FHV-1 results – only low numbers of viral particles are shed intermittently, so although PCR is more sensitive than viral isolation, more than one sample might be required.

TREATMENT OPTIONS – MEDICAL

Unless deep or refractory corneal ulceration is present, the treatment for FHV-1 keratitis is usually medical. Unfortunately, the response to medication is often unpredictable and can be disappointing with recurrences occurring commonly. However, the response to acute flare-ups is usually better than in chronic cases of stromal keratitis. Virtually all potential treatment options are unlicensed for use in cats and owners must be made aware of this. For any treatment prescribed the balance between achieving a clinical improvement while not unduly stressing the patient must be met.

If dendritic ulcers are present and an acute flare-up is suspected, these are the cases most likely to respond to topical antiviral agents. Aciclovir when applied topically in cats is usually disappointing and a better choice is trifluorothymidine, which can be obtained from the pharmacy at Moorfields Eye Hospital. Initially the drops

need applying six to eight times daily for 3–4 days before reducing to four times daily. Topical hypersensitivity reactions can occur (Figure 31.4). The demanding treatment regimen can be difficult for owners to administer and is stressful for cats (which can trigger further viral recrudescence!). In conjunction with the antiviral, topical lubricants can promote ocular comfort while broad spectrum topical antibiotics should be considered if secondary bacterial infections are suspected.

Oral antiviral agents show promise and famciclovir appears to be the most efficacious, with reasonable safety, in cats. Other agents such as aciclovir can cause bone marrow suppression when given orally and are not advised. However, since controlled clinical trials have not been performed for any of the systemic antiviral agents used in people, their use in cats in general practice situations cannot be readily justified at present. Nevertheless, they do offer the advantage of less frequent application, which is less stressful for the patient, and hopefully their use will become more acceptable in the near future.

Oral L-lysine, as mentioned in the section on feline conjunctivitis, competes with arginine during viral replication and seems to reduce both the clinical symptoms and the duration of the disease. It can also be safely used prophylactically but again controlled clinical trials are lacking. Doses of between 250 and 500 mg once or twice daily have been recommended.



Figure 31.4 Topical hypersensitivity to trifluorothymidine in an adult Burmese cat. Note the corneal ulceration and stromal vascularization typical of FHV-1 keratitis plus the hyperaemic thickened eyelids and slight chemosis which reflect the hypersensitivity. The latter settled after 72 hours of topical lubricants alone.

Other possible treatments for herpetic keratitis include alpha interferons, either topically or systemically. Although they appear to do no harm, whether they confer any benefit to the patient is debatable.

Some veterinary ophthalmologists advocate the careful use of topical steroids in cats with severe stromal keratitis. The rationale for this is to treat the immune-mediated component of the disease. However, since viral reactivation is well recognized following topical and systemic administration of steroids, their widespread use cannot be justified. If they are employed it should always

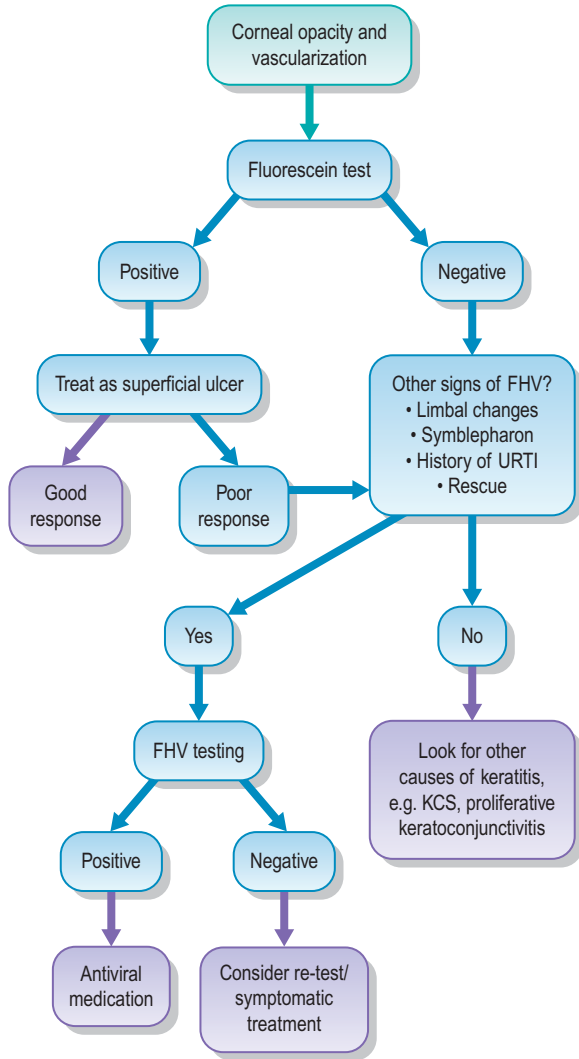


Figure 31.5 Decision-making tree – chronic feline keratitis. KCS – keratoconjunctivitis sicca; FHV – feline herpes virus; URTI – upper respiratory tract infection.

be in conjunction with antiviral therapy and careful re-examinations and discussion with the owner about the possible detrimental effects of such treatment must be undertaken.

TREATMENT OPTIONS – SURGICAL

Recurrent or indolent superficial corneal ulcers due to herpetic keratitis can require superficial keratectomy as mentioned for the treatment of non-healing ulcers in dogs. If complications such as sequestrum formation occur, then surgery is frequently necessary. Should a keratectomy be performed it is sensible to submit a sample of the cornea for FHV-1 PCR. Traditional postoperative treatment with contact lenses or conjunctival grafts is required depending on the depth of the keratectomy and whether any concurrent bacterial infection is present. There is some merit in the logic that removing the superficial stroma in cases of recurrent ulceration and stromal keratitis can remove the site of some viral latency and replication and thus improve the clinical outcome. However, achieving rapid healing of the keratectomy site, with minimal scarring, can be a challenge.

PROGNOSIS

The prognosis for a complete cure is guarded and the response to treatment can be variable. The high carrier state and rapid return of clinical signs on reactivation of the virus can be debilitating for the cat and frustrating for owner and clinician alike. Repeated bouts of ulceration and stromal keratitis will lead to severe corneal scarring which can compromise vision. Both permanent epiphora, due to symblepharon blocking the nasolacrimal punctae and canaliculi, or chronic keratoconjunctivitis sicca can be sequelae from FHV-1. Corneal sequestrum formation and proliferative keratoconjunctivitis sicca are also not uncommon sequelae and deciding on the most appropriate treatment regime for each individual patient can be challenging.

CASE EXAMPLE 31.1

Patient details: Domestic short haired cat, 5 years old, male (neutered)

PRESENTATION AND HISTORY

The cat had been rescued from a feline charity 6 months earlier and had always had a 'funny eye'. It had been cloudy but did not bother the cat until he became unwell following a cat fight and an abscess at the base of his tail – when debilitated from this, he started to close the right eye and it started to water. The owner mentioned this when the cat was returned for a check of the abscess.

CLINICAL SIGNS

The abscess was healing well and the cat was not pyrexic. The left eye was normal. The right was cloudy with increased lacrimation. A patchy stromal opacity was noted throughout the cornea and superficial branching stromal vascularization was observed. The limbus was irregular and the demarcation between cornea and sclera was blurred (Figure 31.6). No ulceration was noted – even with rose Bengal staining. Intraocular contents were unremarkable.

WORK-UP

FHV-1 PCR was performed on conjunctival and oropharyngeal swabs and the conjunctival swab was positive.



Figure 31.6 Five-year-old male (neutered) domestic short haired cat with typical herpetic keratitis – note the increased lacrimation, corneal opacity due to stromal scarring, fine vascularization and disrupted limbus.

TREATMENT

Oral L-lysine was started, 250 mg daily in food. The owner was offered topical antiviral agents but declined this since the cat was improving and was difficult to medicate. Lysine was used for a month.

OUTCOME

Two weeks later the eye was comfortable again but still cloudy – the owner was not bothered about this. Four months later the eye became very sore and a superficial corneal ulcer was present centrally. Schirmer tear tests were normal. This was treated with fusidic acid only and healed after 3 weeks. Oral lysine was restarted and continued long term. A second ulcer developed only a month later and this time topical antiviral medication was used in conjunction with once daily fusidic acid. The cat was hospitalized for 5 days and trifluorothymidine was used eight times daily for 2 days then six times daily, with the owner continuing three times daily at home. The ulcer healed rapidly and the antiviral was continued for 3 weeks altogether. It was a year before the cat developed another ulcer and was treated similarly.

This case shows that although the cat clearly was a carrier for FHV-1 and the eye was permanently damaged, most of the time it did not cause him any problems. Topical antivirals seemed to reduce the time it took for the ulcers to heal and the continued lysine should have reduced viral replication. The owner understood that the condition was similar to people who have cold sores due to herpes infections and that flare-ups would occur from time to time.

SECTION 5

UVEA

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32 Uveal tract – introduction

The uveal tract makes up the middle layer of the globe, and consists of the iris, ciliary body and choroid. It is usually pigmented and is the vascular layer of the globe. The anterior uvea includes the iris and ciliary body while the choroid comprises the posterior uveal tract.

The *iris* is the visible coloured layer seen within the eye. It is made up of an anterior layer of stroma and iris sphincter muscle, and a posterior layer made up of an epithelial layer (which is pigmented) and dilator muscle (Figure 32.1). The colour of the iris depends on both the number of melanocytes present within the stroma and the thickness of the anterior layer itself. In addition to muscle fibres and melanocytes, the stroma contains numerous blood vessels, nerve endings, fibroblasts and collagen fibres. The main blood vessels entering the iris are the long ciliary arteries (temporal and nasal) which together form the major arterial circle visible in most animals as an undulating ring within the anterior iris stroma. The *pupil* is the central hole in the iris. Normally the iris rests lightly against the anterior surface of the lens. Movement of the iris muscles controls the quantity of light entering the posterior segment through the pupil. The sphincter muscle circles the iris in the dog while in the cat the fibres also criss-cross above and below the pupil allowing it to constrict to a narrow vertical slit. Other species such as horses, sheep and reptiles have different arrangements of this muscle which contribute to pupil shape. Constriction of the pupil is mediated via parasympathetic nerve fibres travelling in the oculomotor nerve (cranial nerve III). The dilator muscle is innervated by sympathetic fibres (which run with the ophthalmic branch of the trigeminal nerve (cranial nerve V)). The dilator muscle radiates out from the pupil margin towards the iris periphery in a similar pattern to bicycle spokes. In addition to regulating the amount of light entering through the pupil, altering its size also affects depth of focus and reduces optical aberrations.

The *ciliary body* is located behind the iris, between the lens and choroid. The outer epithelium of the ciliary body is continuous with that of the iris, but confusingly is non-pigmented rather than pigmented as is that of the iris. The ciliary body can be divided into two areas – one containing the ciliary processes (pars plicata) and the other comprising the ciliary body musculature (pars plana). It also forms part of the iridocorneal angle (ICA). The ciliary processes secrete the aqueous humour – the transparent fluid which fills the anterior and posterior chambers. Aqueous provides nutrients to, and removes waste products from, the avascular structures within the anterior part of the eye, i.e. the lens and cornea. Aqueous drains from the eye mainly through the ICA. Pectinate ligaments span the opening to the ICA. The balance between production and outflow of aqueous creates the intraocular pressure and imbalance can lead to raised pressure (glaucoma) or lowered pressure (hypotony).

The ciliary body epithelium forms part of the 'blood–aqueous barrier' protecting ocular metabolism. Breakdown of this barrier occurs with inflammation, trauma, surgery and some drugs, and results in leakage of proteins and other blood-borne components into the aqueous. In addition to its metabolic functions, the ciliary body provides attachments for the lens (via the lens zonules) and has some effect on accommodation (focussing), although this mechanism is poorly developed in dogs and cats. Spasm of the ciliary body muscle commonly occurs during intraocular inflammation and can be very painful. Cycloplegic drugs that paralyse the muscle (e.g. atropine) are frequently used to help abolish the pain encountered in uveitis, and this function is as important as the mydriasis induced.

The *choroid* is the posterior portion of the uveal tract and lies between the sclera and retina. It is made up of several layers and contains many thin-walled blood vessels together with pigmented support tissues. Within the dorsal portion of the choroid is found the tapetum lucidum or simply the tapetum. This is a mirror-like layer (responsible for the eye shine seen in dogs and cats) which functions to re-stimulate the retinal photorecep-

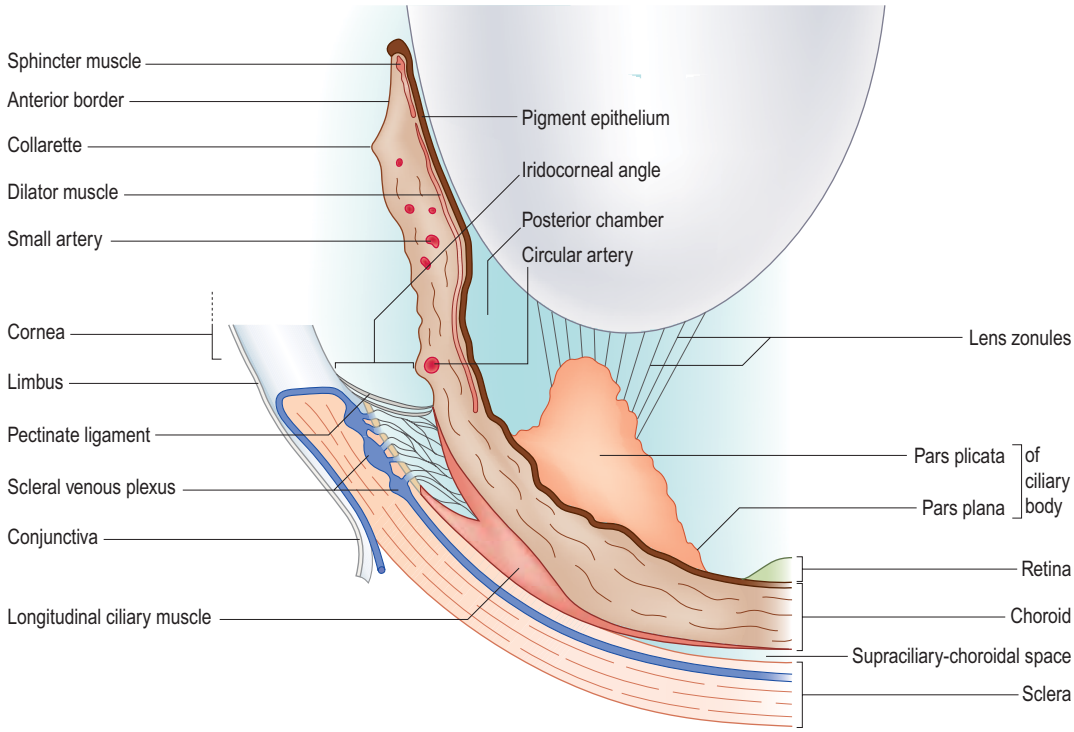


Figure 32.1 Anatomy of the canine anterior uvea and associated structures.

tors when light is reflected back from it. This increases visual sensitivity and assists vision in low light intensity. The choroid also provides glucose and oxygen for retinal metabolism.

Inflammation of the uveal tract is a common finding and is termed uveitis. Due to the intimate continuity of its various components it is unusual to have inflammation affecting just one anatomical part – iritis or choroiditis for example. Far more common is a general anterior uveitis (iris and ciliary body) or posterior uveitis (choroid and ciliary body). Similarly, inflammation of the choroid alone is rare and chorioretinitis, where some retinal involvement is present, is far more common. The uvea is immunologically competent and ocular antigens will stimulate both B and T lymphocytes from the general circulation and both antibody and cell-mediated immune responses thus occur. Subsequent exposure to the same antigen will stimulate a faster and more severe response – the uveal tract can be likened to a lymph node in this respect – and this is a fundamental concept when chronic or recurrent uveitis is encountered. In addition to primary inflammation of the uveal tract, a secondary reflex is often seen when other parts of the eye are inflamed. This reaction is usually mild and self-resolving, for example with a simple corneal ulcer, but can be excessive and result in serious ocular damage on occasions.

The clinical signs of uveitis, such as miosis, aqueous flare, episcleral congestion, low intraocular pressure, corneal oedema and so on, are similar no matter what the cause for the inflammation and are discussed in the following cases. Non-inflammatory uveal disease is less common.

The treatment for uveitis consists of both specific and non-specific agents. Thus, if the underlying aetiology is known, treatment is targeted at removing this, while non-specific anti-inflammatory agents are also usually required. These will be discussed in more detail as we consider some cases affecting the uveal tract.

33 Canine uveitis

INITIAL PRESENTATION

Pain, abnormal appearance – red/cloudy, blind

PRESENTING SIGNS

Dogs are normally presented with a sudden onset painful eye, often with colour change, and sometimes with reduced vision. The pain is manifested by the patient holding the affected eye closed with increased lacrimation and photophobia. The dog might shy away from attention to this side of its face, or may be seen rubbing the eye. Owners will often also comment that the dog is depressed. The condition can be either unilateral or bilateral. Any age of dog can be affected and there is no particular breed predisposition which is a reflection of the wide variety of causes for uveitis in dogs.

CASE HISTORY

Since there are so many potential causes for uveitis the history can be variable. A few common examples are the dog returning from a walk with a sore eye, which might suggest a traumatic uveitis (due to blunt or penetrating injury). A recently vaccinated puppy might present with a 'blue' eye – which despite being a vaccine reaction to the canine adenovirus 1 vaccine is surprisingly often unilateral not bilateral. Thankfully this condition is not commonly encountered with modern vaccines. A history of recent travel abroad, especially to the Mediterranean, might alert one to possible *Leishmania* infection. The patient might have suffered from mammary carcinoma a few months previously which could be relevant to the ocular symptoms. Thus it is very important in all cases of uveitis to take a detailed history. This can point us in the right direction regarding further diagnostic tests and will help to rule out certain causes of uveitis immediately.

CLINICAL EXAMINATION

General clinical examination can be normal but may show abnormalities. For bilateral cases of uveitis an underlying systemic problem is possible and should be considered. Such patients might be pyrexic, have a lymphadenopathy, foci of pain or an abdominal mass. Unilateral cases might be traumatic, and careful evaluation for evidence of injury (e.g. bite wounds around the face, severe bruising, etc.) might be noted.

The typical ophthalmic signs of uveitis are episcleral congestion with conjunctival hyperaemia, corneal oedema, miosis and increased lacrimation (see Table 33.1). Pain is normally present such that the examination might be resented. The ocular discharge is usually serous. Vision might be reduced, especially if there is posterior segment involvement. Aqueous flare is often present, sometimes along with frank hypopyon or hyphaema. The iris will be dull and swollen, with rubeosis iridis, although this is quite difficult to appreciate in dogs with dark irides as most do have; however, it is readily noted in blue-eyed dogs. Redness might also be present around the limbus encroaching on the cornea – ciliary flush – or inflammation of the deep episcleral vessels with growth into the cornea (Figure 33.1).

Fluorescein testing should be performed. Usually this will be negative, unless a traumatic incident has occurred or the patient has traumatized the eye secondarily. Measurement of intraocular pressure (IOP) is important and normally readings will be low – commonly 6–10 mmHg. Evaluation of the posterior segment might not be possible. Indirect ophthalmoscopy is likely to be more rewarding than using a direct ophthalmoscope. Mydriasis will assist with fundus examination, but many cases of uveitis

Table 33.1 Ophthalmic signs of canine uveitis

Acute signs	Chronic signs and complications
• Pain • Increased lacrimation • Visual impairment • Photophobia • Miosis • Aqueous flare • Swollen iris • Conjunctival hyperaemia • Episcleral congestion • Corneal oedema • Lowered intraocular pressure • Hypopyon • Keratic precipitates • Hyphaema • Posterior segment changes	• Severe ongoing pain • Iris darkening • Endophthalmitis • Cataract • Lens luxation • Iris bombe • Blindness • Retinal detachment • Glaucoma • Phthisis bulbi

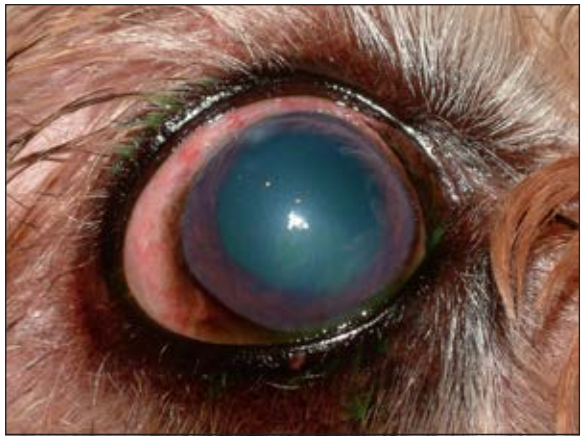


Figure 33.1 Acute anterior uveitis in a Shih Tsu. The pupil has been dilated with atropine but note the episcleral congestion, corneal oedema, out-of-focus appearance to the iris due to aqueous flare and faint fluorescein uptake (ventrally). The dense white spot at 12 o'clock close to the limbus was the site of a traumatic perforation which caused the inflammation. Intraocular pressure was 8 mmHg.

will be slow to dilate with tropicamide such that several drops need to be applied over 30–40 minutes before adequate dilation is achieved. Vitreal haze (hyalitis) might be noted along with retinal detachment or areas of active chorioretinitis. These will be seen as dull grey areas within the fundus. Perivascular oedema might be noted as a result of leaky blood vessels. The optic disc is usually normal on examination.

DIFFERENTIAL DIAGNOSES

- Glaucoma
- Lens luxation
- Corneal ulcer with secondary uveitis
- Lipid-laden aqueous
- Iris bombe
- Lens-induced uveitis (associated with cataract)

CASE WORK-UP

If the cause for the uveitis is identified on clinical examination – a deep corneal ulcer or head trauma for example – then specific work-up for the uveitis is not always required. Unfortunately, the inciting agent is elusive in most cases and extensive investigation can be required to identify the underlying cause. If the cornea or anterior segment is too opaque to allow direct visualization, then ocular ultrasonography should be considered. In unilateral cases this could well outline a uveal tumour for example, or perhaps demonstrate a posterior scleral rupture following blunt trauma.

It is the bilateral cases that can be the most challenging to diagnose. Clearly a systemic problem is likely and routine blood screening with haematology and biochemistry profiles are a minimal baseline. These might highlight an infectious aetiology such that more specific diagnostic tests are indicated or suggest a blood dyscrasia or neoplastic process. Generalized septicaemia such as associated with pyometra can cause uveitis. Therefore, the basic screen might suggest that clotting profiles are indicated, or perhaps *Toxoplasma*, *Ehrlichia*, *Leishmania* or *Borrelia* titres. Mycotic causes for uveitis are rare in the UK but should be considered in immunocompromised patients or those that have travelled through endemic areas. Migrating parasite larvae, such as *Toxocara canis*, *Angiostrongylus vasorum* and, less commonly, *Dirofilaria immitis*, can all trigger an acute uveitis. Faecal examination can be diagnostic in such cases. Some possible causes of uveitis are listed in Table 33.2.

If the uveitis is severe but no cause can be identified, then referral for aqueocentesis or vitreocentesis can be considered while the eye(s) is/are still potentially sighted. By the time a secondary glaucoma has developed the prognosis for saving vision is very poor, and enucleation can be considered both on humane grounds but also as a diagnostic aid. Clearly this is a last resort!

Abdominal ultrasonography is sometimes useful as part of the uveitis work-up – especially if neoplasia is suspected. Most cases of bilateral uveitis which are asso-

Table 33.2 Possible causes of uveitis

Infectious causes		Non-infectious causes	
Bacteria	Brucellosis Lyme disease (borreliosis) Leptospirosis Any septicaemia	Immune mediated	Lens induced (i.e. due to cataracts or lens trauma) Uveodermatological syndrome Coagulopathies, e.g. thrombocytopenia
Fungal	Blastomycoses Coccidioides Cryptococcus Histoplasmosis	Metabolic	Diabetes mellitus (especially if cataract) Hyperlipidaemia
Viral	Adenovirus (canine hepatitis) Distemper Rabies	Drug induced	Excessive miotic use
Parasitic	Leishmaniasis Toxoplasmosis Ehrlichiosis Rickettsial Migrating larvae	Idiopathic	
Algal	Prototheocosis	Trauma	Blunt Penetrating
		Secondary to ulceration	Reflex uveitis
		Neoplastic	Primary (e.g. ciliary body adenoma) or secondary (e.g. myeloma, lymphoma)

ciated with neoplasia are secondary – lymphoma, myeloma or mammary carcinoma for example.

If all diagnostic tests are negative, then one assumes a diagnosis of idiopathic or immune-mediated uveitis. Symptomatic treatment is started, but further tests might be warranted if the patient does not respond as expected or develops other clinical signs.

NURSING ASPECTS

Most cases of acute uveitis are painful and so appropriate analgesia should be considered. Nurses will be well placed to evaluate the degree of discomfort. Attempts at rubbing the eye(s) should be discouraged. The treatment for uveitis usually involves both topical and systemic medication which nurses can administer, as well as ensuring that owners are competent to continue the treatment regimen at home. Some patients with uveitis might be contagious – canine adenovirus for example. The risks of zoonotic infections such as *Leishmania* should not be overlooked and appropriate barrier nursing undertaken.

EPIDEMIOLOGY

Many cases of uveitis occur on an individual basis, but occasional outbreaks of infectious disease in which uveitis is part of the clinical picture do occur in dogs. Thus, unvaccinated patients could be seen with canine adenovirus or leptospirosis, both of which can have ocular signs in addition to the systemic problems. Younger dogs are more likely to suffer from traumatic causes while neoplasia is clearly more frequent in older patients. There are some breed predispositions – for example, Japanese Akitas are predisposed to uveodermatological syndrome, an immune-mediated disease where ocular and dermatological melanocytes are targeted and destroyed with severe localized inflammation.

Whatever the cause, be it exogenous or endogenous, the ocular inflammation develops following breakdown of the blood–ocular barrier. Chemical mediators (such as prostaglandins) result in a leaky barrier, often within the iris endothelium (but also in the choriocapillaris in cases of posterior uveitis), and both proteins and cells (red and/or white blood cells) pass between the gaps into the anterior chamber. Aqueous flare, hypopyon, hyphaema and keratic precipitates are all visible results of the break-

down of the blood–ocular barrier. Miosis occurs via a prostaglandin-mediated iris sphincter muscle spasm and this is particularly painful. The lowered IOP mentioned above results from reduced aqueous production.

If the inflammatory response is mild and localized, healing will occur with minimal scarring. However, small lesions in the eye can result in severe impairment of function – unlike in many other body organs where scarring can be incidental. Thus profound and uncontrolled inflammation can have catastrophic effects on vision. Chronic and recurrent uveitis occur frequently following acute episodes, although the detailed immunological actions resulting in this are complicated and poorly understood (and their discussion is beyond the scope of this text). However, the uveal tract does act rather like an accessory lymph node which is certainly important in recurrent immunological responses.

CLINICAL TIPS

- Check both eyes carefully
- Measure IOP to differentiate from glaucoma
- Eyes with uveitis are slower to respond to mydriatics (e.g. tropicamide) than normal pupils
- Compare the iris colours – usually darker in uveitic eyes
- Carefully examine for other clinical symptoms
- Look at the eye from the side to determine whether clouding is purely corneal or if aqueous flare/hypopyon is also present
- Don't forget to ask about travel history
- Re-examine every few days and check IOP as well as clinical appearance. If the eye is deteriorating, consider further diagnostic tests or referral

TREATMENT OPTIONS – MEDICAL

Uveitis is usually treated medically. Prompt therapy is required to prevent the potentially devastating sequelae of uveitis which are discussed in the section on prognosis. A combination of therapies, both topical and systemic, is normally required and treatment will be divided into specific elimination of the inciting cause, if known, together with symptomatic therapy. Clearly the specific therapy can be variable – particular antibiotics such as clindamycin for toxoplasmosis, or tetracyclines for ehrlichiosis. Otherwise a broad spectrum agent with good ocular absorption such as cefalexin can be chosen. Other specific treatment could be anthelmintics if migrating parasite larvae are identified or antifungal drugs in the rare cases of systemic fungal disease. It is sensible to use systemic antibiotics if the cause is uncertain but systemic steroids are required – the immunosuppression could allow an unidentified bacterial infection to proliferate and cause severe generalized septicaemia.

In severe cases of uveitis, systemic steroids, usually prednisolone at 1–2 mg/kg, will be required. Clearly the potential side effects of these high doses need to be considered, particularly if long-term medication is required – for immune-mediated causes for example. Some patients might even require systemic immunosuppressant drugs such as azathioprine or ciclosporin. Careful management in liaison with both ophthalmologists and internal medicine or oncology practitioners is sensible in such cases. If systemic steroids are contraindicated (e.g. in patients with diabetes mellitus), then systemic NSAIDs should be used instead, but they are not usually as effective at reducing the inflammation.

Thankfully many cases of uveitis are not so severe and do not require systemic medication, apart perhaps from a few days of systemic NSAIDs for pain relief. Topical therapy is centred on the use of anti-inflammatory agents and usually atropine. Topical antibiotics are not usually necessary unless an ulcer or intraocular infection is suspected. Topical steroids are preferred over topical NSAIDs although the latter might be indicated instead if ulceration is present, or as well as topical steroids in very severe cases. The choice of topical steroid is important – agents that are potent and have good intraocular penetration are required. Thus 1% prednisolone acetate and 0.1% dexamethasone are preferred over betamethasone sodium phosphate which has little intraocular activity. The frequency of application will clearly depend upon the severity of the uveitis, as will the duration of treatment. Mild cases might require just twice daily dosing for a week, while severe cases will require six to eight times daily initially, reducing as the condition improves, but many weeks of treatment are often required to prevent relapses. Clearly the underlying cause will affect the long-term outcome as well – immune-mediated causes will require life-long treatment. Occasionally subconjunctival steroids can be injected but they are not a substitute for topical therapy. Methylprednisolone acetate is the most commonly used – 4–10 mg in the bulbar conjunctiva.

Topical NSAIDs are used if corneal ulceration is present (and thus topical glucocorticoids are contraindicated) and in severe cases in conjunction with steroids where

their combined effects are additive. The most frequently used topical agents are flurbiprofen and ketorolac trometamol, usually two to four times daily.

Topical atropine is an important agent to use in dogs with uveitis. This parasympatholytic drug is both mydriatic and cycloplegic. By dilating the pupil synechia formation should be reduced, and paralysing the ciliary body muscle relieves the pain associated with spasm of this muscle. There is also a non-specific stabilization of the blood–aqueous barrier. Care should be exercised if secondary glaucoma is a risk – in severe cases of uveitis where posterior synechiae are already present, and in breeds which suffer from primary goniodysgenesis such as springer spaniels and flat coated retrievers for example. Atropine will lower aqueous tear production so its use must be cautious in patients with low Schirmer tear test readings. These should be checked at each re-examination if borderline. Treatment is to effect, i.e. frequently enough to maintain a dilated pupil. This might be just once daily in mild cases, or three to four times in more severely affected animals. Atropine can last a long time in non-inflamed eyes. Therefore do not be surprised if the pupil is still slightly dilated a week after stopping treatment when the eye is quiet.

TREATMENT OPTIONS – SURGICAL

Surgery is only required in a very few specific cases of uveitis. Clearly, if a pyometra is present, an ovariohysterectomy is required along with systemic antibiotics, topical corticosteroids and atropine. If an aberrant parasite larva is present in the anterior chamber this might need surgical removal prior to treating with anthelmintics – the dead larva can cause a very severe inflammatory reaction with disastrous consequences for the eye and so removing it while still alive and following this with appropriate treatment is usually advised. Referral should be considered in such cases.

Most cases of uveitis which are secondary to intraocular neoplasia are not amenable to surgery but a few can be – these are discussed in the section on intraocular neoplasia. If the uveitis has resulted in secondary glaucoma, then enucleation might be advised. Clearly histopathology is important in such cases.

PROGNOSIS

As with the causes and treatment regimens for uveitis in dogs, the prognosis is also extremely variable. A simple case which is secondary to corneal damage – ulcer or foreign body – will resolve fully with no scarring, no relapse and normal vision. At the other end of the spectrum, a panuveitis with retinal detachment due to multiple myeloma will not just be blinding, but life threatening. The chronic sequelae of uveitis include posterior synechia, cataract formation, secondary glaucoma and phthisis bulbi, all of which can be blinding and some of which can require enucleation. Long-term treatment is frequently required in immune-mediated cases – chronic usage of topical steroids can predispose to corneal ulceration and owners must be made aware of this and asked to stop the drops should any signs of ulceration – a wet, painful eye for example – develop. Systemic steroids should be reduced to the lowest possible dosage regime.

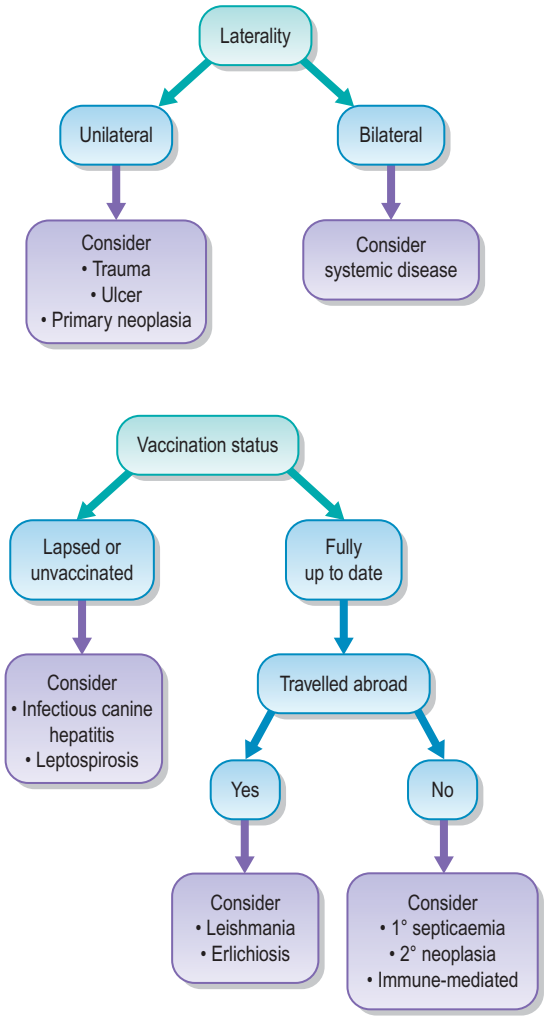


Figure 33.2 Decision-making tree – canine uveitis.

CASE EXAMPLE 33.1

Patient details: Dalmatian, 7 months old, female

PRESENTATION AND HISTORY

The dog was presented as an emergency. She had been completely fine the previous day, but on waking she appeared blind, both eyes were very cloudy and she held them closed and tried to shy away from the owner. She did not eat her breakfast. No previous ocular or systemic problems were reported. She had not travelled abroad and had completed her full vaccinations 3½ months previously.

CLINICAL SIGNS

Both eyes were similarly affected. Vision was absent but dazzle responses were present. Severe corneal oedema was present bilaterally, along with marked episcleral congestion and miosis (Figure 33.3). No corneal ulceration was present. Intraocular pressures were 6 mmHg bilaterally. Fundus examination was not possible. The dog was clinically depressed and pyrexic (39.5°C) although no other systemic abnormalities were detected.

WORK-UP

Blood was taken for routine haematology and biochemistry but surprisingly no abnormalities were detected.

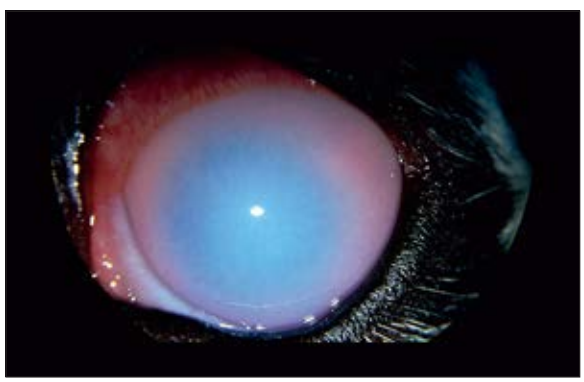


Figure 33.3 Acute uveitis in a 7-month-old Dalmatian showing corneal oedema, episcleral congestion and miosis.

Ocular ultrasonography showed debris in the anterior chamber, no lens abnormalities and no evidence of vitreal disease or retinal detachment. It was decided to treat the dog symptomatically initially and, depending on her response to treatment, to consider further laboratory investigation.

TREATMENT

The dog was started on prednisolone acetate drops hourly for 6 hours then every 2 hours plus atropine drops hourly for three doses then three times daily. Carprofen was given by injection together with intravenous cefalexin (although the bloods did not show any evidence of bacterial infection it was possible that the anterior chambers were infected and it was considered that the antibiotic would do no harm!).

OUTCOME

Twenty-four hours later the dog was dramatically improved. Vision was good from both eyes, the corneal oedema was clearing and the pupils were well dilated. Intraocular pressures were 12 mmHg in the right eye and 14 mmHg in the left. Fundus examination by indirect ophthalmoscopy revealed no problems. Rectal temperature was back to normal and the dog was bright and eating. The dog was sent home on six times daily prednisolone acetate, twice daily atropine drops and oral cefalexin and carprofen at standard doses. Five days later, only minimal conjunctival hyperaemia remained and treatment was reduced to Pred Forte only, four times daily. This was continued for 2 weeks then reduced to twice daily for another week before stopping. No relapses occurred and the dog did not develop any other symptoms whatsoever.

This is a slightly unusual case in that the eyes were very severely inflamed yet no abnormality was present on initial blood screening and she made a dramatic recovery such that further investigation was not justified. Bilateral trauma is unlikely to cause such symmetrical changes. The pyrexia could have been pain related, but a viral aetiology was also possible.

CASE EXAMPLE 33.2

Patient details: Labrador retriever, 10 years old, female (neutered)

PRESENTATION AND HISTORY

The dog presented with painful, cloudy eyes. The owner had noticed that they had been a bit red for a few days prior to suddenly deteriorating. Vision was reportedly OK but the dog was lethargic with a reduced appetite. No previous ocular or systemic problems were reported. She was fully vaccinated and had not travelled outside the UK.

CLINICAL SIGNS

Ophthalmic examination was bilaterally similar. Menace responses were present. Pupils were slightly miotic and slow to constrict further on stimulation with bright light. Some episcleral congestion was noted together with slight corneal oedema. No corneal ulceration was present. The eyes were very hazy in appearance due to marked aqueous flare which had a slightly reddish tinge. Fundus examination was not possible – some vitreal haze was also present. Intraocular pressures were 10 mmHg bilaterally. General clinical examination revealed that the dog was depressed but in good condition. Several petechial haemorrhages were noted along the gum margins and some bruising was present on the ventral abdomen. There was no history of recent treatment with any systemic medication.

WORK-UP

A systemic problem was suspected. Blood was taken for haematology, biochemistry and clotting profiles. These showed a severe thrombocytopenia with 8000 platelets/μl and a moderate regenerative anaemia. Prothrombin time and activated partial thromboplastin times were normal. Biochemistry was unremarkable. Urinalysis showed mild proteinuria but no haematuria. An antiplatelet antibody test was performed and was positive and so bone marrow aspirates for direct immunofluorescence tests were not considered necessary. Survey chest and abdominal radiography was unremarkable with no evidence of neoplasia. The diagnosis was immune-mediated thrombocytopaenia.

TREATMENT

Topical treatment was started with prednisolone acetate drops six times daily and atropine drops three times daily initially. Oral prednisolone at 2 mg/kg was started.

OUTCOME

A week later the eyes were improving well. The hyphaema had cleared well although a small area of vitreal haemorrhage was noted (Figure 33.4). Fundus examination revealed several pinpoint retinal haemorrhages (Figure 33.5). Clinically the dog remained lethargic and was polydipsic, polyuric and polyphagic from the steroids. Topical treatment was reduced to twice daily prednisolone acetate for a month and once daily atropine for a week. The systemic steroids were reduced to 1 mg/kg/day. Her platelet count at this stage had increased to 15 000/μl. The eyes fully resolved, although the retinal haemorrhages took several weeks to reduce.

The dog's general health improved slightly but then she became acutely lame on her right hind, with a painful swollen hock joint. A spontaneous haemorrhage was suspected. This resolved, and 2 months later the

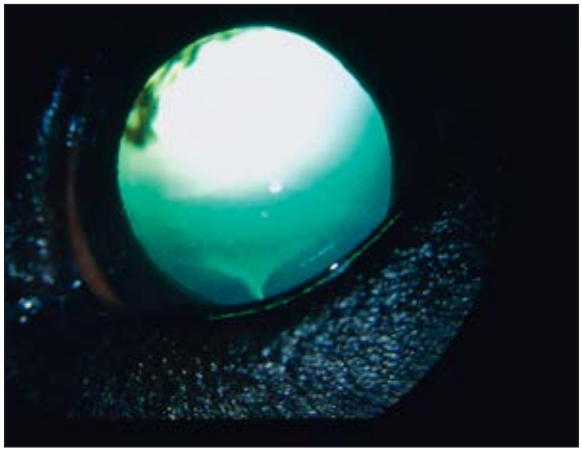


Figure 33.4 One week after starting treatment. The pupil is dilated from atropine use. Note the area of haemorrhage from 9–11 o'clock. This is in the anterior vitreous and will have come from the ciliary body.

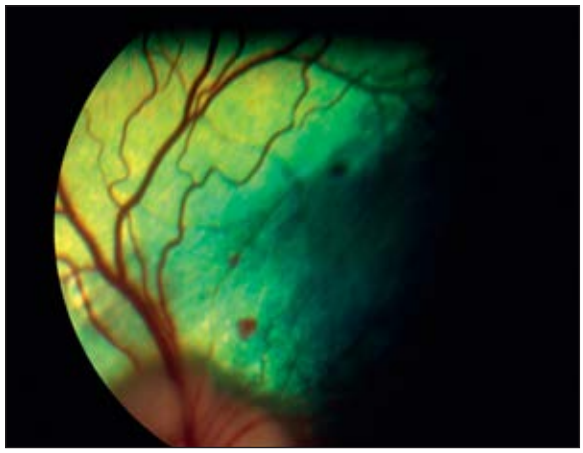


Figure 33.5 Fundus view 1 week after starting treatment showing retinal haemorrhages.

dog was bright and comfortable with no ocular changes (apart from the resolving retinal haemorrhages). Platelet counts returned to the normal range. She was maintained on oral prednisolone 0.5 mg/kg/48 hours for a further 2 months.

This case illustrates that what might appear to be a purely ocular problem should always be fully investigated since a serous systemic aetiology might be present.

34 Feline uveitis

INITIAL PRESENTATION

Abnormal appearance – red/cloudy

PRESENTING SIGNS

Cats with uveitis are normally presented because the owner has noticed a change in appearance of one or both eyes. This is usually a colour change, cloudy or red being most common. Sometimes there is blepharospasm and increased lacrimation but this is less frequently encountered than in dogs, mainly because uveitis in cats is more often insidious in onset and chronic by the time it is presented, unlike the acute episodes we see in dogs. Occasionally the cat is unwell but usually no other symptoms are reported.

CASE HISTORY

The history can be quite variable. Often there is nothing specific, just the change in appearance of the eye(s). Sometimes there is a history of a traumatic incident – a cat fight for example – when we can assume that the uveitis is secondary to this. Affected cats are more often outdoor types, possibly hunters or fighters, so it is worth asking the owner about the cat's lifestyle when history taking. It is also important to ascertain whether any previous illnesses have occurred and whether the cat has suffered from any ocular problems in the past – sometimes a traumatic ulcer will have been treated, but a low grade uveitis could have been undetected at the time and slowly progressed without the owner, or veterinary surgeon, being aware that the two conditions were linked.

CLINICAL EXAMINATION

A general clinical examination should check for evidence of trauma, particularly if only one eye is affected. If both eyes are involved then systemic disease is more likely and a detailed physical examination is required. Ophthalmic examination will follow the standard format. Most cats are visual. Light reflexes are often sluggish and pupils can be fixed due to secondary synechia formation. As such, a miotic pupil, considered typical in acute uveitis, might not be present.

Some conjunctival hyperaemia and/or episcleral congestion will be present but the outside layers of the eye are not as dramatically red as in the case of canine uveitis. Instead the redness is normally inside the eye. The iris will be inflamed; rubeosis iridis is engorgement of the iridal blood vessels and neovascularization of the iris. This is noticeable as redness in a gold iris, but might manifest as a change in iris colour – from blue to green for example in cats with blue irides. Hyphaema might also make the eye appear red, but again this is less common in chronic cases.

The eye will appear cloudy. This can be due to a combination of aqueous flare, keratic precipitates, hypopyon, cataract and vitreal opacification (Figure 34.1). Oblique examination of the eye, but with the light source directed perpendicular, will assist in locating the site of opacity. Keratic precipitates tend to accumulate ventrally on the corneal endothelium. When the cat is on the examination table and you are looking down at it these opacities might not be obvious. However, if you get the cat to sit at the edge of the table and look down, while you squat on the floor and look up into its eyes, the ventral half of the eye will be more easily examined. Also when looking for keratic precipitates it is important to check both eyes. Many cases of uveitis present unilaterally but on careful examination it is clear that both eyes are affected to differing degrees.

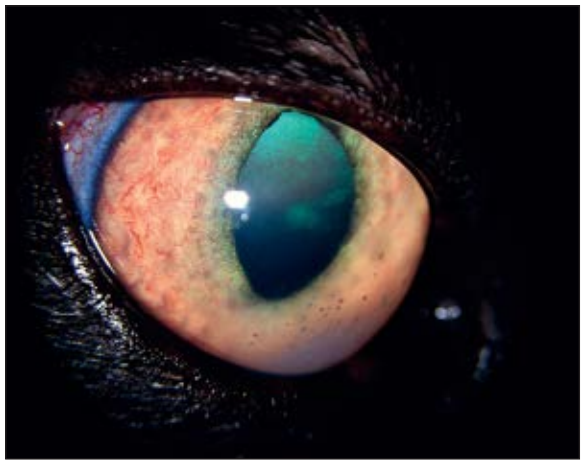


Figure 34.1 Idiopathic uveitis in a 6-year-old domestic short haired cat. Note the rubeosis iridis, aqueous flare and keratic precipitates (which are denser ventrally). Mild conjunctival hyperaemia is present along with some vitreal haze.

Aqueous flare will vary from just a slight haze to the anterior chamber through to full blown hypopyon obscuring further intraocular examination. Similarly, most cats will have a clear lens, but in chronic cases a total mature cataract can be present. Vitreal inflammation, hyalitis, is quite common in chronic uveitis cases and will most frequently be centred behind the lens – a pars planitis. Without careful examination this can be confused with cataract. Some cases of anterior uveitis will also have chorioretinitis as well. Thorough fundus examination, after pupil dilation with tropicamide, might reveal both active and inactive chorioretinal lesions.

The cornea should be routinely stained with fluorescein – traumatic incidents can result in ulceration, plus it is important to ensure that no active corneal damage is present since this could alter the treatment regime. Intraocular pressures should also be checked if possible. Uveitis results in a lowered intraocular pressure but chronic cases in cats frequently develop secondary glaucoma which might not be obvious on the ophthalmic examination alone.

DIFFERENTIAL DIAGNOSES

- Uveitis:
  - Traumatic
  - Associated with systemic disease: feline leukaemia virus (FeLV), feline immunodeficiency virus (FIV), *Toxoplasma*, feline infectious peritonitis (FIP), any septicaemia
  - Neoplastic
  - Idiopathic
- Corneal ulceration
- Hypertension
- Neoplasia
- Retinal detachment
- Lipaemic aqueous

CASE WORK-UP

If the case appears traumatic, minimal further work-up is required. However, most cases do not present with an obvious cause and, as such, diagnostic tests are normally required. The clinical appearance is the same regardless of cause, and thus one cannot determine which virus is involved, for example, on ophthalmic appearance alone. A baseline investigation would therefore include full haematology and biochemistry (looking for evidence of infection, renal disease, chronic anaemia for example) and viral screening.

FeLV and FIV status should always be checked. *Toxoplasma* titres (both IgM and IgG) are important in cats which hunt, fight or show any retinal changes. If the cat is FeLV positive then it should be carefully evaluated for evidence of systemic lymphoma – abdominal ultrasound should be performed for example. FIP titres and a coronavirus screen should be considered in young animals and cats which are systemically ill, but if only ocular changes are present in adults then this is a less likely cause for the uveitis.

Other infectious agents such as *Bartonella henselae* can be considered if the preliminary screening does not provide any definitive answers. Thankfully systemic fungal infections in cats in the UK are very rare, but can be considered if the cat has travelled from endemic countries. Unfortunately, despite extensive diagnostic testing, many cases remain idiopathic. On occasion it is useful to perform aqueocentesis to try to establish a definitive cause but such cases should be referred to a suitably qualified specialist – the risks of further iatrogenic damage are high even in experienced hands.

NURSING ASPECTS

Since most cats are systemically well the nursing relates only to the application of medication and ensuring that owners are able to follow the treatment regimen.

EPIDEMIOLOGY

Since many of the causes of feline uveitis are infectious, the lifestyle of the cat plays an important role regarding the risk of contracting uveitis. Outdoor type male or neutered male cats carry the highest risk of FeLV, FIV and toxoplasmosis. Multi-cat households will be more likely to contract FIP-related uveitis. Infectious causes tend to be more common in younger cats, with idiopathic causes or secondary neoplasia more likely in older patients.

Any infectious process (e.g. a cat bite abscess or bacterial cystitis) could, in theory, contribute to the development, or recurrence, of chronic uveitis in cats and should not be overlooked.

CLINICAL TIPS

- Carefully examine both eyes even if the presentation appears initially to be unilateral
- Examine from below to look for ventral keratic precipitates
- Check intraocular pressure
- Uveitis patients are often slow to respond to tropicamide and might require several applications to achieve sufficient mydriasis for fundic examination
- Don't forget to fluorescein-stain the eye
- There are no pathognomonic signs to suggest the aetiology for infectious causes of chronic feline uveitis

TREATMENT OPTIONS – MEDICAL

The medical treatment for chronic feline uveitis can be divided into specific and non-specific management. Thus, if the definitive cause is established, then appropriate medication is instigated:

- For cats with positive titres to *Toxoplasma*, clindamycin (25 mg/kg bid for 2 weeks) is advised.
- *Bartonella henselae* is sensitive to doxycycline (5 mg/kg bid for 2–4 weeks).
- If a cat bite abscess is present, a broad spectrum antibiotic such as cefalexin is advised.
- If FeLV positive and lymphoma is diagnosed, various chemotherapy protocols can be followed.

Unfortunately, in many cases we have to rely on non-specific treatment. This usually consists of topical corticosteroids and atropine. Providing no ulceration is present, prednisolone acetate and dexamethasone are good choices which penetrate the intact cornea well. The frequency of application will depend upon the severity of signs, but three to six times daily is typical. If ulceration is present then a topical NSAID can be given but these do not appear to be as effective clinically. Atropine is given to minimize the development of posterior synechiae, stabilize the blood–aqueous barrier and relieve the pain associated with ciliary spasm. Ointment formulations are preferable to drops in cats, since they tend to salivate profusely as the bitter tasting drops drain down the nasolacrimal duct. Atropine is given to effect – usually twice daily for a few days, then once daily and reducing as required. In a non-inflamed eye atropine will cause mydriasis lasting several days and owners should be warned that the pupil will remain dilated for some time after discontinuing this treatment. Topical antibiotics are not usually necessary in most cases of chronic uveitis unless a penetrating injury is suspected.

Some cats benefit from systemic steroids – prednisolone at 1 mg/kg twice daily for a week and then reducing can be used, especially if the cat is not amenable to frequent topical medication. Subconjunctival steroid injections can be given for the same reason, but clearly care should be taken if an infectious aetiology is suspected.

Glaucoma in cats is most frequently secondary to chronic uveitis. Indeed the cat might not be presented until in the late stages of both diseases. Some cats respond well to a combination of carbonic anhydrase inhibitors and steroids, both topically. Atropine should clearly be avoided if secondary glaucoma is suspected.

TREATMENT OPTIONS – SURGICAL

Surgery is not indicated in the treatment of uveitis with a few exceptions. Aqueocentesis, or rarely iridal biopsy, can be performed for diagnostic purposes. If the eye has developed secondary glaucoma and is painful, enucleation can be considered which will also provide useful information from histological examination. If the lens

has become cataractous such that the eye is blind, phacoemulsification can successfully restore vision, although ongoing medical treatment will be required for the uveitis. Some cats suffer from lens luxation associated with chronic uveitis and these can benefit from lendectomy. Clearly referral to a suitably qualified specialist is required for these latter procedures.

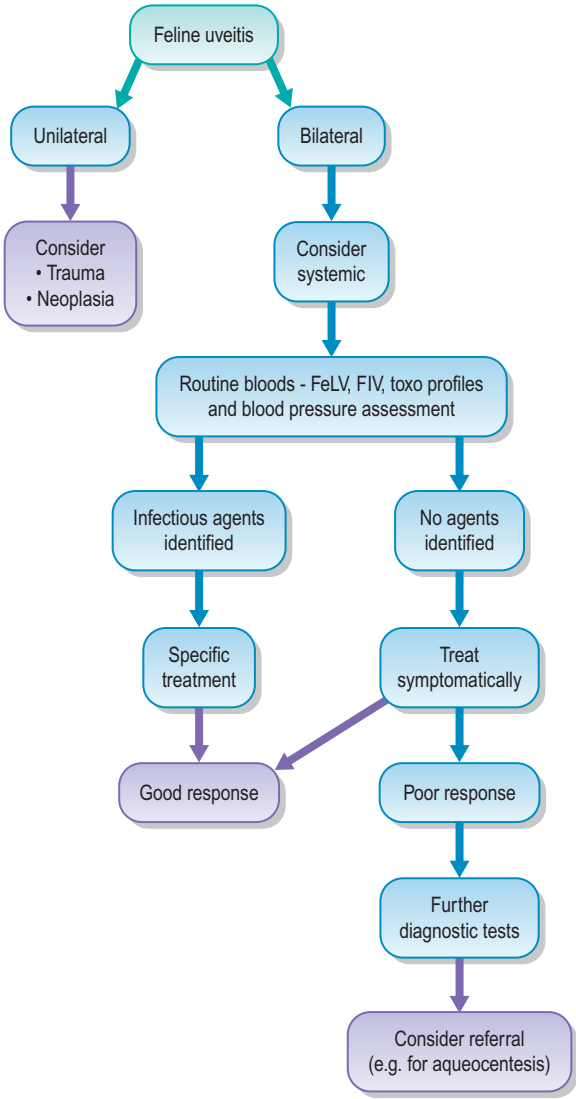


Figure 34.2 Decision-making tree – feline uveitis.

PROGNOSIS

The prognosis for feline uveitis will obviously vary according to the cause and severity. Long-term medication is often required.

Chronic sequelae of uveitis in cats include:

- refractory uveitis with poor response to medication
- recurrent episodes of inflammation
- posterior synechia formation
- cataract
- lens luxation (more commonly anterior)
- chorioretinitis and retinal detachment
- iris nodules
- corneal ulceration
- glaucoma
- phthisis bulbi
- intraocular sarcoma formation.

Some cats will make a full recovery, particularly if a treatable condition is diagnosed, although mild intraocular scarring could remain (Figure 34.3). However, many will be idiopathic and, despite a good initial response to treatment, can suffer from relapses which result in further intraocular damage. Thus posterior synechiae and cataractogenesis are common. Total cataracts can be surgically removed to restore vision. Some lenses will luxate as the lens zonules are damaged by the ongoing inflammation (Figure 34.4).

If glaucoma develops and is not diagnosed and treated quickly, it can result in serious optic disc and retinal degeneration such that vision is gradually and

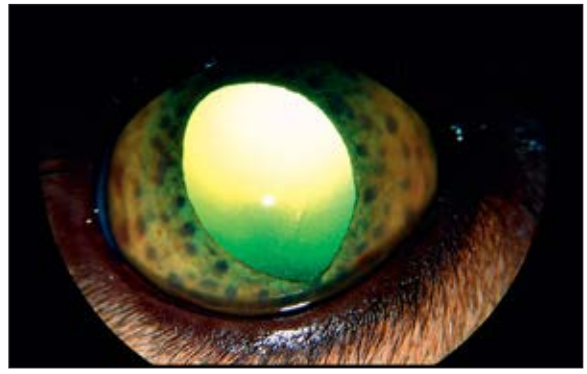


Figure 34.3 Iris nodules remain as an indication that this cat has suffered from uveitis although the eye is currently not inflamed.



Figure 34.4 Chronic uveitis in an elderly cat. The lens has a hypermature cataract which has luxated into the anterior chamber. Hyphaema and aqueous flare are both present, the pupil is miotic and some corneal vascularization is also present. Intraocular pressure was 6 mmHg.

permanently lost. Providing the cat is not in pain this can be managed medically, but enucleation is required in some cats. If a systemic disease is present, such as lymphoma, the prognosis will clearly be more guarded.

One other very important complication of chronic uveitis in cats is the development of intraocular sarcoma. Lens damage is required for this – which can be the result of traumatic damage or purely the result of chronic inflammation. The lens epithelial cells undergo neoplastic transformation and a sarcoma develops. These are both locally invasive (up the optic nerve to the brain) and can metastasize. They often do not develop for years after the inciting damage. Thus cats do require frequent monitoring, particularly if cataract is present with the chronic uveitis, and early enucleation with histopathology should be considered if there is any change to the appearance of the eye.

CASE EXAMPLE 34.1

Patient details: Domestic short haired cat, 16 years old, female (neutered)

PRESENTATION AND HISTORY

The cat had a long history of ocular problems. It had been diagnosed with uveitis in the right eye several years previously, and had received intermittent courses of steroid drops for this. However, no investigations had been undertaken as to the cause. Recently both eyes had changed in appearance – the right became cloudy while the left was red and cloudy and the owners sought veterinary attention again.

CLINICAL SIGNS

The cat appeared blind. It was slightly thin but otherwise general clinical examination was unremarkable. The right eye was comfortable while slight blepharospasm was present in the left. Menace responses were absent and pupils were fixed – the right was dilated and the left miotic. Dazzle reflexes were present bilaterally. The right lens was cataractous and luxated into the anterior chamber. No active inflammation was noted although the iris appeared mottled, suggesting iridal follicles from previous inflammation. The left showed conjunctival hyperaemia, corneal oedema, aqueous flare, a dyscoria (with posterior synechiae) and rubeosis iridis. Slight fluorescein uptake occurred. Intraocular pressures were 19 mmHg in the right eye and 8 mmHg in the left eye (Figures 34.5 and 34.6). Fundus examination was not possible. A diagnosis of chronic uveitis with a recent flare-up in the left eye was reached.

WORK-UP

Blood was taken for routine haematology and biochemistry which showed mild renal insufficiency. The cat was FeLV positive but FIV negative. Systemic blood pressure

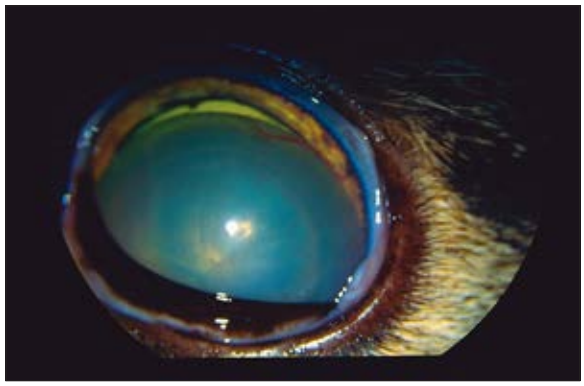


Figure 34.5 Right eye of 16-year-old cat with cataractous anterior lens luxation.

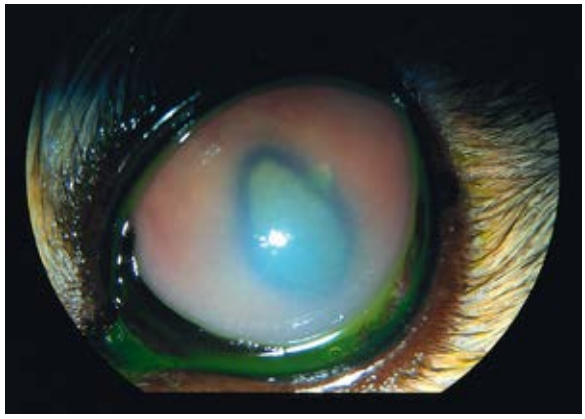


Figure 34.6 Left eye of the same cat as in Figure 34.5 with typical signs of severe anterior uveitis.

was 180 mmHg (systolic) – considered normal. Ultrasonography was offered to look for evidence of lymphoma but this was declined. Thus, although the FeLV status was probably associated with the uveitis, this could not be definitively determined.

TREATMENT

The owners were reluctant for major intervention, and so although lendectomy in the right eye would probably restore vision, this was not undertaken. Instead the cat was placed on atropine ointment twice daily and fusidic acid once daily to the left eye, together with prednisolone 5 mg daily for 5 days then 2.5 mg daily. After a week the left eye was fluorescein negative and prednisolone acetate was started three times daily with the atropine reduced to once daily. Her diet was changed to a renal support diet which she readily ate (perhaps because of the steroids!).

OUTCOME

The right eye was unchanged. The left improved with clearing of the oedema and aqueous flare, reasonable pupil dilation and a return of some useful vision. She was maintained on 2.5 mg prednisolone every other day and prednisolone acetate twice daily to the left eye (no treatment to the right). After 6 weeks the left eye became painful with increased lacrimation and a superficial corneal ulcer was diagnosed. Fusidic acid was started twice daily with the anti-inflammatory drops being swapped to ketorolac (an NSAID) twice daily. Prednisolone was stopped. The ulcer healed after 2 weeks but the uveitis worsened. Prednisolone was restarted: 2.5 mg daily for a week then back to every other day. She was maintained on NSAID drops and no further ulcers developed in the following 3 months.

This case shows how cats can suffer from chronic uveitis for many years, sometimes just in one eye, but it is not unusual for the second eye to become affected. Long-term medication can be necessary to keep the eyes visual and comfortable. However, complications are frequent – shown with the lens luxation in the right (which did not cause any secondary glaucoma – unlike dogs) and the ulceration in the left.

CASE EXAMPLE 34.2

Patient details: Domestic short haired cat, 6 years old, male (neutered)

PRESENTATION AND HISTORY

The cat was presented 48 hours following a routine dental with a cloudy left eye. Several extractions had been necessary although there were no difficulties associated with this. The cat was on oral clindamycin. The owners noticed that the left eye became cloudy and red overnight but the cat was otherwise well and was eating.

CLINICAL SIGNS

The right eye was normal. The left showed marked hypopyon and some hyphaema. It was open and comfortable with no ocular discharge. The pupil was miotic (Figure 34.7). No menace response was present.

WORK-UP

It was assumed that the cat had developed uveitis as a result of the dental disease and initially no work-up was carried out.

TREATMENT

Chloramphenicol drops were started four times daily plus once daily atropine ointment. Clindamycin was continued orally.



Figure 34.7 Left eye of a 6-year-old domestic short haired cat with marked hypopyon and some hyphaema.

OUTCOME

Three days later the eye was no better. A more thorough examination at this time showed no direct pupillary light reflex or a consensual to the right eye. No dazzle reflex was present. These findings suggested severe ocular disease and irreparable blindness. Intraocular pressure was 32 mmHg in the left eye and 15 mmHg in the right. Ocular ultrasonography revealed marked debris in the posterior segment and a suspected retinal detachment. The shape of the globe was distorted ventrally – the outline of the sclera could not be followed. It was suspected that a posterior scleral rupture had occurred at the time of the dental, resulting in severe panophthalmitis. Enucleation was performed and this confirmed a 3 mm rent in the ventral sclera with severe bruising around it and significant blood and pus in the posterior segment. Regrettably, histology was not performed, although this would be expected to reveal a pyogranulomatous inflammation and bacteria would probably have been present in the vitreous.

The globe of cats sits very close to the maxillary dental arcade, and it is possible to penetrate the ventral sclera with periosteal elevators and other dental instruments quite easily. Care should always be exercised since the outcome for the globe will be disastrous. In some cases the ocular damage will be immediate, as in this case, but small scleral perforations can seal with minimal immediate inflammation, only to develop a refractory panuveitis several weeks or even months later, where the bacteria slowly multiply in the vitreal cavity and eventually wreak their havoc.

35 Rabbit uveitis

INITIAL PRESENTATION

Abnormal appearance – red /cloudy, pain

PRESENTING SIGNS

Owners will present rabbits with uveitis in one of two ways – either because the eye is red and painful, or because they have noticed a small white lesion within it. There is normally no history of trauma and in most cases the rabbit is systemically well. Younger animals tend to be affected and it is normally just one eye that is involved. It is unusual for the owner to have noticed any visual impairment.

CASE HISTORY

Often there is no prior general or ocular history, although occasionally the rabbit will have suffered from previous problems – urinary infections or dental disease for example. It is unusual for the owner to have tried any treatment before presentation. Normally the owner will have been aware of the ocular changes for days to weeks before presentation – the lack of systemic illness makes it unlikely for them to present the rabbit as an emergency.

CLINICAL EXAMINATION

The rabbit is normally systemically well but occasionally other systemic signs might be present. These could include weight loss, urine scalding, head tilt, dental disease and so on. Other ophthalmic lesions such as a dacryocystitis or retrobulbar abscess can be present but this is not typical. Normally one eye is completely normal on examination. The affected eye may or may not be visual, demonstrated by normal or absent menace responses, and if blind in one eye the animal is likely to be nervous and 'jumpy' when approached on the blind side.

The most obvious lesion is usually a white mass within the anterior chamber, attached to the iris and lens at the pupil margin. The pupil is distorted as a result (dyscoria). Local uveal inflammation with rubeosis iridis will be appreciated in animals with a pale coloured iris (Figure 35.1). The lens is often opaque with total or partial cataract formation. Some episcleral congestion and conjunctival hyperaemia are to be expected but the eye is often non-painful (unless affected with secondary glaucoma). Ulceration is not a feature.

DIFFERENTIAL DIAGNOSES

- Uveal neoplasia
- Lens-induced uveitis (phacoclastic) due to *Encephalitozoon cuniculi*
- Uveal abscess (e.g. *Pasteurella multocida* infection)
- Iris cyst
- Granulomatous uveitis
- Traumatic uveitis (e.g. cat scratch)

CASE WORK-UP

A thorough ophthalmic examination should narrow the differential list to either phacoclastic uveitis due to *E. cuniculi* or an abscess due to a bacterial infection such as *Pasteurella multocida* or *Escherichia coli*. General clinical examination should determine whether any systemic involvement is present. A minimal work-up should



Figure 35.1 Uveitis due to *Encephalitozoon cuniculi* in a young dwarf rabbit. Note the bulging iris dorsally, with rubeosis iridis, plus the dyscoria and white lesion within the pupil – the lens is cataractous.

include serology for *E. cuniculi* (antibody detection), the results of which need interpreting in conjunction with clinical signs, since a positive titre demonstrates exposure to the organism but not necessarily active infection which can be attributed to the clinical symptoms. The organism is excreted intermittently in urine, but collection is not usually easy from pet rabbits and false negatives can occur if the sample happens to be taken during a period of non-shedding. It is unusual for a more thorough laboratory work-up to be undertaken in pet rabbits – response to treatment is more frequently used to assist with definitive diagnosis.

NURSING ASPECTS

The general nursing of lagomorphs should be addressed – keeping warm, feeding from appropriate containers, water dispensers with which they are familiar and so on. Topical and systemic medication might both be required, and if anaesthesia is required the specific risks of this in rabbits need considering.

EPIDEMIOLOGY

Encephalitozoon cuniculi is an obligate intracellular microsporidium parasite which principally affects rabbits, but can be found in other species, including, on occasion, immunocompromised human beings. Infection is via ingestion of food contaminated with infected urine, and the spores extrude a polar filament when in the gastrointestinal tract of the rabbit. Host cells become infected as sporoplasm is transferred though the polar filament and replication occurs in the cell. This then ruptures, releasing spores which invade further cells throughout the host. Chronic inflammatory reactions, particularly granulomatous lesions, develop as a result of host cell rupture and spore release.

Infection can occur vertically in utero, and it is thought that this is how the eye, and particularly the lens, become affected. In the adult the lens capsule is too thick for the polar filament to penetrate, but in embryos and fetuses the capsule is thin or non-existent, allowing spores to penetrate and cause disease in early adulthood.

E. cuniculi is involved in a number of clinical symptoms in addition to phacoclastic uveitis. These include vestibular disease and urinary tract infections most commonly, but a wide variety of presentations – from subclinical carriers to sudden death – occur. The degree of infectivity is difficult to determine; certainly if one rabbit is affected it is likely that others housed in the same environment will be seropositive, but it is not fully understood how this might relate to clinical disease. All breeds can be affected, but the condition has been reported in dwarf rabbits more commonly than in other breeds. No sex predilection exists.

Once the lens is infected with spores, these infect lens epithelial cells and result in rupture of the anterior lens capsule. This triggers a granulomatous, phacoclastic uveitis, and cataract formation also occurs. Histologically the inflammation is centred around the lens capsule rupture which is normally at its thinnest point axially.

Pasteurella multocida and *Escherichia coli* can also cause a similar clinical picture, with low grade uveitis and granuloma/abscess formation in the iris or anterior chamber, such that differentiation on clinical examination is not possible. However, it appears that with these causes a more florid uveitis can be expected, with more severe aqueous flare or hypopyon and a painful globe. A generalized septicaemia and ill rabbit can also be encountered.

CLINICAL TIPS

- Check for evidence of trauma
- Carefully look to see where the 'abscess' is centred – on the front of the lens close to the pupil is typical of *E. cuniculi*
- Check the other eye
- Check for evidence of systemic disease
- Consider the age of the rabbit

ANATOMY AND PHYSIOLOGY REFRESHER

The uveal tract and lens of the rabbit are very similar to other mammals with a couple of exceptions. The uveal tract is highly labile – the blood–aqueous barrier is more permeable and thus uveal inflammation is common. In addition, lens rupture leads to an inflammatory and granulomatous reaction rather than the proliferative reaction seen in dogs (which leads to fibroplasia and synechia formation). These factors contribute to the type of uveitis encountered with *E. cuniculi* which is totally different from the catastrophic phacoclastic uveitis seen in dogs with similar lens injury.

TREATMENT OPTIONS – MEDICAL

Medical management for cases of *E. cuniculi* includes topical and systemic medication. Topical corticosteroids will reduce some of the associated inflammation but would not be expected to reduce the size of the granuloma or prevent the cataract from progressing. However, they can be beneficial if surgery is not being considered. Topical atropine can be used if significant uveitis is present but caution should be exercised in rabbits due to the risk of ileus.

Systemic antibacterials are rarely successful, but if a bacterial infection is suspected systemic enrofloxacin is licensed for use in the species and can be helpful. Systemic anthelmintics such as albendazole and more commonly fenbendazole have been used and do seem to eliminate the organism, but do not reverse the ophthalmic changes present.

In a unilateral case, where the rabbit is systemically well, a combination of topical anti-inflammatory agents and systemic anthelmintics is a sensible approach. Isolating the rabbit from fellow companions can be considered but since the disease is not particularly contagious this might not be necessary. The owners should be aware that the eye will never return to normal, but so long as the rabbit is well and the eye is comfortable, this approach is acceptable.

TREATMENT OPTIONS – SURGICAL

There are two possible surgical treatments for phacoclastic uveitis – enucleation or phacoemulsification of the lens together with removal of the granuloma. If the eye is painful and not responding to topical anti-inflammatory agents (together with a course of fenbendazole), then enucleation is a viable option. Care should be exercised since the rabbit possesses a large orbital venous plexus and severe haemorrhage (even death) can occur

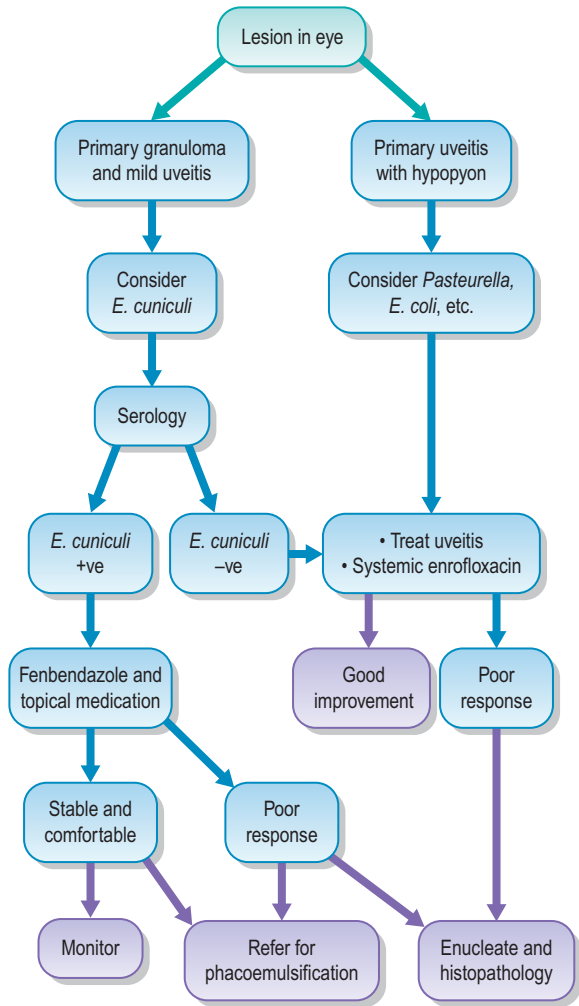


Figure 35.2 Decision-making tree – rabbit phacoclastic uveitis.

if this is damaged. Thus a transconjunctival rather than transpalpebral approach to the enucleation (where it is possible to keep the dissection very close to the globe) is a safer surgical choice in this species. Histopathology should be considered, particularly to differentiate *E. cuniculi* from bacterial causes of the uveitis and lens damage.

If the rabbit is clinically well, and the owner does not wish for enucleation, or in bilateral cases, referral for phacoemulsification can be considered. Clearly the risks of lengthy anaesthesia are greater than for other species, and the financial costs are high, but this surgery can be very successful, resulting in good vision and no recurrence of the uveitis.

PROGNOSIS

The prognosis is generally reasonable for cases of phacoclastic uveitis providing the rabbit is clinically well. Obviously patients with neurological or severe urinary tract disease carry a guarded prognosis but in most rabbits with ocular signs, these are the only clinical symptoms, and remain so. Thus stabilization of the globe with anti-inflammatory medication, together with systemic anthelmintics, can control, but not cure, the ocular disease. Enucleation will cure the ocular signs but the fellow eye could become involved. Rabbits will adjust to blindness, especially if kept with a companion, but some owners request euthanasia of blind animals.

36 Hyphaema

INITIAL PRESENTATION

Abnormal appearance – red

PRESENTING SIGNS

Hyphaema is blood in the anterior chamber, and therefore presents as a red eye! Normally it is acute in onset. The owners might be aware of a traumatic incident – road traffic accident, kick in the face by a horse or accidental blow to the eye by a football for example. However, there might be no antecedent event known. The patient might be well in themselves, or have other clinical signs depending on the underlying cause for the bleeding. One or both eyes might be affected and vision can clearly be impeded. Many patients show no particular signs of ocular discomfort, but some exhibit the typical signs of ocular pain – namely blepharospasm, photophobia and increased lacrimation. Owners will normally present patients to the surgery rapidly since the dramatic appearance of hyphaema is alarming to them, even if the patient seems otherwise well.

CASE HISTORY

The history of patients with hyphaema can be very variable depending on the underlying cause. As mentioned above, a traumatic incident might be known. The patient might have had access to rat poison or similar toxins, or have recently travelled abroad and come into contact with infectious agents such as the tick-borne diseases. A history of previous systemic disease such as hepatic or renal insufficiency might be present, or the patient might have known neoplastic disease elsewhere in the body. It might be undergoing treatment for hypertension or have an underlying cardiac problem. In young animals a congenital abnormality might be present and examination of litter mates and sire and dam could be useful. Thus it is very important to take a thorough case history for any patient presenting with hyphaema – there are numerous potential causes, some of which can be ruled out immediately from the history, while others will require further investigation.

CLINICAL EXAMINATION

A general clinical examination must be performed on any patient presenting with hyphaema. Careful examination for haemorrhages elsewhere in the body – gums, skin, into the gastrointestinal tract for example – should be undertaken. The patient should be checked for signs of trauma – bruising around the face, scuffed nails, other injuries including skull fractures and so on. Chest auscultation and abdominal palpation can be informative. Occasionally the patient will be pyrexic if an infectious aetiology is present.

Ophthalmic examination will include careful assessment of the globe for evidence of trauma – blunt or penetrating. Subconjunctival haemorrhage is frequently present following blunt trauma, while a corneal injury might be noted following sharp trauma. Fluorescein testing should be performed. Vision should be assessed. The amount of blood present in the anterior chamber will determine the appearance – a small bleed will settle to the ventral anterior chamber and a fluid line will be noted, while a more severe bleed can present with the anterior chamber full of blood such that no intraocular structures can be evaluated (Figure 36.1).

If the pupil can be seen, reflexes should be checked. If it cannot be seen, then the indirect response in the fellow pupil is important since this can indicate whether

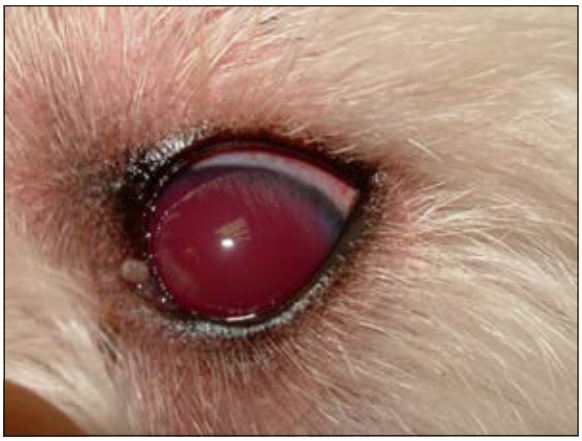


Figure 36.1 Total hyphaema in a 4-year-old West Highlight White terrier. The dog ran into the door jamb while chasing a cat out of the kitchen the previous day, and thus a traumatic aetiology was suspected.

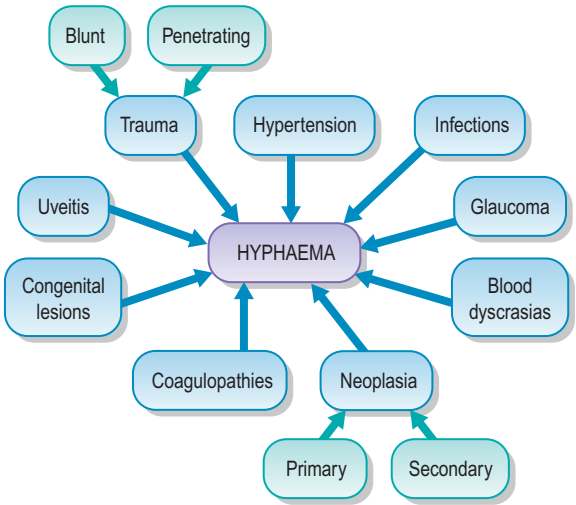


Figure 36.3 Decision-making tree – causes of hyphaema.

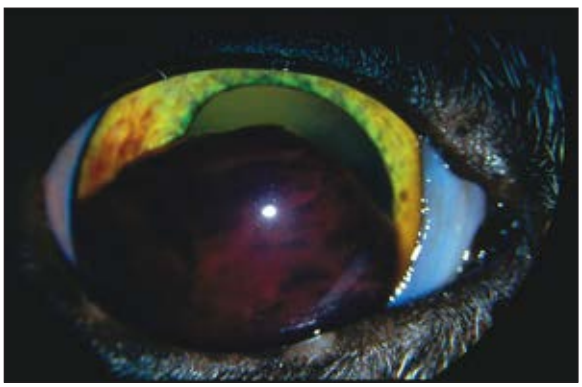


Figure 36.2 Clotted hyphaema in a hypertensive cat.

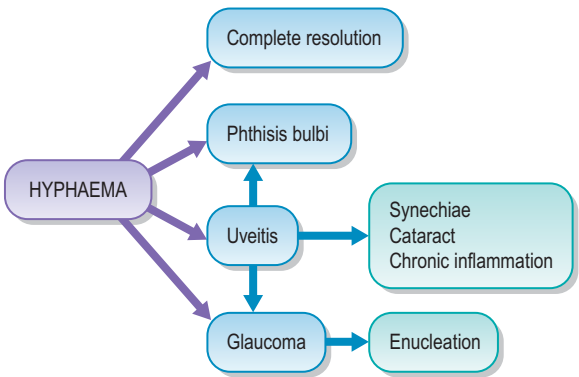


Figure 36.4 Sequelae of events in hyphaema.

the fundus and optic nerve in the affected eye are responsive to light. If there is no constriction of the fellow pupil when light is shone into the affected pupil, this might suggest severe posterior segment damage and the prognosis for any return of vision is guarded. The dazzle reflex should be similarly checked and carries comparable prognostic information.

The colour of the blood can give an indication regarding chronicity – in acute bleeds it will be bright red, while if present for several days it becomes much darker in colour. If clotted there might be 'clumps' seen in the pupil, mixed with fibrin and other inflammatory mediators (Figure 36.2), while unclotted blood will sink to the ventral anterior chamber.

The fellow eye should be evaluated. If trauma is not the cause for the hyphaema, then a systemic disease could be present. In many such cases there will be bilateral ocular abnormalities, although these are not necessarily symmetrical. Small petechiae might be visible on the iris providing it is not very darkly coloured when appreciation of such haemorrhages is much more difficult. Fundus examination might reveal retinal haemorrhages or vessel abnormalities suggestive of a vasculitis or hypertension. Retinal detachment might be present. If vitreal haemorrhage is present, this will be seen as strands or clumps of blood obscuring the view of the fundus (see Case 33.2 and Figure 33.4).

Intraocular pressure should be measured, if possible, and can be normal, reduced or elevated in cases of hyphaema. Treatment choices will vary according to intraocular pressure and hence measurement is important.

DIFFERENTIAL DIAGNOSES

There are many potential causes for hyphaema (see Figure 36.3):

- Trauma – blunt or penetrating
- Coagulopathies – inherited clotting problems, following dicoumarin poisoning, acquired with renal or hepatic disease
- Blood dyscrasias – hyperviscosity, anaemia, polycythaemia, thrombocytopenia of various causes
- Hypertension
- Vasculitis – due to tick-borne diseases (e.g. ehrlichia)
- Intraocular neoplasia
- Associated with uveitis, glaucoma or retinal detachment where neovascularization has occurred
- Associated with congenital abnormalities – collie eye anomaly, persistent hyperplastic primary vitreous, persistent hyperplastic tunica vasculosa lentis, retinal dysplasia.

CASE WORK-UP

If there is total hyphaema, such that intraocular examination is not possible, then ultrasonography should be performed. This is of particular help following blunt trauma, where evaluation of intraocular structures can be essential regarding the prognosis for return of vision, or even saving the eye. Thus the globe outline should be evaluated. With severe blunt trauma posterior scleral rupture can occur and such eyes should undergo early enucleation. If the globe appears intact, evidence of vitreal haemorrhage or debris, retinal detachment and lens damage can be assessed.

The prognosis for purely anterior chamber bleeding is much better than if the posterior segment is also involved. In addition to checking for traumatic damage, neoplastic processes might be identified on ocular ultrasound – a ciliary body mass, for example, can cause quite severe haemorrhage. If suitable probes are not available for ocular examination (7.5–12 Hz normally), or if the practitioner is not familiar with the interpretation of ocular ultrasonograms, then referral should be considered.

If there is evidence of trauma to the globe, skull radiography might be warranted.

If there is no suggestion of trauma, from the history and clinical examination, and especially if the condition is bilateral, laboratory investigation is necessary. Blood samples for complete haematology and biochemistry (including electrophoresis) and clotting profiles should form the baseline evaluation. Blood pressure should be measured. Depending on the findings, further investigations such as urinalysis, abdominal ultrasonography and radiography, serology for specific infectious agents and bone marrow biopsy might be advised.

NURSING ASPECTS

All patients with hyphaema should be kept quiet. If allowed to bark or get excited the blood will mix with the aqueous again and resorption will be reduced. Activity levels certainly affect the distribution of blood – owners will frequently comment that the eye looks better in the morning, after the pet has been asleep and settled, but becomes more red during the day purely as a result of ocular and general movement and not because there has been further bleeding.

Thus cage rest (with sedation if required) is advised. Topical medication will normally be prescribed. Some patients have serious, potentially life-threatening systemic disease and general supportive as well as specific nursing duties will be applicable.

EPIDEMIOLOGY

Traumatic events commonly cause hyphaema but are sporadic single events. Blunt trauma will frequently cause more severe damage than sharp trauma as a result of compression and expansion of the globe followed by oscillations between the two in aftershock ripples. These cause alternate crushing and stretching of the iris, lens, vitreous, choroid, retina and optic nerve which can have catastrophic sequelae.

Sharp trauma will cause corneal damage and iridal haemorrhage. Providing the lens has not been damaged (see Case example 24.1), then the cornea can be repaired (surgically if necessary) and the prognosis is generally reasonable.

Many systemic causes of hyphaema result from primary haematological disease – thrombocytopenia, clotting abnormalities, anaemia, hyperviscosity and so on, and it is beyond the scope of this book to go into details regarding the pathogenesis of each specific possible cause.

Erythrocytes released into the anterior chamber are phagocytosed by cells within the trabecular meshwork of the drainage angle. The anterior iris releases fibrolysin which helps to dissolve clots such that the cells can be removed via the drainage angle. Very little blood is actually resorbed through the iris.

CLINICAL TIPS

- Examine the eye carefully for evidence of trauma
- Check the fellow eye – including fundus examination
- Look for bleeding elsewhere in the body
- In the absence of trauma consider systemic disease
- The underlying systemic disease could be life threatening and potentially much more serious than the hyphaema
- Measure systemic blood pressure

TREATMENT OPTIONS – MEDICAL

The general aims of treatment are to identify the cause, prevent recurrent bleeding, control the uveitis and limit the sequelae of the uveitis.

Most treatment options for hyphaema are medical. If there is an underlying systemic disease, clearly treating this is of utmost importance. For example, a case of ehrlichiosis might require doxycycline, warfarin poisoning might require vitamin K and supportive treatment, immune-mediated thrombocytopenia might require immunosuppressive doses of systemic steroids (see Case example 33.2), hypertensive patients would benefit from amlodipine and so on. We will not go into specific treatment regimens since these will vary tremendously from case to case.

Specific treatment for the hyphaema is generally symptomatic and few controlled studies have been undertaken to test the efficacy of the different medications available. Certainly there is some controversy amongst ophthalmologists regarding the merits or otherwise of the different agents available. Rest (with sedation if required) is always helpful.

Providing there is no corneal damage, the use of topical corticosteroids is beneficial and provides the mainstay for hyphaema treatment. Prednisolone acetate and dexamethasone two to four times daily are good choices. Occasionally subconjunctival steroids can be used. Topical NSAIDs are avoided due to their effects on platelet function and clotting – their systemic use is also not recommended routinely for the same reason. If the condition is painful, then opiate analgesics are more appropriate. Mild cases of hyphaema will resolve gradually in 1–2 weeks.

Topical atropine drops are of benefit in some patients, but care must be exercised due to the risk of glaucoma. A decision must be made, balancing this risk of glaucoma against that of increased uveitis and pain, plus sequelae such as iris bombe if atropine is not used. Frequent intraocular pressure measurements (once or twice daily) should be undertaken in cases of severe hyphaema.

The use of topical pilocarpine is controversial in hyphaema patients. Some people advocate its use – the miosis induced will open the trabecular meshwork, thus facilitating the removal of the erythrocytes and providing a greater surface area of iris for the release of fibrinolytics, enhancing clot resolution. However, pilocarpine also causes ciliary muscle spasm, causing ocular discomfort, and by causing dilation of iridal vessels it can predispose to further bleeding. Alternating pilocarpine with sympathomimetic agents such as phenylephrine is supposed to reduce the risk of synechia formation but this is of dubious benefit. Their vasoconstrictor effect is also thought to counteract the blood vessel dilation caused by pilocarpine but again the benefits are questionable.

If there is no evidence of systemic infection, then systemic steroids such as prednisolone are frequently prescribed. These will not speed up the removal of the blood from the anterior chamber, but should reduce the associated uveitis and thus the risk of complicating sequelae is lessened.

Where intraocular pressure is raised, or if the risk of secondary glaucoma is considered high but frequent intraocular pressure measurement is not possible, then anti-glaucoma medication might be warranted. Topical carbonic anhydrase agents such as dorzolamide and brinzolamide are most frequently used, and are normally applied three times a day.

Fibrinolytic agents have some value in dissolving blood clots, thus facilitating the removal of erythrocytes. Tissue plasminogen activator (tPA) is most frequently used, but referral to a specialist is advised. Intracameral

(into the anterior chamber) injection is required and the timing of treatment and close post-medication monitoring are essential to the success of this agent.

TREATMENT OPTIONS – SURGICAL

Unfortunately the most frequent surgical procedure for cases of hyphaema is enucleation. This is indicated in cases of severe trauma. Blunt trauma with rupture of the sclera (most commonly just behind the limbus or at the optic nerve head – these are the weakest parts), lens rupture, and severe vitreal haemorrhage with retinal detachment all require globe removal. Enucleation might also be advised following extreme sharp trauma – particularly where the lens has been badly damaged or loss of intraocular contents has occurred through the wound. The long-term sequelae of hyphaema, which mainly relate to chronic uveitis and glaucoma, might also necessitate enucleation.

Enucleation is also indicated if the hyphaema is due to intraocular neoplasia. This is frequently primary, most often due to ciliary body masses such as adenocarci-

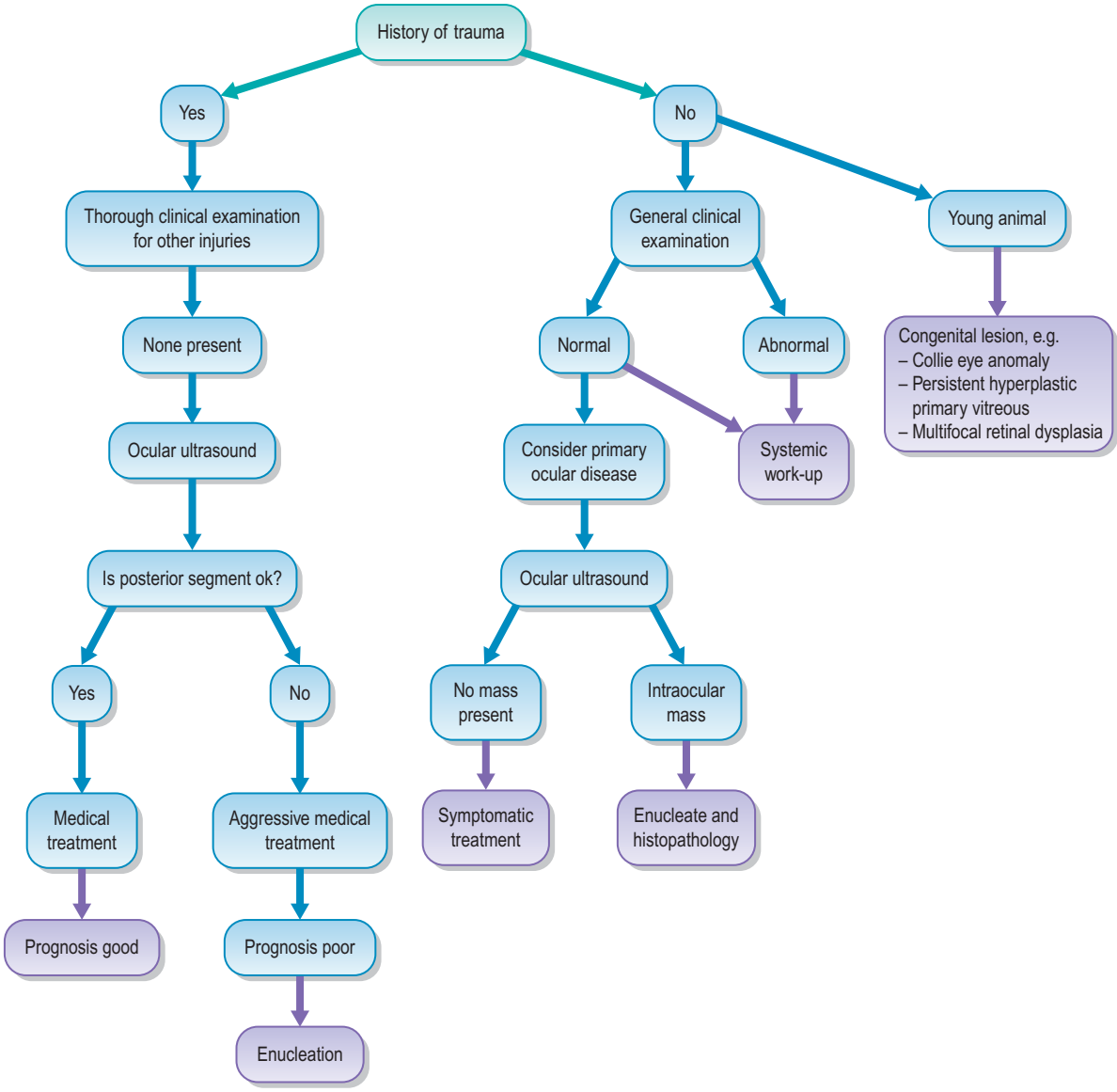


Figure 36.5 Unilateral hyphaema.

noma, and the patient can be cured by ocular removal. However, secondary neoplasia can also present as an intraocular mass with hyphaema (such as from mammary carcinoma) and survey ultrasonography and/or radiography should always be considered. Histopathology should also be performed on all enucleated globes, particularly if the cause for the hyphaema is not immediately obvious.

Another surgical option, which is rarely indicated and best left to specialists, is the drainage of the blood. Gentle flushing of the anterior chamber with concurrent aspiration (e.g. using the irrigate–aspirate handpiece of a phacoemulsification machine) is occasionally of benefit.

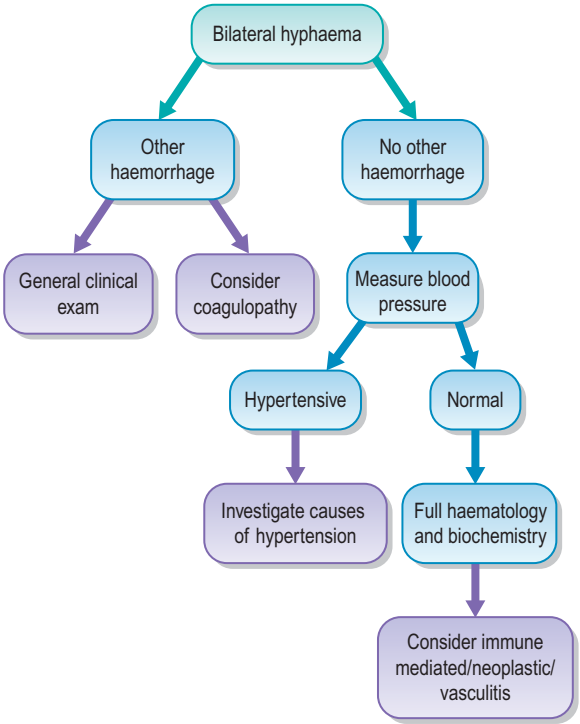


Figure 36.6 Bilateral hyphaema.

PROGNOSIS

The prognosis for cases of hyphaema depends on the cause and the degree of haemorrhage. Mild cases with less than half of the anterior chamber affected, and no posterior segment damage, should have a good prognosis. Treatment with topical steroids and a few days of topical atropine should result in a gradual clearing of the blood in 1–2 weeks and complete recovery. If the hyphaema is total and has not started to clear after 7–10 days, the prognosis becomes worse. Chronic uveitis will set in, with synechia formation, including iris bombe, secondary cataracts and glaucoma all potential outcomes. The prognosis for saving the eye, let alone any restoration of vision, is guarded in such cases.

Patients with underlying systemic disease need to have this assessed before a prognosis can be given. Patients with infectious disease might be cured, those with hypertension for example might be controlled and vision preserved, while those with immune-mediated disease or neoplasia (such as lymphoma and multiple myeloma) might have life-threatening disease, in which case the hyphaema is the least of their problems.

CASE EXAMPLE 36.1

Patient details: Shih Tsu, 10 years old, female (neutered)

PRESENTATION AND HISTORY

The patient was presented with sudden onset red eyes and blindness. She had been a bit quiet for a couple of days. Two weeks previously she had been acutely lame on her right hind with pain in the stifle. This resolved fully after two injections of carprofen and strict rest. No other history was reported and the owner was sure that the dog had had no access to rat or other poisons.

CLINICAL SIGNS

The dog was totally blind and clearly frightened. Menace responses were absent and pupillary light reflexes could not be evaluated; however, dazzle reflexes were good. Both eyes were similar. Total hyphaema prevented intraocular examination – only the edge of the iris could be seen (Figure 36.7). Conjunctival hyperaemia and some subconjunctival haemorrhage were noted. A mucoid discharge was present but no ulceration and Schirmer tear test readings were normal. Intraocular pressures were



Figure 36.7 Total hyphaema and subconjunctival haemorrhage in a 10-year-old Shih Tsu.

10 mmHg bilaterally. On general clinical examination the dog had a grade 3/6 systolic cardiac murmur which had been present from a young age and was subclinical; there were no other abnormalities detected.

WORK-UP

Blood was taken for routine haematology, biochemistry and clotting profiles. Apart from very mildly raised liver enzymes these were normal. Systolic blood pressure was normal. Ocular ultrasonography revealed no evidence of retinal detachment or vitreal abnormalities.

TREATMENT

Symptomatic treatment was instigated using prednisolone acetate drops four times daily and once daily atropine.

OUTCOME

Four days later the dog was much improved. The haemorrhage had clotted and was retracting in the anterior chamber. Some vision had returned. Intraocular pressures were 14 mmHg in the right eye and 12 mmHg in the left eye. Indirect ophthalmoscopy revealed no fundus changes. Atropine was stopped but the prednisolone acetate was continued four times daily. After another week the clots were 50% smaller again and in the coming month they resolved completely. There were no recurrences and the dog remained systemically well.

This case illustrates a single episode of severe hyphaema for which the cause was unknown. Possibilities include mild warfarin poisoning which did not result in long-term clotting problems but was enough to allow a single bleed, choking or neck trauma (foxes were present in the garden but the dog had no other apparent injuries), or a strange idiosyncratic reaction to the previously administered carprofen. The owner was warned that if further haemorrhage occurred then repeated blood tests would be required – a progressive haematological disorder could be present or even underlying neoplasia. Clearly from a veterinary standpoint it is frustrating that the cause was never identified, but as we know this is not infrequent in veterinary medicine, and at least the patient was fine!

37 Canine intraocular neoplasia

INITIAL PRESENTATION

Abnormal appearance – red/change in shape

PRESENTING SIGNS

Most cases present because the eye has become red or the owner has noticed a change in appearance. Some blepharospasm and ocular discharge might be present but this is not consistent, especially early in the course of the disease. It is more common to encounter unilateral cases, associated with primary intraocular neoplasia, but bilateral cases, normally associated with metastatic disease, do occur. These latter patients are more likely to present with a uveitis rather than just change of appearance, and the patient might be systemically unwell. Such cases are discussed more in the section on uveitis. Thus, in most dogs, there are no other clinical symptoms which the owner has noticed. Sometimes the owner has not been aware of any problem at all and the lesion is identified by the veterinary surgeon during a consultation for something else. Affected dogs are typically 8–10 years and older.

CASE HISTORY

In most cases there is no previous history of ocular disease, or relevant systemic disease.

CLINICAL EXAMINATION

Since most cases of intraocular neoplasia will be primary rather than secondary it is likely that the general clinical examination will be normal. Obviously if both eyes are affected or the patient is unwell, then metastatic disease is more likely. However, the majority of cases will be unilateral. As such, the abnormal eye can be compared to the normal one during examination which is helpful.

The affected eye might be blind but usually vision is present. Thus a menace response is likely to be normal. Some conjunctival hyperaemia and episcleral congestion might be present but not always. Normally a discoloured lesion will be present. This can be on or in the iris, or behind it poking thorough the pupil. Its colour can vary from dark brown to pale pink and various shades in between (Figure 37.1). The pupil is frequently distorted (dyscoria) but pupillary light reflexes remain. Mild uveitis, with aqueous flare and episcleral congestion, can be present and occasionally the patient is not presented until the eye is painful and enlarged through secondary glaucoma.

If the neoplasm is based in the iris, then the discolouration will be visible within the iris, and can be raised above it (Figure 37.2). Thus looking at the eye from the side can be useful to determine if the lesion protrudes out from the plane of the iris. Ciliary body-based masses are more difficult to see initially; however, by dilating the pupil the lesion should be more easily visualized through the pupil, especially when looking tangentially across the eye. Vitreal haze is often present with ciliary body neoplasms. Fundus neoplasia is rare but occasionally choroidal extension of uveal melanomas does occur.

The most commonly encountered uveal tumour in dogs is the iris melanoma/melanocytoma, with ciliary body adenoma/adenocarcinoma the next most frequent. However, many tumour types can occur – haemangiosarcoma, medulloepithelioma, primary lymphoma and so on, and although the clinical examination can suggest the tumour type, it is only on histological examination or cytology that this can be confirmed.

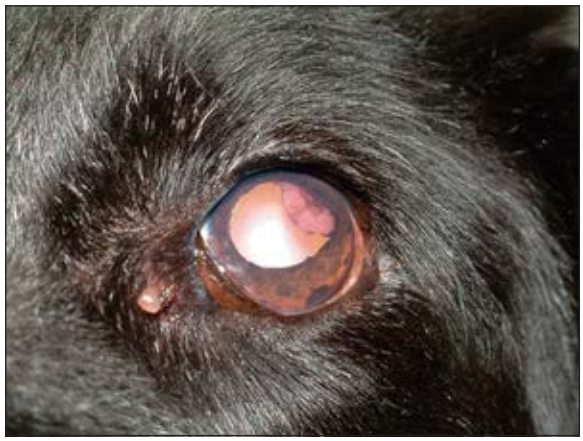


Figure 37.1 Ciliary body mass in an elderly Labrador cross. The non-pigmented mass can be seen at 1–3 o'clock behind the pupil. Apart from some dyscoria due to the mass, plus age-related iris atrophy and nuclear sclerosis, the eye is otherwise unremarkable.



Figure 37.3 Iris cyst in a Labrador. The pupil has been dilated. The free floating cyst can be seen to be hollow and is not attached to the iris.

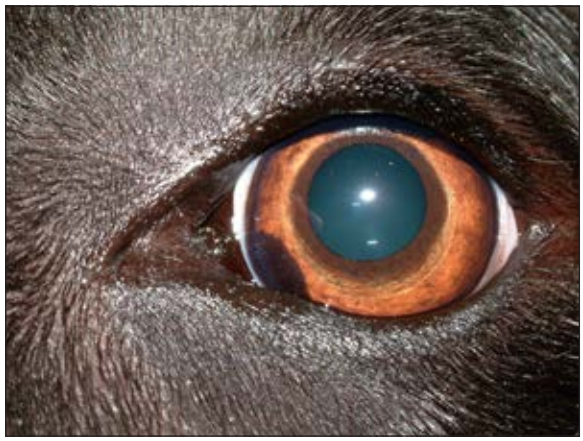


Figure 37.2 Uveal melanoma in a Labrador. The pigment is close to the iris base and slightly raised above the plane of the iris.

DIFFERENTIAL DIAGNOSES

- Non-neoplastic proliferations (e.g. iris freckles and pigment dispersal syndrome)
- Iris darkening following uveitis (diffuse hyperpigmentation)
- Intraocular extension from extraocular mass (e.g. limbal melanoma; see Case example 56.1)
- Perforating injury with iris prolapse
- Uveal cyst (Figure 37.3)

CASE WORK-UP

If an ocular mass is suspected a thorough clinical examination is required to try to establish whether this is primary or secondary. Thus if there are no changes in the fellow eye, nor on general examination, it is appropriate to assume the lesion is primary – especially if it presents as a mass rather than a diffuse uveitis.

Gonioscopy can be useful to determine whether extension into the drainage angle has occurred (which could indicate an increased risk of metastasis as tumour cells escape from the eye into the general circulation). Ocular ultrasonography can be considered but this might not provide further information about the lesion (although it can differentiate a solid mass from a cyst). Chest and abdominal radiography and/or ultrasonography should be undertaken if there is any suspicion of metastatic spread – for example, in advanced cases where there is not only a visible mass but also associated uveitis and secondary glaucoma.

Fine needle aspirates of draining lymph nodes can also be performed should metastatic disease be suspected. However, since many cases of ocular neoplasia in dogs are primary, and metastasis is uncommon, extensive work-up is not usually required unless there is an unusual aspect to the presentation.

NURSING ASPECTS

There is little specific nursing required in cases of uveal neoplasia in dogs. Some cases might require topical medication, or since enucleation is frequently performed, nursing care during and following surgery will be necessary but should be routine.

EPIDEMIOLOGY

Primary ocular neoplasia is not particularly common in dogs, but will be encountered in general practice from time to time, especially in older dogs. Uveal melanomas are the most common neoplasm encountered and normally behave in a benign manner – metastasis is via the bloodstream and occurs in less than 4% of cases. This is different from the situation in cats where malignancy with metastasis is more common. As with most tumours, histological examination can assist prognosis regarding the likelihood of metastasis.

Local extension – through the sclera, into the choroid or even down the optic nerve – is more likely in cases which have not been presented early. It should be remembered that uveal melanomas can occasionally be secondary – metastasis from oral or skin tumours does occur and so if other pigmented lesions are discovered on clinical examination it is probably more likely that the lesion in the eye is secondary to these. Uveal melanomas occur in dogs more frequently than in other domestic species, and darkly pigmented breeds such as the boxer and German shepherd dog are more frequently affected. Lines of Labrador retrievers can also suffer from the condition, suggesting an inherited component in this breed. In addition, affected Labradors are often younger – 3–6 years rather than the 8–10 years normally cited for other breeds. No sex predilection has been demonstrated

The second most common primary intraocular mass derives from the ciliary body – usually adenoma or adenocarcinoma, although medulloepitheliomas, originating from undifferentiated embryonal tissue, are occasionally encountered. Adenoma and adenocarcinoma behave clinically in a similar manner and rarely metastasize, even though the histological appearance may suggest different levels of malignancy. As with the melanoma, the German shepherd dog seems predisposed, and possibly the American cocker spaniel as well, but again, no sex predilection is reported. Older dogs are more frequently affected – 8 years and above most commonly. Most ciliary body neoplasms arise from the non-pigmented ciliary epithelium and are pinkish in colour, but they can develop from the pigmented epithelium and, as such, clinical examination cannot accurately differentiate them from melanoma.

Various other primary intraocular tumours have been reported – haemangioma and leiomyosarcoma for example – but these are extremely rare. Secondary neoplasia is sometimes seen in dogs, and is more often bilateral, presenting as a diffuse uveitis rather than a distinct mass. However, on occasions, a discrete mass will be secondary and emphasizes the importance of histological examination.

CLINICAL TIPS

- Transilluminate any mass to determine if solid (more likely neoplastic) or hollow (probably a uveal cyst)
- Examine the eye from the side to look through the pupil to the ciliary body
- Determine if limbal or iris mass by examining the drainage angle
- Dilate the pupil
- Check the fellow eye
- Check history for any previous neoplasia – e.g. mammary carcinoma

TREATMENT OPTIONS – MEDICAL

Since most primary intraocular tumours are clinically benign (even if histology shows signs of malignancy), and it is normally older patients that are affected, in some cases a 'watch and see' approach is appropriate. If the lesion is causing no discomfort and the eye is sighted, then it can be logical to examine the patient on a regular basis – every 1–2 months for example – and consider enucleation only if the mass starts to enlarge or cause uveitis. In very elderly patients, where the risks of anaesthesia are significant, especially if the dog has other medical problems such as chronic cardiac disease, topical steroids might help if localized inflammation occurs. However, should a secondary glaucoma develop, then enucleation would be recommended. Delaying removal of malignant masses could increase the risk of metastasis.

TREATMENT OPTIONS – SURGICAL

Enucleation remains the most appropriate treatment for many intraocular neoplasms and if a moderate uveitis or secondary glaucoma has developed it is definitely advised. All globes should be sent for histopathological examination. Sometimes the globe can be salvaged and so if the owner is not willing for enucleation, or if the mass is small and localized, referral should be considered. Small uveal melanomas (such as shown in Figure 37.2) are amenable to laser destruction and the prognosis for such patients is good. Occasionally ciliary body masses can be removed surgically, saving a visual eye.

PROGNOSIS

Generally the prognosis for primary ocular neoplasia in the dog is good, and enucleation is usually curative. However, histological examination of all removed eyes is recommended since the occasional mass is not what it appeared and thus there might be potential repercussions to which the owner should be alerted. Owners are often concerned that the tumour could spread from one eye to the other – this is extremely unlikely if the mass is primary, but can occur in metastatic disease – normally with spread to other tissues as well.

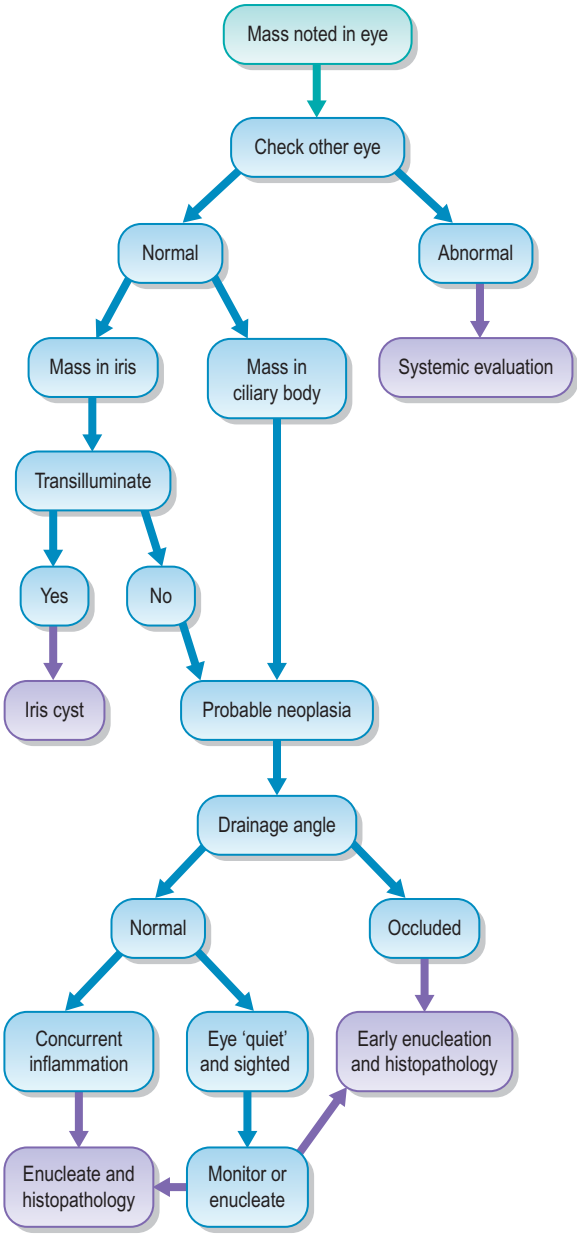


Figure 37.4 Decision-making tree – uveal tumour in dog.

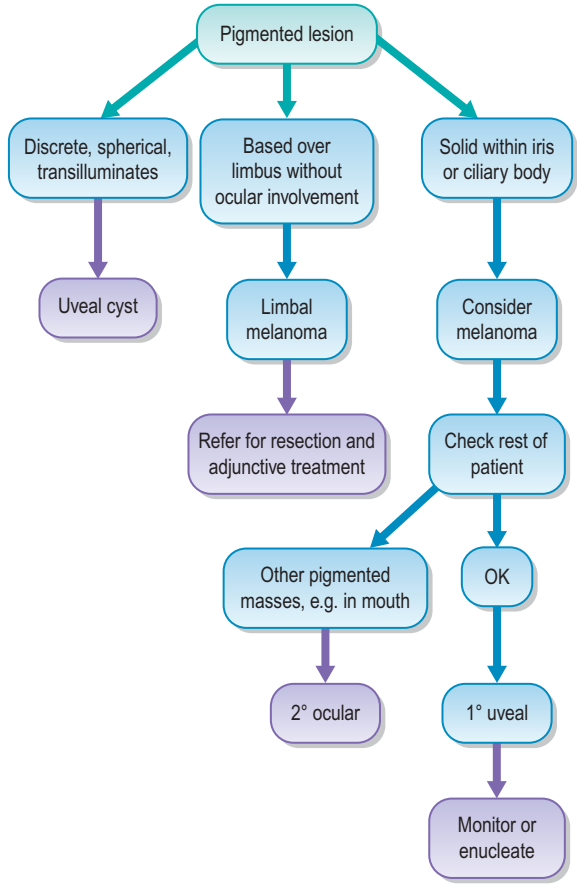


Figure 37.5 Decision-making tree – pigmented lesion.

CASE EXAMPLE 37.1 (SEE ALSO CHAPTER 45)

Patient details: Springer spaniel/collie cross, 15 years old, male (entire)

PRESENTATION AND HISTORY

The owner had been aware of problems with the dog's left eye for many months – it had been cloudy and 'odd-looking' but had not caused any discomfort. However, recently the eye had started to bulge and the dog could not see from it. The dog was very elderly and the owner did not want him 'messed about with'.

CLINICAL SIGNS

On general clinical examination the dog was underweight, weak on the hind legs and had severe dental disease. He was blind in the left eye with a fixed dilated pupil and no menace response. No consensual pupillary light reflex to the right eye was present. The left globe was enlarged and distorted laterally where a large pigmented mass was visible in the sclera; there was also some corneal pigment dorsolaterally (Figure 37.6). This was seen to be an extension from a large ciliary body mass. The iris was thickened and dark. The lens was cataractous and totally luxated into the anterior chamber with some pigment attached to the lens capsule. No ulceration was noted. Intraocular pressures were 32 mmHg in the left eye and 14 mmHg in the right. Apart from dense nuclear sclerosis the right eye was unremarkable. A ciliary body mass with scleral extension, chronic uveitis and secondary glaucoma was diagnosed.

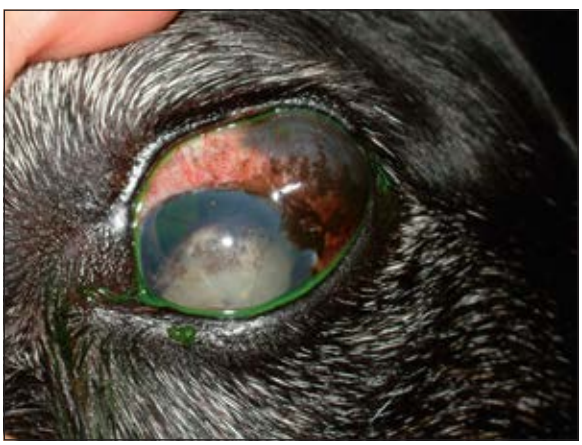


Figure 37.6 Large episcleral pigmented lesion which is an extension from a ciliary body mass (although from the angle of the photograph this cannot be seen). The globe is enlarged and distorted, chronic uveitis has been present, the lens is luxated into the anterior chamber and is a hypermature cataract and the pupil is widely dilated.

WORK-UP

The owner was advised that enucleation was the best option – the eye was irreparably blind from the glaucoma and although it did not seem particularly painful, it was thought that some discomfort must be present, and which would worsen as the mass continued to enlarge. However, the owner was totally against surgical intervention and did not want the dog's general deterioration investigated either.

TREATMENT

Medication was started with prednisolone acetate and dorzolamide drops, both three times daily.

OUTCOME

A week later the eye looked unchanged on examination, but intraocular pressure had reduced to 26 mmHg. The owner was happy that the dog was in no discomfort and wanted to continue. The drops were reduced to once daily prednisolone acetate but dorzolamide continued three times daily. The eye remained stable for the following 6 months until the dog went off his hind legs and was euthanized.

This patient was presented very late in the course of the disease and early enucleation could have prevented the secondary glaucoma. However, he was very elderly, and the owner's reluctance for surgery was understandable – this was one reason for the delay in presentation. It was possible to prevent suffering through ocular discomfort by controlling the glaucoma medically for the dog's last few months.

38 Leishmaniasis

INITIAL PRESENTATION

Colour change – red, blindness, change in appearance – swellings, discharge

PRESENTING SIGNS

Both dogs and occasionally cats can be infected with *Leishmania* and the clinical presentation can be extremely variable. Thus patients can be presented due to red eyes, blindness, ocular or periocular swellings, abnormal appearance to one or both eyes or just an ocular discharge. Systemic illness is frequently also present, most notably skin disease but also general malaise, weight loss, lameness or signs of hepatic or renal disease. Indeed it is common for the owner to present the patient due to the systemic signs with the ocular symptoms only noted during the clinical examination. Any age of dog can be affected although young to middle-aged adults are perhaps more commonly encountered with the disease.

CASE HISTORY

The case history again is very variable. However, one reliable factor is that the animal will have travelled through an endemic area. Areas where *Leishmania infantum* and the vector *Phlebotomus* spp. (sand flies) are found include the Mediterranean shore, parts of east Africa and India, while *Leishmania chagasi* is located in parts of Central and South America. Thus the travel history of the patient should alert one to the possibility of this disease. The patient might be acutely presented but more frequently there is a history of illness for up to several weeks. The skin lesions, if present, tend to be progressive, scaly and non-pruritic.

CLINICAL EXAMINATION

General clinical examination might be normal, but this is the exception. Careful attention to the skin should be made, as areas of alopecia, frequently with seborrhoea and desquamation, might be present. These might be just in the periorbital area or on the head, but can be more widespread. The patient might be thin, have renal or liver pain on abdominal palpation, or might show lameness. Thus there can be a variety of systemic abnormalities present.

Ophthalmic examination can be similarly variable. In some cases the eyes will be normal on initial examination (but ocular changes can develop later in the course of the disease, even after treatment has been instigated). Some patients will have thickened, inflamed eyelids – in either a diffuse or a nodular pattern (Figure 38.1). Some will have marked chemosis and conjunctivitis, with or without corneal involvement. Nodular swellings at the limbus might be noted. Corneal ulceration and vascularization are occasionally present. Anterior uveitis is common, with miosis, aqueous flare and iridal swelling (Figure 38.2). Such patients will be in pain and can be blind. If the posterior segment is visible then signs of chorioretinitis might be present, but the anterior segment is more frequently the area of the uveal tract which is most inflamed. Occasionally orbital cellulitis will be present and the patient will have a painful exophthalmos. Schirmer tear test readings should be performed – some dogs will have keratoconjunctivitis sicca. Clearly fluorescein testing is mandatory. If tonometry is available, intraocular pressures should be assessed – secondary glaucoma can be a feature. Most cases are bilateral but not necessarily symmetrical.



Figure 38.1 Severe blepharitis in a Rottweiler. Some keratitis was also present in this dog. Image courtesy of Teresa Peña, College of Veterinary Medicine, Autonomous University of Barcelona, Spain.



Figure 38.2 Severe keratouveitis. Corneal vascularization, granulation tissue and some pigment are present along with corneal oedema and aqueous flare. Detailed intraocular examination is not possible. Image courtesy of Teresa Peña, College of Veterinary Medicine, Autonomous University of Barcelona, Spain.

DIFFERENTIAL DIAGNOSES

Since the clinical presentation is so variable, the differential list is exhaustive! Basically any cause for blepharitis, keratoconjunctivitis and uveitis should be considered. Systemic infections (including fungal infections) and neoplasia (e.g. lymphoma) should be on the list of differential diagnoses, along with immune-mediated conditions (e.g. uveodermatological syndrome where periocular, oral and nasal depigmentation is present along with uveitis). Due to the diversity of clinical presentation the condition can be difficult to diagnose in some patients, but the travel history clearly provides a clue.

CASE WORK-UP

An initial work-up includes full haematology and biochemistry, together with urinalysis. Depending on whether the leishmaniasis is purely cutaneous, ocular or visceral (or frequently a combination of these), the initial blood results can be normal or markedly abnormal, with renal and hepatic changes, hyperglobulinaemia and anaemia commonly seen. If *Leishmania* is suspected on initial presentation, then serology using ELISA tests can be considered (but in endemic areas many dogs are seropositive, suggesting subclinical infection and so serology results must be interpreted carefully). Biopsy is necessary to confirm the disease in most cases. Skin lesions, limbal nodules or conjunctiva can all be sampled and the organism can be identified on cytology or histopathology. Lymph node or bone marrow aspirates can also be examined. A third diagnostic test is polymerase chain reaction (PCR) which is performed on skin biopsies or bone marrow smears.

Although leishmaniasis is less common in cats than in dogs, it should be considered in this species and some cats can be immunosuppressed. Thus feline leukaemia virus (FeLV) and feline immunodeficiency virus (FIV) testing is necessary and other coexisting infections such as toxoplasmosis can complicate the issue in this species.

NURSING ASPECTS

Animals with leishmaniasis are frequently systemically ill and require careful general medical nursing as well as specific ocular treatment. They are often on complicated medical regimens and nurses should follow these and ensure that owners understand and are capable of following the treatment instructions at home.

EPIDEMIOLOGY

In the Mediterranean, the subspecies responsible for leishmaniasis is *Leishmania infantum* and infections are

common in this region. It is transmitted by sand flies (*Phlebotomus* spp.) and after the vector has bitten the animal the amastigotes of *Leishmania* infect the dermal macrophages. If the host does not elicit a protective immune response to this, the organism then multiplies and disseminates throughout the body, causing disease – the visceral form will have multi-organ involvement as a result of infection of the reticuloendothelial system. The organism depresses T cell immunity while increasing B cell activity. The incubation period varies from months to years, and any age of dog can be affected, although it is possibly more common in young adults. Ocular manifestations are thought to be due to a combination of direct organism infiltration (e.g. in periocular and conjunctival nodules) and as a result of the immune reaction to the organism. Widespread immune complex formation, hyperglobulinaemia and antinuclear antibody formation are typically encountered in leishmaniasis, and since the uveal tract is particularly sensitive to immune-mediated disease, it is easy to see how anterior uveitis is a common finding in these patients.

Subclinical infections are common in endemic areas, and the ability of some animals to tolerate this, while others develop severe disease, is not fully understood.

The majority of patients with leishmaniasis present due to systemic or cutaneous disease. Of these, approximately 25% have ocular signs as well, although the reported incidence of ocular involvement varies from 16% to 80%. Approximately 15% of cases are presented with ophthalmic disease only – and no evidence of systemic involvement at all.

CLINICAL TIPS

- Always ask about travel history
- Consider biopsy early if the presentation is unusual or progressive
- There is no pathognomonic sign for leishmaniasis

TREATMENT OPTIONS – MEDICAL

The treatment for leishmaniasis is medical, expensive and long term. A combination of systemic and topical treatments is normally required. Systemic agents which target the organism include subcutaneous injections of *N*-methylglucamine antimoniate (80 mg/kg daily for 30 days), together with oral allopurinol (10 mg/kg twice daily for 6–12 months or even longer).

Treatment for the ocular signs is symptomatic and a combination of topical steroid drops (dexamethasone or prednisolone acetate) and atropine is used in cases of uveitis. Specific therapy for keratoconjunctivitis sicca might be required, or antibiotics if any corneal ulceration is present. Anti-glaucoma medications might also be required. If there is ulcerative blepharitis, then systemic antibiotics such as cefalexin or potentiated amoxicillin might be necessary. Occasionally systemic prednisolone will be used to help treat the uveitis if it is severe or poorly responsive to the topical treatment.

PROGNOSIS

The prognosis for leishmaniasis must be guarded. Many cases do respond well to initial treatment, but relapses are frequent. Sometimes the clinical picture will change

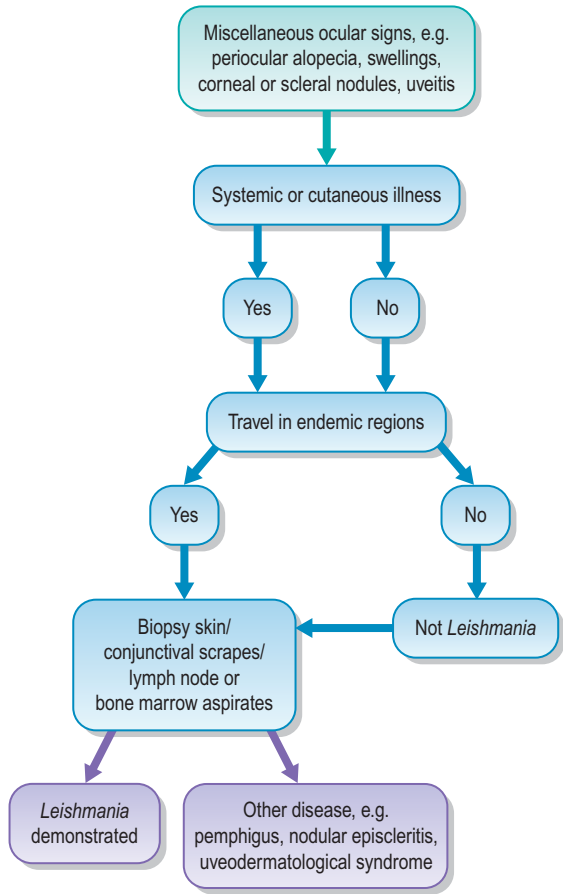


Figure 38.3 Decision-making tree – leishmaniasis.

with the relapse – for example, patients might develop ocular symptoms when they had none previously, or the visceral form might develop cutaneous lesions. Sometimes these different presentations will not manifest for months to years after the diagnosis and instigation of treatment. The ocular signs can lead to blindness, secondary glaucoma and even enucleation (where histopathology might demonstrate the amastigotes in the uveal tract). The prognosis for the ocular signs is generally better if they are adnexal (blepharitis, limbal nodules) rather than if uveitis is present. The recurrence of ocular signs is estimated to be approximately 30% while systemic signs can recur in 75% of cases in 2 years. Euthanasia of debilitated patients is necessary.

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SECTION 6

LENS

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39 Lens – introduction

The lens can be involved in primary disease (e.g. cataract or lens luxation) or can exhibit pathological change secondary to other ocular disease such as uveitis and glaucoma. An understanding of its anatomy and physiology is necessary to appreciate its role in ocular disease.

The lens is a biconvex, transparent and refractive structure suspended by zonular fibres, situated behind the iris, dividing the anterior and posterior segments of the eye. The lens consists of a capsule, lens epithelium and the lens fibres. The lens is subdivided into a central nucleus and surrounding cortex, and these can be further subdivided clinically into anterior and posterior portions. The equator is the peripheral area of the lens and these different anatomical regions are useful when describing the position of lesions within the lens (Figure 39.1). It is held in position by lens zonules which originate on and between the ciliary processes of the ciliary body and insert onto the lens capsule, both anterior and posterior to the lens equator around the full 360 degrees of its circumference (see Figure 32.1 as well as Figure 39.1). Muscular action within the ciliary body alters the tension on the zonules, thus altering the lens shape and therefore its optical power. This process is known as dynamic accommodation and only occurs to a limited degree in dogs and cats.

The anterior capsule of the lens is in contact with the posterior iris, which glides over it as the pupil size alters. The posterior capsule has attachments to the vitreous (as the hyaloideocapsular ligament). The adult lens consists of primary and secondary lens fibres. The primary ones differentiate from the posterior lens epithelium at an early stage of eye development and this differentiation is induced by the embryological retina. These primary fibres elongate and fill the lens vesicle to form a solid sphere. In the adult these primary fibres form the central 'nucleus' of the lens. Abnormalities of this early differentiation will manifest as nuclear cataracts. The posterior lens epithelium disappears as the lens matures, leaving just a very thin posterior lens capsule. The anterior epithelial cells are located beneath the anterior lens capsule and actively divide throughout life. As they multiply, they move towards the lens periphery and when they reach the equatorial region they start to elongate to form the secondary lens fibres, one arm of which stretches into the anterior cortex and the other into the posterior cortex (Figure 39.2). New secondary fibres are constantly produced throughout life, resulting in progressive compression of the lens nucleus. The anterior lens capsule is produced by the anterior epithelium throughout life and gets thicker as the animal ages. The secondary lens fibres are partially responsible for the normal ageing change of senile nuclear sclerosis. Sclerosis of the lens occurs in the older animal and is not as obvious in the cat as in the dog. It should be differentiated from a true cataract.

The regular arrangement of secondary lens fibres confers transparency to the lens as does their long narrow shape and relative lack of cellular organelles. On careful examination it is possible to visualize the site at which the tips of the lens fibres meet. These areas are known as the lens suture lines. Those within the posterior cortex form an inverted 'Y' (Mercedes sign) and are often more visible than the anterior ones, which are in the shape of an upright 'Y'. These are a normal finding and not cataract.

The adult lens is made up of 65% water and 35% protein – the latter divided into soluble crystallins (90%) and insoluble crystalloids. It has a very high concentration of glutathione.

While the lens is developing it derives nutrition from the hyaloid artery (which comes from the optic nerve to the back of the lens) and the tunica vasculosa lentis – a network of blood vessels which are found both anterior and posterior to the growing lens. These should regress fully but remnants can remain into adulthood. Persistent hyaloid vessels are seen as incidental findings occasion-

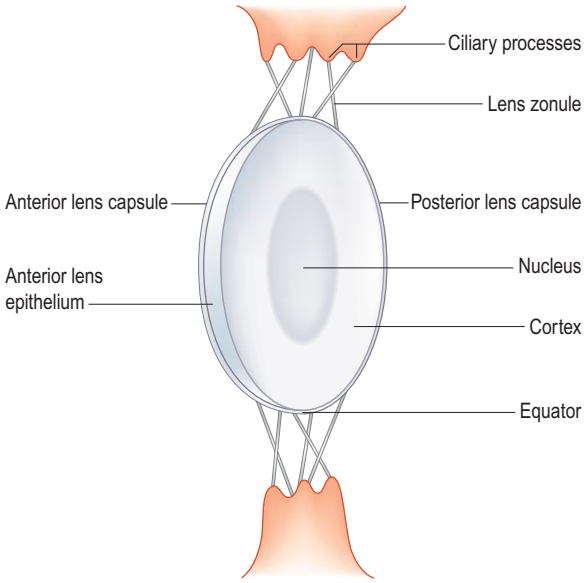


Figure 39.1 The anatomy of the lens.

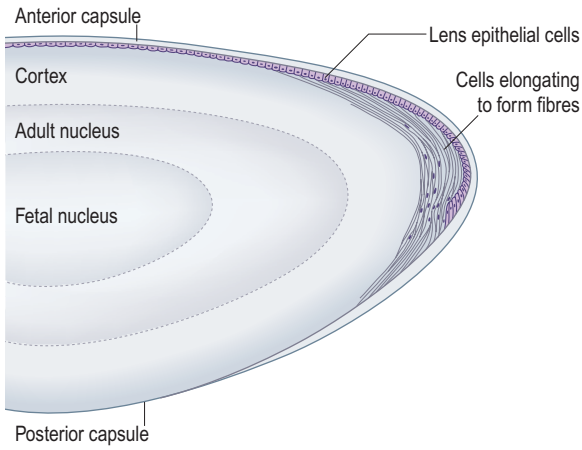


Figure 39.2 Lens anatomy showing the formation of lens fibres.

ally – as pale tags of tissue on the back of the lens, or even complete to the disc containing blood. More severe remnants – the persistent hyperplastic tunica vasculosa lentis – are inherited in Dobermann and Staffordshire bull terrier breeds but can occur sporadically in any breed. These have a wide range of clinical appearance, from a small pigmented patch on the posterior capsule to extensive vascular networks with cataract and abnormal lens shape.

Normally the adult lens is devoid of vascular supply. Its nutrition and removal of waste products are provided by the aqueous. Metabolism is mainly by anaerobic glycolysis. Any abnormalities in the aqueous, such as associated with uveitis, will have a deleterious effect on lens metabolism and can initiate cataract formation.

There are few disorders of the lens. Congenital abnormalities can occur affecting just the lens (e.g. cataract or coloboma) or in conjunction with multiple ocular abnormalities, including microphakia, persistent pupillary membrane remnants, lenticonus (abnormal lens shape) and retinal dysplasia. Some breeds of dog, such as the cocker spaniel and miniature schnauzer, suffer from such multiple ocular abnormalities. Adult lenses can develop cataract, an opacity of the lens and/or capsule, and there are several causes of cataract formation – inherited forms are common in dogs while cats frequently develop opacification of the lens secondary to chronic uveitis. Lastly the lens can dislocate – fully as in a total luxation, or partially (subluxation) – and this can be primary or secondary to other ocular disease; in each case the lens zonules weaken and break, causing the lens to wobble and move from its normal position. We will consider these lens abnormalities in the following cases. Since all lens problems will require referral for expert evaluation and surgery if indicated, only general case discussions are included in this section, rather than specific examples.

40 Diabetic cataract in a dog

INITIAL PRESENTATION

Abnormal appearance – cloudy, blind

PRESENTING SIGNS

Dogs with cataracts due to diabetes mellitus can present in two different ways. Firstly, they can present with sudden onset blindness, where the owner was unaware that the dog was suffering from any clinical disease, but on examination was found to have the typical signs of diabetes mellitus. In these cases the patient can be quite ill, and clearly the blindness is not life threatening and so must take second priority. The other way patients can present is with, again, a rapid deterioration of vision in a known diabetic patient. These can be both poorly and well-stabilized patients, although more likely in the former. With both presentations the owners will have noticed that the dog suddenly became disoriented and started bumping into things, and at the same time they noticed that the eyes looked cloudy (or 'with a film over them').

CASE HISTORY

If the patient has presented due to the visual loss only, in addition to the ophthalmic problems it will have the typical history associated with diabetes mellitus – polydipsia and polyuria, weight loss despite polyphagia, and possibly even dehydration, weakness and collapse and so on if in a state of diabetic ketoacidosis. Known diabetics might have recently had a period of instability or another illness – urinary tract infection most commonly – and a few days to weeks afterwards the owner noticed the clouding and vision loss. Sometimes there will be a more gradual loss of vision, particularly in very well stabilized patients, and the progress of the cataract might have been noted for several weeks. However, it is far more common for the dog to progress from normal vision to total blindness within a very short space of time – just days.

CLINICAL EXAMINATION

General clinical examination will reveal the typical signs of diabetes in non-diagnosed patients, while those currently on treatment might have symptoms attributable to other disease processes which have affected their insulin metabolism. Thus they should be carefully examined for evidence of urinary tract infection, dental disease and signs of hyperadrenocorticism or hypothyroidism for example.

Ophthalmic examination will reveal reduced or absent menace responses and poor vision. Pupillary light reflexes are often normal, but occasionally are slightly sluggish. Some conjunctival hyperaemia or episcleral congestion can be present. The iris might be uniformly dark. However, the most striking abnormality is the presence of bilaterally symmetrical total mature cataracts. Frequently the lenses appear slightly 'swollen' (intumescent) and water clefts along the suture lines are apparent (Figure 40.1). Fundus examination is rarely possible. If intraocular pressure can be measured it is frequently slightly below normal (e.g. 8–12 mmHg). Lens-induced uveitis is very common in sudden onset cataracts, particularly those induced by diabetes mellitus. Schirmer tear test readings should be recorded and the cornea stained with fluorescein to check for ulceration.

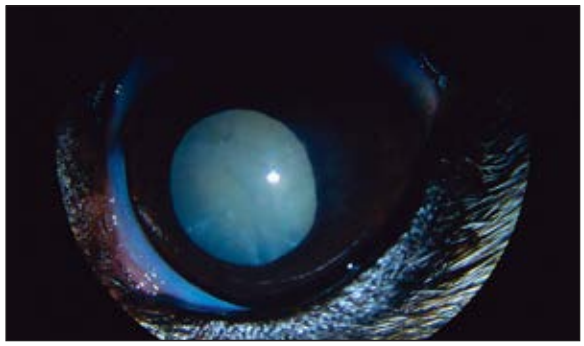


Figure 40.1 Typical sudden onset cataract in a diabetic dog. Note the total opacity with 'water clefts' along the anterior suture lines and swollen appearance to the lens. The iris is very dark, suggesting some lens-induced uveitis.

DIFFERENTIAL DIAGNOSES

- Other causes for cataract, e.g. inherited, age-related, post-uveitis, secondary to progressive retinal degeneration
- Other causes for opacity – corneal endothelial dystrophy, glaucoma
- Other causes for blindness – retinal detachment, central lesion

CASE WORK-UP

Obviously the patient needs to be worked up for the diabetes and this should be stabilized as much as possible. Once the diagnosis is made via urine and blood glucose measurements, and the patient started on insulin therapy, discussions about referral for cataract surgery can be considered. Most ophthalmologists will only consider surgery when the patient is stable, with blood glucose curves and fructosamine assays used in addition to clinical parameters to assess this. Clearly full laboratory evaluation should be undertaken to ensure that they are free from other metabolic disease (such as hyperadrenocorticism) and severe dental disease is a contraindication for elective cataract surgery – this should be treated prior to elective phacoemulsification. However, some patients are difficult to stabilize, and since the surgical success is higher before complications such as severe lens-induced uveitis (with lens capsule rupture) have occurred, early surgical intervention is occasionally advocated. It is sensible to discuss the patient with your ophthalmologist as soon as the cataracts are diagnosed so that liaison can take place for the best outcome for the patient.

NURSING ASPECTS

The details of nursing the ill diabetic patient are beyond the scope of this text. However, ocular medication will be required with cataract surgery and owners should be taught how to do this.

EPIDEMIOLOGY

Cataract formation is extremely common in patients with diabetes mellitus. Reports range from 50% developing them within a year of the onset of diabetes mellitus to 80% of dogs eventually forming cataracts. Thus client education is important and, along with the initial instructions regarding management of the diabetic patient, the likelihood of their pet developing cataracts as a complication of the diabetes should be discussed. In this way owners will not be shocked and dismayed when their dog does go blind.

Glucose is the principal energy source for the lens. During normal euglycaemia only 5% of glucose is metabolized via the sorbitol pathway, the vast majority using the hexokinase pathway to convert the glucose to lactic acid. During hyperglycaemia this pathway is overwhelmed and the extra glucose is shunted into the sorbitol path, metabolized by aldose reductase. This promotes a sudden osmotic gradient as sorbitol accumulates and water is drawn into the lens from the aqueous humour. This rapidly alters the lens fibre architecture (swelling and rupture) and cataract results. Some oxidative damage is also thought to occur.

The sudden change in the lens triggers a lens-induced uveitis. Sometimes when the lens rapidly becomes cataractous, the imbibition of water causes such lens swelling (intumescence) that the lens capsule ruptures – typically at the equator. Such patients are frequently poorly controlled with respect to their diabetes and the uveitis which results from the lens damage can be severe and difficult to manage.

Diabetes mellitus also has other effects on the eye. Tear production can be reduced, as can corneal sensitivity, and both can lead to recurrent or refractory ulceration.

CLINICAL TIPS

- Alert owners of diabetic dogs that cataract formation is likely to occur
- Check that the diabetes is stable before considering surgery
- Remember to treat the lens-induced uveitis promptly
- Close liaison with the veterinary ophthalmologist is essential if surgery is considered
- Always examine the eyes on each check for the diabetes – consider referral immediately any evidence of cataract is detected
- Cats are resistant to the development of diabetic-induced cataracts

TREATMENT OPTIONS – MEDICAL

The only successful treatment for cataract is surgical removal of the lens. However, most patients with sudden onset cataracts due to diabetes mellitus will have some lens-induced uveitis and early recognition of this, and subsequent treatment, will offer the best prognosis if surgery is undertaken (Figure 40.2). Topical steroids are the main choice for lens-induced uveitis, and although some systemic absorption will occur, in theory potentially causing some insulin resistance, in a clinical situation this is very rarely of any consequence. Patients should be maintained on their normal insulin regimen.

If surgery is indicated, the patient will almost certainly be medicated with topical steroids postoperatively. Prednisolone acetate is commonly used, two to three times daily into both eyes. Careful monitoring for evidence of ulceration is required – diabetic patients are more prone to corneal ulceration than non-diabetics and topical steroids do not help! An alternative to their use is NSAID drops. Ketorolac and flurbiprofen can be employed in mild to moderate cases of uveitis, again at a dose of one drop two to three times daily. These have no risk of affecting the insulin requirements. Some patients will require both, and even topical atropine to effect mydriasis and stabilize the blood–aqueous barrier, as well as reducing synechia formation.

Keeping the patient well controlled with respect to the diabetes will help reduce the severity of uveitis both pre- and post-surgery. The blood–aqueous barrier is unstable both in diabetics and following intraocular surgery, and recurrent episodes of hyperglycaemia seem to result in a more severe uveitis and thus lower success rate.

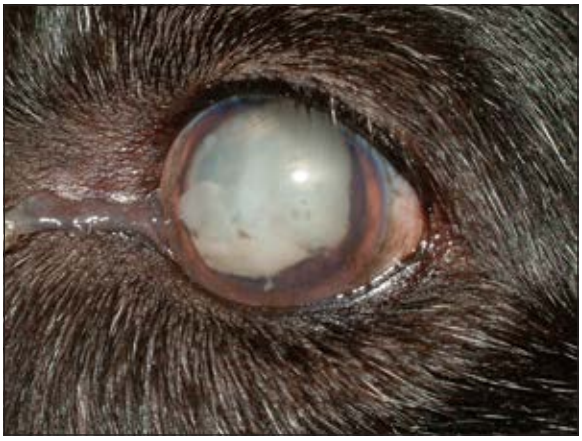


Figure 40.2 Severe lens-induced uveitis in a poorly controlled diabetic crossbred with concurrent hyperlipidaemia. Although this patient has been treated with topical steroids and atropine, some uveitis remains along with posterior synechiae and leakage of lens material into the anterior chamber probably following a rupture in the lens capsule. The patient is not suitable for cataract surgery and both eyes rapidly developed phthisis bulbi.

TREATMENT OPTIONS – SURGICAL

Phacoemulsification is the surgery of choice for diabetic-induced cataracts, with or without intraocular lens implantation (see Figure 40.3), which depends on surgeon preference, the degree of uveitis and subse-

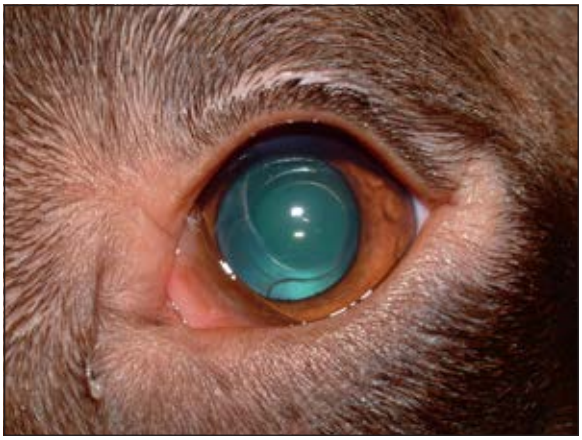


Figure 40.3 Labrador 2 weeks following phacoemulsification for diabetic cataract and intraocular lens implantation. The eye is quiet with good vision.

quent scarring preoperatively, finances and various other factors. In general, the success rate is good for diabetic patients – 85–90% do very well. The surgery is technically easier in younger patients – older ones will have lens sclerosis (hardening) which makes the surgery more time consuming and can increase the complication rate. Rapidly progressed intumescent lenses with uveitis and lens capsule rupture can also present difficulties during surgery. The main potential complications with cataract surgery in diabetics include ongoing uveitis, corneal ulceration, keratoconjunctivitis sicca and sometimes secondary glaucoma or retinal detachment.

It is sensible to consider referral sooner rather than later if owners are interested in cataract surgery, and liaison with the ophthalmic specialist regarding any particular medication or further work-up which they might require is useful (see Table 40.1).

Once referred, the ophthalmologist might perform further tests such as ocular ultrasonography (checking both for lens capsule rupture and retinal detachment) and electroretinography (to assess photoreceptor function) prior to considering surgery. Any owner wishing to have cataract surgery on their dog must be committed – the after-care is very time consuming and frequent re-examinations with the ophthalmologist are required.

PROGNOSIS

Generally, the prognosis for restoring reasonable vision in patients with diabetic cataracts is good. However, there are several potential complications. Uveitis is inevitable, and usually can be well controlled, although some dogs require life-long topical medication. In most patients the uveitis does not have a major deleterious effect on vision but in some it can even result in blindness. Diabetics are more prone to corneal ulceration, slow healing, wound breakdown and infections following surgery compared to non-diabetics. The risks of glaucoma and retinal detachment are similar in diabetic and non-diabetic patients, and can affect up to 20% of dogs long term. Thus owners must be aware of the pros and cons of any surgical intervention and will have discussion with the ophthalmologist regarding this.

It should be remembered that not all dogs which develop diabetic cataracts are suitable for, or even need, surgery. Patients with other health issues might not be

Table 40.1 Checklist for consideration prior to referral for cataract surgery

- Stabilize the diabetes mellitus – clinical impression, glucose curves and fructosamine assay
- Consider lens-induced uveitis and treat as necessary (usually topical steroids)
- Check for concurrent systemic disease – hyperadrenocorticism, hypothyroidism, severe dental disease
- Ensure owner has time to commit to medication and postoperative checks with ophthalmologist
- Discuss costs with owners
- Liaise with referral ophthalmologist regarding any further requests

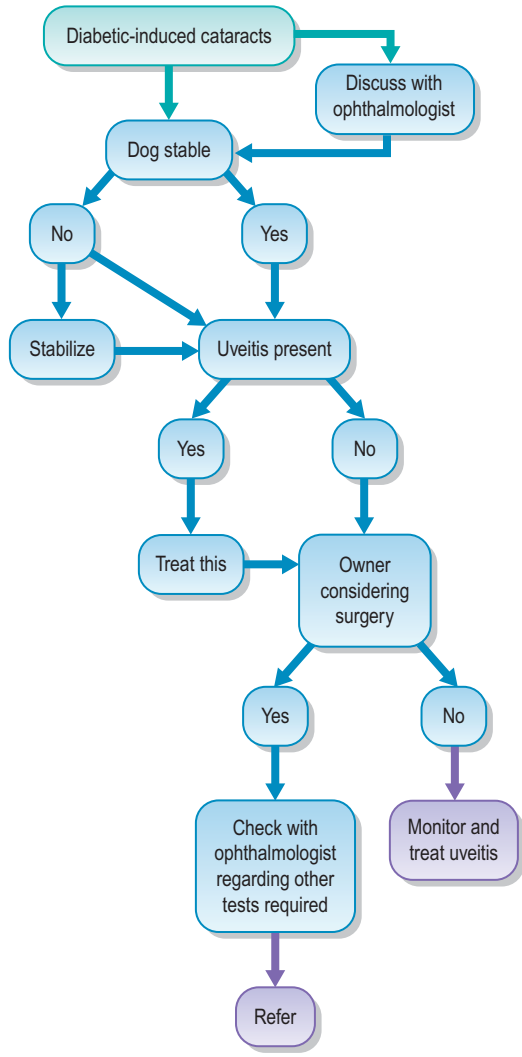


Figure 40.4 Decision-making tree – diabetic cataract.

appropriate anaesthetic risks, or might have too high a complication rate to warrant subjecting the dog to elective surgery. Many dogs that go blind adjust very well and still lead happy lives. Since the diabetic-induced cataracts progress so rapidly, it is very disorientating for the dog initially. However, after only a few weeks they have learned their way around and a good quality of life is resumed. Often the depressed behaviour first noted by the owner is as a result of the dog feeling unwell due to the hyper- or hypoglycaemia, and stabilizing this is what returns the dog to normal – despite still not seeing!

Even if cataract surgery is not performed, the eyes still need monitoring. If left untreated, the chronic, low grade lens-induced uveitis can result in secondary glaucoma which will be painful. The lenses will rapidly become hypermature, exacerbating the uveitis, and on occasion the lenses can luxate following inflammatory damage to the lens zonules. Therefore, even if surgery is not an option, the eyes should not be forgotten!

41 Cataract in a young dog

INITIAL PRESENTATION

Abnormal appearance – cloudy

PRESENTING SIGNS

A young adult pedigree dog could be presented because the owner has noticed that one eye looks cloudy. Occasionally both will be affected and the dog could have missed catching its ball, or walked off with the wrong owner when out in the park. Occasionally the owner will have noticed nothing at all and the ocular abnormalities will have been detected on clinical examination for some other problem or at routine vaccination.

CASE HISTORY

In most cases there is no previous history of ocular disease or relevant systemic disease. In pedigree dogs, especially those breeds listed in Table 41.1, the owner should be asked whether the sire and dam had their eyes tested prior to breeding. Unfortunately this is not performed as often as one would like and, as such, there is a fairly high incidence of inherited cataract in some breeds of pedigree dog.

CLINICAL EXAMINATION

General clinical examination is normally unremarkable. On ophthalmic examination menace responses can be normal or reduced. Pupillary light reflexes are usually normal (providing no retinal disease is present – the breeds which suffer from inherited cataracts can also suffer from progressive retinal atrophy). Unless the lenses have suddenly developed maturing cataracts there is unlikely to be any ocular inflammation, but evidence of episcleral congestion, iris darkening or thickening, and mild miosis can suggest lens-induced uveitis. Opacification of one or both lenses will be apparent on distant direct ophthalmoscopy and can be examined in more detail with the direct ophthalmoscope at a setting of +8 to +12D (Figure 41.1).

It is common for both eyes to be affected but to differing degrees – such that the owner (and sometimes general practitioner) is unaware that the condition is bilateral. For full evaluation of the lens and posterior segment, pupil dilation with tropicamide is suggested. It is helpful to locate the opacity anatomically within the lens (e.g. anterior cortical, posterior subcapsular). Fundus examination is frequently normal.

DIFFERENTIAL DIAGNOSES

- Other causes of cataract (see Table 41.2)
- Corneal endothelial dystrophy
- Corneal scarring
- Persistent hyperplastic primary vitreous
- Retinal detachment
- Hypopyon
- Lipid-laden aqueous

CASE WORK-UP

Minimal work-up is usually necessary. Blood and urine glucose should be checked to rule out diabetes mellitus. General health profiles should be considered and referral for specialist evaluation and prognosis regarding progression and whether cataract surgery is warranted should be discussed with the owners. If a suspected inherited cataract is diagnosed, the owner should be asked to alert the breeder and the health officer of the local breed club.

Table 41.1 Inherited cataracts in dogs (UK list)

Type of cataract/ inheritance	Breed
Congenital	Miniature schnauzer
Early onset with progression	Boston terrier Cavalier King Charles spaniel German shepherd dog Leonberger Miniature schnauzer Old English sheepdog Staffordshire bull terrier Standard poodle Welsh springer spaniel
Posterior polar (upside down 'Y' or triangle) Sometimes progressive	Alaskan malamute Chesapeake Bay retriever Golden retriever Labrador retriever Large Munsterlander Siberian Husky Belgian shepherd dog Norwegian Buhund
Other forms	American cocker spaniel Boston terrier (different from early onset cataract)
Suspected to be inherited	Many breeds including Bichon Frise, Border collie, Border terrier, French Bulldog, giant schnauzer, Tibetan terrier, Yorkshire terrier

NURSING ASPECTS

Initial nursing is minimal. If there is any lens-induced uveitis, this will be treated topically and nurses should ensure that owners are competent to do this.

EPIDEMIOLOGY

Inherited cataracts are quite common in pedigree dogs and juvenile animals are most frequently affected. If the



Figure 41.1 Immature cataract in an 11-month-old Staffordshire bull terrier. The condition is bilateral but not quite symmetrical.

Table 41.2 Different methods of classification of cataracts

Method of classification			
Age of onset	**Cause**	**Position**	**Stage of development**
Congenital	Inherited	Nuclear	Incipient (very early changes with no effect on vision)
Juvenile	Secondary to other ocular disease (uveitis, generalized progressive retinal atrophy, glaucoma)	Cortical (anterior or posterior)	Immature (tapetal reflex still visible)
Senile	Trauma (blunt or penetrating)	Capsular (anterior or posterior)	Mature (entire lens opaque, eye blind)
	Metabolic (diabetes mellitus, hypocalcaemia)	Equatorial (positioned at the periphery of the lens)	Hypermature (altered density of lens and leakage from lens causing lens-induced uveitis)
	Toxic or drug induced Radiation (following treatment for tumours on the head)		

inheritance has been established it is usually autosomal recessive, although some breeds have an incomplete dominant mode of transmission. Each affected breed tends to have a typical age of onset, appearance of the cataract and rate of progression. Posterior polar subcapsular cataracts are found in Labrador and golden retrievers, large Munsterlanders and Irish red and white setters among others. They can be seen at any age from a few months to 8 years, but two peaks of incidence are seen in the retriever breeds – between 6 and 18 months and again at about 6–7 years of age.

The cataracts are seen as a triangle or 'Mercedes sign' at the posterior pole and are usually bilateral. In younger dogs the risk of progression to cause visual loss is higher than in older dogs. Approximately 5% are suggested to progress and this can be quite rapid over a few weeks. Staffordshire bull terriers get rapidly progressing nuclear and cortical cataracts at 6–18 months of age which render them blind if not surgically removed. A DNA test is available in this breed and the Boston terrier, and further tests are likely to be developed as the genes causing the disease are identified in the various breeds. For the majority of breeds the diagnosis of inherited cataract in an individual dog is made circumstantially – the age of dog, breed and location of cataract, together with absence of any other predisposing cause, make inheritance the most likely aetiology.

For breeds in which genetic testing is not yet available, control rests with eye testing of potential breeding stock, and up-to-date information regarding affected breeds and where to arrange eye testing is available from the British Veterinary Association under the BVA/KC/ISDS scheme. Similar schemes are available in Europe, the USA and Australia.

CLINICAL TIPS

- Always check both eyes carefully – the cataracts are not always symmetrical
- Consider inheritance in any young pedigree dog unless evidence of another aetiology is convincing
- Advise against breeding from affected dogs and their siblings/sire/dam
- Consider early referral – do not wait until the dog is completely blind and the cataracts 'ripe' – this is very old fashioned and does not provide the best care for the patient

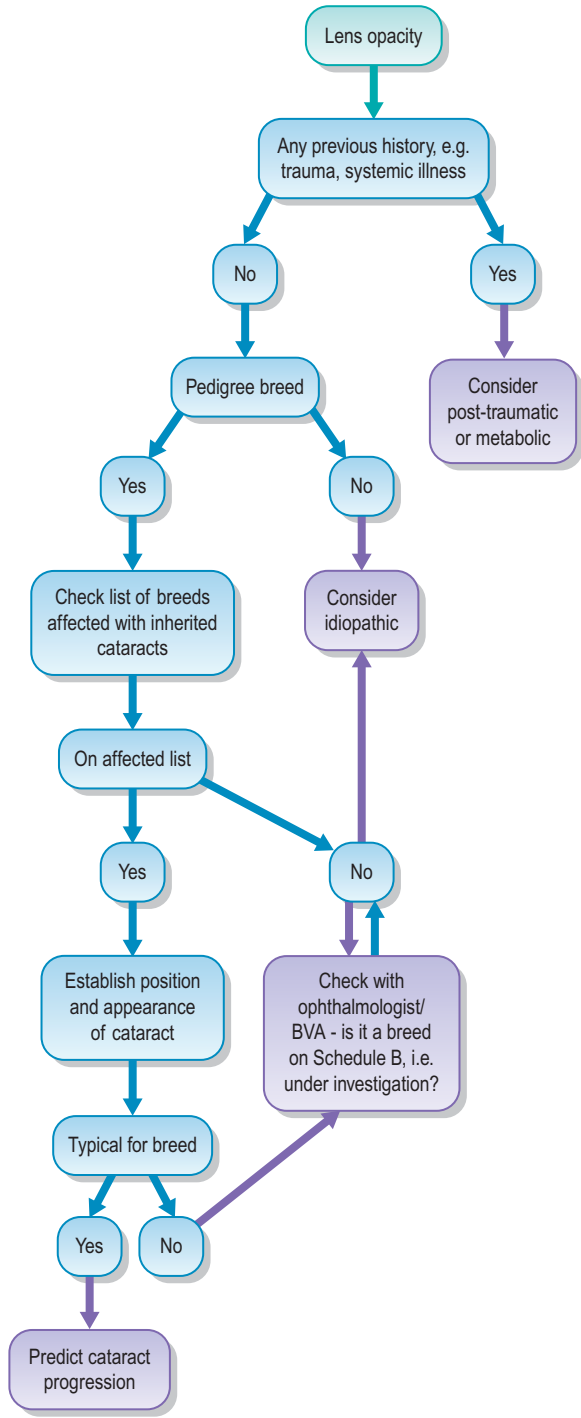


Figure 41.2 Decision-making tree – cataract in a young dog.

TREATMENT OPTIONS – MEDICAL

Many inherited forms of cataract in dogs do not progress to cause significant visual impairment and, as such, monitoring is all that is required. However, some cataracts can be rapidly blinding. If there is any doubt about the diagnosis, referral to an ophthalmologist should be considered. They can also be contacted for advice regarding the likelihood of progression in a particular breed if necessary, although there is much printed information available in standard ophthalmic texts and available on various websites.

There is no medical treatment for cataract. Recently, antioxidants (e.g. carnosine) and aldose reductase inhibitors have been developed and marketed for use in both humans and dogs, claiming to slow or reverse the cataractous change. Unfortunately, no clinical benefit has been demonstrated in controlled studies and, as such, their use cannot be advocated. Therefore, in cases where the cataract is progressing and affecting vision, surgery remains the only option in preventing blindness.

TREATMENT OPTIONS – SURGICAL

If the cataracts are progressing, affecting vision, or in a breed known to have progressive cataracts (e.g. Staffordshire bull terrier, Bichon Frise, American cocker spaniel and several others), referral for specialist advice and surgical removal should be considered. Since these dogs are typically young and active they do benefit from surgery (usually phacoemulsification with intraocular lens implantation) and committed owners should be encouraged to proceed with referral.

PROGNOSIS

The prognosis for young dogs with juvenile inherited cataracts is generally good, although owners must be made aware of the fact that their dog does not have, and never will have, normal eyes or normal vision. In some breeds, such as the retriever family, the majority of affected dogs do not demonstrate any visual impairment and live out their lives with the cataracts as a blemish and nothing more. If the cataracts do require surgery, most do well.

Young dogs with immature cataracts have soft lenses which are amenable to phacoemulsification (± intraocular lens implantation) but owners must be committed to the long-term care of these dogs. After the initial postoperative period they should still be examined by an ophthalmologist every 6–12 months indefinitely to check for evidence of long-term complications such as posterior capsular opacification, where there is fibrosis of the posterior capsule (so-called after-cataract) which is more common in young dogs and can reduce vision.

Glaucoma is also a potential complication and unless it is diagnosed early will result in blindness. Thus, although the initial success rate in juvenile cataracts is approximately 90%, this reduces over time, and after 2 or 3 years the successful visual outcome can drop to 50–60%. However, even if the dog does succumb to secondary glaucoma and blindness, it will have had vision for significantly more of its life, during its young, most active stage, than had surgery not been performed.

42 Canine lens luxation

INITIAL PRESENTATION

Pain, abnormal appearance – cloudy

PRESENTING SIGNS

Dogs are presented with a painful eye when suffering from acute anterior lens luxation, the most frequently encountered form in dogs. Normally the owner notices a sudden onset of blepharospasm and increased lacrimation and the eye also appears both red and cloudy. Vision is frequently reduced but owners might not be aware of this if the other eye is normal. Terriers are most commonly affected and young adults, 4–6 years, are most at risk. Affected animals can be depressed and unwilling to eat due to the pain from the condition. Most owners realize that there is something wrong and bring their pet to the surgery quickly, but unfortunately this is not always the case. Breeders will be aware of the condition and as such are more likely to present affected dogs as a matter or urgency.

CASE HISTORY

In most cases there is no previous history of ocular disease, or relevant systemic disease. The dog might have been out playing, such that the owner suspects a traumatic incident, but this is rarely witnessed (and usually has not occurred!). Sometimes the owner will have been aware that the eye was a bit red and watery for a few days before it suddenly became acutely worse.

CLINICAL EXAMINATION

On general clinical examination the patient might be subdued and unwilling for the face to be touched – especially on the affected side. Mild pyrexia can be present due to the pain of the condition but otherwise the examination is unremarkable.

Ophthalmic examination is likely to reveal reduced vision on the affected side. A moderate serous ocular discharge might be present, along with blepharospasm and photophobia. It might be difficult to open the eye for examination, especially in moderately enophthalmic breeds such as the miniature English bull terrier. Application of topical anaesthetic drops might assist the examination. Corneal oedema is frequently present, sometimes affecting the whole of the eye (when severe secondary glaucoma has set in) or just in the subcentral area, with a cloudy patch visible. Episcleral congestion will be present. Ulceration is not normally a feature unless the patient has self-traumatized.

The lens can be visualized in the anterior chamber between the back of the cornea and the front of the iris (Figure 42.1). By viewing the patient from the side it should be possible to see the iris bowing back away from the cornea and the structure of the lens should be apparent. The size of the pupil is variable (if visible!) but is normally mid sized or dilated, and often fixed or poorly responsive to light. If fundus examination is possible it could be normal, or optic disc cupping might be noted (discussed further in the cases on glaucoma). Intraocular pressure measurement should be undertaken if possible, since this will have an important effect on the prognosis and management of the cases.

Positive diagnostic tests which can be performed in practice are simple – if there is a positive dazzle reflex in the affected eye, and a consensual pupillary light response in the unaffected eye, i.e. on shining a light

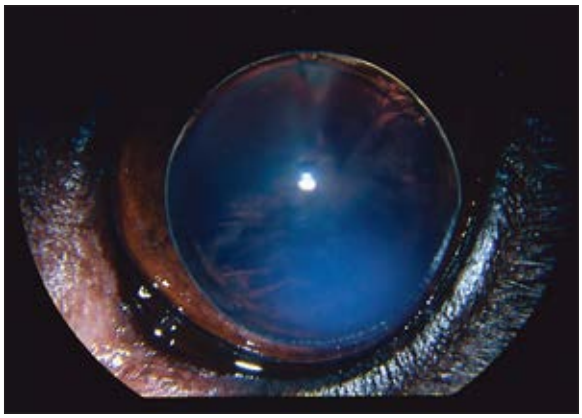


Figure 42.1 Anterior lens luxation in a 4-year-old Jack Russell terrier. The lens can be seen sitting in the anterior chamber and a subcentral patch of corneal oedema is present where the lens is resting against the corneal endothelium.

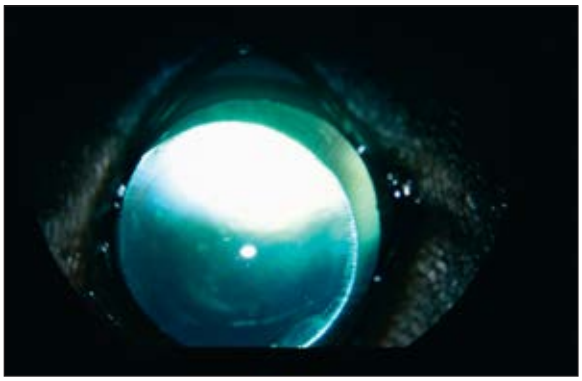


Figure 42.2 Subluxated lens in a 5-year-old miniature English bull terrier. An aphakic crescent can be seen dorsomedially with stretched lens zonules just visible in this area. The pupil has been fully dilated to appreciate this lens instability although iridodonesis was present clinically.

into the affected eye the fellow pupil constricts, these both indicate that vision might be preserved with rapid referral for lendectomy. If neither test is positive, then the prognosis for vision is very poor, and any emergency surgery is only likely to salvage the globe.

The fellow eye should be very carefully evaluated since primary lens luxation is a bilateral condition, although it is rare for both lenses to luxate simultaneously. The contralateral lens is frequently subluxated – this can be appreciated as iris wobble (iridodonesis) or lens wobble (phacodonesis) as the eye moves. The anterior chamber might be deep or shallow, depending on where the lens has slipped, and strands of vitreous are frequently visible in the anterior chamber. Pupillary dilation with tropicamide might allow the edge of the lens to be visualized – normally it slips slightly ventrolaterally, and stretched lens zonules can sometimes be noted (Figure 42.2). The worse affected eye (with the full anterior luxation) should not be dilated with tropicamide since this could both exacerbate any glaucoma and allow the lens to fall into the vitreous, making surgical retrieval much more difficult.

Some patients will not have an anterior but a posterior lens luxation. Typically with these the owner reports the eye was very sore and cloudy, but then became more comfortable, although it still did not look normal. In such cases the subcentral patch of corneal oedema will still be present but the lens is not visible in the anterior chamber (Figure 42.3). The iris normally wobbles (iridodonesis) on globe movement and the anterior chamber is often deep. By looking though the pupil from above, the lens can normally be seen lying in the ventral posterior segment. The different subtypes of lens luxation are shown in Figure 42.4.

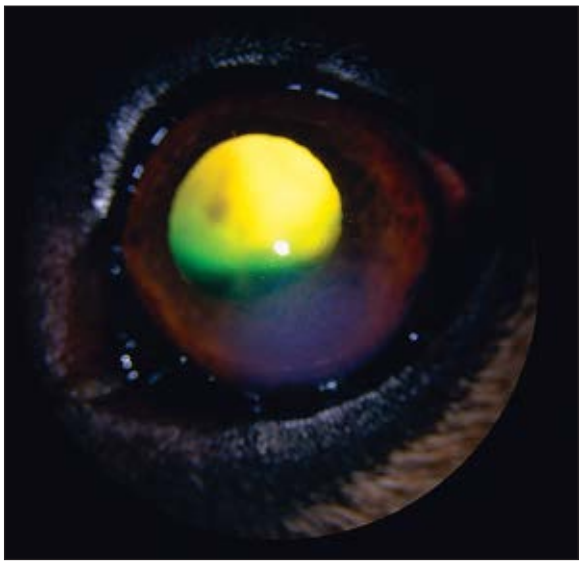


Figure 42.3 Posterior lens luxation in an elderly German shepherd dog. There is a patch of corneal oedema but no lens in the anterior chamber or pupil – with careful examination it will be found ventrally in the vitreous.

It should be remembered that chronic glaucoma will cause globe enlargement and can result in lens sublux-

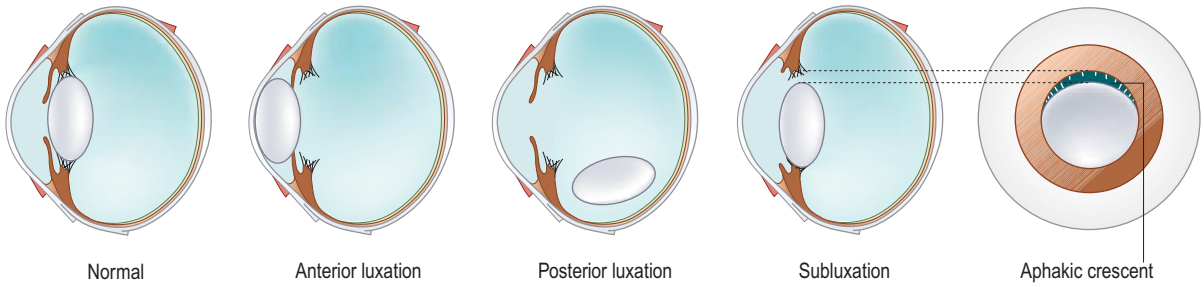


Figure 42.4 Diagram showing position of the lens with subluxation and luxation.

ation or complete luxation. This needs to be differentiated from primary lens luxation. Consideration of the breed, previous ocular history and careful evaluation of the fellow eye should assist.

DIFFERENTIAL DIAGNOSES

- Lens luxation:
  - Primary
  - Secondary (to glaucoma or uveitis)
- Glaucoma
- Corneal endothelial dystrophy
- Sudden onset cataract

CASE WORK-UP

Since many cases of primary lens luxation will require referral for emergency surgery, there is little work-up required once the diagnosis has been reached. If the corneal oedema is so severe that intraocular contents cannot be examined, ocular ultrasonography will delineate the abnormal lens position.

Emergency treatment pending referral

- Analgesia – systemic NSAID or opiate.
- Anti-glaucoma medication – topical carbonic anhydrase inhibitor (e.g. dorzolamide or brinzolamide).
- Possible prostaglandin analogue – latanoprost – but check with your ophthalmologist since some do not recommend this in cases of lens luxation.
- Consider mannitol if severe glaucoma and any delay in referral.

NURSING ASPECTS

These patients will be in pain and so gentle handling and careful medication are required. Since the treatment for the condition is frequently surgical, details of the nursing care for this will not be discussed here as it is a specialist procedure.

EPIDEMIOLOGY

Primary lens luxation does have an underlying inherited basis. In the Tibetan terrier it has been shown to be an autosomal recessive form of inheritance, and this is assumed for other breeds as well. Eye testing for prospective breeding animals is recommended for at-risk breeds under the British Veterinary Association/Kennel Club/International Sheep Dog Society (BVA/KC/ISDS) scheme. Similar schemes exist in Europe, USA and Australia. The condition should always be considered in any young adult terrier presenting with an acutely painful eye.

Breeds affected by primary lens luxation include:

- Jack Russell terrier
- Lancashire heeler
- Wire haired fox terrier
- Miniature bull terrier
- Sealyham terrier
- Tibetan terrier
- Terrier types
- Border collie
- Shar Pei.

The condition is bilateral, and owners must be warned that similar problems are likely in the fellow eye. Indeed

it is very common to find abnormalities in the supposedly 'normal' eye on examination. The lens zonules are weak and abnormal, and, with time, they fracture, allowing the lens to wobble before it dislocates completely. Unfortunately, the condition is not as straightforward as initially assumed – lens luxation leading to secondary glaucoma. Intraocular pressure (IOP) is frequently elevated before the lens luxates completely, suggesting that there are drainage problems present even when the lens is subluxated. Indeed, careful ophthalmoscopy of the fundus in subluxated cases will frequently reveal optic disc damage associated with high pressures (either pressure spikes or sustained elevations). As such, vision can be reduced well before the lens dislocates completely, meaning that even with early surgical intervention sight is not preserved. The exact mechanisms for this complex problem are not fully understood, but do make the condition very serious for the patient and sometimes frustrating to manage.

Once the lens luxates fully into the anterior chamber a pupil block occurs whereby aqueous cannot flow from the posterior chamber through the pupil and out through the drainage angle. This results in a sudden rise in IOP which causes the acute pain, and can result in permanent blindness. The presence of the lens also directly blocks the drainage angle, and any vitreous which moves forward with the lens further reduces drainage. The lens touches the cornea, and the pressure from it on the corneal endothelium results in local damage to the endothelial pump and a patch of oedema. As the IOP rises, this oedema spreads to include the entire cornea and the whole surface becomes cloudy. If the lens falls into the posterior segment, the pressure will reduce somewhat as the aqueous can flow once more, providing the drainage angle or pupil is not still blocked with vitreous. Repeated episodes of the lens moving anterior then posterior will cause an inflammatory reaction and this can further trigger the secondary glaucoma.

CLINICAL TIPS

- Signalment – any adult terrier or terrier type presenting with an acutely painful eye should be examined for potential lens luxation
- Examine the eye from the side to visualize the lens in the anterior chamber
- A subcentral patch of corneal oedema could suggest lens luxation – if you cannot see the lens, look in the posterior segment
- Iridodonesis is pathognomonic for lens luxation
- ALWAYS check the fellow eye – strands of vitreous in the pupil and iridodonesis are common
- MEASURE IOP!
- Check dazzle reflexes and consensual pupillary light response
- Decide if this is primary lens luxation or secondary to previous glaucoma
- Consider urgent referral

TREATMENT OPTIONS – MEDICAL

Emergency medical management aims to lower the IOP such that permanent damage can be reduced, and to alleviate pain prior to referral for lendectomy.

Posterior lens luxation cases can be managed medically – sometimes successfully for a long time. Treatment here aims to keep the pupil miotic, thus reducing the risk of the lens falling forwards again by providing a solid diaphragm with the iris, and also in maintaining IOP within the normal range. Pilocarpine has been used in the past but has mainly been superseded by prostaglandin analogues such as latanoprost and travoprost. Although expensive, these drugs are effective at maintaining a miotic pupil and increase uveoscleral outflow, thus lowering IOP. The miosis can be profound, such that tunnel vision is all the animal has, but compared to being totally blind (or even enucleated) this is an acceptable compromise. Some patients with subluxated lenses can also benefit from this approach to treatment, particularly if surgery is not wanted.

Any patient on such medication requires frequent re-examination and IOP measurement, and continued liaison with a veterinary ophthalmologist will offer the best long-term prognosis for these patients. If the lens does move anteriorly despite the medication, then surgical removal is required. Some ophthalmologists prefer to remove posteriorly luxated lenses as well as those in the anterior chamber but the risk of complications both during and after surgery is higher with posterior rather than anterior luxations.

TREATMENT OPTIONS – SURGICAL

In general, emergency lendectomy is the only treatment for primary lens luxation if there is any chance of maintaining or regaining vision. This will require referral to a suitably qualified colleague as a matter of urgency. An open sky intracapsular extraction remains the most frequently performed method of lens extraction, but phacoemulsification with vitrectomy is possible in some cases, providing the lens can be stabilized sufficiently to allow the phacoemulsification (which is a less traumatic method of surgery and seems to carry a slightly improved prognosis).

If the eye has been painful for more than 1–2 days it is likely that the secondary glaucoma will have caused excessive damage such that vision has been permanently lost. The absence of a dazzle response in the affected eye and of a consensual pupillary light reflex to the fellow eye are both negative prognostic indicators, and enucleation might be necessary if the IOP is high and the eye irreparably blind. Referral for evaluation of the contralateral eye is still advised in such cases. Some owners will request lendectomy, even knowing that no vision can be restored (particularly to avoid enucleation of the second eye). Frequently patients with lens luxation require life-long medication, hopefully to maintain vision but also to prevent the pain associated with the glaucoma, and owners should be fully aware of the fact that the dog will never be normal.

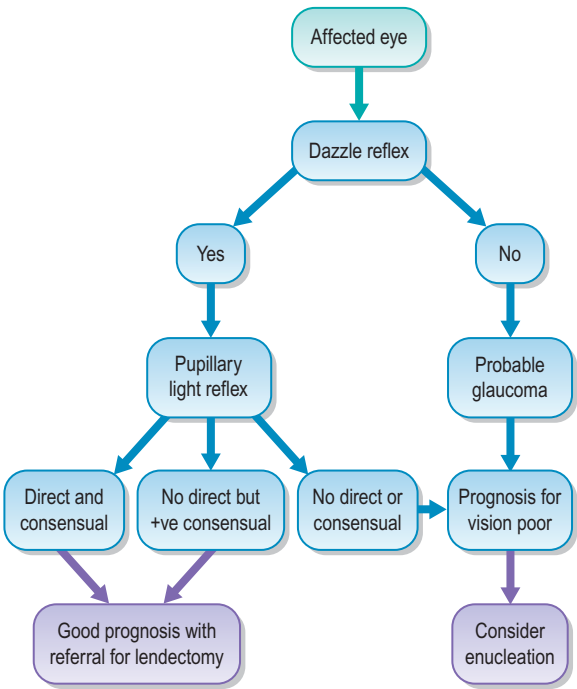


Figure 42.5 Decision-making tree – lens luxation.

PROGNOSIS

Primary lens luxation is a horrible disease and the prognosis is guarded in every case. Early diagnosis and rapid removal of the anteriorly luxated lens can maintain vision but topical therapy with anti-glaucoma ± anti-inflammatory medication will be necessary long term. Unfortunately, risks of blinding glaucoma still exist even with early lendectomy. Retinal detachment is the other main complication – as the lens falls forwards and pulls the vitreous with it, this in turn pulls the neurosensory retina away from the retinal pigment epithelium. The resulting retinal detachment is not treatable and results in permanent blindness. Some ophthalmologists perform laser or cryoretinopexy prior to lendectomy in an attempt to 'spot weld' the retina in position but the effectiveness of this procedure remains to be established. Dogs with retinal detachment are normally comfortable albeit blind.

Some surgeons will advocate surgery on subluxated lenses but others prefer monitoring since the risk of retinal detachment is present with surgery on these eyes. Many eyes with primary lens luxation end up being enucleated. Frequently owners will accept this with the first eye, but when the second eye becomes affected wish to try all treatment options before resorting to enucleation (or intraocular prosthesis). In general, the prognosis for the second eye is slightly better than for the first, since the diagnosis is made earlier and the owner is more aware of what signs to look for and is usually committed to saving the eye.

With primary lens luxation, prevention is definitely preferred. Owners of affected dogs should inform the breeder in all cases, and the health officer for the breed club as well if possible. Only by avoiding breeding from affected or at-risk animals can we reduce the incidence of this debilitating disease. Genetic testing should be available in the future.

SECTION 7

GLAUCOMA

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43 Glaucoma – introduction

Glaucoma is the term given to the group of diseases in which a raised intraocular pressure leads to damage to the optic nerve and the retinal cells. It is a painful, often blinding, condition and is a very serious disease which can be difficult to treat. There are many causes of glaucoma and the choice of treatment often depends on the cause, the duration of the disease and the potential visual outcome.

The maintenance of normal intraocular pressure depends on a balance between production and drainage of aqueous humour. The ciliary body produces aqueous from the posterior non-pigmented epithelial cells by a process of both active secretion and passive ultrafiltration, and it flows through the pupil into the anterior chamber. It drains out of the eye through the iridocorneal angle (drainage or filtration angle). This pathway is shown in Figure 43.1. Once in the drainage angle, the aqueous passes through the pectinate ligaments and into the uveal trabecular meshwork where it is absorbed into the venous plexus vessels and out into the episcleral and scleral drainage veins (Figure 43.2). There is a second route of aqueous drainage – the uveoscleral or unconventional pathway. Here aqueous is drained via the ciliary body interstitial cells into the suprachoroidal space and choroidal and scleral vessels. The percentage of aqueous following this alternative pathway varies between species – it is more important in the horse than in dogs for example.

Most cases of glaucoma result from impaired drainage rather than from overproduction. Tonometry, the measurement of intraocular pressure, is essential for the diagnosis and management of glaucoma. Normal intraocular pressures are listed in Table 43.1. Without the appropriate instruments to do this, it is impossible to diagnose and treat successfully, and therefore referral to an ophthalmologist is advised should a tonometer (Schiotz, Tonopen or TonoVet) not be available in the surgery. Since glaucoma is such a common ophthalmic problem, representing approximately 10% of ocular problems in one survey, it is strongly recommended that each veterinary practice has access to tonometry. In addition to measuring the intraocular pressure, gonioscopy – the examination of the iridocorneal (drainage) angle – is frequently necessary. The techniques for both tonometry and gonioscopy are discussed in Chapter 1. The latter technique requires practice to master and so is not routinely performed in general practice.

The clinical signs of glaucoma will vary depending on the species of animal, the cause, the speed of onset and the duration, together with how high the intraocular pressure has become. For ease of description the signs are usually divided into those seen with acute glaucoma and those in chronic cases – these are listed in Table 43.2. In general terms, the condition usually presents unilaterally and acute onset is common in dogs. Cats more frequently suffer from a chronic insidious onset. In dogs, pain, corneal oedema, episcleral congestion and blindness are the most consistent presenting signs whereas in cats it might just be a dilated pupil which is noted.

The causes of glaucoma are broadly divided into primary and secondary (see Figure 43.3). Congenital glaucoma is also encountered albeit rarely. Primary glaucoma usually involves goniodysgenesis – an abnormality of the drainage angle which is visible on gonioscopy. It is a bilateral, but not usually symmetrical, disease and there can be a delay of weeks to years before the second eye is affected. It is discussed in the following section. Secondary glaucoma is more commonly encountered in dogs and cats, as well as in horses. It develops due to some other ocular abnormality leading to problems with the circulation and drainage of aqueous humour – for example, at the pupil due to adhesions between the iris and lens, or due to a tumour blocking the drainage angle – more details follow in Chapter 45.

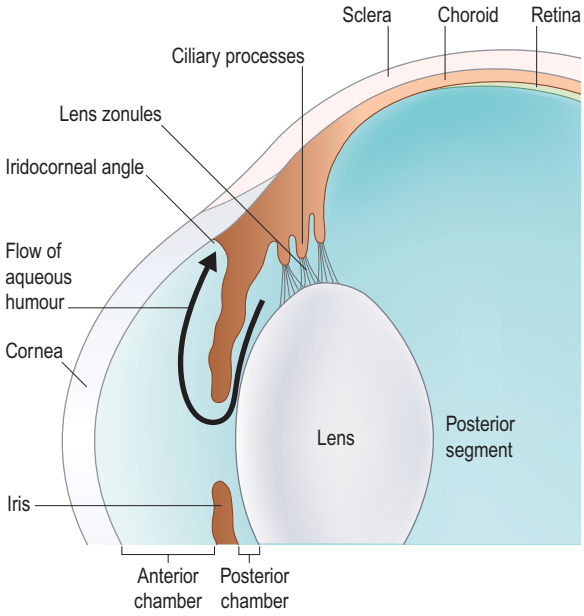


Figure 43.1 Pathway of aqueous production and drainage in the eye.

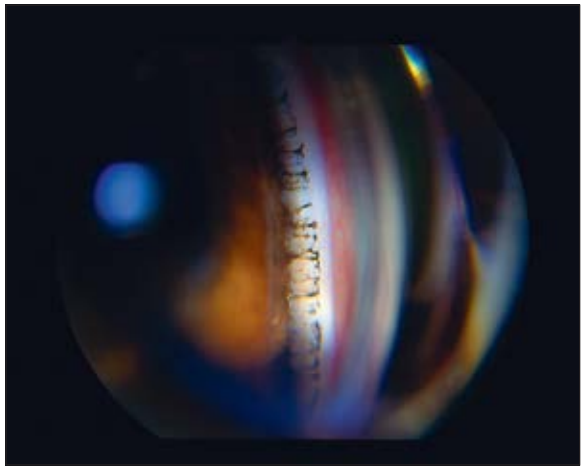


Figure 43.2 Drainage angle as seen on gonioscopy – the pectinate ligaments can be clearly seen spanning the angle.

Table 43.1 Normal intraocular pressures in dogs, cats and rabbits

Dog	Cat	Rabbit
15–25 mmHg	16–27 mmHg	16–20 mmHg

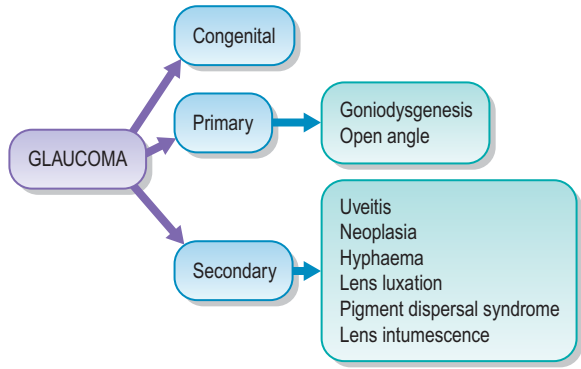


Figure 43.3 Classification of glaucoma.

Table 43.2 Clinical signs of glaucoma

Acute glaucoma	Chronic glaucoma
• Pain • Increased lacrimation • Blepharospasm • Protrusion of third eyelid • Corneal oedema • Episcleral congestion • Dilated pupil (mydriasis) • Peripheral corneal vascularization • Blindness	• Enlarged globe • Superficial and deep corneal vascularization • Optic disc cupping (posterior bowing of optic disc from pressure) • Corneal ulceration • Corneal (Haab's) striae (grey lines on cornea from tears in Descemet's membrane) • Lens subluxation/luxation • Retinal atrophy

TREATMENT OF GLAUCOMA

The treatment of glaucoma can be medical or surgical and often a combination of both is required. Clearly the underlying cause needs to be determined since this will influence the choice of treatment. If the glaucoma is secondary to an intraocular mass, then enucleation is the treatment of choice, while primary glaucoma is more amenable to medical or surgical management. The aim of treatment is to lower the intraocular pressure to a level which is comfortable and does not promote further optic nerve or retinal damage. If the eye is still visual, or has the potential for a return of some vision, then this is also a major aim – to prevent deterioration in eyesight. Unfortunately, many eyes will be irreparably blind on presentation. If the cause is primary glaucoma, then treatment must be targeted to prevent the same outcome for the second eye.

Emergency treatment for glaucoma should be considered in all acutely presented cases regardless of cause.

Alleviation of the pain associated with the elevated intraocular pressure will give time for further investigation such that a long-term management plan can be devised. Osmotic diuretics such as mannitol solution are given intravenously. Care must be taken to ensure that renal function is adequate before using these agents since they can trigger renal failure in compromised patients. The pressure-lowering effect of osmotic diuretics is only short lived, and so a longer term treatment plan involving medication or surgery must be arranged.

In addition to lowering the pressure, specific pain relief should be provided, e.g. with opiates or NSAIDs. Long-term medication usually involves the use of topical carbonic anhydrase inhibitors such as dorzolamide (Trusopt, MSD) and brinzolamide (Azopt, Allergan). These lower intraocular pressure by reducing the production of aqueous humour at the ciliary body. Topical prostaglandin analogues such as latanoprost (Xalatan, Pharmacia & Upjohn) are used for primary glaucoma but should not be used if uveitis is present. They work by improving the outflow of aqueous, and are often used in conjunction with the topical carbonic anhydrase inhibitors. Other agents such as miotics and beta-adrenergic blockers are also sometimes prescribed (see Table 43.3).

Surgery for glaucoma can include procedures to reduce the production of aqueous such as laser and cryosurgery (cyclophotocoagulation and cyclocryotherapy, respectively) and procedures to increase outflow which involve either drainage implant surgery or scleral trephination and peripheral iridectomy. Clearly these are specialist procedures and referral is required to assess whether a patient might be suitable. Unfortunately, enucleation is frequently necessary, particularly if the eye is blind and painful.

Table 43.3 Medical treatment of glaucoma

Emergency treatment	Long-term treatment
Mannitol – 10–20% solution by intravenous infusion over 20–30 minutes	Topical anhydrase inhibitor, e.g. dorzolamide (Trusopt MSD)
Analgesia – systemic NSAID or opiate	Topical prostaglandin analogue, e.g. latanoprost (Xalatan, Pharmacia & Upjohn)
Paracentesis (removal of a small volume of aqueous)	Topical miotic, e.g. pilocarpine
Systemic carbonic anhydrase inhibitors, e.g. acetazolamide occasionally used	Topical beta-adrenergic blockers, e.g. timolol

44 Primary canine glaucoma

INITIAL PRESENTATION

Pain, abnormal appearance – cloudy, blind

PRESENTING SIGNS

Dogs with primary glaucoma will be presented with a painful, cloudy eye. Increased lacrimation is normally present as well. Owners might be aware of visual loss in the affected eye, but frequently this is not something which they have noticed. The patient is likely to be depressed and may be inappetant. In addition, the owner might report seeing their pet make attempts at rubbing or hiding their face. The presenting signs normally develop very quickly. Affected animals are usually pedigree dogs, and aged 5–8 years, although some breeds develop primary glaucoma at a younger age – discussed in more detail later.

CASE HISTORY

In most cases there is no previous history of ocular disease or relevant systemic disease. Indeed, the patient is usually completely normal one day, then suddenly develops a very sore, cloudy eye – perhaps noticed when the owner gets up or when they come in from work. Occasionally there is a history of some intermittent slight clouding and redness on a few occasions which settled before the owner became particularly worried, but this is the exception rather than the rule.

CLINICAL EXAMINATION

General clinical examination is unremarkable, although a mild, pain-induced pyrexia is sometimes present. The dog might resent examination of the face and the affected eye can be difficult to examine due to the severe blepharospasm. However, with patience, gentle handling, and the use of topical anaesthesia if necessary, a rewarding ophthalmic examination should be possible.

The affected eye is likely to be blind, or with severely reduced vision, demonstrated by a reduced or absent menace response. Corneal oedema will manifest as a blue–grey corneal discolouration. In addition to the clouding, the eye will also be red – episcleral congestion is usually marked and peripheral corneal vascularization develops which manifests as a red fringe around the limbus (ciliary flush) (Figure 44.1). The pupil can be assessed using a bright light source and is usually mid sized or dilated with very limited constriction when illuminated. A dazzle reflex remains if the pressure has not got too high. The indirect or consensual pupillary light reflex should be assessed and this can help with the prognosis regarding any return of vision – if there is no constriction of the fellow pupil there is less chance of a return of any useful eyesight in the affected eye.

Detailed intraocular examination is hampered by the corneal oedema but the use of indirect ophthalmoscopy will be more informative than direct ophthalmoscopy since it provides better imaging through semi-opaque media. Thus, on fundus examination, cupping of the optic disc might be appreciated – seen as a small, dark disc with the blood vessels seeming to dip down into it from the retina (Figure 44.2). Peripapillary retinal degeneration is sometimes seen, along with more widespread hyper-reflectivity and blood vessel attenuation in advanced cases.

Measurement of intraocular pressure is essential to reach a definitive diagnosis, to provide prognostic advice, to draw up a treatment plan and to monitor the effectiveness of the treatment. Without tonometry it is very



Figure 44.1 Primary glaucoma showing severe corneal oedema and episcleral congestion. The pupil is mid-dilated. Intraocular pressure was 38 mmHg.

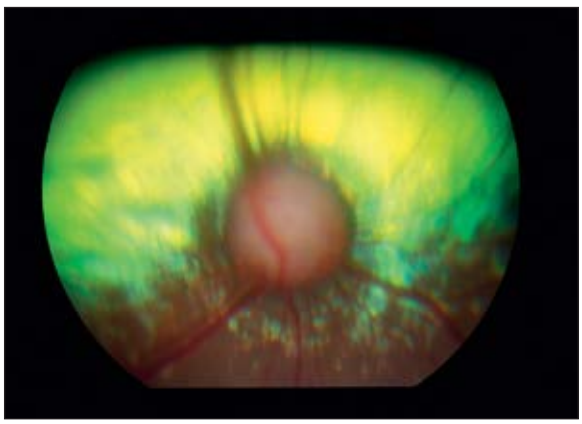


Figure 44.2 Optic disc cupping. The canine optic disc is small and round, with the blood vessels appearing to dip down into it.

difficult to assess the patient properly. If a tonometer is not available, then referral for pressure measurement should be considered. The technique for tonometry, using a Schiotz tonometer, Tonopen or TonoVet, is detailed in Chapter 1. Remember that there are several factors which can affect the intraocular pressure readings (Table 44.1).

Breeds affected by goniodysgenesis (UK list) include:

- American cocker spaniel
- English cocker spaniel
- English springer spaniel
- Welsh springer spaniel
- Basset hound
- Bouvier des Flandres
- Labrador retriever
- Flat coated retriever
- Golden retriever
- Great Dane
- Welsh terrier
- Dandie Dinmont terrier
- Siberian husky
- Samoyed.

DIFFERENTIAL DIAGNOSES

- Corneal endothelial dystrophy
- Uveitis
- Exophthalmos
- Episcleritis
- Secondary glaucoma

CASE WORK-UP

Measurement of the intraocular pressure will give the diagnosis of glaucoma but does not tell us the cause for the raised pressure. Primary glaucoma develops in the absence of other intraocular disease and is an inherited condition in several breeds of dog. Goniodysgenesis, or malformation of the drainage angle, is the most frequent cause of acute primary glaucoma in dogs, and is a bilateral condition (although usually presented as a unilateral problem). Thus it is necessary to evaluate the contralateral eye. Gonioscopy is the examination of the drainage angle and referral for this procedure should be considered in all dogs which present with acute glaucoma. It is rarely possible to perform gonioscopy on the affected eye (due to the corneal oedema, peripheral corneal vascularization and pain for example) but examination of the fellow eye will reveal evidence of goniodysgenesis and thus the second eye is also at risk from the development of glaucoma.

Other tests which can be performed as part of the case work-up include ultrasonography. This will allow detailed visualization of intraocular structures despite the opaque ocular media and can help to differentiate primary from secondary glaucoma. For example, an iridal mass or lens luxation could be detected, which would suggest that the raised intraocular pressure developed secondary to other intraocular disease. Ultrasonography can also allow measurement of the globe diameter to determine if it has been stretched and enlarged (hydrophthalmos) as a result of the increased pressure.

Table 44.1 Factors that can affect intraocular pressure (IOP) readings

Factor	Effect
Breed	Higher in terriers and fluctuates more in these breeds as well
Age	Decreases with age
Degree of restraint required	Pressure around the neck will result in higher readings
Position of head	Higher with the head back and elevated
Instrument used	Slight variations between types of tonometer
Amount of pressure used to measure IOP	Pressure on the globe (including the forced lid opening required to access the cornea) will increase readings

EPIDEMIOLOGY

Primary glaucoma is most commonly associated with goniodysgenesis but a second category, open-angle glaucoma, also occurs. Goniodysgenesis is considered inherited in several breeds, as listed above. The exact relationship between the abnormal drainage angle and the development of glaucoma is unknown. Indeed, some dogs with severely malformed iridocorneal angles do not develop glaucoma at all, while others with relatively mild changes can have severe disease in one or both eyes. If gonioscopy reveals an abnormal angle, then the animal is certainly at a higher risk from developing glaucoma than the general population, but unfortunately at present we cannot quantify this risk. The glaucoma seen with goniodysgenesis is acute and serious. The dog exhibits a sudden onset, red, cloudy, painful, often blind eye and intraocular pressure measurement is mandatory in any of the breeds listed above which present with these signs. Screening programmes, such as the British Veterinary Association/Kennel Club/International Sheep Dog Society (BVA/KC/ISDS) eye scheme, provide gonioscopy to breeders and it is recommended that dogs affected with goniodysgenesis are removed from breeding circles.

Primary open-angle glaucoma is less common, but can be encountered in the Norwegian elkhound and beagle. Here the condition is slow in onset and vision can be maintained until quite late in the course of the disease. The increase in intraocular pressure is gradual and the eye can tolerate these changes with less pain and globe damage than with goniodysgenesis. Thus patients are presented late in the course of the disease, with visual loss, dilated pupils and enlarged globes as the most notable symptoms.

NURSING ASPECTS

General nursing to make the patient comfortable and prevent self-trauma is required, along with the application of various topical (and sometimes also systemic) medications.

CLINICAL TIPS

- Remember the list of breeds affected with primary glaucoma and always consider this condition if an at-risk breed presents with an ophthalmic complaint
- Compare intraocular pressures – but remember what can affect your readings (Table 44.1)
- Refer for tonometry and gonioscopy if you cannot perform these essential diagnostic tests
- Optic disc cupping is a sign of glaucoma – if the disc of the dog starts to look like that of a cat (i.e. small and round), this could suggest cupping

TREATMENT OPTIONS – MEDICAL

The medical treatment for acute primary glaucoma is divided into emergency treatment and long-term control (see Table 43.3 for summary). Only once the pressure has been reduced and stabilized should the long-term management be considered. Most medical approaches to treatment involve expensive topical agents and owners must be able to administer these effectively and should be aware of the ongoing costs of treatment.

Pain relief should be a priority in acute glaucoma. Systemic NSAIDs in combination with an opiate are most frequently administered.

Traditionally the emergency treatment for reducing intraocular pressure has relied on osmotic diuretic drugs

such as mannitol. Given intravenously this will draw fluid from the aqueous and vitreous into the extracellular space. Renal function should be assessed prior to its use since it can lead to kidney failure in compromised patients. Mannitol is given at a dose of 1–2 g/kg over 20 minutes, and a 20% solution is usually administered. This will result in a reliable reduction of intraocular pressure which lasts for about 6 hours. This provides time for the other pressure-lowering drugs to take effect. Clearly tonometry should be performed regularly (e.g. hourly) following the administration of mannitol. Glycerol is another hyperosmotic agent that can be given orally but can induce vomiting and is not used as frequently as mannitol.

Another emergency treatment which is gaining favour is the use of topical prostaglandin analogues such as latanoprost and travoprost. These reduce intraocular pressure in 15–30 minutes and the reduction will last up to 12–24 hours.

Carbonic anhydrase inhibitors are routinely used for both emergency and maintenance treatment of primary glaucoma. Topical agents have largely superseded the systemic preparations. Dorzolamide and brinzolamide are most widely used, although several other products are becoming available, some combined with beta-blocking drugs (which are effective in human patients but less so in our species).

The long-term medical treatment of primary glaucoma in dogs usually involves a topical carbonic anhydrase inhibitor (e.g. dorzolamide three times daily) plus a prostaglandin analogue (e.g. latanoprost once daily in the evening). The carbonic anhydrase inhibitor reduces the production of aqueous while the prostaglandin analogue improves its drainage and the drugs work synergistically. Topical beta-blocking agents are widely available, and although they can reduce intraocular pressure by a small amount in our patients, they are certainly far less effective in cases with goniodysgenesis than in the cases of open-angle glaucoma which are frequently encountered in human ophthalmology.

Treatment of the second eye is frequently undertaken, although this will not prevent the eye developing full-blown glaucoma. However, it might provide some protection during the insidious pressure spikes which are known to occur before the eye develops sustained elevated intraocular pressure. Typical prophylactic treatment would be a twice daily topical carbonic anhydrase inhibitor.

TREATMENT OPTIONS – SURGICAL

Surgical options for primary glaucoma include globe-saving procedures or enucleation. The former include techniques for reducing the production of aqueous, such as cyclophotocoagulation (laser ablation of the ciliary body) and cyclocryoablation (cryosurgery to freeze and destroy the ciliary body epithelium), together with techniques involving drainage implants to redirect aqueous outflow. Sometimes more than one procedure is combined. Newer techniques such as ciliary body endolaser show promise, but their cost and availability are limiting factors. Clearly referral is required for all of these techniques.

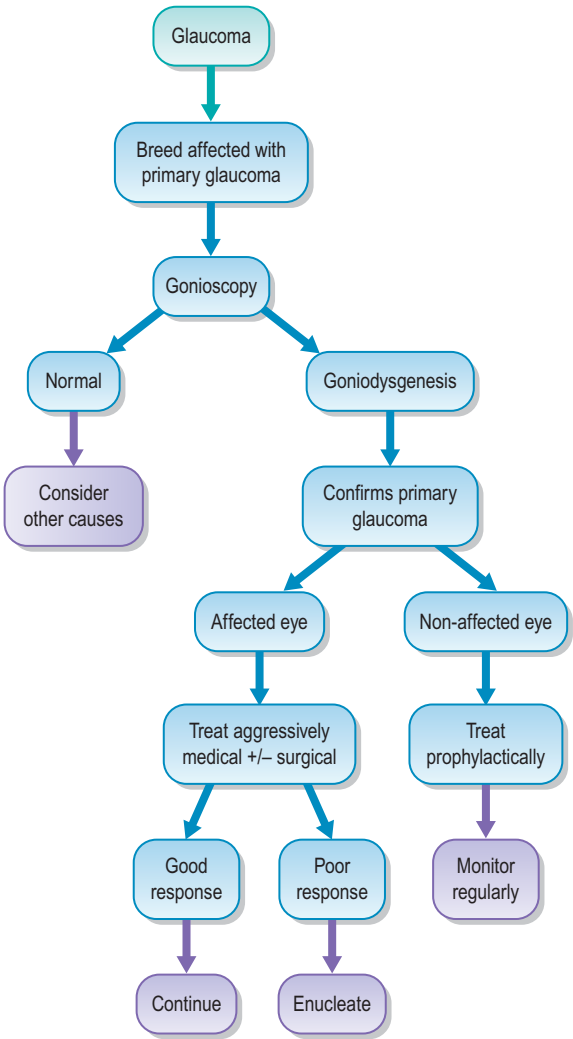


Figure 44.3 Decision-making tree – canine primary glaucoma.

In cases of severe glaucoma, where the eye is blind and painful, enucleation is frequently necessary and owners report that the dog is much happier and more playful and so on after the surgery, indicating that chronic pain was present prior to globe removal. Histopathological examination of enucleated globes should always be considered to confirm the diagnosis of primary glaucoma. The use of intrascleral prosthetics is becoming a more popular alternative to enucleation. Here the contents of the globe are removed (evisceration), leaving the sclera and corneal shell, and a silicone ball is placed into the empty globe. This can provide a more cosmetically acceptable appearance to the owners, but the procedure carries no benefit to the patient whatsoever and has the potential to cause problems such as corneal ulceration and extrusion of the implant. As such, the ethics of performing what is essentially cosmetic surgery for the benefit of the owner should be questioned.



Figure 44.4 Chronic glaucoma showing corneal striae (Haab's striae), some corneal pigmentation and episcleral congestion. Intraocular pressure was 32 mmHg.

PROGNOSIS

The prognosis for primary glaucoma is always guarded, both in terms of saving vision and in saving the eye. Owners should be aware from the start that the condition has the potential for both eyes to become affected and that blindness can be total. Often the first eye to become affected is permanently blinded, even if the owners present the dog quickly. The damage caused by the sudden rise in intraocular pressure can be severe and thus the aim with the first eye is often to keep the patient comfortable. The acute episode will become chronic, and other ocular changes such as globe enlargement (hydrophthalmos), corneal striae (breaks in Descemet's membrane, also called Haab's striae), corneal ulceration and pigmentation from exposure keratitis, secondary lens luxation and so on can all develop (Figure 44.4). Some globes eventually become phthitic and then non-painful but it can take several years for this to occur. Many globes are ultimately enucleated.

Once gonioscopy has been performed and it is established that the second eye is at risk from developing full-blown glaucoma, efforts should be made to reduce this risk. Prophylactic medication can delay the onset of glaucoma but we cannot predict which patients will benefit from prophylactic medication. Some dogs will never develop glaucoma despite goniodysgenesis, and thus the use of topical beta-blockers or carbonic anhydrase inhibitors prophylactically is an expensive waste of time. However, since the condition is so severe, the use of such drugs can probably be justified. They will 'buy some time' and provide some protection should the pressure suddenly rise. Owners should be educated to check their pet themselves and to bring it for regular ophthalmic examinations which include tonometry. They should check the pupil and look for any ocular redness (episcleral congestion) or subtle corneal oedema.

Many cases of primary glaucoma have wide variations in intraocular pressure with high pressure spikes prior to the onset of full-blown glaucoma, and observant owners can detect these. Regular ophthalmic examinations might reveal areas of retinal infarction or early optic disc cupping which resulted from short pressure spikes. These findings would indicate that the patient is starting with glaucoma and aggressive medical management or surgical intervention is warranted at this stage. With early diagnosis and thorough treatment the prognosis can be improved slightly, but the condition is still a very serious one which we are not able to manage particularly well. Prevention, by screening at-risk breeds with gonioscopy and removing affected animals from breeding pools, is to be encouraged.

CASE EXAMPLE 44.1

Patient details: Dandie Dinmont terrier, 8 years old, male (entire)

PRESENTATION AND HISTORY

The dog was presented with sudden onset pain, redness and clouding of the left eye. The owner was a breeder and was aware of glaucoma in the breed, but had not had her dogs examined under the BVA/KC/ISDS eye scheme. However, she was worried that this could be what was wrong with the dog and presented him to the surgery quickly. With hindsight she did concede that the eye had been a bit red and watery several times over the previous 2 months but she had not been worried at that stage.

CLINICAL SIGNS

On general examination the dog was subdued and head shy on the affected side but otherwise in good condition. The left eye was uncomfortable. No menace response was noted. There was marked episcleral congestion but only mild corneal haze. The pupil was dilated and unresponsive although a consensual response to the fellow eye was present. A reduced dazzle reflex was noted. Fundus examination revealed a very small, pale optic disc and some vascular attenuation (Figure 44.5). Intraocular pressures were 46 mmHg in the left eye and 18 mmHg in the right, which was ophthalmoscopically normal. Thus glaucoma was diagnosed, and assumed to be primary.

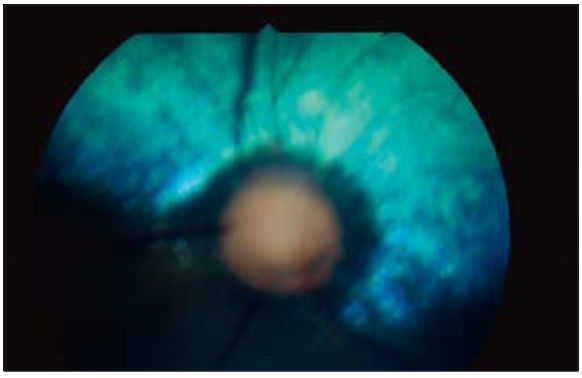


Figure 44.5 Fundus view of an 8-year-old Dandie Dinmont terrier. Marked optic disc cupping is apparent.

WORK-UP

Gonioscopy was performed on the right eye. This revealed a very narrow drainage angle, with sheets of tissue spanning it rather than discrete pectinate ligaments. This confirmed goniodysgenesis as the cause for the primary glaucoma.

TREATMENT

The left eye was treated topically with four times daily dorzolamide and twice daily latanoprost. Twice daily dorzolamide was also started in the right eye. The dog was given an injection of buprenorphine initially followed by oral carprofen.

OUTCOME

The intraocular pressure reduced to 32 mmHg 4 hours after initiating treatment and the patient was comfortable, albeit still blind in the left eye. Over the following 3 weeks the pressure varied between 28 and 42 mmHg despite continued medication. The owner decided that, since no vision had returned even when the pressure was lower, enucleation would be best for the dog. This was performed a month following diagnosis and certainly he was much more comfortable afterwards. The right eye was monitored on a monthly basis and the owner was fully aware that glaucoma could develop at any time. She diligently checked the eye and was prepared for surgery (probably laser rather than a drainage implant) as soon as either the pressure was above 25 mmHg or any evidence of optic disc or retinal disease was noted on the monthly check-ups. Early surgical intervention does offer the best prognosis but most ophthalmologists will not intervene until some indication of glaucoma has developed – each surgical technique carries the risk of complication (including blindness) and therefore is not usually undertaken prophylactically.

The dog had been used at stud. The owner had two of his offspring and both were found to have goniodys-

genesis. The breed club arranged a testing session at one of their dog shows and any dogs which were affected with goniodysgenesis were not used for further breeding.

This case illustrates that, even with early diagnosis, the prognosis for the first eye is frequently hopeless. However, the second eye does have a better chance of remaining visual and pain free for some time, although even with aggressive intervention blindness can ensue. The incidence of this debilitating disease can be reduced with selective breeding.

45 Secondary canine glaucoma

INITIAL PRESENTATION

Abnormal appearance – red/cloudy, pain, blind

PRESENTING SIGNS

Most dogs with secondary glaucoma will present with a painful eye and colour change – redness or grey/blueing of the cornea. The owner might or might not be aware of visual impairment in the affected eye. Since the condition is normally unilateral the reduction in vision in the affected eye is not always apparent to owners. The dog can be any breed, including crossbreeds, and is usually middle aged or older. The owner might have noticed the dog rubbing and the eye looking red or simply 'different' for days or weeks prior to seeking veterinary advice. The dog might also be slightly 'depressed' and quiet. Occasionally the presentation will be very acute, with no previous ocular changes, although this is less common with secondary than with primary glaucoma.

CASE HISTORY

Many dogs will have a history of previous ocular disease, although this is not always the case, and the dog might not have received veterinary treatment despite the owner being aware that there was something 'not quite right' with the eye for a while before it became red and painful. The most frequent previous ocular problem will be anterior uveitis – traumatic, immune-mediated or infectious – which responded to treatment initially but on cessation of anti-uveitis medication the eye deteriorated again a few days or weeks later. Sometimes owners will have restarted the previously prescribed medication but this did not improve the eye and so they returned for another examination. The cases with an acute history might be associated with lens luxation, so this should be considered in all terrier breeds (see Chapter 33 on uveitis and Chapter 42 on lens luxation).

CLINICAL EXAMINATION

On general clinical examination the dog might be slightly depressed with a mild pyrexia. It might be head shy and resent examination of the affected eye. A thorough general examination should be undertaken since the ocular problems might be associated with systemic disease (see Chapter 33). The presentation is normally unilateral for secondary glaucoma, but if a bilateral uveitis has been present, then both eyes can develop secondary glaucoma more or less simultaneously.

On ocular examination the affected eye might be noticeably enlarged. This is more common in long-standing cases or in younger dogs and puppies where the globe is able to stretch more easily (see Figure 37.5). The eye is likely to be red, with conjunctival hyperaemia and episcleral congestion; some peripheral corneal vascularization (ciliary flush) is also likely to be present. The eye is probably blind with no menace response, and the pupil is normally dilated and unresponsive, although if a previous uveitis has been present the pupil might be miotic, especially if extensive posterior synechiae are present. Alternatively, the pupil might be misshapen (dyscoria) (Figure 45.1). Thus the actual size of the pupil is not always useful in assisting with the diagnosis of secondary glaucoma.

The cornea will not be normal. Some corneal oedema is likely, giving a steamy appearance, such that detailed intraocular examination might be difficult. Corneal

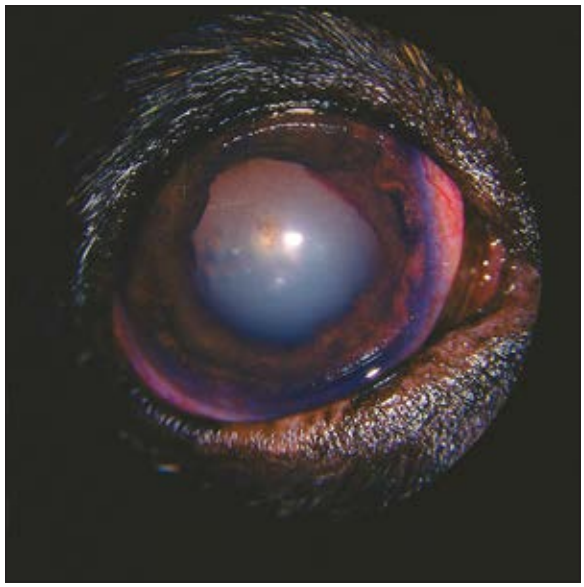


Figure 45.1 Glaucoma secondary to uveitis – note the dyscoria, posterior synechiae, pigment disruption, cataract and episcleral congestion.

DIFFERENTIAL DIAGNOSES

- Primary glaucoma:
  - Goniodysgenesis
  - Open angle
- Secondary glaucoma:
  - Uveitis
  - Neoplasia
  - Hyphaema
  - Intumescent or hypermature cataract
  - Retinal detachment
  - Lens luxation
  - Pigment dispersal syndrome
  - Post cataract or lens luxation surgery
- Uveitis
- Intraocular mass

vascularization, pigmentation and ulceration are all possible (often associated with exposure keratitis). In addition, corneal striae (Haab's striae; see Figure 44.4) might be present. These appear as a double white line on the cornea, almost like narrow train tracks, and are due to breaks in Descemet's membrane which form as the globe stretches. If there is any ongoing uveitis, this might manifest as aqueous flare, hypopyon or hyphaema.

Careful examination of the iris is important as neoplasia with a uveal mass is a common cause of secondary glaucoma in older dogs. If a mass is suspected, it is important to look tangentially through the pupil to see if any mass is present in the vitreous extending from the ciliary body – what is visible on the anterior surface of the iris is often just the tip of the iceberg, with a large ciliary body component also present. If fundus examination is possible this is likely to reveal optic disc cupping and possibly some retinal degeneration.

Measurement of intraocular pressure is essential in any suspected cases. Referral should be offered to the client if this is not possible in the practice. Both the absolute reading and the comparison between the two eyes are considered.

CASE WORK-UP

It is important to try to establish the underlying cause which triggered the secondary glaucoma. If a uveal mass, an anteriorly luxated lens or an intumescent cataract is present, this might be obvious on ophthalmic examination. However, ultrasonography is frequently required since the cornea is often opaque and detailed intraocular examination is thus not possible. In addition to examining for evidence of intraocular neoplasia, the size and position of the lens should be evaluated, the depth of the anterior chamber assessed and the posterior segment checked for evidence of retinal detachment. If there is a history of previous uveitis, this has hopefully been investigated already, and the secondary glaucoma is an unfortunate complication from the uveitis. This is one reason why it is important to accurately diagnose anterior uveitis cases and treat them appropriately – assuming that a 'red eye' is purely conjunctivitis, i.e. a superficial disease, can have disastrous consequences. Chest and abdominal radiography together with full haematology and biochemistry are warranted if the cause is not obvious. Many eyes require enucleation and histopathology should always be considered, both for diagnostic and prognostic indicators.

NURSING ASPECTS

Since dogs with secondary glaucoma will be in some discomfort they should be given suitable analgesics while awaiting decisions on more specific treatment. They should be made as comfortable as possible and be prevented from self-traumatizing. Both topical and systemic medication might be necessary and some dogs will require surgery.

EPIDEMIOLOGY

As with primary glaucoma, most secondary cases develop a raised intraocular pressure through problems with aqueous drainage, rather than due to overproduction. The flow of aqueous can be blocked at the pupil – posterior synechiae, where the iris is stuck on the anterior lens capsule, will cause this if extensive (total posterior synechiae will result in iris bombé, where the whole of the pupil margin is attached to the lens and a forward bowing of the iris results). A lens which is displaced anteriorly – either subluxated or totally luxated – will also result in pupil block, as will an intumescent (swollen) cataract. The sudden onset of total cataract seen in diabetic patients is the most frequently encountered intumescent cataract and can lead to secondary glaucoma, often exacerbated by lens-induced uveitis. Checking the depth of the anterior chamber can help to determine if the lens is swollen or shifted forwards, although ultrasonography can be more revealing. Pupil block can also occur with displaced vitreous – again associated with lens luxation/subluxation or following cataract extraction surgery.

The other area where aqueous flow is impeded is at the drainage angle. Forward movement of the iris will result in closure of the angle and occurs with intumescent cataracts, subluxated lenses and uveal neoplasia. Blockage of the angle with inflammatory, pigment or neoplastic cells, with red blood cells in cases of hyphaema, or with vitreous is also encountered, while both anterior uveitis and neoplasia can result in neovascularization of the anterior iris face (pre-iridal fibrovascular membrane formation) and this can also restrict aqueous drainage. Occasionally glaucoma can develop secondary to perforating injuries, where the iris moves anteriorly to plug the defect and the resultant anterior synechiae disrupt aqueous flow. However, the uveitis and sequelae of that are usually more to blame than the actual position on the iris in these cases.

CLINICAL TIPS

- Pupil size can be variable in secondary glaucoma, especially if uveitis or a uveal mass is involved
- Compare intraocular pressure between the two eyes
- Accurate diagnosis and treatment of uveitis will reduce the risk of secondary glaucoma
- Always measure intraocular pressure with uveitis cases on each recheck – secondary glaucoma can creep up unawares otherwise
- Look at the position of the lens and iris from the side to compare the depth of the anterior chamber between the two eyes

TREATMENT OPTIONS – MEDICAL

Medical management of secondary glaucoma is directed towards treatment of the underlying cause as well as the raised intraocular pressure. However, many cases are not responsive to medical management and enucleation is frequently required. Moderately raised intraocular pressures associated with primary uveitis are worth treating medically, with carbonic anhydrase inhibitors topically and both topical and systemic anti-inflammatory agents – typically steroids but sometimes NSAIDs. Prostaglandin analogues and miotic agents such as pilocarpine are both contraindicated in cases of secondary glaucoma associated with an inflammatory process. As the uveitis settles the pressure will come down, providing there is not excessive damage at the drainage angle. Frequent rechecks are necessary and life-long medication can be required.

TREATMENT OPTIONS – SURGICAL

In a blind, painful glaucomatous eye enucleation should be considered regardless of the cause. Sometimes it is not possible to determine the cause until histopathology is performed. Cases of uveal neoplasia which have

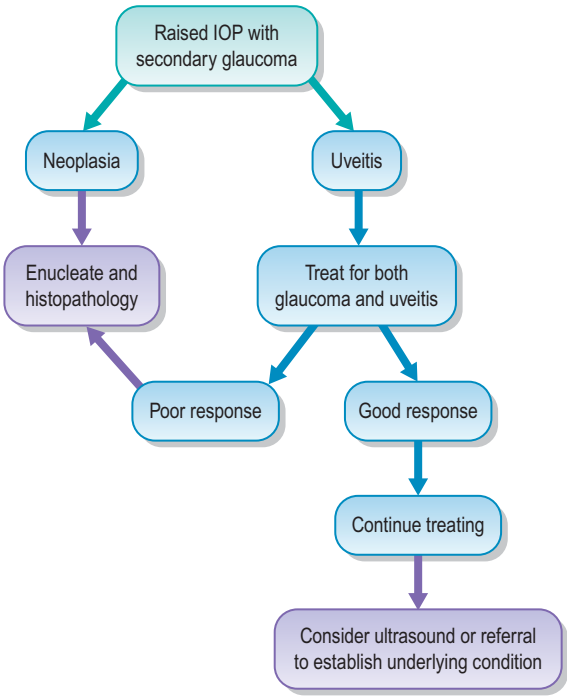


Figure 45.2 Decision-making tree – secondary canine glaucoma.

resulted in secondary glaucoma should be enucleated, as should any eye with a very high pressure which does not respond to medication. If an acute lens luxation is the reason for the raised pressure, then lendectomy may be beneficial, and early referral for this is advised (see Chapter 42). Other surgical procedures for glaucoma such as drainage implants and laser ablation of the ciliary body are usually disappointing in secondary cases.

PROGNOSIS

The prognosis for the affected eye with secondary glaucoma is unfortunately poor, and most cases do end up with the eye being removed sooner or later. Acute lens luxation with emergency lendectomy is the only real exception to this, although even here the prognosis remains guarded with respect to a return of vision. However, the prognosis for the patient as a whole is normally good. Most cases of primary intraocular neoplasia in the dog do not metastasize (although survey radiography and ultrasonography are still advised, and histopathology should always be performed). Uveitis due to trauma with secondary glaucoma similarly should be cured on enucleation.

CASE EXAMPLE 45.1

Patient details: Cairn terrier, 8 years old, female (neutered)

PRESENTATION AND HISTORY

The patient had been treated for non-responsive conjunctivitis for several months before the owners changed veterinary surgeons. The dog had received several different antibiotic agents topically plus steroid drops, none of which made much difference. The owner changed practices when she noticed that the dog started bumping into things. The eyes were both still red, but the owner was not aware of any other changes to the eyes. However, the dog did have its fur covering the eyes most of the time and the owner had not looked closely!

CLINICAL SIGNS

The dog was clinically well with only ocular abnormalities. A menace response was present in the left eye but not the right. Pupillary light reflexes were poor. Dazzle reflexes were present in both eyes. Marked episcleral congestion was present along with patches of episcleral pigment. Corneal pigment was also noted and both irises were very dark. The pupils were irregular and dilated. Pigment was present on the anterior lens capsules. The left lens was subluxated. Asteroid hyalosis was noted bilaterally (Figure 45.3). Fundus examination showed cupping of both optic discs, with some retinal vessel attenuation in the right. No ulceration was present. Intraocular pressures were 48 mmHg in the right eye and 40 mmHg in the left.

WORK-UP

The clinical examination led to the diagnosis of glaucoma secondary to pigment dispersal syndrome and no further work-up was performed. The owners had this dog's sister. They were not concerned about her eyes at



Figure 45.3 Right eye of Cairn terrier with pigment dispersal syndrome and secondary glaucoma. Note the episcleral pigment, dyscoria and asteroid hyalosis.

all but examination revealed mild pigment changes and early glaucoma in her as well.

TREATMENT

Treatment was started with brinzolamide (Azopt, Alcon) three times daily bilaterally and dexamethasone (Maxidex, Alcon) twice daily bilaterally. The dog did not appear in pain so no analgesia was given.

OUTCOME

Intraocular pressures reduced to the mid 30s in both eyes and some improvement in vision was noted in the left eye, although the right remained blind. After 3 months of treatment the dog developed a corneal ulcer in the right eye. This did not improve on topical antibiotics and intraocular pressure increased to 48 mmHg again. Enucleation of the right eye was performed 1 month later. Histopathology confirmed severe pigment proliferation and secondary glaucoma. The left eye maintained limited vision and no further increases in intraocular pressure for another 3 months, although further pigment deposition occurred. The dog continues to be monitored.

This case was misdiagnosed as conjunctivitis for several months prior to the correct diagnosis being reached. Pigment dispersal syndrome is a recognized breed-related problem and Cairn terriers are particularly susceptible. Treatment aims to reduce the inflammation associated with pigment deposition and reduce intraocular pressure, but unfortunately nothing has been discovered which will reduce the relentless pigment accumulation. Surgical treatment with drainage implants or laser ablation of the ciliary body are disappointing. However, if this dog had been correctly diagnosed in the first place the prognosis for saving vision for a much longer period would certainly have been better.

46 Feline glaucoma

INITIAL PRESENTATION

Abnormal appearance – enlarged

PRESENTING SIGNS

Cats presenting with glaucoma tend to have much more subtle signs than dogs. Owners may notice a dilated pupil for example, or a colour change and even an enlarged globe, but no sign of pain or visual disturbance. Sometimes it is the change in appearance of the eye – a colour change for example – that the owner is concerned about. Sometimes both eyes can be affected simultaneously, which again is unlike dogs. Cat owners might report that the pet is quieter than previously, but since the condition is most common in older animals, this change is often attributed to simple ageing.

CASE HISTORY

In some cases there is no previous history of ocular disease or relevant systemic disease. However, many cats do have a long history of previous uveitis which has been treated with variable success for months or even years prior to the onset of the glaucoma. A chronic, low grade uveitis is sometimes overlooked and the condition gradually progresses, causing various intraocular changes which lead to secondary glaucoma. The clinical manifestation of feline glaucoma, often characterized by the dilated pupil, alters the appearance of the eye and alerts the owner to a change in the disease status and thus the presentation is frequently late in the course of the disease. Secondary glaucoma is also a feature of feline intraocular melanoma, and in such cats the owner may have been aware of a gradual colour change – darkening of the iris – for many months or even years prior to presentation.

CLINICAL EXAMINATION

General clinical examination is frequently unremarkable. One or both eyes can be affected. The pupil is normally dilated and the direct pupillary light reflex absent or sluggish and incomplete (Figure 46.1). Moderate episcleral congestion is usually present but this can be difficult to appreciate since the globe fits tightly into the socket and there is little exposed sclera or episcleral tissue. Sometimes chemosis may be noted. Corneal oedema is not a common feature of feline glaucoma.

Globe enlargement can be present but is often subtle and needs to be differentiated from exophthalmos. Looking down on the eyes from above will help since retropulsion is minimal in cats and thus can be difficult to appreciate, while looking down from above one can visualize if the globe fits in the socket or is pushed forwards while also comparing the corneal curvature of the two eyes – this will be altered if the globe is truly enlarged. Sometimes the globe enlargement will lead to an exposure keratitis – corneal ulceration and vascularization may be present and fluorescein testing should always be undertaken.

Signs of chronic uveitis might be present, and these could include keratic precipitates, rubeosis iridis, iris lymphoid nodules, posterior synechiae and iris rests (Figure 46.2).

The lens can be cataractous and is sometimes luxated into the anterior chamber. Another dissimilarity with dogs is that the luxated lens does not tend to further exacerbate the glaucoma and develops as a result of the uveitis damaging the lens zonules, not due to primary lens instability. Lens luxation in cats is less painful than in dogs since the anterior chamber can accommodate

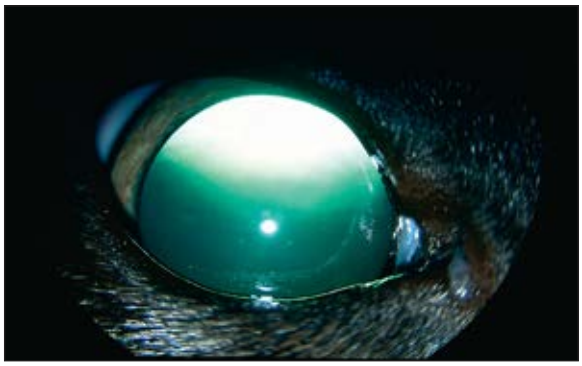


Figure 46.1 Chronic glaucoma in a cat – the widely dilated pupil is the only obvious abnormality. Intraocular pressure was 40 mmHg.

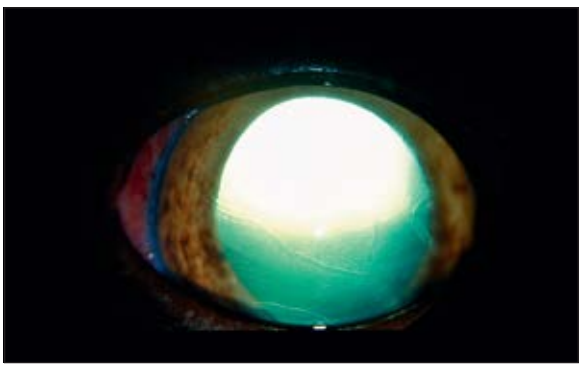


Figure 46.2 Severe chronic glaucoma secondary to uveitis in a 6-year-old Burmese. Note the episcleral congestion, corneal striae (rare in cats), dilated pupil and iridal changes. Intraocular pressure was 64 mmHg.

the dislocated lens without further exacerbating the glaucoma.

If possible, fundus examination should be performed and this will frequently reveal diffuse tapetal hyperreflectivity and blood vessel attenuation as signs of chronic retinal degeneration. The optic disc will be cupped but this can be difficult for general practitioners to appreciate since the feline disc is small and dark anyway, and the signs of feline disc cupping are much more subtle than in dogs.

Intraocular pressure measurement is essential and access to tonometry should be made available. Cats can have dramatically high intraocular pressures – in the 60s mmHg sometimes – yet show remarkably little pain and some will even maintain some useful vision despite the very high readings. Always compare the readings in the two eyes since the difference between them can help to diagnose borderline cases. For example, a cat with an intraocular pressure of 28 mmHg in one eye but 8 mmHg in the other, where both eyes have signs of uveitis, would certainly have secondary glaucoma, while a cat with readings of 26 mmHg in both eyes, and no other ocular changes, might simply have high normal pressures.

If there are no signs of previous or active uveitis, then assessing the depth of the anterior chamber can be very useful. A rare form of feline glaucoma manifests with a shallow anterior chamber following misdirection of aqueous into the vitreous gel, forcing the iris and lens more anterior. The drainage angle can be easily assessed in cats without the need for goniolenses – simply shining a pen torch across the cornea and looking at the angle between the iris and cornea will reveal the pectinate ligaments as the opening to the uveoscleral drainage angle. Goniodysgenesis is not a feature in feline glaucoma, but the angle can be narrowed if the iris is pushed forwards, and is frequently blocked by inflammatory cells or peripheral anterior synechiae following anterior uveitis.

DIFFERENTIAL DIAGNOSES

- Primary glaucoma – aqueous misdirection syndrome (rare)
- Chronic uveitis
- Uveal melanoma
- Retinal degeneration or optic atrophy resulting in a dilated pupil
- Exophthalmos

CASE WORK-UP

If the glaucoma is secondary to anterior uveitis, as is most common in cats, then the cause for the uveitis should be investigated and this is discussed in Chapter 34. If a uveal melanoma is suspected, the cat should be thoroughly assessed for signs of metastasis with blood work, radiography and ultrasonography. If there is no evidence of other intraocular disease, then a tentative diagnosis of primary glaucoma is made. It has been seen in Burmese and elderly Siamese cats on occasion, and the rare aqueous misdirection form mentioned briefly above can be seen in any cat breed. If a primary glaucoma is suspected, then referral should be considered, particularly to confirm this diagnosis and provide prognostic information for both eyes.

NURSING ASPECTS

Cats with glaucoma usually require medical therapy in the form of topical anti-inflammatory and anti-glaucoma drops. Since these sometimes require frequent administration both owner and cat compliance can be less than ideal! Nurse consultations showing owners how to medicate, and drawing up tick sheets for the owners to fill out, will assist compliance – unfortunately, however, many cats do not make long-term topical medication easy for their owners!

EPIDEMIOLOGY

As mentioned, most cases of feline glaucoma are secondary, with up to 50% of cases of chronic uveitis developing glaucoma as a complication. It can result from a combination of factors – inflammatory cells blocking the drainage angle, peripheral anterior synechiae, posterior synechiae and iris bombe, and pre-iridal fibrovascular membrane formation where new blood vessels (seen as part of the rubeosis iridis of uveitis) grow over the anterior surface of the iris and drainage angle, blocking outflow. In addition, any cataract formation can cause lens swelling, pushing the iris anteriorly and functionally closing the drainage angle. If the lens luxates into the anterior chamber, this will further compromise aqueous outflow, although to a lesser extent than in dogs, since the feline anterior chamber is larger and can accommodate the lens more easily.

Uveal melanomas in cats can also lead to glaucoma, through similar mechanisms – with iris thickening, exfoliation of neoplastic cells into the drainage angle and pre-iridal fibrovascular membrane formation all contributing (Figure 46.3). Other intraocular neoplasms such as lymphoma and ciliary body adenocarcinoma can lead to secondary glaucoma but with much less frequency than melanomas. Traumatic uveitis can also lead to secondary glaucoma – a corneal laceration can be plugged by the iris such that aqueous outflow is impeded, while posterior scleral damage, through a cat fight for example, with a tooth penetrating the sclera, can lead to a severe panuveitis, panophthalmitis and rapid glaucoma development.

Primary glaucoma is rare. It has been seen on occasion in older Siamese and Burmese cats and more recently an inherited form has been researched in a colony of North American Siamese cats, but this does not appear to be a problem in the field. The aqueous misdirection form of glaucoma is frequently bilateral, but thankfully rare. As mentioned previously, if the underlying cause for the glaucoma is not immediately apparent, referral should be considered.



Figure 46.3 Secondary glaucoma due to uveal melanoma in the left eye. Note the dark iris and dilated pupil. A small exposure ulcer is also present. Intraocular pressures were 46 mmHg in the left eye and 20 mmHg in the right. Enucleation was necessary.

CLINICAL TIPS

- Carefully look for a primary problem – normally chronic uveitis – to explain the glaucoma
- Look from above the cat to differentiate globe enlargement (hydrophthalmos) from exophthalmos
- Measure intraocular pressures and compare the two eyes!
- Remember the symptoms are less obvious in cats than in dogs!

ANATOMY AND PHYSIOLOGY REFRESHER: DIFFERENCES FROM DOGS

- Cats have a deep anterior chamber and the drainage angle can be easily visualized without the need for a goniolens.
- Cats can tolerate high intraocular pressures with less optic nerve and retinal damage than dogs and vision can be preserved even with elevated pressures.
- Globe enlargement occurs more readily in cats than in dogs.

TREATMENT OPTIONS – MEDICAL

As with all secondary diseases the initial treatment should be directed at the underlying cause. Thus many cats will have chronic uveitis, and investigation as to the cause for this should be considered (but is frequently unrewarding in chronic cases). Topical and systemic anti-inflammatory agents, usually corticosteroids, are advocated. Systemic prednisolone can be used along with topical dexamethasone or prednisolone acetate. The long-term use of topical steroids can lead to elevated pressures in cats (as well as in humans) and so cats on such treatment should have regular tonometry. Since the uveitis is probably more likely to result in glaucoma than the chronic use of topical steroids, this low risk is acceptable, but nonetheless present. Mydriatics are normally used in cases of uveitis but should be avoided if secondary glaucoma is present as they can further compromise aqueous drainage.

Specific anti-glaucoma medications are less reliable than in dogs. Since cats rarely suffer from the severe, acute form commonly encountered in dogs, especially those with goniodysgenesis, emergency treatment with systemic hyperosmotic agents is rarely necessary. Topical carbonic anhydrase inhibitors (e.g. dorzolamide and brinzolamide) are effective at lowering pressures and are the first choice for lowering intraocular pressure. Systemic anhydrase inhibitors are poorly tolerated in cats and should be avoided in favour of topical agents. Topical beta-blocking drugs such as timolol seem more effective in cats than in dogs, and products are available where they are combined with topical carbonic anhydrase inhibitors. This makes owner and cat compliance easier, since frequent and long-term topical medication can be difficult to maintain in cats. Topical miotics (e.g. pilocarpine) are of little value and since they can worsen any uveitis they should be avoided. Topical prostaglandin analogues such as latanoprost seem ineffective in most cases of feline glaucoma.

Ocular lubricants such as carbomer gel should also be considered, particularly if the globe is enlarged and an exposure keratitis is developing. Overt corneal ulceration should be treated with topical antibiotics and lubricants but it can be difficult to achieve healing in view of the multiple pathological processes compromising normal ocular function.

A summary of medical options for treating feline glaucoma is given in Table 46.1.

TREATMENT OPTIONS – SURGICAL

The use of drainage implants can be considered in cats, as can methods to lower aqueous production such as laser cyclophotoablation. However, they are rarely indicated due to the secondary nature of most cases of feline glaucoma. As such, enucleation does remain the most frequently performed surgical treatment for glaucoma. Clearly if a neoplastic process is present, enucleation with histopathology should be performed, with

Table 46.1 Medical options for feline glaucoma

Option	Prescription	Regimen
Anti-inflammatory	Prednisolone acetate Dexamethasone Ketorolac if corneal damage Systemic prednisolone if severe inflammation Consider systemic NSAID, e.g. meloxicam	Two to four times daily 1 mg/kg/day
Anti-glaucoma*	Dorzolamide Brinzolamide Timolol (with above)	Three times daily Twice daily Two to three times daily
Other	Carbomer 940 gel or other lubricant	Two to four times daily

*Avoid systemic carbonic anhydrase inhibitors.

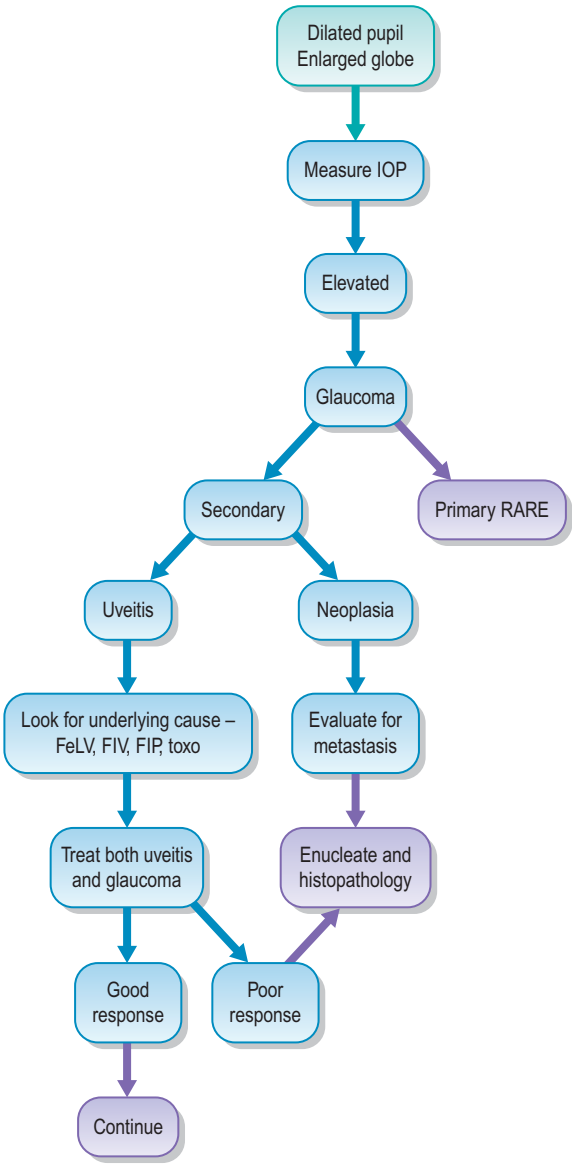


Figure 46.4 Decision-making tree – feline glaucoma.

careful assessment for evidence of metastasis prior to surgery. Histopathology should be performed on all enucleated globes and most will return with a lymphocytic–plasmacytic uveitis as the underlying pathological process. Any cat with an enlarged globe, secondary exposure keratitis and blindness can be considered for enucleation, although since the underlying uveitis is frequently bilateral the second eye requires close monitoring – early intervention with both anti-inflammatory and anti-glaucoma medication can reduce the risk of the second eye going the same way.

PROGNOSIS

The prognosis for cases of feline glaucoma is always guarded. Although vision can be maintained despite high pressures, many cats are not amenable to frequent topical medication and will hide from the owners or stay outside, making it difficult for them to follow treatment regimens. Owners must be very dedicated, and the cats compliant, to achieve reasonable control. Life-long medication is also necessary. Many cats do not show overt signs of pain, but when enucleation has been performed the owners report that the cat is much more lively and playful again, indicating that it must have suffered ongoing discomfort from the glaucomatous globe.

Glaucoma in itself is not a life-threatening condition, but since in cats it can be bilateral, and many owners are not willing to have a bilateral enucleation (even though the cat is blind anyway), some patients are euthanized as a result of the condition.

CASE EXAMPLE 46.1

Patient details: Domestic short haired cat, 8 years old, male (neutered)

PRESENTATION AND HISTORY

The cat had been diagnosed with idiopathic bilateral uveitis 12 months previously. This had initially been treated with systemic and topical steroids plus topical atropine and a good improvement had occurred. For the past 3 months the cat had received just once daily prednisolone acetate bilaterally. The owner re-presented the cat because the left eye had changed in appearance with a dilated pupil. Vision seemed unaffected and the cat was otherwise well.

CLINICAL SIGNS

The right eye had a few tiny keratic precipitates but no active inflammation was noted. The left eye had a dilated pupil which was unresponsive to light, although a menace response and good dazzle reflex were present. Rubeosis iridis was noted, along with multiple keratic precipitates (Figure 46.5). No ulceration was present. Intraocular pressures were 32 mmHg in the left eye and 14 mmHg in the right. No abnormalities were noted on fundus examination.



Figure 46.5 Dilated left pupil in a domestic short haired cat with glaucoma secondary to chronic idiopathic uveitis. Intraocular pressure was 32 mmHg.

WORK-UP

No further work-up was undertaken. The glaucoma was presumed to be secondary to the chronic idiopathic uveitis.

TREATMENT

Prednisolone acetate (Pred Forte, Alcon) was increased to four times daily in this eye. Topical dorzolamide and timolol drops (Cosopt, MSD) were started three times daily in the left eye only.

OUTCOME

A week later the pupil remained dilated but intraocular pressure had reduced to 25 mmHg and there was less active inflammation. The prednisolone acetate was reduced to twice daily. After another month the pupil remained dilated, and posterior synechiae were assumed to have formed. However, the eye was comfortable, with no active inflammation, and intraocular pressure remained in the low 20s. The anti-glaucoma drops were stopped but 2 weeks later the glaucoma had returned (intraocular pressure was 30 mmHg again). Long-term maintenance with twice daily dorzolamide and timolol plus once daily prednisolone acetate kept the eye comfortable and visual.

This case illustrates that chronic glaucoma can develop following uveitis, even if the latter appears to have been fairly well controlled. The owner noted the change in appearance of the eye and by treating both the inflammation and the raised pressure relatively early and aggressively, the secondary glaucoma could be controlled. However, long-term damage had been done and it was not possible to reduce the treatment levels further. Luckily both owner and cat were compliant with this regimen!

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SECTION 8

FUNDUS

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47 Fundus – introduction

The fundus is the part of the posterior segment of the eye which is examined with an ophthalmoscope. It consists of the retina and choroid together with the optic disc (optic nerve head). In some animals, especially those with little pigment (e.g. merle collies), the sclera can also be seen.

The *retina* is a very complicated structure and consists of 10 layers, illustrated in Figure 47.1. Nine of these layers make up the neurosensory retina while the 10th and outermost layer is the retinal pigment epithelium. More than one layer is usually involved in any pathological process but involvement of the photoreceptor layer, i.e. the rods and cones, is frequently what results in impaired vision or even blindness.

The *choroid* is the posterior portion of the uveal tract forming the middle layer of the back of the eye, with the sclera as the outer fibrous tunic. The choroid consists of many blood vessels, pigment and, in some species, the tapetum lucidum. The choriocapillaris is the layer of fine vessels closest to the retinal pigment epithelium and provides nutrition to the retina. Examination of the fundus is by indirect and direct ophthalmoscopy, providing the visual axis is clear. However, in the presence of opacification, such as cataract or hyphaema, other methods of investigation such as ultrasonography and electroretinography are required. Behavioural testing is also essential – in both bright and dim light, with obstacle courses being particularly useful.

The appearance of the normal fundus varies tremendously in dogs, less so in cats (see Figures 47.2–47.5). In most animals the tapetum is present in the dorsal half of the fundus (the reflective layer within the choroid underlying the retina). The non-tapetal fundus makes up the rest of the area and is usually pigmented, due to the presence of melanin within the retinal pigment epithelium which is the outermost layer of the retina. Thus, different colours to the tapetum, absence of the tapetum, different degrees of pigmentation in the non-tapetal fundus, various shapes to the optic disc and a range of vascular patterns are all encountered. The optic disc is usually myelinated in dogs, resulting in a whitish fluffy appearance, whereas in cats it is smaller and darker due to a normal lack of myelin.

The retinal vessels in dogs and cats are holangiotic in distribution. This means that there are three to four pairs of arterioles and venules radiating from the optic disc and then branching. The arterioles and venules are more difficult to distinguish from each other in cats, where they are of similar size and calibre. In dogs the venules are wider, straighter and with darker blood within.

The fundus is not fully mature at birth, nor when the puppy or kitten opens its eyes. It is not until 12–16 weeks that the retina takes on its adult colouration – it is frequently blue–purple in colour in young animals and the blood vessels look quite large compared to adults (Figure 47.6). This means that one must look at lots of eyes to be able to appreciate the wide array of normal appearance before one can appreciate what is abnormal! Ophthalmologists commonly have animals referred to them with suspected retinal haemorrhages for example, only to find that the animal is totally normal – just lacking pigment in the fundus such that the choroidal vasculature is visible (see Figures 47.3 and 47.5). Many general practitioners find fundus examination and interpretation the most difficult aspect of veterinary ophthalmology. Only by examining lots of normal animals and becoming confident at the examination will the abnormalities become easier to detect and interpret. If one only looks at the fundus if the owner is worried that vision is not normal, it can be difficult to understand whether what you can see is normal or not! Thus the importance of practice, practice, practice cannot be overemphasized when discussing retinal problems.

Before we consider specific diseases affecting the fundus, it is important to understand the basic principles of how the area is affected by pathological processes.

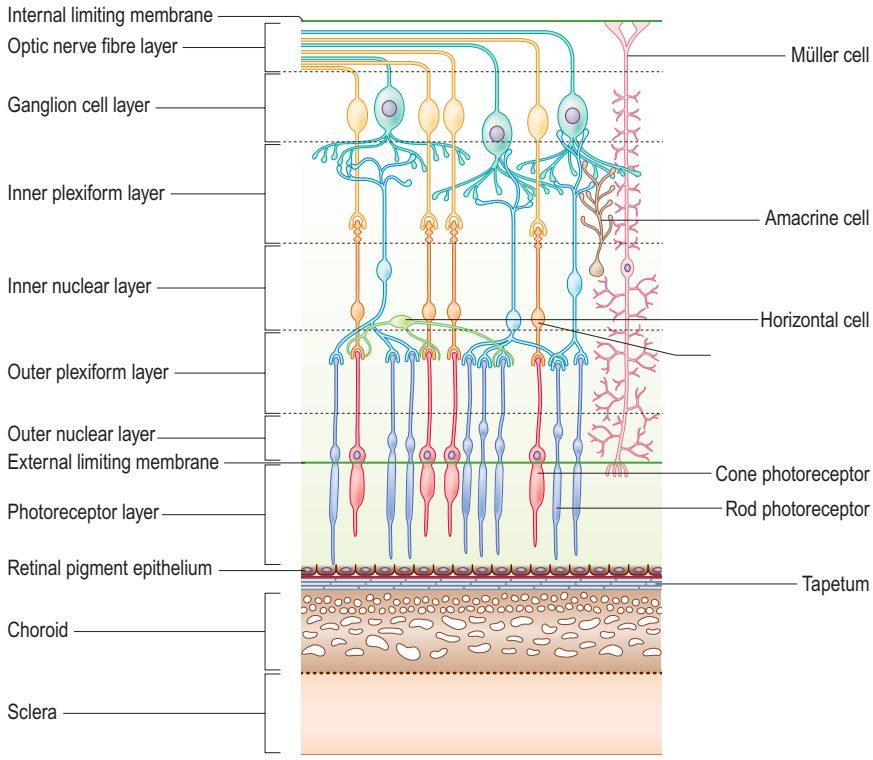


Figure 47.1 The layers of the retina.

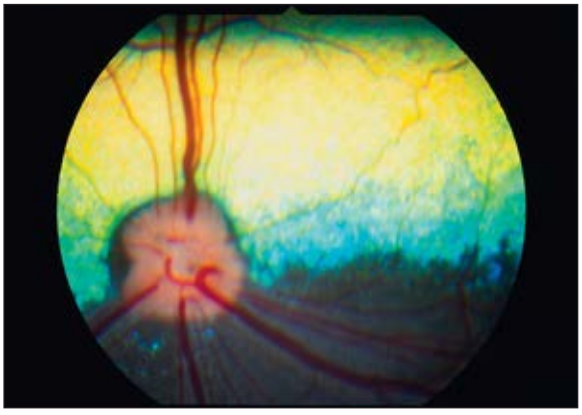


Figure 47.2 Normal fundus in a Labrador.

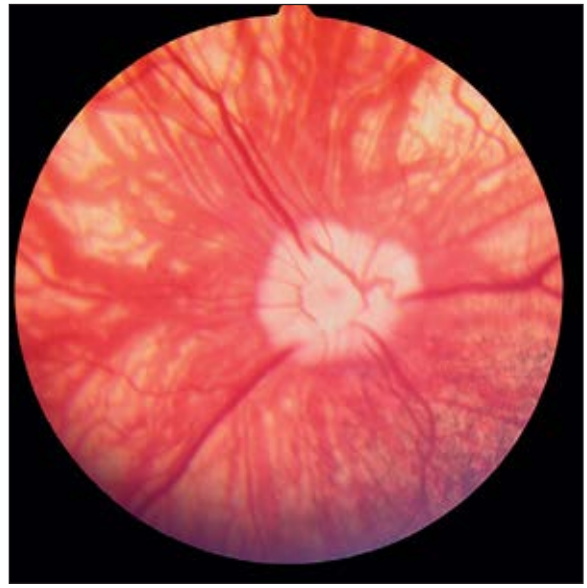


Figure 47.3 Normal fundus in a merle collie.

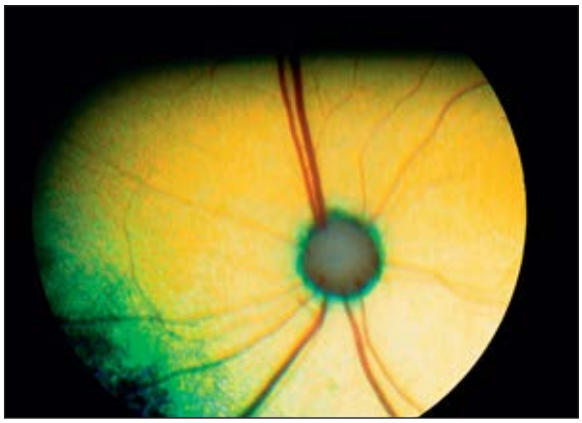


Figure 47.4 Normal fundus in a domestic short haired cat.

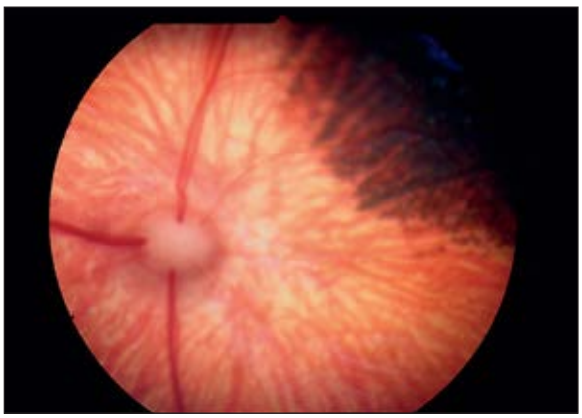


Figure 47.5 Normal fundus in a lilac-point Birman cat.

Abnormalities of the fundus are manifested in a limited number of ways. These are changes in reflectivity of the tapetum, differences of pigmentation, altered vascularization and haemorrhage, and are summarized in Figure 47.7.

- The *tapetal reflex* is increased (hyper-reflectivity) when the retina is thinned – more of the underlying tapetum is seen through the thinner retina. This usually signifies degeneration or atrophy of the retina. The tapetal reflex is reduced by thickening of the retina – for example, due to exudates within it, or retinal detachments. This concept is illustrated in Figure 47.8.
- *Pigmentation* within the retina alters with disease – both decreased and increased amounts occur. Gener-

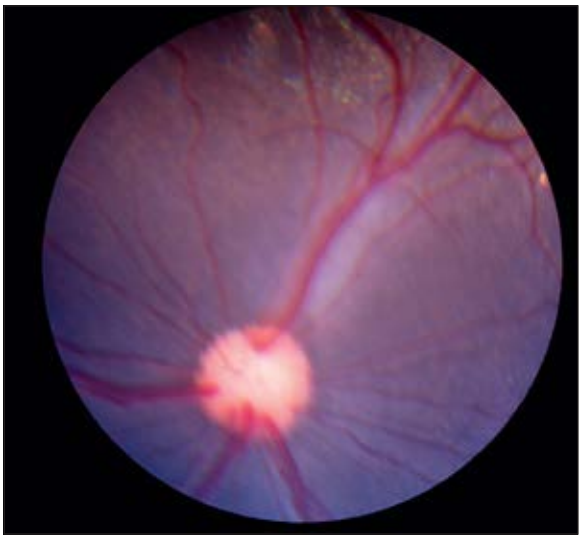


Figure 47.6 Normal fundus in a 10-week-old collie – the purple colouration will be replaced by the adult appearance in the coming weeks.

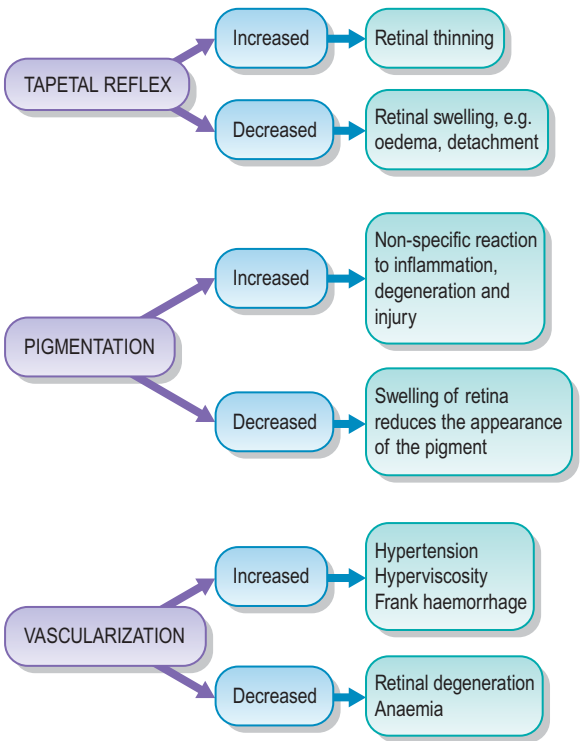


Figure 47.7 Manifestations of pathological processes on the appearance of the fundus.

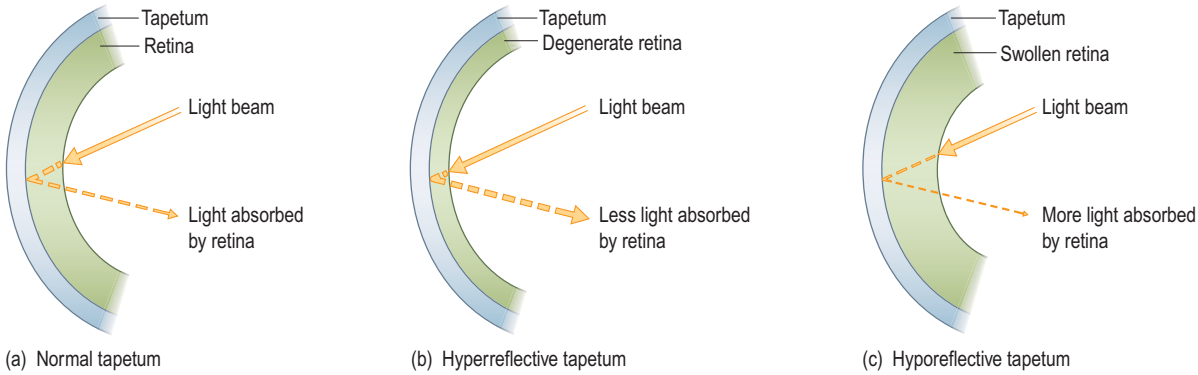


Figure 47.8 Effect of retinal thickness on the tapetal reflex.

ally decreased pigmentation is associated with thickening of the retina (e.g. oedema) such that the retinal pigment epithelium beneath it is less visible and hence paler. Increased pigment in the retinal pigment epithelium is more common, and is a non-specific reaction to disease – inflammation, degeneration and injury can all cause increased pigment both in the non-tapetal and within the tapetal fundus.

- Changes in *vascularization* can include attenuation of the retinal blood vessels (e.g. with retinal degeneration or anaemia), increased volume within the vessels making them appear thicker and more tortuous (e.g. with systemic hypertension or hyperviscosity syndromes) and blurring of the outline of the blood vessels due to inflammation and leakage from them (seen in active chorioretinitis).
- Retinal *haemorrhage* occurs with several diseases and the appearance of the bleeding will vary according to which layer of the retina or choroid is affected. For example, haemorrhages in the nerve fibre layer are typically flame-shaped, those in the choroid are usually puddle-like and dark in colour, while pre-retinal (i.e. between the nerve fibre layer/internal limiting membrane and the vitreous) tend to be described as 'keel' shaped as the red blood cells sink slightly.

The *optic disc* is frequently also involved with pathological processes in this area. It can become swollen and raised above its normal plane – with papilloedema or optic neuritis for example – or can shrink in cases of retinal degeneration or glaucoma. Haemorrhages can occur both on and around the disc (peripapillary). Binocular indirect ophthalmoscopy provides a good way of appreciating the three-dimensional structure of the disc, but careful examination with a direct ophthalmoscope is also rewarding. By paying close attention to how the blood vessels cross the edge of the disc, and by altering the dioptre setting by +2D and –2D, it is possible to tell if the disc is raised, sunken or just in a normal position.

The exact changes noted on fundus examination might in themselves be diagnostic, but frequently will be fairly non-specific such that further investigations are necessary to reach a definitive diagnosis.

48 Generalized progressive retinal atrophy

INITIAL PRESENTATION

Blind, abnormal appearance – cloudy

PRESENTING SIGNS

The owner will present the patient with generalized progressive retinal atrophy (gPRA) because they have noticed that vision is reduced and perhaps the eyes look different. Thus they will have become aware that their pet is reluctant to go out in unfamiliar places, chases the ball less and occasionally bumps into things at home. Normally owners will realize, with hindsight, that things have not been normal for several months. They may report that the eyes look 'cloudy' or 'glassy'. Middle-aged dogs are most commonly affected – certain pedigree breeds such as most spaniel types (especially cocker spaniels) and Labrador breeds (particularly the Labrador retriever) and miniature and toy poodles are most frequently seen. Cats are sometimes affected, most notably middle-aged Siamese.

CASE HISTORY

Some owners feel that the pet suddenly went blind. This is most common if they have been to stay in a different house and noticed that the pet bumps into things, or if it has been in kennels and when brought back home does not seem to be able to see properly. However, when questioned in more detail the owners might be able to recall certain instances where the pet did not react as they would normally do – suddenly barking at a dark rubbish sack on the street, not reacting quickly to another dog or bumping into an open cupboard door in the kitchen for example. Vision is frequently worse at night, but the owners might not have noticed this. Careful questioning – for example, 'Does he still like going for walks when it is dark in the evening?' or 'Will he go out in the garden at night quite happily?' – might elicit answers such as 'I have to keep him on the lead at night otherwise he is frightened/gets lost/sits down and won't move' or 'He is happy if I put the outside light on, but won't go out into the total darkness'. Animals are often presented in the autumn when the clocks go back, and it is darker both in the morning and the evening.

Owners might report that the eyes look different – glazed, glassy, shiny and cloudy are all possible descriptions. Observant owners might have noticed that the pupils are quite dilated in normal light levels. Many notice the clouding, and attribute this to cataract formation which is what they blame for the visual problems. However, it is very important to ascertain whether the eyes looked cloudy before or after they started to notice visual problems.

CLINICAL EXAMINATION

General clinical examination is usually unremarkable, but the ophthalmic examination is not! Dogs might be nervous in the waiting room and can bump into objects on the way into the consulting room. Alternatively, they might walk with their head down, nose to the ground, and have a slightly high stepping gait. Cats will be reluctant to come out of their basket and might put their paws out to feel the edges of the table. Menace responses are likely to be reduced, and in most cases pupillary light reflexes are slow and incomplete. Thus the pupils are likely to be dilated in room light and even with a bright light source full and rapid constriction is unlikely to occur. Cats will retain a better pupillary light reflex than

dogs. Both the menace response and pupillary light reflexes should be checked in bright (photopic) and dim (scotopic) light. Reactions are likely to be markedly reduced in the latter. In addition, the dazzle reflex is frequently reduced – the patient probably will still react to the bright light, but less quickly and with less intensity than a normal animal.

The adnexal examination is usually normal, as is the cornea. Dogs might have cataracts – ranging from incipient cortical opacities to total mature cataracts, depending on how advanced the underlying retinal disease is. Providing any cataract is not too advanced, it should be possible to examine the fundus – this will be easier with indirect ophthalmoscopy if some cataract formation has occurred. Initial distant direct ophthalmoscopy will reveal dilated pupils and an increased tapetal reflex ('eye shine'). This tapetal hyper-reflectivity can be so bright as to almost dazzle the observer when attempting close direct ophthalmoscopy, and reducing the intensity of the ophthalmoscope light will make examination easier. The increased reflectivity is frequently quite uniform over the tapetal area, but in early cases it can be almost 'granular' in distribution.

The retinal blood vessels will be narrowed and the optic disc can be small and pale (Figure 48.1). The non-tapetal fundus should not be forgotten, and the normal even pigment of the retinal pigment epithelium might be broken up into a patchy, 'pavementing' pattern. The changes in the retina are bilateral and symmetrical.

In addition to the ophthalmic examination, an obstacle test is very useful. This can be set up in the waiting room or similar and should consist of a selection of solid (e.g. cardboard boxes) and open (e.g. chairs) obstacles. The pet is held at one corner of the room while the owner calls them from the other. The animal is observed first in normal lighting, when they will often negotiate the room well, and then in dim light, when they are probably much more hesitant, and likely to collide with things. The obstacles should be moved around between tests. For cats, a couple of objects can be placed between the cat and the safety of its basket, and its ability to find its way is similarly observed in differing light intensities.

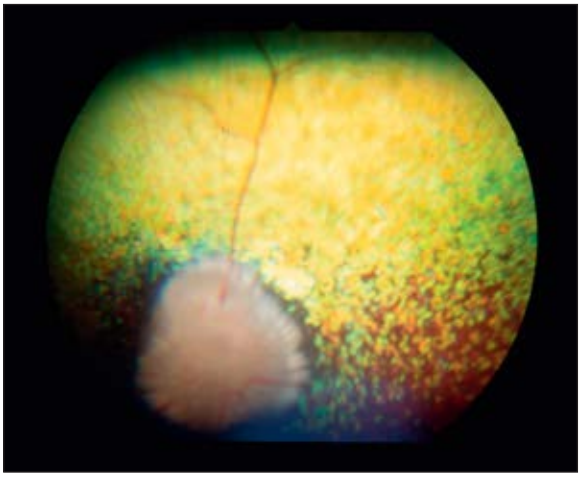


Figure 48.1 Tapetal hyper-reflectivity and blood vessel attenuation typical of generalized progressive retinal atrophy in a 10-year-old miniature poodle.

DIFFERENTIAL DIAGNOSES

- Retinal detachment
- Hypertensive retinopathy
- Severe chorioretinitis
- Retinal pigment epithelial dystrophy (RPED)
- Sudden acquired retinal degeneration (SARD)
- Toxic retinopathy (e.g. enrofloxacin in cats)
- Primary cataract
- Chronic glaucoma
- Chronic keratitis
- Optic neuritis
- Central blindness

CASE WORK-UP

The presentation, history and ophthalmic examination are frequently sufficient to confirm a diagnosis of generalized progressive retinal atrophy (gPRA). However, if cataract formation is marked, it might not be possible to properly evaluate the fundus, even with mydriasis and indirect ophthalmoscopy. These patients benefit from electroretinography and referral is necessary for this. The procedure is usually performed under sedation, and a bright light source is shone into the eyes, while electrodes pick up the retinal response to the light. Recordings can be made at different light intensities, which will allow rod and cone function to be differentiated. Patients with gPRA will have reduced rod responses initially but the condition does progress to involve the cones as well, and all wave amplitudes will be reduced in such cases.

DNA tests are also available in some canine breeds to confirm the diagnosis and as a screening test for potential breeding stock. Blood or saliva samples can be used and results will indicate affected dogs, carriers and clear, unaffected animals. DNA tests are becoming available in

more and more breeds and an up-to-date list of which breeds can be tested is available at www.optigen.com. In addition, both the Animal Health Trust and Cambridge University Veterinary School provide DNA tests for gPRA in some breeds.

NURSING ASPECTS

Since there is no treatment for gPRA the nursing is limited! However, nurses can play an important role in client education. Most owners are upset to learn that their pet is either totally blind or will soon become so. They need reassurance that the pet will adapt very well and retain a good quality of life. Their other senses of smell and hearing will take over, and in familiar environments they will behave normally. Dogs and cats are creatures of habit and this assists with their behavioural changes. Dogs can still exercise off the lead, providing the environment is safe, and playing with a sighted dog will encourage exercise. Cats can go out if the garden is enclosed, but many decide to become indoor cats of their own accord. Nurses can take time to explain to owners that teaching the pet new commands – stop, step, danger and so on – and to consider toys with bells or squeakers so that they can follow the sound will further enhance their quality of life.

Table 48.1 Canine breeds with generalized progressive retinal atrophy in the UK

Breed	Age at which ophthalmoscopic signs become visible
Australian cattle dog	>6 years (quite variable)
Collie (rough)	4 months
Dachshund (miniature long haired)	5 months
Finnish Lapphund	3 years (approximately)
Glen of Imaal terrier	3–5 years
Irish setter	4 months
Irish Wolfhound	9 months (sometimes earlier)
Lhasa Apso	3 years +
Miniature schnauzer	12 months
Norwegian elkhound	12 months
Poodle (miniature and toy)	3 years
Retriever (Chesapeake Bay)	8 months early onset, 4 years later onset
Retriever (Labrador)	4 years
Retriever (Nova Scotia duck tolling)	3 years
Retriever (Golden)	3 years
Spaniel (American cocker)	3 years
Spaniel (cocker)	3 years
Spaniel (English springer)	12 months
Tibetan spaniel	12 months
Tibetan terrier	12 months
Welsh corgi (Cardigan)	3 months

EPIDEMIOLOGY

Generalized progressive retinal atrophy (gPRA) is an inherited disease in several breeds of dog (Table 48.1). It is presumed to be so in the Siamese, and has been shown to be so in the Abyssinian, although in the latter the condition is rarely seen clinically. A similar panretinal degeneration is occasionally seen in younger domestic short haired cats but the actual underlying cause for this has not been identified and we cannot presume that it is a true inherited progressive retinal atrophy in these cases.

Generalized PRA is not a single disease, but a group of different conditions which are breed specific. Thus some types of gPRA are due to retinal photoreceptor dysplasia, where the rods (and in some cases also cones) fail to develop normally and rapidly die away. Normally this manifests in younger animals – at 6–12 months in the miniature long haired dachshund for example. Rod–cone degeneration is more common, and affects older animals – typically from 5 years upwards, although ophthalmoscopic signs can be detected from 3 years – and electroretinography will pick up abnormalities in even younger animals. The clinical and ophthalmoscopic appearance is the same regardless of the underlying photoreceptor abnormality and gene defect. The latter will vary between breeds, and is usually an autosomal recessive, although an X-linked form of gPRA is encountered in the Siberian husky in the USA. The incidence of the condition varies between breeds, and some types are more common in the UK than in Europe or the USA and vice versa.

The retina will degenerate at different rates in different breeds, and even within the same breed, and the same litter, the rates of progression can vary. This makes it more difficult to predict how long it will take for an individual to progress to total blindness. As the retina degenerates it releases abnormal substances which trigger cataract formation. This normally starts in the posterior cortex and progresses to involve the entire lens.

It is frequently the cataract which is blamed for the visual loss, and since many of the breeds which suffer from gPRA also suffer from inherited cataracts, this further complicates the picture. Careful history taking and examination, along with electroretinography (especially if cataract surgery is being considered), are very important. Cats rarely develop cataracts secondary to gPRA.

Since the disease is inherited, the incidence can be reduced by selecting non-affected animals for breeding. Screening programmes such as the British Veterinary Association/Kennel Club/International Sheep Dog Society (BVA/KC/ISDS) eye testing scheme recommends annual testing of affected breeds. However, since the condition frequently does not manifest until the animals have already been used for breeding, this can make control difficult. Thus the introduction of DNA testing is extremely useful for breeders and now many breed clubs recommend that their members have their dog's status checked. This also allows a wider gene pool to be used, since the DNA tests will differentiate between affected,

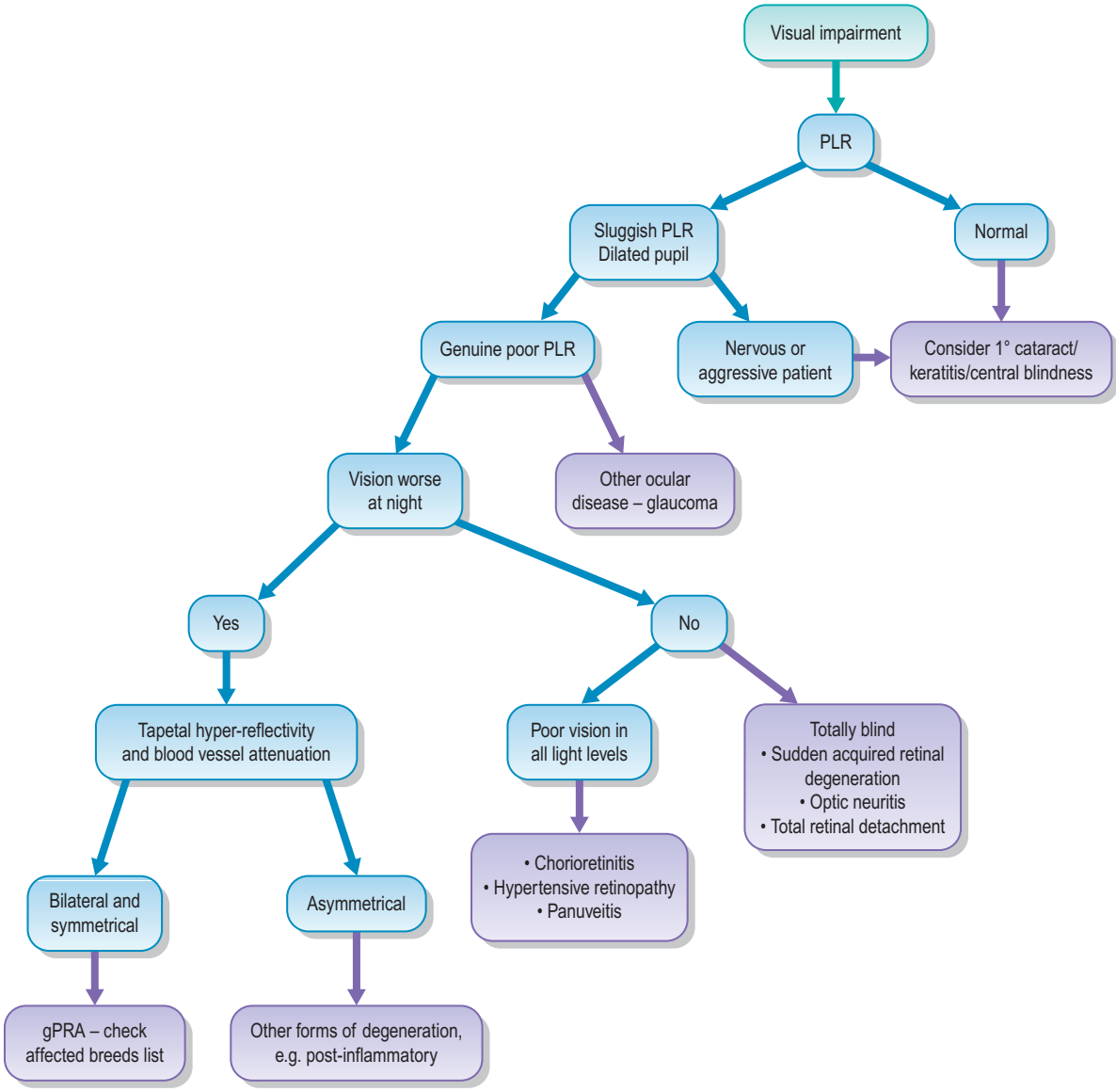


Figure 48.2 Decision-making tree – progressive retinal atrophy.

carrier and non-affected dogs, such that a carrier can be safely mated to a clear dog, knowing that no offspring will be affected, and that eventually the incidence of the abnormal gene will be diminished.

Generalized PRA is a different condition from central PRA, more correctly termed retinal pigment epithelial dystrophy (RPED). Some breeds are affected by both diseases. Thankfully RPED is not particularly common. It is usually associated with an underlying vitamin E problem (poor diet or abnormal absorption or metabolism) which causes abnormalities in the retinal pigment epithelium leading to visual impairment. For further details of this condition, the reader is referred to standard ophthalmic textbooks.

CLINICAL TIPS

- Carefully question the owner's history:
  - With hindsight, what to them was a sudden onset blindness that might turn out to be more gradual?
  - Did the eyes become cloudy before or after they noticed visual problems?
- Pupillary light reflexes:
  - Not always reliable
  - Remain good in cats despite advanced retinal degeneration
  - Should be normal if the cataract is primary
- Check the breeds that suffer from gPRA
- Indirect ophthalmoscopy will allow better fundus visualization if any cataract is present
- Generalized PRA is bilaterally symmetrical

If cataract prevents a good fundus examination, referral should be considered – an ophthalmologist might be able to interpret any lesions more easily and will be able to perform electroretinography if necessary.

TREATMENT OPTIONS

There is no treatment for gPRA. In general, animals with cataracts secondary to gPRA do not benefit from cataract extraction since the underlying retinal degeneration is contributing to the impaired vision.

PROGNOSIS

The prognosis for vision is hopeless – cases of gPRA are progressive as the name implies, and all affected animals eventually go totally blind. However, in some breeds (e.g. cocker spaniels), this can take several years after initial diagnosis. Quality of life, however, should remain good. Since the condition is slowly progressive it gives the affected animal plenty of time to adjust to the change in vision and providing their home environment is not changed too much, they lead full, near-normal lives. As they develop secondary cataracts, dogs should be checked for signs of lens-induced uveitis which, if left untreated, can sometimes progress to secondary glaucoma or lens luxation. Since these affected eyes are blind anyway, enucleation might be required, but this is certainly only necessary in a small number of PRA-affected dogs.

CASE EXAMPLE 48.1

Patient details: Cocker spaniel, 8 years old, female (neutered)

PRESENTATION AND HISTORY

The owner presented the dog because she had suddenly started bumping into things when they went to stay with friends for the weekend. The owner thought she had suddenly gone blind, and looked more closely at the eyes, which were reportedly 'glassy'. No previous systemic or ocular problems were reported. However, on closer questioning, it transpired that the dog had been more reluctant to go out for walks at night for a couple of months, which the owner attributed to the cold, wet weather rather than to any visual problem.

CLINICAL SIGNS

The dog was hesitant in the unfamiliar surroundings of the surgery. Both eyes looked shiny from a distance.

Menace responses were present in good light but when the lights were dimmed these were absent. Pupils were widely dilated and failed to constrict more than 1–2 mm, even with the bright light source of a Finhoff transilluminator. Cortical cataract formation was noted with many cortical vacuoles present (Figure 48.3). On distant direct ophthalmoscopy a bright tapetal reflex was noted and on close examination this was confirmed with marked hyper-reflectivity. Blood vessel attenuation was bilaterally symmetrical and some increased pigment was noted close to the tapetal–non-tapetal junction.

WORK-UP

Clinical examination was sufficient to diagnose gPRA with secondary cataract formation. The only slightly unusual feature was the hyperpigmentation close to the tapetal–non-tapetal junction which was simply attributed to the retinal degeneration. The owner was offered referral for electroretinogram but since this would not alter the prognosis it was deemed unnecessary. Later discussions by the owner with the breeder revealed that the dog's dam and a half sister had both gone blind at similar ages.

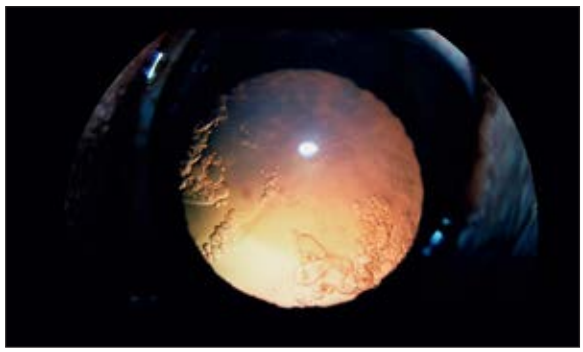


Figure 48.3 Eight-year-old cocker spaniel with early cortical cataract. Note the dilated pupil and 'eye shine'.

TREATMENT

No treatment is possible. Thankfully this dog had not had puppies and the breeder had not had any more litters from the dam.

OUTCOME

The dog adjusted well – clearly she had been losing her sight for several months, and once back in her familiar environment she was very happy. The cataracts progressed over the following 10 months and a year later total mature cataracts were present. The owner acquired another dog, a rescue crossbred, shortly after the diagnosis while the cocker still retained some day vision, and the two dogs bonded and continued to play well together even as total blindness ensued.

This case highlights how a change in circumstance can make a chronic condition appear to develop suddenly. The owner was now aware of the importance of eye testing in pedigree dogs and would not buy another pedigree unless the breeders undertook all health screens advised. This dog retained a good quality of life, but clearly it is preferable to avoid producing animals that will inevitably go blind.

49 Retinal detachment

INITIAL PRESENTATION

Blind

PRESENTING SIGNS

Owners will present the dog or cat because of sudden onset blindness affecting one or, more commonly, both eyes, since many do not notice if their pet is blind in one eye. Adult dogs and cats are normally affected but occasionally young dogs can be presented. The appearance of the eye is sometimes different – cloudy or red for example – and the patient might be depressed or confused. Increased thirst and frequent urination, together with weight loss, might be reported in some animals.

CASE HISTORY

The history is usually brief since the presentation is acute. However, some animals might have a history of recent illness – renal or cardiac disease for example – or might have travelled abroad and thus be susceptible to exotic infections.

CLINICAL EXAMINATION

The animal will be blind in one or both eyes if the detachment is widespread, but if partial in one eye this may be asymptomatic. The affected pupil will be dilated and non- or poorly responsive to constriction in bright light. Thus the patient might have bilateral mydriasis. If unilateral, the normal pupil will constrict on illumination, whereas the abnormal eye will remain dilated on direct illumination but its pupil will constrict if the light is shone in the normal eye. This indirect or consensual light reflex is an indicator of afferent lesions and is termed a positive swinging flashlight test.

Most eyes will be 'quiet' with no pain or ocular redness. Some might have cataract formation which could preclude fundus examination – indeed the owner might believe that the patient is blind due to the cataract alone. The eye might also look opaque due to the retinal detachment itself – if a total bullous or rhegmatogenous detachment is present this can be visualized as a grey 'veil' floating immediately behind the lens (Figure 49.1). It can be seen on direct illumination with a pen torch without the need for an ophthalmoscope in many cases. If the retina has torn from its dorsal attachments at the periphery behind the lens, it will fall forward over the optic disc, obscuring it, while the tapetum will be more exposed and extremely hyper-reflective above the retinal detachment (Figure 49.2). There will be a complete absence of retinal vessels dorsal to the disc but they may be visualized out of focus in the grey detached ventral retina.

Less severe detachments will require ophthalmoscopy and will manifest as out-of-focus areas of the retina and a reduction in reflectivity due to increased thickness of the retina as it becomes oedematous and fluid accumulates between the retina and tapetum (Figure 49.3). Perivascular cuffing (exudates around the blood vessels) might be noticeable along with retinal, and sometimes vitreal, haemorrhage as the detached vessels are stretched and torn. If the patient is blind in just one eye with a total detachment, the other fundus should be carefully evaluated for early signs of problems – for example, collie eye anomaly, retinal dysplasia, chorioretinitis, vascular anomalies and so on could be present.



Figure 49.1 Total bullous retinal detachment in a hypertensive cat. The retina can be seen floating behind the dilated pupil like a veil. Exudative retinal detachment.

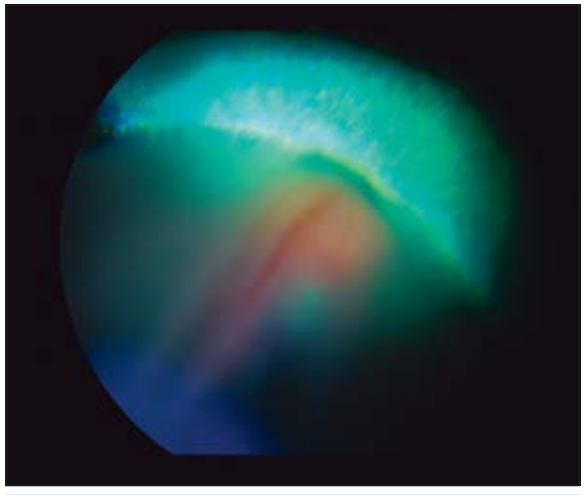


Figure 49.2 Rhegmatogenous retinal detachment of unknown aetiology in a Manchester terrier. The grey retina can be seen draped over the optic disc, while the tapetum above is highly reflective with no blood vessels over it.

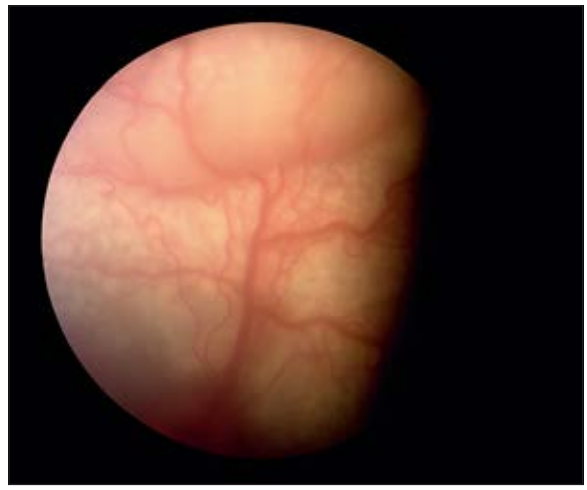


Figure 49.3 Dorsal retinal detachment in a pointer with hyperviscosity syndrome – note the retina bulging forwards dorsally, the out-of-focus retina (due to retinal oedema), the perivascular cuffing and engorged, tortuous vessels.

DIFFERENTIAL DIAGNOSES

- Congenital lesions – collie eye anomaly and retinal dysplasia
- Hypertensive retinopathy
- Chorioretinitis with inflammatory exudates (see Table 49.1 for differential list)
- Hyperviscosity syndrome
- Vitreal degeneration and liquefaction
- Hypermature cataract
- Post-surgery – cataract and lens luxation
- Idiopathic or steroid-responsive retinal detachment
- Solid detachment from adjacent pressure (e.g. retrobulbar neoplasia)
- Severe trauma

CASE WORK-UP

A fairly extensive work-up can be required to reach a definitive diagnosis in cases of retinal detachment, particularly in adults with serous detachments. Blood samples should be taken for haematology and biochemistry screening. These might show a neutrophilia suggesting infectious disease or hyperviscosity syndromes for example, such that serum electrophoresis is

warranted, along with bone marrow biopsy. Renal and hepatic disease might be present. Urinalysis should be considered. Systemic blood pressure should always be taken since hypertension is a common cause of retinal detachment in both dogs and cats. If chorioretinitis is suspected, specific serological testing for the various causes of this can be considered (Table 49.1).

If the fundus cannot be directly visualized, for example because of the presence of a total cataract, then ocular ultrasonography will be invaluable in detecting a retinal detachment. General abdominal and cardiac ultrasonography might also be indicated depending on the preliminary investigation results – looking for cardiac disease or adrenal masses in cases of systemic hypertension for

Table 49.1 Causes of chorioretinitis in dogs and cats

Cause		Dog	Cat	Diagnostic test
Infectious	Viral	Distemper Herpes	Panleukopenia FIP FeLV FIV	Specific serology if available, demonstration of organism (e.g. blood smears or faecal samples), radiography, urinalysis, history of exposure in endemic regions, ELISA testing
	Bacterial	Septicaemia Brucellosis Tuberculosis	Tuberculosis	
	Protozoal	Toxoplasmosis Neosporosis Leishmaniasis	Toxoplasmosis Leishmaniasis	
	Parasitic	Toxocariasis Angiostrongylus Migrating dipteran larvae	Toxocariasis Migrating dipteran larvae	
	Rickettsial	Ehrlichiosis Rocky mountain spotted fever		
	Mycotic	Aspergillosis Blastomycosis Histoplasmosis Cryptococcosis Coccidioidomycosis	Cryptococcosis	
	Algal	Protothecosis		
Immune mediated		Thrombocytopenia Anaemia Uveodermatological syndrome	Thrombocytopenia Anaemia	Appropriate haematology, Coombs' test Skin biopsy for UDS
Vascular disease		Hypertension	Hypertension	Measure blood pressure, identify underlying aetiology, e.g. renal or cardiac disease
Neoplasia		Hyperviscosity syndromes (paraneoplastic syndrome, e.g. with multiple myeloma) Lymphoma Metastatic tumours	Hyperviscosity syndromes (paraneoplastic syndrome, e.g. with multiple myeloma) Lymphoma Metastatic tumours	Haematology, biochemistry, electrophoresis, lymph node aspirate, bone marrow biopsy, urinalysis
Trauma		Proptosis Surgery Foreign body	Proptosis Surgery Foreign body	History and clinical examination

FeLV, feline leukaemia virus; FIP, feline infectious peritonitis; FIV, feline immunodeficiency virus; UDS, uveodermatological syndrome.

example, or for the location of a primary tumour in hyperviscosity syndromes. Unfortunately, in some instances, the underlying aetiology might not be revealed despite extensive work-up.

Cases of rhegmatogenous retinal detachment do not always require much case work-up since the cause is more frequently ophthalmic rather than systemic. Previous cataract or lens luxation surgery and vitreal degeneration will predispose to large retinal tears and in these situations minimal investigation is required.

NURSING ASPECTS

Animals with sudden onset blindness will be frightened and confused and require sympathetic handling and nursing. Small dogs should be carried as this will be less disorienting for them, while larger dogs should be kept on a short lead when moved. Cats should always be moved in a basket where they will feel safer and be less likely to panic and try to escape. The animal should always be spoken to as it is being approached so that it is less frightened when handled. As these patients require quite extensive diagnostic testing, and frequently are put on multiple treatment regimens, nurses should try to make this as stress-free as possible for the animal.

EPIDEMIOLOGY

Retinal detachments occur as the neurosensory retina separates from the underlying retinal pigment epithelium (RPE) rather than a complete detachment of the entire retina from the underlying choroid. They can develop in one of three ways (Table 49.2):

Table 49.2 Types of retinal detachment

Type	Pathological description
Exudative	Retinal effusion or occasionally solid cells push neurosensory retina from underlying retinal pigment epithelium
Rhegmatogenous	Tear in peripheral retina allows vitreous to move between neurosensory retina and retinal pigment epithelium
Traction	Retina pulled off by vitreal traction bands

- exudative, where effusions, exudations or rarely solid accumulations of cells push off the neurosensory retina
- rhegmatogenous detachments where a tear or hole in the retina causes disinsertion, allowing liquefied vitreous to accumulate under the tear
- traction detachments where proliferation of preretinal (i.e. vitreal) membranes pulls the neurosensory retina away from its underlying RPE.

The actual mechanism of detachment is often not fully understood, although microtrauma, vascular abnormalities and tension on the peripheral retina are involved. Once there is loss of structural integrity there is rapid degeneration of the photoreceptors in the affected area – they cannot function when not in close association with the RPE. Thus, even if treatment is successful in causing retinal reattachment, a return of functional vision does not necessarily ensue.

Most cases of retinal detachment are sporadic in dogs and cats. Congenital abnormalities such as collie eye anomaly, coloboma and retinal dysplasia are frequently inherited and puppies can be presented blind at only a few weeks of age, or subsequently go blind at a few months as the abnormal retina detaches. Litters of puppies of at-risk breeds should be examined at 6–8 weeks of age by an ophthalmologist and even mildly affected animals should not be used for breeding.

Since many causes of retinal detachment will have an underlying systemic abnormality – hypertension, hyperviscosity syndrome and so on – the predisposing factors relating to these specific aetiologies will be appropriate. Some cases of bullous retinal detachment are idiopathic. A syndrome of steroid-responsive retinal detachment is encountered mainly in young adult German shepherd dogs, and manifests as a sudden onset total bilateral bullous detachment in an otherwise healthy dog. The condition is poorly understood but is suspected to be immune mediated. Rhegmatogenous retinal detachments are more common in Shih Tsu, whippet and Italian greyhound breeds where a primary vitreal degeneration and liquefaction predisposes to peripheral retinal tears and sudden, total detachment. Usually one eye is affected before the other. The cause for the initial vitreal liquefaction is unknown but since the condition is more prevalent in these few breeds an inherited component is likely. Rhegmatogenous detachments are also seen in those breeds that suffer from lens luxation and are additionally associated with hypermature cataracts (whether or not these undergo surgical removal) in any breed.

CLINICAL TIPS

- Pupil will be dilated and non-responsive in affected eye(s)
- If a cataract is present the pupillary light response should be normal unless retinal pathology is present
- Ultrasound the eye if you cannot see the retina!
- A pen torch is often sufficient to visualize a totally detached retina as a veil behind the lens
- Rule out systemic disease
- Think of congenital causes in young dogs

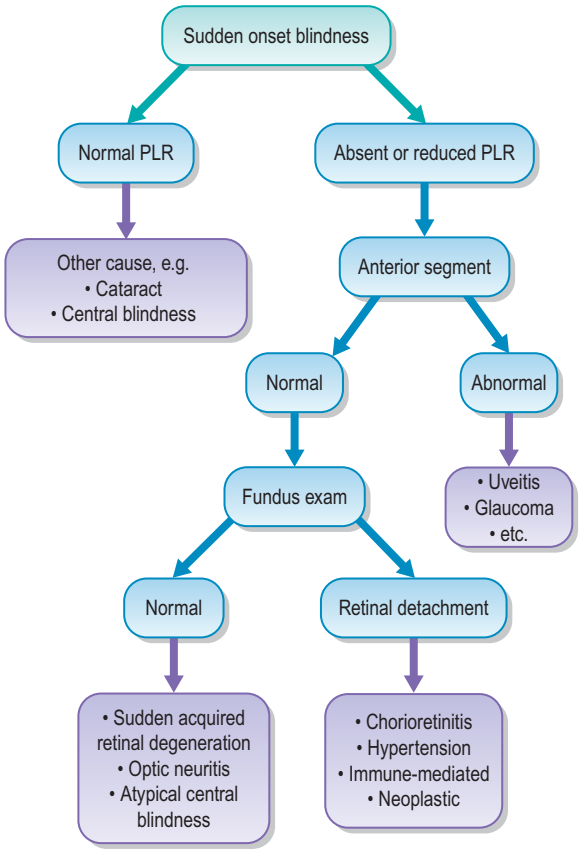


Figure 49.4 Decision-making tree – retinal detachment and sudden onset blindness.

TREATMENT OPTIONS – MEDICAL

For total rhegmatogenous retinal detachment there is no treatment and the eye remains blind; serous detachments, however, can be amenable to treatment. This is first directed at correcting the underlying systemic condition – treating the hypertension or infectious chorioretinitis for example. If no underlying cause is identified, then empiric treatment with immunosuppressive doses of steroids is started – usually prednisolone at 1–2 mg/kg once or twice daily. Partial detachments are more likely to reattach but some total bullous detachments can do very well. The steroids are gradually reduced as the patient improves, but low dose, alternate day therapy can be required for many months. Systemic diuretics have been used historically but do not seem to be particularly efficacious.

TREATMENT OPTIONS – SURGICAL

Surgery to reattach retinas is rarely performed in veterinary ophthalmology, even by specialists. However, a few centres do offer laser surgery which will delineate small detachments (both serous and rhegmatogenous) in an attempt to prevent them spreading. Unfortunately, most patients are too advanced by the time they are presented to the general practitioner for any salvage surgery to be attempted. The risk of retinal detachment following surgery – for anterior lens luxation or hypermature cataracts for example – can be reduced by performing transscleral laser retinopexy surgery before the intraocular surgery and is recommended by some ophthalmologists in high-risk breeds.

PROGNOSIS

The prognosis for retinal detachment is guarded. Rhegmatogenous and traction detachments are not amenable to treatment and the blindness is permanent. However, these eyes are not usually painful and providing the animal is systemically well, then quality of life can be maintained.

Bullous detachments can reattach once the underlying systemic illness has been treated. However, the reattached areas might not be functional, and frequently develop signs of degeneration with hyper-reflectivity and pigment abnormalities in the affected areas. As such, some patients will regain some vision after an acute detachment, but vision will gradually deteriorate again with time as the degeneration sets in.

CASE EXAMPLE 49.1

Patient details: Boxer, 10 years old, male (entire)

PRESENTATION AND HISTORY

The dog was presented with sudden onset blindness. The owner had noticed that the left eye had looked red and glazed for a few days and the dog was hesitant at times. He had also lost weight recently, was lethargic and slightly inappetant. No relevant previous history was noted.

CLINICAL SIGNS

Menace responses were markedly reduced in both eyes but not completely absent. Pupillary light reflexes were reduced and the pupils were dilated in ambient light. The left eye had mild episcleral congestion and a dark solid mass could be seen through the pupil (Figure 49.5). This was dorsomedial and appeared to involve the retina. The right eye (Figure 49.6) had some vitreal haze but a large serous retinal detachment was noted on fundus examination. On general clinical examination the dog was slightly underweight with a dry, scurfy coat. A moderate generalized lymphadenopathy was noted.

WORK-UP

Blood was taken. Routine haematology was unremarkable apart from a mild anaemia, but on biochemistry there were elevations in creatine kinase (CK) and aspartate transaminase (AST), a mild hypoalbuminaemia and moderate hyperglobulinaemia. A fine needle aspirate of the submandibular lymph node revealed many small immature lymphocytes and a diagnosis of multicentric lymphoma was reached. Abdominal ultrasonography revealed thickening of the small intestine and enlarged mesenteric lymph nodes.

TREATMENT

Topical prednisolone acetate was started twice daily bilaterally. The owner elected to try chemotherapy, and a standard protocol using vincristine, cyclophosphamide and prednisolone was started.

OUTCOME

Initially the dog did quite well. His demeanour improved, the lymph nodes reduced in size, the retinal detachments reduced and vision improved. However, 4 weeks after starting chemotherapy he became very depressed and totally anorexic and the owners elected for euthanasia.

This case illustrates how an animal can present with apparent ophthalmic disease but actually have a very serious underlying condition. Any pet presenting with bilateral ocular signs should have a thorough work-up since a life-threatening neoplastic process could be present.

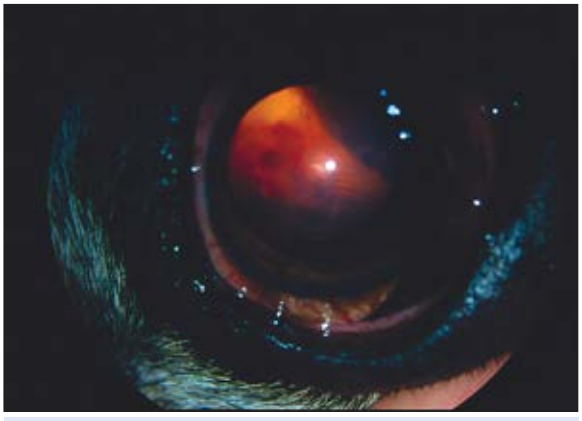


Figure 49.5 Left eye of boxer showing dilated pupil and dorsomedial mass visible in vitreous.

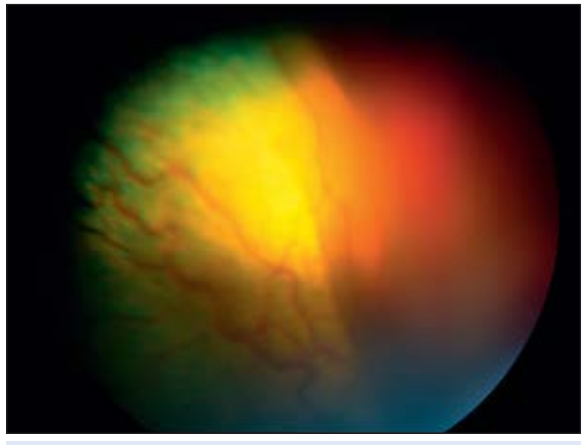


Figure 49.6 Right eye of the same boxer as in Figure 49.5 showing exudative retinal detachment.

50 Sudden acquired retinal degeneration

INITIAL PRESENTATION

Blind

PRESENTING SIGNS

Owners will present the patient because the animal has suddenly gone blind. This may have occurred literally overnight, or may have progressed over a few days. The patient is likely to be distressed and slightly disoriented, and might be depressed or clinically unwell – with the owners reporting increased appetite and thirst. However, the patient might be totally normal except that they are bumping into things. The owners might have also noticed a change in the appearance of the eyes – shiny, glassy or perhaps just looking different. Small breed, overweight female dogs are most frequently presented. The condition has not been described in cats.

CASE HISTORY

In most cases there is no previous history of ocular disease or relevant systemic disease. However, the patient might have recently gained weight and might show signs of polydipsia and polyuria. A few cases will have a history of low grade hyperadrenocorticism which is either not being treated or not controlled with treatment.

CLINICAL EXAMINATION

General clinical examination will reveal a lethargic, slightly overweight or even obese patient but no other specific abnormalities. The patient will be obviously totally blind – reluctant to walk, bumping into objects, head down smelling the floor and with a high stepping gait. There will be a 'starey' appearance to the eyes, due to the presence of dilated pupils. Menace responses will be absent. Pupils will be dilated in room light and in the dark, and will be symmetrically so. Pupillary light reflexes are usually totally absent, although slight pupil constriction might occur with a very bright light source. Dazzle reflexes will be absent.

Intraocular examination will be normal – one would expect fundus abnormalities but this is not the case – the retina and optic nerve look normal initially. However, if the patient is not presented for several weeks the early signs of retinal degeneration, with tapetal hyperreflectivity and blood vessel attenuation, might have developed.

DIFFERENTIAL DIAGNOSES

- Sudden acquired retinal degeneration (SARD)
- Optic neuritis
- Retinal detachment
- Central blindness (for causes, see list below)
- Hypertensive retinopathy
- Toxic retinopathy
- Other causes of acute blindness – glaucoma, uveitis

Possible causes of central blindness include:

- optic neuritis (retrobulbar)
- brain tumour (e.g. lymphoma, meningioma, pituitary tumour)
- hydrocephalus
- hepatic encephalopathy

- encephalitis (e.g. distemper, feline infectious peritonitis (FIP), *Toxoplasma*, *Neospora*)
- meningitis (viral, bacterial, fungal)
- toxicity (ivermectin, lead, levimasole)
- brain trauma (e.g. road traffic accident)
- anoxia (anaesthetic induced)
- cerebrovascular accident (e.g. hypertensive cats)
- lysosomal storage disease
- granulomatous meningoencephalitis (GME).

CASE WORK-UP

In patients which present with sudden onset blindness in the absence of ocular abnormalities, other than dilated non-responsive pupils, the main differentials are SARD, optic neuritis (retrobulbar – such that no optic disc swelling or haemorrhages are present on fundus examination) and central blindness – mainly that due to lesions located close to the optic chiasm. The easiest way to differentiate between these is by electroretinography, and referral for this should be offered to owners. The procedure is performed under sedation and topical anaesthesia, and bright light is shone into each eye in turn, while electrodes pick up the summed response of the retina. A normally functioning retina will elicit a typical response – seen with cases of optic neuritis and central blindness – while SARD patients will have no retinal response and thus a 'flat line'. If the response is normal, then further diagnostic tests would include a complete neurological examination and possibly MRI. Table 50.1 summarizes these findings.

In addition to electroretinography, patients should have blood taken for routine haematology and biochemistry, with further evaluation for hyperadrenocorticism if the initial blood screens suggest this – there is a tenuous link between SARD and Cushing's disease in some patients.

NURSING ASPECTS

Patients who go suddenly blind are frightened and disoriented. Gentle handling and frequent, familiar, reassuring voices will help to settle such patients. Small dogs should be carried as this will be less disorienting for them, while larger dogs should be kept on a short lead when moved. The animal should always be spoken to as it is being approached so that it is less frightened when handled. These patients require quite extensive diagnostic testing, and nurses should try to make this as stress-free as possible for the animal.

EPIDEMIOLOGY

SARD is seen as a sporadic condition in dogs. Smaller breeds such as the dachshund varieties, miniature schnauzer and small crossbreds are seen with more frequency than larger breeds, but it can occur in any breed or cross. Dogs tend to be middle aged or older (8–10 years median), with a definite preponderance of females (both neutered and entire – although numbers of the latter are generally low at this age!). It has not been reported in cats. It has been called 'silent retina syndrome' and toxic retinopathy in the past, but the underlying cause of the condition is still unknown, despite considerable research.

There is a sudden, severe bilateral loss of all photoreceptors (i.e. both rods and cones), with the outer segments dying by apoptosis followed by degeneration of the remainder of the retina. Abnormal retinal proteins and neurotoxins have been postulated to be involved in the pathogenesis but conflicting reports abound. Systemic signs of polyphagia, weight gain, polydipsia and polyuria are reported in 40–50% of patients, but only a few of these test positive for hyperadrenocorticism.

Table 50.1 Differential diagnoses for SARD, optic neuritis and central blindness

	SARD	Optic neuritis	Central blindness
Pupillary light reflex	Absent	Absent	Normal (usually)
Fundus examination	Normal	Swollen optic discs and peripapillary haemorrhages *or* normal	Normal
Electroretinogram	Flat line	Normal trace	Normal trace
MRI	Normal	Swollen optic nerves	Central lesion, e.g. brain tumour or lesions suggestive of granulomatous meningoencephalitis visible

CLINICAL TIPS

- Did the patient really go suddenly blind – carefully question the owner
- Pupils are dilated and usually fixed – ensure this is not just because the patient is terrified!
- The fundus looks normal
- Definitive diagnosis only reached by electroretinography
- 'The dog sees nothing; the ophthalmologist sees nothing'

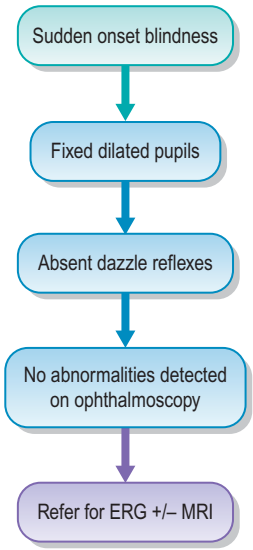


Figure 50.1 Decision-making tree – sudden acquired retinal degeneration. See also Figure 49.4.

TREATMENT OPTIONS

There is no treatment for SARD but affected patients should be checked for evidence of hyperadrenocorticism and treated appropriately. This can be present at the time of SARD diagnosis but sometimes does not develop for several months.

PROGNOSIS

The prognosis for any return of vision is hopeless – affected dogs go blind and stay blind. However, they do adjust to their disability and can lead full, happy lives. The initial confusion and disorientation subside after a couple of weeks as the dogs learn their way around the environment using smell, sound and tactile cues. The condition is not painful and certainly far less disabling to the patient than other causes of sudden onset bilateral blindness such as brain tumours.

51 Hypertensive retinopathy

INITIAL PRESENTATION

Blind, abnormal appearance – red

PRESENTING SIGNS

The presentation in cases of hypertensive retinopathy is variable. Owners might have noticed a sudden or gradual onset blindness, a change in appearance of the eye(s) such as dilated pupils, a clouding or redness, sudden onset hyphaema, or a general change in the pet's demeanour, perhaps with increased drinking and urination together with some weight loss. Cats are presented more commonly than dogs and they are normally older – 10 years or more.

CASE HISTORY

In some patients there will be no previous history, with the pet presented suddenly with the ocular complaint. On questioning the owner it might be apparent that some abnormalities have been developing for a while – polydipsia, polyuria and weight loss despite a good appetite being the most frequent. The pet might be less active and sleep more, but this is frequently attributed to increasing age. Some animals might already have known systemic disease such as hyperthyroidism, renal insufficiency or cardiac disease for which they are being treated. Occasionally the hypertension is picked up on a routine check-up – on a senile health check for example – or if the pet is presented for vaccination and a thorough ophthalmic examination is performed.

CLINICAL EXAMINATION

General clinical examination might reveal abnormalities, but might be normal. Patients might be thin, with poor coats, small, hard kidneys, an elevated heart rate and cardiac murmur or a palpable thyroid gland for example.

Ophthalmic examination can be similarly variable. One or both eyes can be affected. Some patients will be blind, with no menace response and dilated poorly responsive pupils. Remember that cats can retain a brisk pupillary light reflex despite advanced retinal disease, so these should not be relied upon in this species. Dazzle reflexes are usually reduced. The conjunctiva is occasionally slightly hyperaemic but no ocular discharge or corneal ulceration is usually present. Some hyphaema or vitreal haemorrhage might be present, both of which render fundus examination impossible (see Figure 36.2). However, it is unusual for both eyes to have severe haemorrhage simultaneously and so some fundus evaluation should be possible.

If any opacification of the ocular media is present, indirect ophthalmoscopy will be more rewarding than direct ophthalmoscopy. The retina might be completely detached in one or both eyes, and can be seen as a grey veil floating behind the lens (see Figure 49.1). In less advanced cases, the fundus will show various changes – some small blister-like areas of bullous detachment (seen as elevated, out-of-focus dull patches within the tapetal fundus) or retinal haemorrhages – both within the retina itself (flame shaped) and pre-retinal (dark puddle-like areas). The retinal blood vessels are frequently engorged and tortuous with some perivascular cuffing and haemorrhage (Figure 51.1). The vessels often have variable diameter along their length, giving the appearance of a string of sausages. Variable amounts of vitreal haemorrhage can also be present. Although bilateral, the condition is not symmetrical.

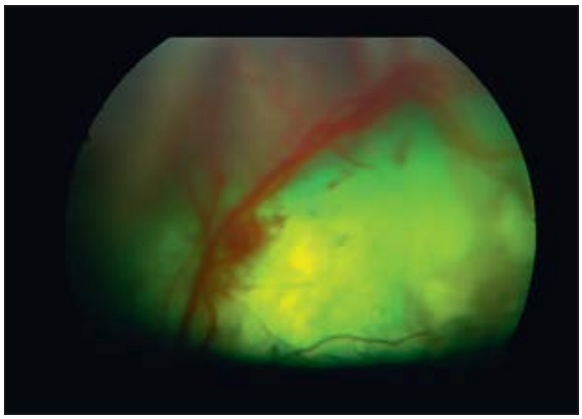


Figure 51.1 Hypertensive retinopathy in a 12-year-old domestic short haired cat. Note the retinal detachment, multiple small haemorrhages and tortuous blood vessels with perivascular cuffing.

DIFFERENTIAL DIAGNOSES

- Other causes of hyphaema (see Chapter 36)
- Other causes of retinal detachment (see Chapter 49)
- Chorioretinitis (see Chapter 49)
- Coagulopathies
- Hyperviscosity syndromes
- Retinal dysplasia

CASE WORK-UP

Routine haematology and biochemistry is essential, together with appropriate tests for hyperthyroidism in cats, but also sometimes hypothyroidism in dogs. Blood pressure should be measured as outlined in Table 51.1. Normal readings are provided in Table 51.2.

Further work-up might include urinalysis to check renal function and look for evidence of diabetes mellitus, plus testing for hyperadrenocorticism in dogs if the initial blood results indicate this is a potential underlying problem. If any cardiac abnormalities are suspected on auscultation, then cardiac ultrasonography is recommended. Adrenal ultrasonography is also helpful in some cases.

NURSING ASPECTS

Animals which suddenly go blind with hypertensive retinopathy are frightened and confused and require sympathetic handling. They will undergo multiple diagnostic tests and need gentle reassurance throughout this. Although topical medication is rarely necessary, they will often require multiple systemic drugs and frequently a change in diet which needs patience on behalf of the nurse and subsequently the owner in achieving success, especially in cats.

Table 51.1 Measurement of blood pressure in dogs and cats

- Doppler or oscillometric monitors can be used although the former are more reliable in cats.
- Take patient to a quiet room.
- Allow patient to acclimatize to room.
- Sometimes the measurement will be more accurate if the owner is present (the patient might be less stressed).
- Choose the site for measurement – forelimb or tail are most commonly used.
- Choose an appropriate cuff size (width of cuff should be 40% limb circumference in dogs and 30% in cats).
- Gentle handling and talking to the patient will calm them.
- Take 5–10 consecutive readings.
- Discard the first two readings which are often elevated and inaccurate.
- If the readings are not consistent, abandon the session and try again later.
- Ideally take 3–4 readings during the day.

Table 51.2 Normal blood pressure readings in dogs and cats

Reading in mmHg	Systolic	Diastolic
Dog	110–190	55–110
Cat	120–170	70–110

EPIDEMIOLOGY

There are many potential underlying causes for hypertension in dogs and cats but the underlying ocular pathology is the same.

Causes of hypertension include:

- renal disease
- hyperthyroidism
- diabetes mellitus
- hyperadrenocorticism
- cardiomyopathy
- hyperparathyroidism
- phaeochromocytoma
- high salt diet
- idiopathic/essential
- megestrol acetate administration
- chronic anaemia
- primary aldosteronism.

Sustained elevations in systolic blood pressure lead to vasoconstriction of the retinal arterioles which are autoregulated. This results in hypertrophy and hyperplasia of the vessel walls, and loss of the elasticity of the vessel membrane and eventually fibrinoid necrosis. Both retinal and choroidal blood vessels are affected. The walls become permeable and plasma and fibrin leak from the blood vessels, followed by frank haemorrhage as the condition progresses. Retinal detachment occurs as a combination of ischaemic damage to the retinal pigment epithelium and subretinal exudation. In addition to the retina, both the kidneys and brain are 'target organs' for hypertensive damage.

Hypertensive retinopathy is a condition of older age – dogs over 8–10 and cats over 10–12 are more frequently presented and regular screening of the older pet population is advised. No sex or breed predilection occurs. The condition is being diagnosed more and more – probably due to a combination of factors including veterinary awareness of the condition, better access to accurate blood pressure monitors and the fact that pets are living longer.

CLINICAL TIPS

- Repeat blood pressure measurement several times
- Make the environment for blood pressure monitoring as stress-free as possible
- Look carefully for a primary underlying cause for the hypertension

TREATMENT OPTIONS – MEDICAL

Treatment for hypertensive retinopathy aims to return or improve vision if possible, while reducing the risk of further target organ damage. Clearly, if an underlying problem is established, this must be treated as well. If the primary problem is resolved with treatment, the blood pressure can reduce, but anti-hypertensive medication should not be withheld while waiting to see if this happens – irreversible blindness could ensue.

In general, anti-hypertensive therapy aims to improve cardiac output and decrease peripheral resistance. This should be combined with rest and a low salt diet. Diuretics are sometimes used, particularly in patients with cardiac disease. They should be avoided in cats with hypertension and renal insufficiency. However, calcium channel blocking drugs and angiotensin-converting enzyme inhibitors, both of which lower peripheral resistance, tend to be the first line of treatment. Amlodipine is used as an initial first line drug in cats, sometimes along with benazepril. Both can also be used in dogs, although the latter is frequently used initially. Alpha blocking drugs such as prazosin and beta-blockers such as propranolol are useful in some patients. Table 51.3 lists the frequently used anti-hypertensive drugs in cats.

Table 51.3 Drugs used to treat hypertension in cats

Drug name	Dose rate	Class of drug	Method of action
Amlodipine Diltiazem	0.0625–0.125 mg/day 1.75–2.4 mg/kg bid–tid	Calcium channel blocker	Stabilize heart rate and vasodilate
Enalapril Benazepril	0.25–0.5 mg/kg/day 0.25–1.0 mg/kg/day	Angiotensin-converting enzyme (ACE) inhibitor	Vasodilation to reduce cardiac workload
Propranolol	0.1–0.2 mg/kg/bid	Beta-blocker	Reduces heart rate
Furosemide	1–2 mg/kg/day	Diuretic	Reduces extracellular fluid volume

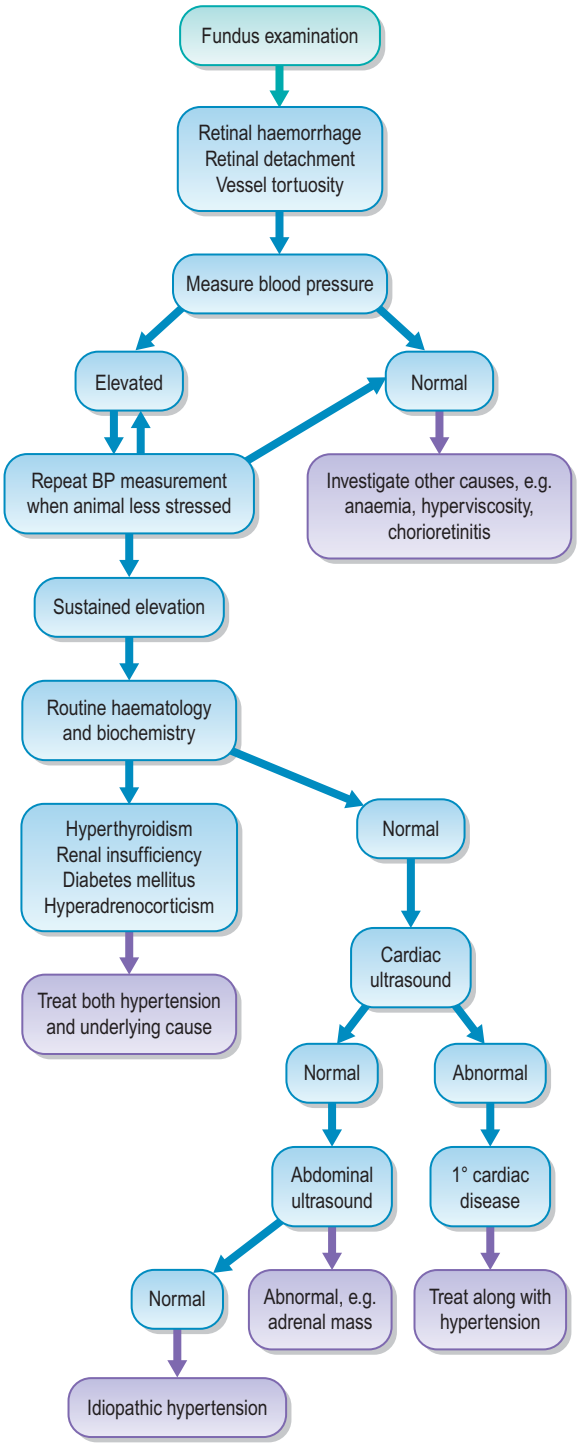


Figure 51.2 Decision-making tree – hypertensive retinopathy.

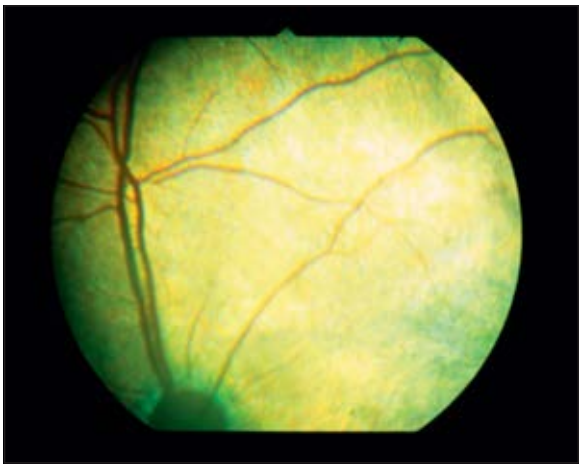


Figure 51.3 Treated hypertensive retinopathy in an 11-year-old domestic short haired cat. The retinal detachments have resolved, leaving tapetal hyperreflectivity and retinal degeneration. No retinal haemorrhages remain but the vessels are segmented. Some vision was present in this eye.

Response to treatment is assessed with a combination of repeated ophthalmic examination, regular blood pressure measurement and repeated laboratory tests as indicated.

PROGNOSIS

The prognosis for patients with hypertensive retinopathy is guarded. Many do regain some vision initially as the retinas reattach as the blood pressure reduces. However, these areas can progress to retinal degeneration and atrophy such that vision gradually deteriorates again (Figure 51.3). Quality of life can nonetheless be improved during this time. Animals with excessive vitreal and retinal haemorrhages do less well with respect to any improvement in vision than those with serous detachments. Life-long medication is necessary and in some patients the stress of repeated oral treatment can be excessive. In addition to improving quality of life by restoring some useful vision, treatment aims to prevent further target organ damage such that cardiac and/or renal failure, plus cerebrovascular accidents, are avoided.

CASE EXAMPLE 51.1

Patient details: Domestic short haired cat, 12 years old, female (neutered)

PRESENTATION AND HISTORY

The cat was presented with sudden onset blindness and a red left eye. She had been sleeping a lot recently and had lost some weight but the owner was not worried until the cat suddenly started bumping into things and the left eye looked red.

CLINICAL SIGNS

On general clinical examination the cat was thin and slightly dehydrated. Her kidneys were not palpably abnormal and no thyroid gland could be palpated. Heart rate was over 220 beats per minute with a gallop rhythm but no murmur was auscultated. Both eyes were blind. The left pupil was fixed and dilated while some direct pupillary constriction occurred in the right, together with minimal consensual constriction in the left. Marked vitreal haemorrhage was noted in the left eye such that fundus visualization was not possible (Figure 51.4). The right fundus showed a partial retinal detachment dorsally, marked vessel tortuosity and multiple retinal haemorrhages.



Figure 51.4 Left eye of same cat as in Figure 51.1 showing dilated pupil and marked vitreal haemorrhage.

WORK-UP

Routine haematology and biochemistry revealed evidence of moderate renal insufficiency and this was confirmed on urinalysis. Systolic blood pressure was measured three times over 4 hours and was consistently 250 mmHg. This confirmed a diagnosis of hypertensive retinopathy, presumed secondary to renal disease (although vice versa is possible). Further investigations such as cardiac ultrasonography were declined in favour of watching response to treatment.

TREATMENT

The cat was started on oral amlodipine 1.25 mg daily and its diet was changed to a proprietary renal support diet, which thankfully the cat ate willingly providing small pieces of chicken were mixed with it!

OUTCOME

Three weeks later systolic blood pressure was 190 mmHg and the cat had gained weight. Renal parameters were unchanged and benazepril was added to the treatment (0.5 mg/kg/day). Vision in the right eye improved but the left was unchanged. Another month later the cat was systemically well and renal parameters had improved. The retinal detachment in the right eye had resolved but some degeneration was noted. The haemorrhages were reduced and no fresh bleeds were noted. The left eye remained blind. The vitreal haemorrhage had started to contract and a total rhegmatogenous retinal detachment was noted – presumably following traction of the vitreal clots. Systolic blood pressure was 180 mmHg. The cat was maintained on the same treatment.

This case shows the typical presentation for cats with hypertension. The left eye remained blind but some functional vision was restored to the right eye and the cat's general demeanour and quality of life were certainly improved with treatment.

SECTION 9

ORBIT AND GLOBE

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52 Orbit and globe – introduction

THE ORBIT

The orbit is the bony fossa which separates the eye from the cranial cavity. Many bones contribute to it – the frontal, lacrimal, maxillary, zygomatic, pterygoid, palatine and sphenoid. The orbit surrounds and protects the globe while providing a route for vessels and nerves to access the eye. In addition to the eye itself, this region contains the extraocular and retrobulbar muscles, optic nerve, lacrimal and nictitans glands, and part of the zygomatic salivary gland, along with the blood vessels and nerves (cranial nerves II, III, IV, V, VI and VII) (Figure 52.1). Connective tissue and fat are also present.

In humans the bony surround is complete, providing excellent protection to the globe from all directions. However, in carnivores there are areas where no bone surrounds the eye – just soft tissue. These areas are mainly ventral to the globe, where the masseter and pterygoid muscles provide the floor of the orbit, and laterally, where the orbital ligament joins the frontal and zygomatic bones. This lack of a complete bony surround is of relevance when considering retrobulbar disease – conditions affecting these muscles of mastication, as well as those in the caudal molars, caudal nasal chambers and zygomatic salivary glands, can all affect the eye due to the close anatomical connection. Thus a dog presenting with a retrobulbar problem needs to have a careful oral examination – a tooth root abscess affecting a molar tooth could easily be contributing to the ocular signs, as could a foreign body which has entered the retrobulbar space via the mouth. Disease processes affecting the frontal and maxillary sinuses can also lead to ocular involvement by local spread of infectious, inflammatory or neoplastic conditions.

The shape of the orbit varies tremendously between canine breeds. Thus in doliocephalic breeds it is a deep concave shape (like an ice cream scoop) while in brachycephalics it can be extremely shallow (more like a teaspoon!). The orbital rim should be palpated as part of the ophthalmic examination and the variation in its position will become apparent. With extreme skull shapes, such as the Pekingese, the orbital rim is markedly posterior to the eyeball itself, and appreciation of this makes it easy to understand how proptosis can occur so easily in such breeds.

The extraocular muscles are located within the orbit. They are attached to the globe and provide ocular motility. Together with the retrobulbar muscle they form a cone shape with the apex at the optic foramen. They are normal striated muscle. There are six extraocular muscles for each eye: the four rectus muscles (dorsal, ventral, medial and lateral – which pull the eye in their respective directions), together with the dorsal oblique (which pulls the dorsolateral aspect of the globe ventromedially) and the ventral oblique (which pulls the eye mediodorsally) (Figure 52.2). The extraocular muscles are innervated by the oculomotor nerve (cranial nerve III), apart from the lateral rectus muscle which is innervated by the abducens nerve (cranial nerve VI) and the dorsal oblique muscle which is supplied by the trochlear nerve (cranial nerve IV). Thus, if there are abnormalities with the cranial nerves, this can alter both the position of the eye within the socket and affect its movement. These findings can aid with the localization of any lesion and form an important part of neuro-ophthalmology.

In addition to the muscles, orbital fat fills the space around the eye within the orbit. This allows the eye to move within the socket when pulled by the extraocular muscles, and also provides protective cushioning (e.g. following blunt trauma). If an animal loses a lot of weight, as in ageing, this fat pad is reduced and can result in a sunken-eyed appearance.

THE GLOBE

The globe is cushioned within the orbit, with the eyelids (including the nictitans membrane) providing anterior

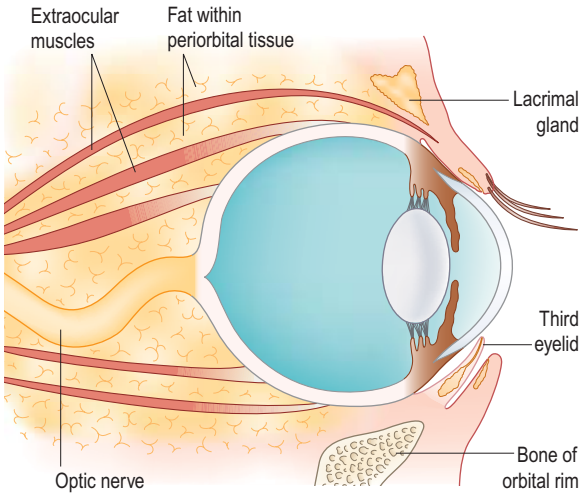


Figure 52.1 Simplified contents of the orbit.

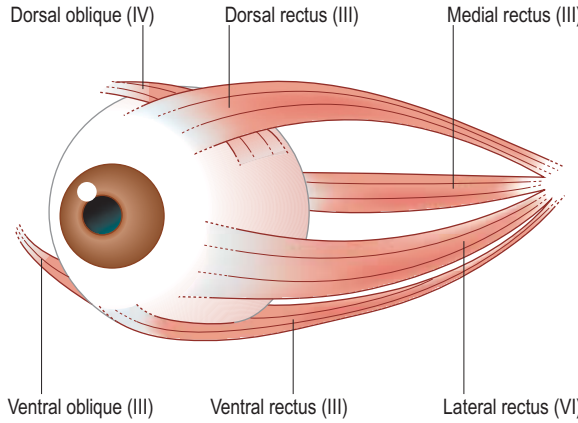


Figure 52.2 The extraocular muscles with their innervation shown in brackets.

protection. It is roughly spherical in shape in dogs and cats, and eyeball size varies much less than breed size! The globe consists of three distinct areas:

- the outer layer, consisting of cornea and sclera, provides structural support and the transparent anterior cornea allows light to enter the eyeball
- the intermediate zone between cornea and sclera is the limbus which is frequently pigmented
- episcleral and conjunctival tissue which cover the anterior sclera.

The optic nerve enters through the sclera, slightly ventrolateral to the midline, while the two long posterior ciliary arteries course from here anteriorly along 3 and 9 o'clock. It is these landmarks which pathologists use if the clinician has failed to indicate whether the enucleated eye sent for histological examination is the right or left globe! The middle layer of the globe is the vascular layer and consists of the uveal tract – iris, ciliary body and choroid – while the inner layer of the lens and neurosensory retina can be considered the optical part of the globe. Only conditions affecting the outer structures are considered in this section – refer to the relevant parts of the text for the other areas.

Conditions which affect the globe and orbit can relate either to the size of the eyeball or to its position. Thus it can be small – microphthalmia is a congenitally small eye, while phthisis bulbi is a decrease in globe size following severe trauma, chronic glaucoma, uveitis or infection for example. Hydrophthalmos is an enlarged globe due to raised intraocular pressure. Buphthalmos is sometimes used synonymously with hydrophthalmos but really should relate to a congenitally enlarged globe only (i.e. one affected by congenital glaucoma). Exophthalmos is the most commonly encountered ophthalmic condition involving the orbit and is a forward displacement of the globe. It can occur in a variety of disease processes, with retrobulbar abscessation/cellulitis and neoplasia being the most frequently seen in general practice. Proptosis, where the globe is displaced forwards in front of the eyelids, which close in spasm behind it, is the most extreme form of exophthalmos. The opposite, enophthalmos, is seen less frequently and can be due to both ophthalmic and neurological disease. Strabismus or squint can also be present, either alone or with other positional abnormalities.

53 Exophthalmos

INITIAL PRESENTATION

Abnormal appearance – position, pain

PRESENTING SIGNS

The owner will present the animal due to the change in appearance of their pet. Frequently the condition is unilateral, but on occasion both eyes can be involved. The owner often thinks that the affected eye is swollen, and in addition there might be some ocular discharge, pain on manipulating the area, prominence of the nictitans membrane and a squint (strabismus). The eyelids and periorbital region might also be swollen and inflamed. Some animals will be clinically unwell, with pyrexia and a reduced appetite – often they will eat soft foods but avoid biscuits and chews. The presentation can be acute, or slow and insidious. The animal can be any age, either sex, and no breed predisposition is present for cases of exophthalmos in general, although some specific conditions are more common in certain breeds as we shall see later.

CASE HISTORY

The history can be quite variable. The patient might have a history of previous or recurrent dental disease, or could be a 'stick chaser' who frequently chews up sticks and similar objects. There is frequently no history of previous ocular disease, and many cases have no systemic history either, although the animal might have suffered from a traumatic incident or even had a neoplastic process treated in the past.

CLINICAL EXAMINATION

On ophthalmic examination the animal will usually have an obviously abnormal appearance. The face will be asymmetrical in unilateral cases. The affected globe might appear enlarged, but on close examination it will be seen to be pushed forwards. Thus, looking at the patient both from above and the side, as well as from in front, will assist in determining the position of the globe (Figure 53.1). The globe might be deviated in position (squint or strabismus) and this is most commonly in a lateral or dorsolateral direction (the deviation will assist in determining the location of any retrobulbar mass). The third eyelid is normally elevated and some ocular discharge is frequent. This can vary from epiphora to a frank purulent discharge. Sometimes it can be blood-stained. Resistance to retropulsion will be present, and this might be painful in some animals. The globe should be gently pushed backwards behind the closed lids and the two sides compared. The amount of normal retropulsion possible varies from breed to breed – it is quite extensive in breeds such as the Great Dane and Rottweiler, while it is minimal in brachycephalic dogs and cats.

The affected eye can be inflamed, with conjunctival hyperaemia and chemosis, exposure keratitis (with or without corneal ulceration – do not forget to use fluorescein!) and periorbital swelling, but in some animals it will appear quite normal. If there is a lot of pressure on the back of the globe, this might result in a dilated pupil, due perhaps to a retinal detachment or damage to the optic nerve, and so full intraocular evaluation is also necessary. Vision will be reduced if the optic nerve is damaged or a retinal detachment is present, but this might be difficult to demonstrate unless the eye is totally blind.

Any animal with exophthalmos should have a thorough oral examination. This will concentrate on evidence

of dental disease, including gingivitis, examination of the back of the mouth behind the last molar (looking for swelling, hyperaemia, discharge and abnormality of the oral mucosa), and checking for wounds or retained foreign bodies. Many patients will exhibit pain when opening the mouth (as the vertical ramus of the mandible impinges on the retrobulbar space). A nasal discharge might be present – for example, if a caudal nasal tumour is causing the exophthalmos. Alternatively, nasal air flow might be reduced and comparison of the two sides can be useful – holding a small tuft of hair in front of each nostril in turn to visualize the air flow on expiration can be helpful.

Some patients presenting with exophthalmos will be pyrexic with a mild lymphadenopathy (especially of draining lymph nodes).



Figure 53.1 Elderly dachshund-cross with right-sided exophthalmos. The right cornea is clearly several millimetres anterior to the left one.

DIFFERENTIAL DIAGNOSES

- Retrobulbar abscess/cellulitis
- Retrobulbar neoplasia
- Masticatory muscle myositis
- Extraocular muscle myositis
- Zygomatic mucocoele
- Orbital haemorrhage
- Orbital emphysema
- Orbital arteriovenous varix
- Glaucoma with hydrophthalmos (globe is enlarged in normal position)
- Proptosis

Table 53.1 provides differential diagnoses for the three most common causes of exophthalmos.

CASE WORK-UP

In addition to the ophthalmic and general clinical examination, further work-up is normally required to reach a definitive diagnosis. If the presentation is acute, then haematology might reveal a neutrophilia, suggesting an active infectious process.

Orbital ultrasonography can be rewarding, and usually can be performed in the conscious patient with topical anaesthesia only, although some patients might require sedation. A 7.5–10 MHz sector transducer is coated with normal viscous ultrasound gel and is placed directly on the desensitized cornea (no stand-off is required). The retrobulbar space will be demonstrated. Imaging through the lids will result in a less clear image as trapped air between the eyelid hairs will cause interference. A

Table 53.1 Differential diagnosis of the common causes of exophthalmos

Abscess or cellulitis	Neoplasia	Myositis
Unilateral	Unilateral	Bilateral
Acute	Chronic and progressive	Acute
Mucopurulent ocular discharge	Minimal discharge	Minimal discharge
Pain on opening mouth	Usually pain free	Pain on opening mouth
Third eyelid protrusion	Third eyelid protrusion	Third eyelid protrusion
No retinal detachment	Occasional retinal detachment	No retinal detachment
No strabismus	Frequent strabismus	No strabismus
Mottled mass on ultrasonography but no globe indentation	Frequently solid mass on ultrasonography, sometimes with globe indentation	No scleral changes and usually no mass present on ultrasonography
No bony changes on radiography	Occasional bony changes on radiography	No bony changes on radiography

normal retrobulbar space will consist of a cone shape of hyperechoic (bright) fat and hypoechoic (dark) muscle extending to the apex of the orbit. Sometimes a dark central line can be demonstrated which corresponds to the optic nerve. Medially the hyperechoic curving structure represents the orbital bone. The retrobulbar space should be examined in both horizontal and vertical planes, with comparison to the fellow eye being useful if one is uncertain of the normal appearance. A retrobulbar mass will appear as a region of hypo- or mixed echogenicity within this cone.

Unfortunately, ultrasonography cannot indicate histological type. However, a solid hyperechogenic mass is more likely to be neoplastic than a mixed echo – where the heterogeneous echo represents both solid and liquid such as is seen with an abscess. If the back of the globe is indented by a mass, this is more common with neoplastic processes than inflammatory ones (Figure 53.2). Occasionally a foreign body can be identified by virtue of the acoustic shadow it casts. Swollen extraocular muscles can be highlighted by ultrasonography in cases of extraocular muscle myositis. Ultrasound-assisted fine needle aspiration can be performed, but this can be dangerous (resulting in inadvertent globe rupture for example) and is best left to specialists.

Radiography is very useful in the investigation of cases of exophthalmos and will require either heavy sedation or, more commonly, general anaesthesia. Interpretation of skull radiographs is difficult, and a normal radiographic atlas and a skull of similar conformation to the patient are invaluable. Lateral, dorsoventral and lateral oblique projections are required, together with intra-oral views. If the retrobulbar pathology is purely soft tissue, radiographs might be normal, or show mild non-specific changes in the orbital soft tissue. However, if there is bony involvement or changes in the sinuses, these will be readily demonstrated and are most frequently caused by neoplastic processes. Thus extension from a nasal tumour into the retrobulbar space is quite common, and close attention should be given to the anatomy of the caudal nasal turbinates and lucency of the frontal sinuses. Occasionally a radiopaque foreign body, such as lead shot, might be identified radiographically. The roots of the cheek teeth should be carefully checked – specific dental radiographs will be useful if dental disease is suspected.

The 'gold standard' for imaging the retrobulbar space is with magnetic resonance imaging or computed tomography, and these should be considered if surgery is being contemplated – to delineate the exact margins of a retrobulbar tumour, or to establish the exact three-dimensional location of a foreign body for example.

Although the history, presentation and clinical examination, together with ultrasonography and/or radiography, will greatly assist in determining whether a case of exophthalmos is inflammatory or neoplastic, samples will be required to reach a definitive diagnosis. Under general anaesthesia samples of tissue can be taken from the retrobulbar space via the mouth – a small incision is made behind the last molar on the affected side. This might allow the exudation of pus or serosanguinous tissue in the case of an abscess or cellulitis. Samples should be taken for cytological examination as well as bacterial culture and sensitivity. A retrobulbar tumour is unlikely to result in any discharge, unless secondarily infected. A fine blunt cannula can be directed upwards towards, but posterior to, the globe, and aspiration with a 10 ml syringe should allow enough cells to be collected for cytological typing. Multiple aspirates should be taken and referral for this might be warranted. For suspected cases of masticatory muscle myositis and extraocular muscle myositis, referral should be considered. Biopsy samples of the muscles involved can be sent for histological examination and autoantibody detection (against type 2M muscle fibres).

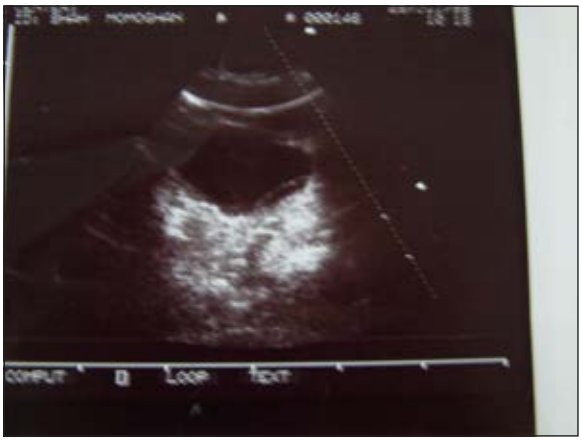


Figure 53.2 Ultrasonogram of a cat with a retrobulbar tumour. The ventrolateral mass can be seen slightly indenting the globe, resulting in a localized retinal detachment.

NURSING ASPECTS

Patients with acute exophthalmos due to abscess, cellulitis or, less frequently, myositis will be depressed and in pain. Cool compresses onto the affected area with the gentle removal of accumulated discharge will be beneficial. Ophthalmic lubricants will reduce the risk of ulceration due to exposure keratitis. Systemic antibiotics, analgesics and anti-inflammatory drugs will be necessary. Patients should be encouraged to eat soft food.

EPIDEMIOLOGY

Exophthalmos is the prominence of a normally sized globe and is caused by a space-occupying retrobulbar lesion of some type. The most common cause of exophthalmos is due to cellulitis or abscessation. There is little difference between the two, although with an abscess the patient is likely to be in more pain and the exophthalmos perhaps more pronounced. Both can be caused by a retrobulbar foreign body such as a fragment of stick or a grass seed and is seen most commonly in younger dogs. Frequently the foreign body is assumed rather than specifically identified. Alternatively, the presumed bacterial infection can reach the orbit haematogenously. Dental disease can also cause inflammatory exophthalmos and is more common in older dogs and cats.

Neoplastic causes of exophthalmos are more common in older dogs (over 9 years) and cats. The presentation is usually more gradual, and strabismus is far more common with cases of neoplasia than with inflammatory causes. The direction of squint can help to localize the mass – a dorsolateral squint might be due to a mass extending through the caudal nasal chamber into the orbit, pushing the globe up and out for example. Masses which arise within the muscle cone (e.g. directly from the optic nerve) will result in forward displacement of the globe without strabismus. Since there are so many tissue types in the orbit and surrounding area, the variety of different tumours which develop is a reflection of this. Thus osteosarcoma, fibrosarcoma, undifferentiated sarcoma and meningioma are all encountered, along with lymphosarcoma and nasal carcinoma. Most orbital tumours are malignant.

The most severe form of exophthalmos is proptosis, with anterior prolapse of the globe out of the orbit. This is more common in brachycephalic breeds, frequently as young adults, but can be seen following severe trauma in any breed of dog or cat. It is discussed more fully in Chapter 55.

Immune-mediated causes of exophthalmos, such as masticatory and extraocular muscle myositis, are rare. They can occur in young dogs, particularly German shepherd dogs, Weimaraners and golden retrievers among other large breeds. They can be recurrent in nature. Other abnormalities such as salivary mucocoeles and arteriovenous fistulae, for example, are even less common – if any of these conditions is suspected, referral is advised.

CLINICAL TIPS

- Look at the patient from above and the side as well as from directly in front, to determine the position of the globe
- A patient with glaucoma and hydrophthalmos (globe enlargement) will be blind in the affected eye; with exophthalmos this is less likely (see Table 53.2)
- Don't forget retropulsion
- Examine the fundus – there might be a solid retinal detachment for example
- Check the teeth!

TREATMENT OPTIONS – MEDICAL

Medical management is indicated for cases of cellulitis and the less frequently encountered myositis. For the former, a broad spectrum antibiotic such as cefalexin is given orally for 7–14 days, usually along with a systemic NSAID such as carprofen. Ocular lubrication is essential, with a bland ophthalmic ointment such as soft white paraffin if there is no ulceration, but using an antibiotic ointment such as chloramphenicol or polymyxin B in the face of any exposure keratitis. An ointment formulation is preferred to drops or gels since it will provide a longer lasting lubrication. The exophthalmos should improve within 24–48 hours and resolve fully in a week. The treatment for retrobulbar abscessation is similar, although surgical drainage should also be attempted as described below.

Immune-mediated cases of myositis require high dose immunosuppressants to control the disease. Clearly a definitive diagnosis should be reached before considering this treatment, particularly since patients are nor-

Table 53.2 Differential diagnosis of exophthalmos and hydrophthalmos

	Exophthalmos	Hydrophthalmos
Vision	Normal	Blind
Position of third eyelid	Prolapsed across globe	Often in normal position or less exposed than normal
Corneal curvature	Normal	Increased due to enlarged globe size
Position of cornea when viewed from above	Deviated anteriorly	Normal
Pain	Variable, can be present on opening mouth	Present in acute cases
Pupil size	Normal	Dilated and poorly light responsive
Lens position	Normal	Can be subluxated secondary to globe enlargement
Appearance of fundus and optic disc	Normal or some fundus indentation if neoplasia present	Optic disc cupping and retinal degeneration
Oral examination	Swelling can be present behind last molar	Normal
Intraocular pressure	Normal	Elevated

mally young adults. Thus 1–2 mg/kg prednisolone and/or azathioprine at similar doses will be required initially before reducing this over several weeks. The patient will require careful monitoring for systemic side effects. Cases of masticatory muscle myositis will also require physiotherapy, with frequent exercises to open and close the mouth and thus reduce the risk of muscle fibrosis as the disease subsides. Referral should be considered for such cases.

TREATMENT OPTIONS – SURGICAL

Surgical drainage of retrobulbar abscessation is recommended and should be performed under general anaesthesia. A gag can be used to keep the mouth open and the throat should be packed. A small incision is made with a scalpel blade through the oral mucosa behind the last molar on the affected side. This should not be inserted more than a few millimetres, but extended gently with a fine pair of curved haemostats. These can be used as a probe and gently opened to try to locate the focus of infection. The surgeon should be aware of the position of the globe at all times and sharp-tipped instruments should not be used for probing as inadvertent damage to the globe or perforation of one of the many branches of the maxillary artery could occur.

It is rewarding when a gentle flow of pus is established, especially if it contains fragments of foreign body, but unfortunately this is rarely encountered, particularly if the exophthalmos is due to cellulitis and not frank abscessation. Instead the discharge is frequently serosanguinous and disappointingly scant. However, samples should still be taken for bacterial culture and sensitivity – a sterile swab can be inserted though the oral incision. The teeth should be carefully checked and extractions might be required. Postoperative treatment with broad spectrum antibiotics and anti-inflammatory agents is required. The former might need changing once the culture and sensitivity results are received.

Surgery can be recommended for cases of retrobulbar neoplasia but several factors need considering. Most orbital tumours in dogs and cats are malignant and many involve other structures – extension from the oral or nasal chambers for example. In these instances even radical surgical excision (including enucleation) might not extend the animal's survival rate even if followed with radiotherapy. Thus the patient needs careful evaluation, with radiography and ideally more advanced scanning techniques such as magnetic resonance imaging (MRI) to fully delineate the mass prior to surgery. Presurgical biopsy is also beneficial as this can give useful prognostic information. If the retrobulbar mass is contained within the orbit, such as a meningioma, then its successful removal can be possible. This is most commonly via enucleation, where exenteration – the removal of all the orbital contents – is performed. In a few cases the mass can be removed via a lateral orbitotomy approach, and the globe can be spared in such cases, but referral for this type of specialized surgery is advised.

PROGNOSIS

The prognosis for cases of exophthalmos varies according to the cause. Thus dogs and cats with cellulitis,

retrobulbar abscess and dental disease carry a good prognosis, with complete resolution and no recurrences. The prognosis for rabbits is always guarded. If a retained foreign body is present, the patient might have several episodes of presumed cellulitis before further diagnostic tests reveal its presence, but once surgically removed the outcome is normally very good.

Unfortunately, the prognosis for cases of orbital neoplasia is guarded, with more than 90% of affected dogs and cats dead within a year of diagnosis, despite various therapies including surgical removal, radiotherapy, chemotherapy and palliative treatment. However, since some primary orbital neoplasms are benign, a definitive diagnosis is necessary as these patients do carry a good prognosis with complete surgical excision of the tumour (and usually the globe).

The prognosis for the less common causes of exophthalmos, such as myositis and mucocoeles, is generally good. If the patient does not respond to treatment as expected, then other potential causes for the exophthalmos should be considered. If the condition appears to be cellulitis but fails to reduce with appropriate medication, then perhaps there is an underlying tumour or mucocoele, or even a fungal granuloma. Such cases might benefit from referral.

CASE EXAMPLE 53.1

Patient details: Labrador retriever, 2 years old, male (entire)

PRESENTATION AND HISTORY

The dog had suffered from acute diarrhoea 48 hours previously which the owner had treated by withholding food for 24 hours and then introducing small bland meals. The dog had not been unwell and seemed fine until it suddenly developed swollen eyes and was very depressed. The owner sought veterinary attention immediately.

CLINICAL SIGNS

The dog was quiet and depressed – not a normal bouncy young male Labrador at all! Marked periorbital swelling and mild exophthalmos were present bilaterally (Figure 53.3). The dog was painful on examination of the face and tried to shy away. Severe chemosis was noted bilaterally and retropulsion was reduced and resented. Menace responses were good and pupillary light reflexes were normal. No abnormalities were detected on examination of the eyes themselves. The dog was mildly pyrexic (39.5°C) and a slight generalized lymphadenopathy was noted. No abnormalities were detected on abdominal palpation.

WORK-UP

Blood was taken for routine haematology and biochemistry which was normal. Conjunctival cytology and biopsy samples were taken together with fine needle aspirates from both the submandibular and popliteal lymph nodes. The conjunctival samples suggested an inflammatory reaction only (no aetiology for this was apparent), while the lymph node aspirates both revealed reactive cells but no evidence of neoplasia. Further work-up could include investigation of extraocular muscle myositis (more common in golden retrievers than Labradors, but similar acute presentation); however, it was decided to see how the dog fared with treatment.



Figure 53.3 Two-year-old male Labrador with sudden onset periocular swelling and exophthalmos.

TREATMENT

The dog was started on symptomatic medication. An injection of buprenorphine was given and oral cefalexin and carprofen were started at standard doses. Twenty-four hours later the dog was brighter and the chemosis was reduced. Treatment was continued for another week.

OUTCOME

The dog made a complete recovery in a week and there were no recurrences. Clearly he was suffering from acute cellulitis but the aetiology of this was unclear. It could have been linked with the diarrhoea but this would suggest a systemic problem of which there was no evidence on the blood samples, although no further investigations were undertaken since the dog got better! Bilateral acute cellulitis is uncommon and an underlying neoplastic process such as lymphoma was a potential differential. Thankfully this was not the case, although it is frustrating that the aetiology remained obscure.

CASE EXAMPLE 53.2

Patient details: Golden retriever, 11 years old, male (neutered)

PRESENTATION AND HISTORY

The dog had been suffering from an intermittent sore right eye for several months. This manifested as marked conjunctival hyperaemia and blepharospasm but no cause had been established. The dog improved with topical antibiotic/dexamethasone drops and oral carprofen but the eye had not been fully normal for about 2 months. The owner then went away for a week and the dog was put into kennels. On collection the owner was told that the right eye had flared up again but she soon realized that the eye looked different from before – swollen – and returned the dog to the surgery immediately.

CLINICAL SIGNS

Right-sided exophthalmos was noted, with periorbital swelling and protrusion of the nictitans membrane (Figure 53.4). The menace response on this side was slightly reduced but pupillary light reflexes appeared normal. Moderate episcleral congestion was noted. Fundus examination revealed a large, solid ventromedial retinal detachment. The left eye was unremarkable. The dog was bright but clearly in some discomfort when the right side of the face was examined, and he did not like his mouth being opened. No nasal discharge was present or reported.

WORK-UP

A retrobulbar mass was suspected, and since ophthalmoscopy revealed the retinal detachment, this was considered more likely to be neoplastic than inflammatory. Ultrasonography would not provide further information and so under general anaesthesia skull radiographs were taken. These revealed both soft tissue infiltration and bony lysis of the posterior right nasal chambers (Figure 53.5). A diagnosis of nasal neoplasia with extension into the retrobulbar space was made. The owner was contacted and offered biopsy to confirm tumour type but declined.



Figure 53.4 Nictitans protrusion.



Figure 53.5 Intra-oral radiograph of the dog in Figure 53.4 showing destruction of the nasal turbinates and soft tissue infiltration on the right side.

TREATMENT

No treatment was given other than pain relief and topical ocular lubricant. The owner was fully aware of the guarded prognosis and did not want to put the dog through extensive surgery or radiotherapy.

OUTCOME

Four weeks after the diagnosis was made the dog was euthanized.

This case is slightly unusual in that the dog had non-specific ocular signs for several months prior to the development of exophthalmos and the subsequent diagnosis of nasal chamber neoplasia. Earlier diagnosis would not have changed the outcome, and certainly skull radiography is not indicated in cases of conjunctivitis. There had been no evidence of orbital disease initially and, as such, it is presumed that the conjunctivitis was a result of localized inflammation due to the development of the nasal mass.

54 Enophthalmos

INITIAL PRESENTATION

Abnormal appearance – sunken

PRESENTING SIGNS

The owner will present the patient due to an abnormal appearance – they notice that the affected eye is sunken with elevation of the nictitans membrane. They might think that the animal is holding its eye fully or partly closed. The condition might be present from a very early age but normally is acquired later. The patient may or may not show signs of ocular pain or systemic disease.

CASE HISTORY

In most cases there is no previous history of ocular disease or relevant systemic disease. Young animals might be presented with congenital lesions but adults are more frequently affected. In these cases there might be some history – for example, a recent traumatic event (e.g. a fight with another dog or cat), a previous episode of uveitis or chronic glaucoma with a recent change in appearance and apparent sinking of the eye. The patient might have recently been systemically ill and lost a lot of weight, resulting in a bilateral presentation, or might have undergone a surgical procedure to the head or neck. The owner (and indeed the veterinary surgeon) might not link the two episodes and therefore careful questioning is required along with examination of the patient.

CLINICAL EXAMINATION

On ophthalmic examination the first thing to determine is whether the eye is truly enophthalmic, i.e. sunken back into the socket, or whether it is just smaller than it should be. Comparison of the two eyes will assist, as will viewing the animal from above as well as from directly in front. A small eye can be present in young animals; sometimes it will be normally developed and visual, but just smaller than expected, but other ocular abnormalities, such as cataract, nystagmus and persistent pupillary membrane, might be present (Figure 54.1).

One or both eyes can be affected. If bilateral, it might be a breed-related normal enophthalmos. For example, rough collies have very long noses, with small, deep set eyes, and some degree of enophthalmos is thus inevitable – even desired by breeders. The rest of the ophthalmic examination will be totally normal in such cases (unless they also have collie eye anomaly, but that's another story!).

An acquired small globe develops following severe intraocular damage and is termed phthisis bulbi. It can follow severe trauma, uveitis or chronic glaucoma (Figure 54.2). A severely phthitic eye will usually be blind and grossly abnormal, with conjunctival hyperaemia, episcleral congestion and frequently severe keratitis such that detailed intraocular examination is not possible. Attempts at measuring intraocular pressure in such patients will be difficult since the globe becomes very soft such that obtaining accurate readings is problematical.

The patient should be examined for evidence of ocular pain which can cause retraction of the globe. Thus a corneal lesion such as an ulcer or laceration, and severe uveitis or a lens luxation can all cause sufficient ocular pain to contract the retractor oculi muscle such that the globe sinks and the nictitans membrane becomes elevated. An ocular discharge is likely to be present in painful eyes – varying from serous epiphora to overt purulent material. This should be differentiated from the accumulation of mucus seen at the medial canthus in



Figure 54.1 Microphthalmos in a young Old English sheepdog. The globe is small with the sclera more prominent, a prolapsed nictitans membrane plus cataract formation. Multiple ocular abnormalities such as these are currently being investigated by the BVA/KD/ISDS eye panel as the condition is suspected to be inherited in this breed.



Figure 54.2 Shrunken right eye (phthisis bulbi) following a road traffic accident several months previously. Hyphaema and vitreal haemorrhage had occurred at the time and although the inflammation settled the eye gradually started to shrink.

patients with deep set eyes (see Case example 12.1 on medial canthal pocket syndrome). Pupil size should be checked – a miotic pupil in an enophthalmic eye should suggest Horner's syndrome, especially if the eye is not inflamed with no evidence of uveitis. The patient should be examined in bright and dim light, and the degree of anisocoria assessed. It will be more noticeable in the dark, as the affected pupil fails to dilate. The other signs of Horner's syndrome, namely ptosis and elevation of the nictitans membrane, will usually be present, together with the enophthalmos and miosis, and all result from damage to the sympathetic supply to the eye.

General clinical examination might be normal in most cases of enophthalmos, but the patient should be checked thoroughly nonetheless. If Horner's syndrome is suspected, a thorough aural examination should be performed. A neurological examination should also be undertaken.

DIFFERENTIAL DIAGNOSES

- Microphthalmia
- Multiple ocular abnormalities
- Physiological (i.e. breed related such as rough collies)
- Phthisis bulbi
- Ocular pain
- Horner's syndrome
- Dehydration or sudden weight loss
- Following temporal muscle atrophy
- Tetanus

CASE WORK-UP

For congenital cases of enophthalmos due to microphthalmia, or purely physiological due to breed, there is clearly no further work-up required. The same is true for cases of phthisis bulbi, where the previous history and clinical examination of the patient are diagnostic. If a source of ocular pain is identified, then the work-up will be directed at determining the cause of this – and is discussed in the case examples on corneal ulceration, uveitis, lens luxation, etc. Cases due to temporal muscle atrophy will similarly be obvious from the clinical examination and previous history. Thus the most frequent cases of enophthalmos which require significant further investigation is when they are due to Horner's syndrome.

Signs of Horner's syndrome include:

- miosis
- protrusion of the nictitans membrane
- enophthalmos
- ptosis
- (ipsilateral conjunctival and dermal vasodilation).

Interruption to the sympathetic supply to the head can occur anywhere along the three-neurone pathway (Figure 54.3). Patients with central denervation usually

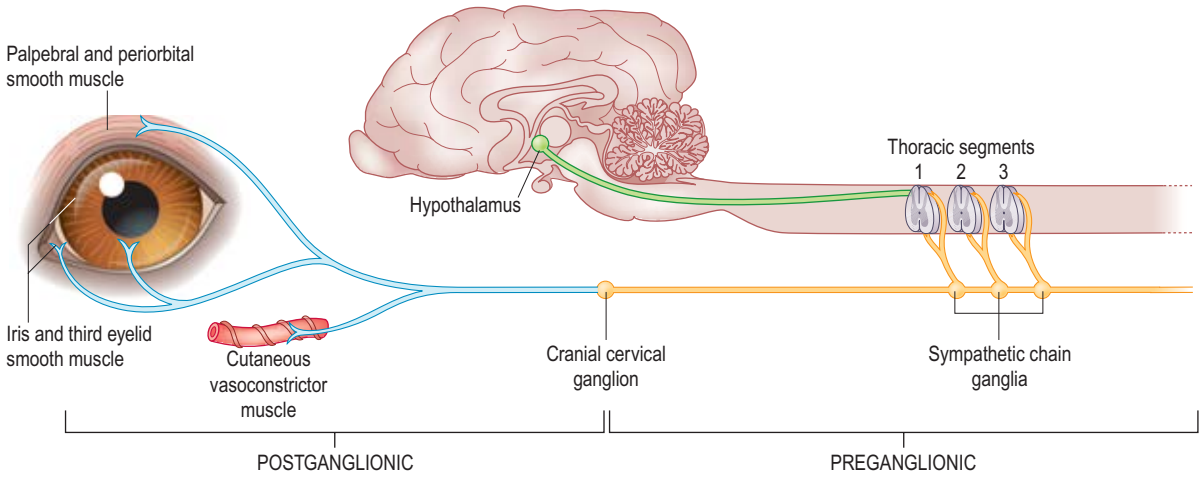


Figure 54.3 Diagram showing the sympathetic pathway to the eye which, if disrupted, results in Horner's syndrome.

Table 54.1 Causes of Horner's syndrome in dogs and cats

Cause	Approx. % canine cases	Approx. % feline cases
Idiopathic	45	25
Road traffic accident	20	25
Bite wounds	10	14
Cervical disc protrusion or surgery	10	–
Otitis media	13	12
Foreign body	–	12
Iatrogenic	2	12

have other neurological symptoms, which are frequently more obvious than the ocular signs. Patients with preganglionic lesions are often otherwise asymptomatic, whereas those with third order or postganglionic Horner's syndrome can be variable – vestibular disease and evidence of otitis media might be present – but many are subclinical with no other symptoms.

Pharmacological testing is a useful way to differentiate between central or preganglionic lesions and third order or postganglionic lesions. It relies on the phenomenon of denervation hypersensitivity – postganglionic fibres which have been deprived of their normal transmitter will react more quickly and to a greater degree than a normal nerve fibre. Phenylephrine, a direct acting sympathomimetic agent, is used; normally the commercially available 10% solution is diluted down to 1% in sterile saline. One drop is applied to both eyes (the contralateral eye acts as the control) and the patient is observed at 5-minute intervals. The size of the pupils and degree of third eyelid elevation are the most readily monitored features. In a postganglionic (or third order) Horner's syndrome the affected pupil will dilate within 20 minutes; the normal pupil does not dilate in this time but takes 60–90 minutes to do so (the same as central (first order) and preganglionic (second order) causes).

Once the order has been established, further diagnostic tests can be directed to the area in question. Thus radiography of the cervical spine, chest and anterior thorax can be useful and, if postganglionic, skull radiography to investigate middle ear disease can be performed. The chest and neck should be carefully examined for bite wounds and other injuries, clipping the fur if necessary. Blood tests should be considered – some cases can be associated with hypothyroidism due to a peripheral neuropathy for example. Unfortunately, many cases of Horner's syndrome remain idiopathic despite extensive work-up (Table 54.1).

NURSING ASPECTS

If the animal has enophthalmos due to anterior segment pain, then sympathetic nursing with the use of appropriate analgesics is essential. However, most cases of enophthalmos are pain-free. This is certainly true of Horner's syndrome and owners need reassuring that the eye is not painful to their pet – its sunken, droopy appearance and pinkness due to the nictitans membrane elevation can make owners think that their pet is in discomfort.

EPIDEMIOLOGY

Enophthalmos which is due to microphthalmos can be inherited. The breeds of dog which have very small eyes, such as rough collies, are at risk. Multiple ocular abnormalities, with microphthalmia as well as nystagmus, cataract and so on, can also be inherited. Miniature schnauzers, West Highland white terriers, cocker spaniels, dobermanns, golden retrievers and Old English sheepdogs are all at-risk breeds in the UK. However, sporadic cases can occur in any breed, and can be due to intrauterine damage to the developing fetuses. Cases of phthisis bulbi are rare and sporadic, and tend to occur in older patients with a history of ongoing ocular disease.

Horner's syndrome can be iatrogenic – following aural or neck surgery or with severe swelling and bruising after jugular venepuncture for example. Young dogs that get into fights and are attacked on the neck can develop Horner's syndrome, as well as other traumatic episodes such as road accidents and foreign bodies. Breeds that suffer from vestibular disease and middle ear infections could be over-represented with Horner's syndrome. An idiopathic form is encountered in golden retrievers, where it is most common in middle-aged male dogs. It is second order (or preganglionic) and usually unilateral, and tends to resolve gradually 8–12 weeks after initial presentation.

CLINICAL TIPS

- Examine the orbit carefully to differentiate a small eye (microphthalmos) from a sunken eye (enophthalmos)
- Look for evidence of ocular pain – entropion, ectopic cilia, corneal ulceration, uveitis, acute glaucoma, lens luxation and so on can all result in enophthalmos
- Check pupil sizes – affected pupil is smaller with Horner's syndrome and uveitis
- Look for other signs of Horner's syndrome – miosis, ptosis and nictitans elevation
- Do not forget tetanus – if bilateral, third eyelid protrusion and enophthalmos are present – look at the facial expression and examine carefully for wounds

TREATMENT OPTIONS – MEDICAL

Microphthalmic eyes and those with multiple ocular abnormalities rarely require treatment (with the exception of those with severe cataracts). If the enophthalmos is due to ocular pain, then treatment of the underlying cause for this will relieve the symptoms.

Treatment for Horner's syndrome will be directed towards the cause, if one is identified. Since many third order (postganglionic) cases are due to middle ear infections, a course of broad spectrum systemic antibiotics (e.g. 3–4 weeks of cefalexin) will be curative and the signs of Horner's will gradually reduce. If due to trauma such as a road traffic accident or iatrogenic surgical trauma, then the symptoms should also improve with time as the post-surgical swelling decreases and normal nerve function can return. Cases of idiopathic Horner's syndrome in the golden retriever gradually improve and resolve in 8–12 weeks. Owners will notice that the dog's eye looks better in the morning and when it is alert but worsens during the day as it becomes more tired before eventually resolving completely.

Topical treatment is rarely necessary. Diluted phenylephrine will abolish the clinical signs of miosis, ptosis, enophthalmos and nictitans membrane protrusion, and can be used in the rare bilateral cases if vision is compromised. However, the drops frequently sting, and once owners have been reassured that the condition is not painful despite its appearance, they are usually happy to allow the symptoms to gradually resolve more naturally.

TREATMENT OPTIONS – SURGICAL

The majority of cases of enophthalmos will either not be treated at all or have medical management. Sunken eyes due to phthisis bulbi might require enucleation,

particularly if painful or if conjunctival infections occur. Enucleation in cats with phthisis bulbi is recommended to prevent intraocular sarcoma formation later in life. In rare cases of microphthalmos, or enophthalmos following illness and loss of the retrobulbar adipose tissue, the patient might be visually compromised by the profound elevation of the nictitans membrane occluding the visual axis. These patients might benefit from a partial resection of the nictitans membrane, but since the function of this structure is essential in maintaining normal corneal health, the surgery should never be undertaken lightly, and specialist advice should be sought.

PROGNOSIS

The prognosis for cases of enophthalmos is reasonable. Small eyes might have other abnormalities which affect vision, but the enophthalmos itself is unlikely to cause too many problems other than allowing an accumulation of mucus in the ventral conjunctival fornix which requires regular removal to prevent bacterial conjunctivitis from developing. Occasionally entropion will be present which can easily be corrected with surgery. Many eyes with phthisis bulbi should be removed, but some can settle such that, although grossly abnormal, they cause the patient no ocular pain nor predispose to repeated infections and so on.

Providing there is no sinister condition underlying the Horner's syndrome – a spinal tumour or mediastinal mass for example – then the prognosis is generally favourable. Most cases resolve with time, even in the absence of a definitive diagnosis, and the patient is generally well despite their appearance. Owners are usually happy to wait and see once they understand that there is nothing wrong with the eye itself. If the condition does not resolve, or other symptoms develop, then referral to an ophthalmologist or neurologist should be considered for specialist investigation.

CASE EXAMPLE 54.1

Patient details: Domestic short haired cat, 18 months old, male (entire)

PRESENTATION AND HISTORY

The cat had been missing for 48 hours and returned with an abnormal eye. The owner presented him immediately. Apart from two previous cat bite abscesses there was no other history.

CLINICAL SIGNS

General clinical examination was unremarkable. The right eye was enophthalmic, with protrusion of the third eyelid, ptosis and miosis (Figure 54.4). The anisocoria was more pronounced in the dark as the right pupil failed to dilate. However, pupillary light reflexes were present and intraocular contents were normal.

WORK-UP

It was decided to try to establish the order of the Horner's syndrome. One drop of 1% phenylephrine was placed in each eye, and checks were made every 5 minutes, looking at pupil size and third eyelid position. After 45 minutes the right pupil dilated, followed by the left 5 minutes later, and both nictitans membranes were in their normal position at 45 minutes. This led to a diagnosis of preganglionic (i.e. first or second order)



Figure 54.4 Horner's syndrome in the right eye (compare to Figures 54.2 and 5.18 which initially look very similar).

Horner's syndrome. Blood samples were to be taken for routine haematology and biochemistry (looking for a focus of infection for example) plus feline leukaemia virus (FeLV) and feline immunodeficiency virus (FIV). On clipping the fur from the neck ready for venepuncture, several scabs were removed and a small hard swelling was felt just under the skin. The blood tests were unremarkable.

TREATMENT

The cat was anaesthetized and the neck swelling was investigated. It was excised and found to be a small granulomatous lesion with a centre of inspissated pus. No foreign body was identified. Histopathological examination was not deemed necessary. Permission was given to castrate the cat. He was treated with oral amoxicillin/clavulanic acid tablets for a week.

OUTCOME

The neck wound healed routinely. Three weeks later the Horner's syndrome started to improve and after another month had fully resolved.

This case of Horner's syndrome was second order. The cause was not initially obvious but on closer examination the wound on the neck clearly suggested a cat fight which had caused local swelling and temporary damage to the sympathetic nerves in the neck. Clearly careful examination of all cats presented with similar symptoms is indicated! Had the neck not been clipped for venepuncture, the bite wounds might not have been identified.

55 Proptosis

INITIAL PRESENTATION

Abnormal appearance – red, swollen

PRESENTING SIGNS

Owners will present patients with proptosis as an emergency. The sudden alteration in appearance will be the most striking abnormality, and most will be aware that the eye has come out of the socket (they frequently state on the telephone that the eye has fallen out). Usually only one eye is affected but occasionally both are involved. Some animals will have other injuries, especially if they have been involved in a road traffic accident or a fight. Brachycephalic breeds are predisposed.

CASE HISTORY

A traumatic incident is usual. This is most commonly a dog fight or a road traffic accident but in some animals, particularly brachycephalic dogs, the trauma can be minimal – restraint for a nail clip for example.

CLINICAL EXAMINATION

On ophthalmic examination the forward displacement of the globe will be obvious. However, the patient should be fully examined and evaluated since other injuries might be present – indeed these might be life threatening. It follows that if a patient has a prolapsed eye following a road accident they have most likely been hit on the head, and skull fractures, concussion and so on might be present. Bite wounds might be severe and in some animals the prolapsed eye can be the least of their problems.

Once the eye is prolapsed the periorbital tissues swell, bruising and haemorrhage ensue and both chemosis and conjunctival haemorrhage occur in response to the injury so these will all be present on ophthalmic examination. The eyelids tightly close around the retrobulbar tissue which impedes venous drainage and further exacerbates the swelling and redness. The eye should be assessed as far as possible in order to provide some prognostic indicators. If the optic nerve and multiple extraocular muscles are severed, then the prognosis is hopeless and enucleation should be performed. If the cornea or sclera is ruptured the same is true, but corneal ulceration does not preclude globe replacement. Pupillary light reflexes should be assessed but are not necessarily reliable. The affected pupil might be miotic (e.g. due to concurrent uveitis) or can be dilated (optic nerve damage). A lack of any light reflex cannot be readily interpreted; however, if a direct and/or consensual constriction is noted, these are favourable signs. Many patients will have hyphaema which can preclude any intraocular examination and indicates severe intraocular trauma. Table 55.1 provides some prognostic indicators for proptosis.

DIFFERENTIAL DIAGNOSES

The diagnosis is obvious from clinical examination and can easily be differentiated from exophthalmos by the fact that the eyelids are closed behind the protruding globe.

CASE WORK-UP

If the patient has suffered from severe trauma then it must be fully stabilized and further injuries assessed before attempting to replace the globe. Rushing in to

Table 55.1 Prognostic indicators in cases of proptosis

Factor	Positive prognosis	Negative prognosis
Breed	Brachycephalic	Doliocephalic dog, cat
Optic nerve	Stretched but intact	Ruptured
Extraocular muscles	Not visible or swollen	Visibly ruptured
Sclera and cornea	Intact	Ruptured
Ocular tension	Normal	Soft – suggests posterior scleral rupture
Corneal ulceration	Superficial or absent	Deep or infected
Intraocular haemorrhage	Absent	Present
Pupillary light reflex	Pupil size unreliable but positive direct and/or consensual reflex are positive signs	No direct reflex or consensual response

sort out the eye without realizing that the patient has a pneumothorax or depressed skull fracture for example is definitely not in the patient's best interests. Thus intensive care, fluid therapy, radiography and so on might be required before surgery.

NURSING ASPECTS

The prolapsed globe must be kept moist to maintain ocular health as much as possible. This can be done with soft saline-soaked swabs, lubricating ointment or jelly or bland ophthalmic ointment. The patient must be prevented from further traumatizing the eye – they can be cuddled and held if appropriate but might require an Elizabethan collar. Surgery is advised as soon as the patient is sufficiently stable.

EPIDEMIOLOGY

Proptosis is the anterior prolapse of the globe beyond the bony orbital rim, with resultant spasm and closure of the eyelids behind it. Canine breeds with shallow orbits and large palpebral fissures such as Pekingese and Pugs are more commonly affected as it takes very little for these globes to become displaced – they have little keeping them in place! Palpating the orbital rim of an extremely brachycephalic dog will reveal how exposed the globe is. Doliocephalic breeds and cats require far more trauma to induce eyeball prolapse. There is no age or sex predisposition and the condition occurs as a sporadic problem.

CLINICAL TIPS

- Tell the owner to keep the eye moist while travelling to the surgery – a damp flannel or tea towel is ideal
- Check the rest of the patient carefully
- Consider replacement rather than immediate enucleation unless severe damage is obvious

TREATMENT OPTIONS – SURGICAL

Proptosis is a surgical emergency. It is preferable to replace the globe if at all possible rather than enucleate immediately – the latter can be performed later if necessary but some eyes will settle back in position surprisingly well. The globe should be kept lubricated until replaced under general anaesthesia. Minimal clipping is required since the lids will already be swollen and bruised and further damage should be avoided. The globe and adnexa are flushed with copious amounts of sterile saline. Sometimes it is possible to gently push the globe back behind the lids but most cases are too advanced, with excessive retrobulbar swelling such that this is not successful and certainly the globe should not be forced.

The standard way to replace the globe is to perform a lateral canthotomy to widen the palpebral fissure initially – it makes the surgery much easier if this is done first! Sutures are pre-placed in the upper and lower lids with long ends which are pulled out over the globe as the latter is gently pushed back towards the socket (a tongue depressor is a useful device for this, or failing that the handle of a pair of thumb forceps or similar can be used). The surgery is more likely to be successful if

the lids are gently pulled out and back over the globe rather than trying to push the globe back into the already swollen retrobulbar tissue. A spey hook can be useful to evert the lid margins and this will make placement of the sutures easier (Figure 55.1). Some texts advise suturing a third eyelid flap before the tarsorrhaphy but this is not always possible. The lid sutures should be placed two-thirds thickness so that they cannot rub on the cornea. If the lids are very swollen, stents can be used. The canthotomy should be repaired. Topical ophthalmic ointment can be placed between the sutures at the time of surgery.

Postoperative treatment with systemic antibiotics and anti-inflammatory agents is routine. Either NSAIDs or occasionally steroids can be used – sometimes an initial dose of dexamethasone will be given by injection, followed 48 hours later by 5 days of a systemic NSAID. Topical ointment – a broad spectrum antibiotic such as chloramphenicol or polymyxin B – can be applied two to three times daily via the medial canthus in cooperative patients. Topical atropine ointment is also useful but it is important not to 'clog up' the tarsorrhaphy site with excessive stale medication. The sutures are removed after about 10 days and a decision can then be made as to whether the globe is functional, or at least salvageable. Further surgery such as repair to a ruptured medial rectus muscle, or permanent partial tarsorrhaphy to reduce exposure keratitis and also the risk of future proptosis, can also be considered.

PROGNOSIS

Proptosis carries a guarded prognosis. Less than 30% of dogs with traumatic ocular proptoses will gain any return of vision, while the figure is significantly lower in cats. Skull shape and species will affect the prognosis – it is better in brachycephalic patients, but many dogs with a 'normal' skull conformation and most cats will require enucleation.

Once the globe has been repositioned the most frequent complications are blindness, lateral strabismus and exposure keratitis. The blindness is due to optic nerve or severe intraocular damage and is normally irreversible. The lateral strabismus occurs because the medial rectus muscle is the shortest and thus the most readily ruptured as the eye proptoses. Occasionally it can be identified and repaired after the initial replacement surgery and once the swelling has subsided, but this surgery is not easy. If the muscle is stretched and not

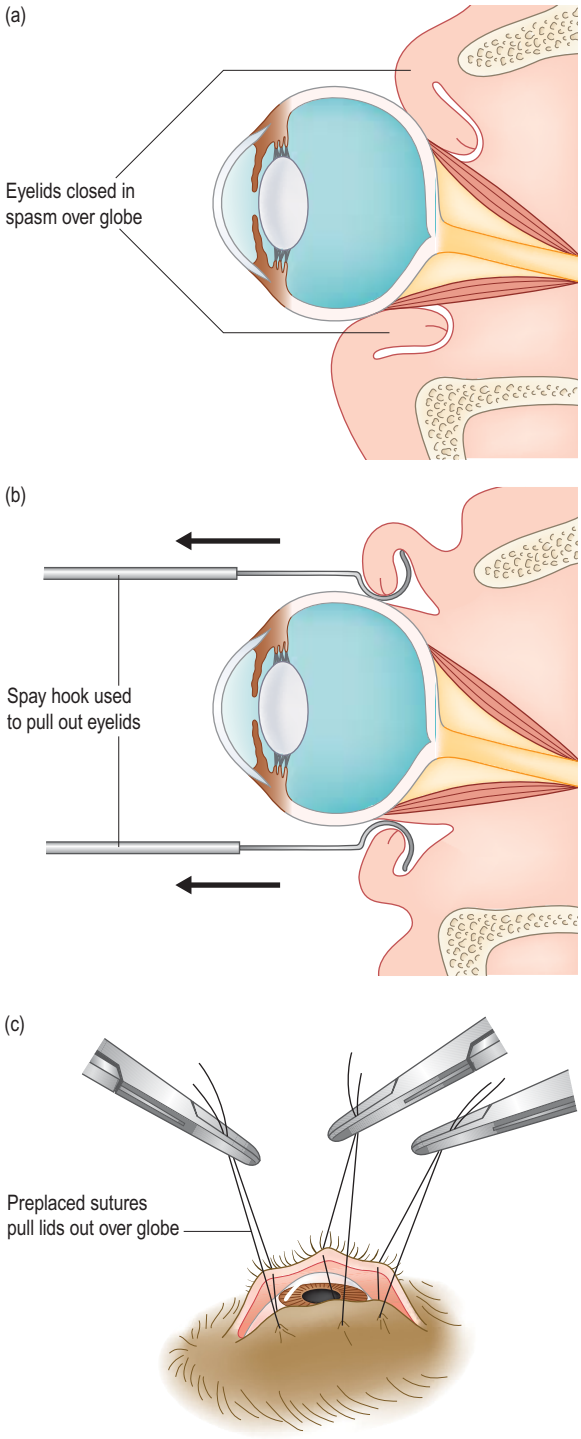


Figure 55.1 Diagram illustrating how to replace a proptosed globe.

fully torn, some improvement in the exotropia can occur after a few months. The exposure keratitis develops following compromise to vascular and nervous supply, with a combination of reduced corneal sensation and lagophthalmos. Some keratoconjunctivitis sicca can also occur.

Frequent ocular lubrication is required to prevent ulceration and some patients will benefit from a partial permanent lateral or medial tarsorrhaphy. The latter will be more appropriate if a lateral strabismus is present as it will protect the exposed sclera and give the eye a more normal appearance. A partial tarsorrhaphy will also provide some globe support, thus reducing the risk of subsequent prolapse. Some patients will require enucleation despite valiant attempts at initial globe replacement.

56 Limbal melanoma

INITIAL PRESENTATION

Abnormal appearance – swollen/colour change

PRESENTING SIGNS

The condition can be seen in dogs (particularly German shepherd dogs and Labrador retrievers) and less commonly in cats, and owners present the patient because they have noticed a change in appearance to the eye. Normally only one eye is affected. They have noticed a dark lesion at the junction between the white and clear parts of the eye which is slightly raised and enlarging. Most commonly the lesion is at 12 o'clock but it can be anywhere around the circumference of the cornea. The lesion has usually been noted a few days or even weeks prior to presentation but since the pet shows no signs of discomfort or visual impairment the owners do not tend to present them immediately on noticing the lesion. Indeed, sometimes they will have been aware of it for many months and only point it out to the clinician when the pet is presented for another reason such as vaccination.

CASE HISTORY

The patient is normally clinically well with no relevant current or previous systemic or ocular problems. No treatment has usually been given for the ocular lesion and the owner has been aware of it slowly enlarging.

CLINICAL EXAMINATION

General clinical examination is normal. On ophthalmic examination the patient has normal menace responses and vision is unaffected. Mild localized conjunctival hyperaemia can be present but the eye is certainly not red or inflamed in appearance. No significant ocular discharge is present. A smooth, shiny, raised pigmented mass is present at the limbus. Occasionally it is only poorly or non-pigmented but most are very dark in colour. The lesion covers the limbus and extends into the cornea, with some localized corneal oedema around it. Sometimes an arc of corneal lipid is also present distal to the lesion (lipid keratopathy). It is usually 3–5 mm in diameter on presentation. It is most frequent between the 12 and 3 o'clock position but can be situated anywhere around the limbus – ventral and ventromedial lesions are often much larger before being presented simply because they are more difficult to see. Pupillary light reflexes are normal and no dyscoria is present. The iris is unaffected – it is of normal colour and shape. Viewing the patient from the side with a pen torch will reveal that the lesion is outside the globe and does not include the iris. No uveitis or other intraocular changes are present. Intraocular pressure is normal.

DIFFERENTIAL DIAGNOSES

- Uveal melanoma (see Figure 37.2)
- Extension of ciliary body melanoma (see Figure 37.6)
- Corneal/scleral laceration with iris prolapse (see Figure 24.1)
- Nodular episcleritis
- Pigmentary keratitis
- Foreign body
- Feline sequestrum (see Figure 30.1)

CASE WORK-UP

The main differential for limbal melanoma is extension from a uveal (i.e. iris or ciliary body) neoplasm. This can be achieved by ultrasonography. A limbal melanoma will be seen as a raised mass on the outside of the globe – a stand-off is necessary to allow visualization of such superficial lesions (a surgical glove filled with water or ultrasound gel works very well). A uveal melanoma will show thickening, and usually a discrete mass, within the iris/ciliary body, which then extends outwards towards or even through the sclera. In addition, other ocular signs are more commonly present with uveal tumours (see Table 56.1).

Gonioscopy is also recommended and referral for this should be considered. With a limbal melanoma there will be no involvement of the drainage angle, while uveal melanomas normally result in pigment deposition and closing of the iridocorneal angle in the affected sector.

Biopsy might be necessary if there is any doubt over the pathology involved, especially if the lesion is poorly or non-pigmented, and thus similar in appearance to nodular episcleritis. Although this can be done with sedation and topical anaesthesia, it is frequently more sensible to anaesthetize the patient.

NURSING ASPECTS

Patients with limbal melanoma are normally surgical candidates and require topical medication postoperatively. Nurses should ensure that owners are capable of applying this but no other specific care is required.

Table 56.1 Differentiation between uveal and limbal melanoma

Uveal melanoma	Limbal melanoma
Dyscoria can be present	Pupil normal
Darkening of iris	Iris normal colour
Mass visible within iris	Mass on surface of globe
Uveal inflammation common	No uveitis
Intraocular pressure can be low	Intraocular pressure normal
Drainage angle can be affected	Drainage angle normal
Difficult to biopsy	Relatively easy to biopsy
Relatively common	Uncommon
Low risk of metastasis in dogs (4%), higher in cats (60%)	Metastasis not reported

EPIDEMIOLOGY

Limbal melanomas are also called epibulbar melanomas or melanocytomas. Heavily pigmented breeds such as the German shepherd dog and black Labrador retrievers have a predisposition to the development of melanomas and this certainly applies to limbal melanomas. Some reports suggest that females are at a slightly greater risk than males. However, any breed, and either sex, can be affected and rarely the condition is also observed in cats.

Two distinct age groups are seen with the condition – young dogs aged 2–4 years and an older group aged 8–11 years. The condition is potentially more serious in the younger age range since they tend to grow more rapidly and can be locally invasive. Thus, if left untreated, they can result in intraocular extension and all the consequences that this entails. In older dogs they normally behave in a much more benign manner and can be stationary or only very slowly growing. Metastasis has not been reported.

Histopathologically limbal melanomas consist of a predominance of plump melanocytes. Although the condition is not particularly common, it is very important to recognize and differentiate from uveal melanomas since treatment and prognosis are very much better for limbal melanomas.

CLINICAL TIPS

- Differentiate from uveal melanoma (Table 56.1)
- Look at the eye from the side, shining a light into the eye – the mass will be outside the globe. Looking only from in front will be confusing since the shadow from the lesion might mimic uveal involvement
- Consider ultrasonography
- Refer if unsure of diagnosis – do not enucleate!!

TREATMENT OPTIONS – MEDICAL

Although there is no medical management for this condition, in older patients with slowly growing lesions, a 'wait and see' approach can be acceptable. Since the lesions tend to be benign in older dogs, and rarely progress to affect vision, patients can be checked at regular intervals (initially every month then reducing to every 3

months). The lesions should be accurately measured with callipers, and diagrams or photographs used to document any changes. Should the lesion start to enlarge, then surgery is appropriate. Surgery is usually advocated in younger dogs and it might be prudent to refer such cases.

TREATMENT OPTIONS – SURGICAL

Surgical removal of limbal melanomas is advised in younger patients and in lesions which are enlarging. If equipped with microsurgical instruments and magnification this can be performed in general practice but referral should be considered. The lesion is removed via superficial keratectomy under general anaesthesia. The corneal portion of the lesion is delineated 2 mm beyond its leading edge with a scalpel blade (Beaver type) and the cornea is grasped with delicate forceps and dissected beneath the pigment towards the limbus. Significant haemorrhage will occur as the limbus is transected and sterile cellulose spears soaked in 10% phenylephrine will help to reduce this. The conjunctival portion of the mass can then be excised. All resected tissue should be submitted for histopathological examination. Normally some pigment will be left around the limbus.

Adjunctive therapy such as photocoagulation (using a diode laser), cryosurgery or β-irradiation has been advocated to destroy any remaining neoplastic cells and most ophthalmologists recommend this. Simply debulking with surgery will not prevent regrowth. The alternate approach is to surgically remove all pigmented tissue. However, this will usually leave a much weakened limbal area (sometimes even removing a full thickness section) and further support in the form of grafts of nictitans cartilage, corneal transplants or adjacent corneal transposition will be required. These are obviously specialist procedures!

Postoperative treatment usually consists of topical antibiotics until the cornea has epithelialized, followed by a few days of topical steroids to reduce any vascular reaction during healing. Patients should be checked every 6 months for signs of regrowth.

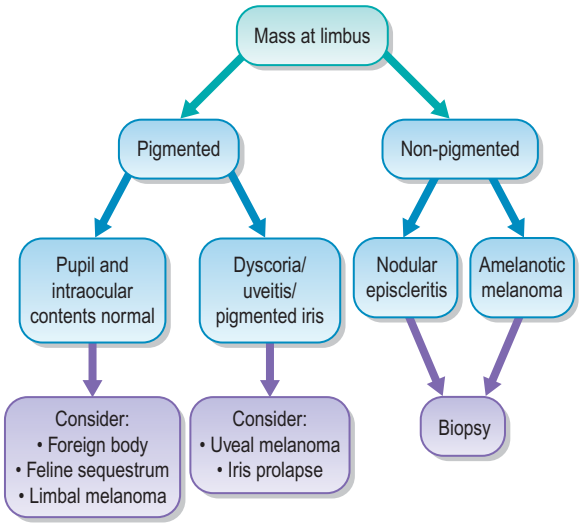


Figure 56.1 Decision-making tree – limbal melanoma.

Enucleation is not necessary in most cases of limbal melanoma – only in cases which have been neglected or mismanaged such that intraocular extension and subsequent uveitis/glaucoma have developed. It should never be suggested as a first line treatment.

PROGNOSIS

The prognosis for limbal melanomas is good. Older patients can be monitored and many do not require surgery since the lesions tend to be benign. With surgical excision, or extensive debulking followed by laser photoablation, cryosurgery or occasionally local irradiation, younger patients are normally cured of the disease. If left untreated, the more aggressive form in younger dogs will result in intraocular extension and resultant uveitis and secondary glaucoma. These eyes will require enucleation but with early recognition and appropriate management this can be avoided. Metastasis from limbal melanomas has not been reported.

CASE EXAMPLE 56.1

Patient details: Border collie, 5 years old, female (neutered)

PRESENTATION AND HISTORY

The owner had been aware of a black lesion ventrally in the right eye for several months but the dog was not bothered by the lesion and it was only when the owner noticed that it had enlarged in the previous month that she presented the dog. No other problems were reported.

CLINICAL SIGNS

General clinical examination was unremarkable. Vision was good. Abnormalities were confined to the left eye where a black shiny mass was noted at the ventrolateral limbus, measuring approximately 3 × 4 mm (Figure 56.2).

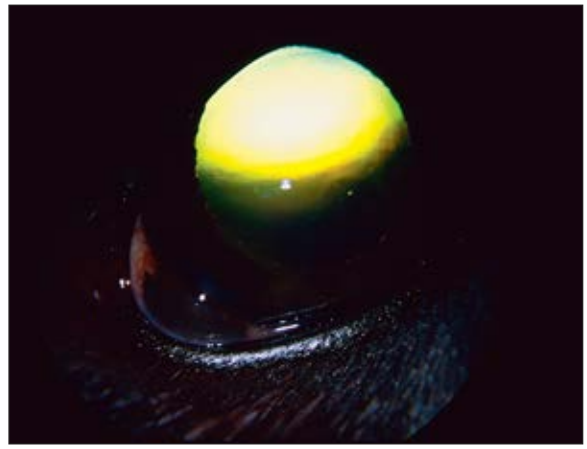


Figure 56.2 Ventrolateral limbal melanoma in a 5-year-old collie.

WORK-UP

No work-up was undertaken initially but the patient was examined again a month later, and the lesion had grown. The patient was referred. Gonioscopy revealed a normal drainage angle and a diagnosis of limbal melanoma was reached.

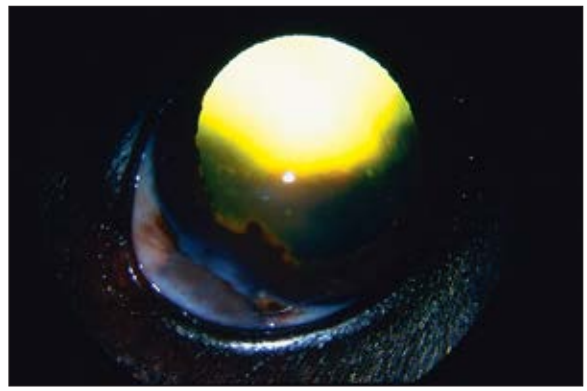


Figure 56.3 Same eye as pictured in Figure 56.2, 12 months following resection and laser ablation. Some pigment scarring is present but there is no evidence of tumour regrowth.

TREATMENT

Since the dog was young and the lesion was growing, surgery was advised. The mass was removed via superficial keratectomy followed immediately by laser photoablation of the tumour base. Histopathology confirmed a limbal melanoma. Topical chloramphenicol was dispensed four times daily for 5 days along with oral carprofen. After 5 days the cornea was fluorescein negative and dexamethasone drops (Maxidex, Alcon) were supplied twice daily for 10 days.

OUTCOME

The dog did very well. The surgical site healed quickly and although some pigment remained it was flat within the episclera and did not alter in the 12 months of follow-up (Figure 56.3).

This case illustrates that with accurate diagnosis and appropriate treatment the prognosis for limbal melanoma is very good.

57 Episcleritis

INITIAL PRESENTATION

Abnormal appearance – red

PRESENTING SIGNS

Episcleritis is seen in dogs rather than in cats and the animal will be presented because of a red eye – usually affecting only one eye, but sometimes both. Often the owner thinks that the dog has conjunctivitis. The condition is not usually painful and the owner reports no apparent visual disturbance.

CASE HISTORY

In most cases there is no previous history of ocular disease or relevant systemic disease. The condition is usually insidious in onset, such that there has been some intermittent ocular redness for days to weeks before the animal is brought to the surgery – the owner may notice that this is worsening. Usually there is minimal discomfort – perhaps mild blepharospasm only – and the owner will not be worried by this.

CLINICAL EXAMINATION

General clinical examination is unremarkable. On ophthalmic examination the most obvious abnormality is the red eye. Marked episcleral congestion will be present, together with some overlying conjunctival hyperaemia. Thus the superficial conjunctival vessels – the thin bright red branching ones – will be engorged, while the deeper, darker, straighter episcleral vessels will be thickened and more tortuous than usual. There will be some diffuse swelling in the area but this will not be excessive (not true chemosis) (Figure 57.1).

The episcleral congestion can be diffuse, affecting large quadrants of the globe, or can be a discrete nodular swelling. With the latter, the lesion is not usually firmly attached to the underlying sclera and can be moved slightly with a moistened cotton bud. However, if the nodule extends into the cornea it will be less mobile. Some peripheral corneal oedema will be present, giving a blue tinge to the cornea adjacent to the limbus. This will be denser and affect slightly more of the cornea where the episcleral involvement is more severe. Fluorescein testing is negative.

The eye will be visual (unless very advanced disease is present involving the posterior segment but this is unusual) with a normal menace response, pupillary light reflexes and no evidence of intraocular disease. Intraocular pressures will be normal. In most cases fundus examination is normal, but in very severe cases there might be evidence of active chorioretinitis (with fuzzy, grey retinal elevations and perivascular cuffing as a direct extension from the outer episcleral inflammation) and even optic neuritis in a few cases.

DIFFERENTIAL DIAGNOSES

- Conjunctivitis
- Chronic superficial keratitis
- Uveitis
- Panophthalmitis
- Healing peripheral corneal ulcer
- Glaucoma
- Limbal neoplasia (for nodular form)
- Retrobulbar cellulitis



Figure 57.1 Mild episcleritis in a golden retriever. Note the slight episcleral swelling and hyperaemia with corneal vascularization and corneal oedema.

CASE WORK-UP

The condition can usually be diagnosed on clinical examination alone. However, in some patients, particularly if suffering from the nodular form of the disease, biopsy is recommended to differentiate from neoplastic disease. This requires heavy sedation or general anaesthesia, and the biopsy should be deep enough to include the episcleral tissue, not just the conjunctiva (it is for this reason that general anaesthesia is advised).

NURSING ASPECTS

Apart from treating with topical and sometimes systemic medication, there is no specific nursing required with cases of episcleritis.

EPIDEMIOLOGY

Episcleritis is a local inflammatory condition affecting the episcleral tissue close to the limbus and can be divided into diffuse and nodular forms. It can also occur secondarily to other ocular diseases such as uveitis, glaucoma and panophthalmitis, but in these instances evidence of other ocular abnormalities will be present. The cause for the disease is unknown, but it is more common in certain breeds – collies and their crosses in particular, but also cocker spaniels, golden retrievers and Shetland sheepdogs. It tends to affect young adults, 4–8 years of age. The condition has not been reported in cats as a primary lesion. It is certainly thought to have an immune-mediated aetiology but further understanding is lacking.

In the collie breeds there are often multiple episcleral nodules and both eyes can be affected. There are various confusing synonyms for this nodular form – nodular fasciitis, limbal granuloma, non-necrotizing scleritis, nodular granulomatous episclerokeratitis for example. Histologically the condition is characterized by a mix of inflammatory cells – histiocytes, lymphocytes, plasma cells and neutrophils. The diffuse form has the same framework of inflammatory cells.

CLINICAL TIPS

- Check carefully for intraocular involvement
- Consider the breed!
- Measure intraocular pressures to help distinguish from uveitis
- Biopsy if uncertain, especially in nodular forms

ANATOMY AND PHYSIOLOGY REFRESHER

The sclera is the fibrous outer tunic of the globe and provides shape and support to the intraocular contents as well as a route of entry for the nerves and blood vessels. It is composed of a meshwork of branching collagen fibres with scattered fibroblasts and melanocytes. The limbus is the transition zone between the sclera and anterior cornea, and the collagen fibres become aligned in a parallel fashion, assisting in corneal transparency. The density of melanocytes is higher at the limbus. The episclera is the thin collagenous, elastic and vascular layer which overlies the sclera and is itself covered with conjunctiva. Any pathological process affecting the episcleral tissue will also involve the overlying conjunctiva, and sometimes the underlying sclera in severe cases.

TREATMENT OPTIONS – MEDICAL

Generally medical management is appropriate for cases of diffuse episcleritis, and many cases of the nodular form. Topical steroids are the first choice of treatment but require frequent administration. Thus prednisolone acetate or dexamethasone alcohol should be applied four to eight times daily initially, reducing the frequency as the condition subsides. Mild cases such as the dog shown in Figure 57.1 would be expected to respond well to this approach. Long-term, low doses may be required to keep the condition in remission.

If the condition does not respond after 2 weeks of topical medication, then systemic drugs should also be introduced. These can be steroids at immunosuppressive levels (e.g. prednisolone 1–2 mg/kg/day) or azathioprine at the same dose rate. Again the dose should be reduced as the clinical symptoms reduce to the lowest level possible to prevent recurrence. Some dogs can be weaned off the medication completely but relapses are possible.

If steroids are not appropriate for the patient, an alternative treatment is to use niacinamide and doxycycline at an empirical dose of 500 mg of each agent three times daily in dogs weighing over 10 kg. This can be reduced to twice daily as the condition resolves. However, some patients do not respond to this regimen.

TREATMENT OPTIONS – SURGICAL

Nodular forms of episcleritis can be treated surgically by excision, with a superficial keratectomy if the cornea is also involved. The excised tissue should be sent for histological examination. Cryosurgery is also effective. Most cases will also require adjunctive medical management as above to prevent recurrences.

PROGNOSIS

The prognosis for control of both diffuse and nodular episcleritis is good, although complete cure is not always possible. Some patients will be self-limiting, some suffer from occasional recurrence in one or both eyes, while others will require permanent medication to keep the condition controlled. If untreated, the condition will progress to affect the full circumference in diffuse cases, and multiple nodules will enlarge in the nodular form. The corneal oedema can progress centrally, and both corneal vascularization and secondary lipid keratopathy can develop – both of which can affect vision.

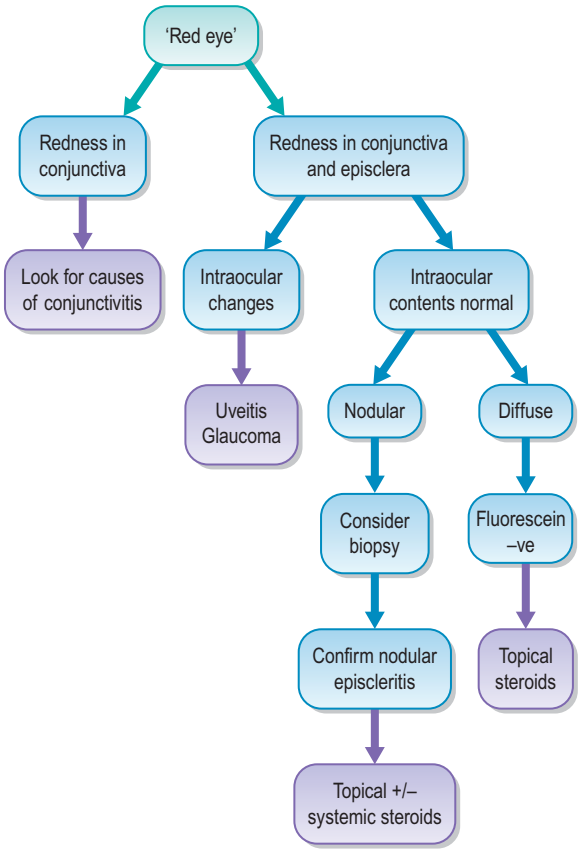


Figure 57.2 Decision-making tree – episcleritis.

CASE EXAMPLE 57.1

Patient details: Collie cross, 7 years old, male (neutered)

PRESENTATION AND HISTORY

The dog had suffered from several bouts of conjunctivitis in the right eye over the previous few months. This had not improved with topical antibiotics alone but when topical steroids were also included the symptoms subsided. No definitive diagnosis had been reached. The owner had noticed that the eye seemed to suddenly become very red, but also cloudy, and brought him back to the surgery.

CLINICAL SIGNS

General clinical examination was normal. The dog was visual and appeared in no discomfort. Severe episcleral congestion and swelling were noted in the right eye, especially from 5 to 1 o'clock laterally, with associated corneal oedema (Figure 57.3). The left eye also had a small episcleral nodule at 2 o'clock (Figure 57.4). No corneal ulceration was present. Intraocular pressures were normal and there appeared to be no intraocular abnormalities.

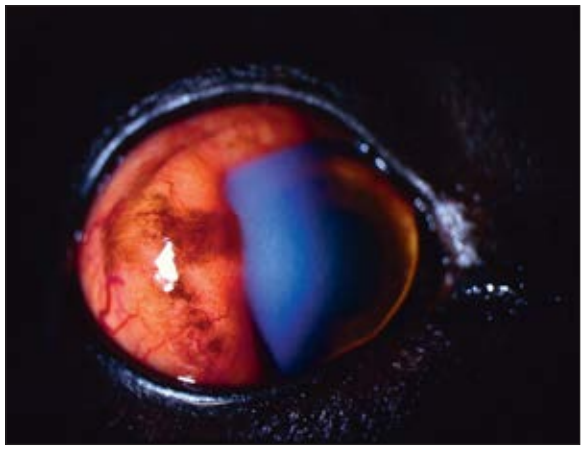


Figure 57.3 Severe episcleral congestion and swelling along with corneal oedema in the right eye of a collie cross.

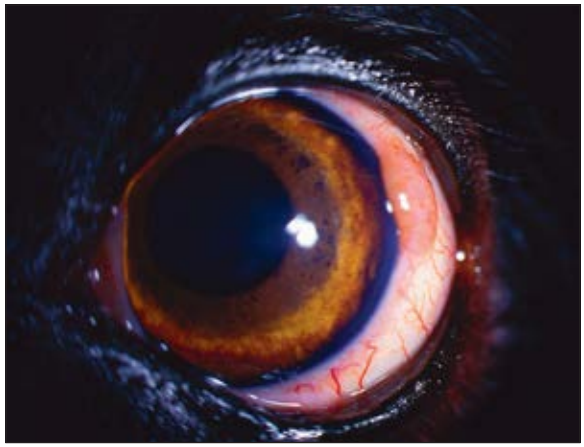


Figure 57.4 Left eye of the same dog as in Figure 57.3 showing a small episcleral nodule.

WORK-UP

A combination of diffuse and nodular episcleritis was suspected. Biopsy was advised to confirm this but the owner was unwilling to agree to anaesthesia for this, and since conjunctival biopsy would not provide a deep enough sample under topical anaesthesia, it was decided to treat the dog symptomatically and biopsy if the response to this was poor.

TREATMENT

Topical prednisolone acetate was started, six times daily in the right eye and three times daily in the left. Prednisolone was started orally at 2 mg/kg/day for 5 days. The eyes were significantly improved after 5 days and the oral steroid was reduced to 1 mg/kg/day for another 2 weeks.

OUTCOME

The good improvement continued, and 3 weeks after starting treatment the oral prednisolone was reduced to 0.5 mg/kg/day and the prednisolone acetate to four times daily in the right eye and twice daily in the left. The steroid tablets were reduced to alternate day dosing another 2 weeks later but the episclera started to become inflamed and swollen again in the right eye so daily medication was resumed. Over the following months the medication was gradually withdrawn to the lowest dose that prevented recurrence. This was oral prednisolone at 0.25 mg/kg every other day and prednisolone acetate twice daily to the right eye and once daily to the left. The dog had blood samples taken every 4 months to check for steroid side effects and the corneas were regularly stained with fluorescein to check for ulceration.

This case is a severe example of episcleritis, and this form is certainly more common in collie types. The early conjunctivitis was clearly the beginning of the disease – it is possible that aggressive medication at that stage could have prevented the progression but since it was assumed to be just conjunctivitis, the use of systemic steroids could not be justified. Additionally, conjunctival biopsy early on would probably not have been helpful since deeper samples are required to identify the episcleral involvement.

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MCQs

OPHTHALMIC EXAMINATION

1. **The menace response involves which nerve pathways?**
   - **(a)** Afferent is cranial nerve V (trigeminal) and efferent is cranial nerve VII (facial)
   - **(b)** Afferent is cranial nerve VII (facial) and efferent is cranial nerve V (trigeminal)
   - **(c)** Afferent is cranial nerve II (optic) and efferent is cranial nerve V (trigeminal)
   - **(d)** Afferent is cranial nerve II (optic) and efferent is cranial nerve VII (facial)
   - **(e)** Afferent is cranial nerve VII (facial) and efferent is cranial nerve II (optic).

2. **Normal Schirmer tear test readings in dogs are:**
   - **(a)** below 10 mm of wetting in 1 minute
   - **(b)** between 10 and 15 mm of wetting in 1 minute
   - **(c)** between 15 and 25 mm of wetting in 1 minute
   - **(d)** above 25 mm of wetting in 1 minute
   - **(e)** extremely variable and have to be evaluated in the light of clinical symptoms.

3. **Distant direct ophthalmoscopy is a useful method to help differentiate between:**
   - **(a)** corneal ulcer and cataract
   - **(b)** retinal detachment and cataract
   - **(c)** nuclear sclerosis and retinal detachment
   - **(d)** corneal ulcer and retinal detachment
   - **(e)** nuclear sclerosis and cataract.

4. **Goniodysgenesis is defined as:**
   - **(a)** raised intraocular pressure
   - **(b)** lowered intraocular pressure
   - **(c)** congenital abnormalities of the drainage angle
   - **(d)** acquired abnormalities of the drainage angle
   - **(e)** normal drainage angle.

THE EYELIDS

5. **Blepharitis can be caused by the following:**
   - **(a)** Bacterial infections
   - **(b)** Immune-mediated disease
   - **(c)** Drug allergies/hypersensitivity
   - **(d)** In conjunction with generalized skin disease
   - **(e)** All of the above.

6. **Surgery for entropion should be considered:**
   - **(a)** only in adult patients
   - **(b)** in animals of any age if they are showing clinical signs
   - **(c)** only when the eyelids rub sufficiently to cause corneal ulceration
   - **(d)** in all Shar Pei dogs
   - **(e)** only in cases of primary entropion.

7. **Eyelid tumours in dogs and cats are:**
   - **(a)** usually benign in both dogs and cats
   - **(b)** frequently malignant in both dogs and cats
   - **(c)** usually benign in cats but are more commonly malignant in dogs
   - **(d)** usually benign in dogs but are more commonly malignant in cats
   - **(e)** of variable behaviour in both species such that no generalizations can be made.

8. **Ectopic cilia are lashes which are located:**
   - **(a)** in the palpebral conjunctiva
   - **(b)** along the eyelid margins
   - **(c)** in the position of normal eyelashes
   - **(d)** both a and b
   - **(e)** a, b and c.

9. Treatment options for prolapsed nictitans glands include:
- **(a)** surgical removal of the gland only
- **(b)** surgical removal of the third eyelid and gland
- **(c)** surgical replacement of the gland
- **(d)** topical anti-inflammatory drops to reduce the swollen gland so that it repositions itself
- **(e)** no treatment is required – it is purely cosmetic.

THE CONJUNCTIVA

10. The bulbar conjunctiva is the area:
- **(a)** covering the globe
- **(b)** overlying the inner aspect of the eyelids
- **(c)** covering the third eyelid
- **(d)** covering the globe and third eyelid
- **(e)** there is no region called the bulbar conjunctiva.

11. The clinical signs of conjunctivitis are:
- **(a)** conjunctival hyperaemia, ocular discharge and corneal ulceration
- **(b)** conjunctival hyperaemia, ocular discharge and corneal oedema
- **(c)** conjunctival hyperaemia, ocular discharge and chemosis
- **(d)** conjunctival hyperaemia, ocular discharge and miosis
- **(e)** conjunctival hyperaemia, ocular discharge and hyphaema.

12. Feline conjunctivitis is most commonly associated with:
- **(a)** primary bacterial infections
- **(b)** primary viral infections
- **(c)** primary *Chlamydophila* infections
- **(d)** both bacterial and viral infections
- **(e)** both viral and *Chlamydophila* infections.

13. Chronic conjunctivitis in cats due to feline herpes virus infection is:
- **(a)** normally bilateral with respiratory symptoms as well
- **(b)** normally unilateral with respiratory symptoms as well
- **(c)** normally bilateral with no respiratory symptoms
- **(d)** normally unilateral with no respiratory symptoms
- **(e)** any combination of the above.

THE NASOLACRIMAL SYSTEM

14. The aqueous portion of the tear film is produced by:
- **(a)** the meibomian glands
- **(b)** the nictitans glands
- **(c)** the lacrimal glands and nictitans glands
- **(d)** the meibomian glands and lacrimal glands
- **(e)** the goblet cells.

15. Treatment for keratoconjunctivitis sicca aims to:
- **(a)** increase normal tear production
- **(b)** supplement the reduced tear production
- **(c)** treat any underlying bacterial infections
- **(d)** reduce any surface inflammation, if present
- **(e)** all of the above.

16. Corneal ulceration secondary to keratoconjunctivitis sicca typically presents as a:
- **(a)** sudden onset descemetocoele
- **(b)** superficial non-healing ulcer of several weeks' duration
- **(c)** mid-stromal ulcer with marked corneal vascularization
- **(d)** melting corneal ulcer
- **(e)** slowly progressive ulcer which is poorly responsive to treatment.

17. Epiphora is defined as:
- **(a)** tear overflow due to increased lacrimation
- **(b)** tear overflow due to reduced nasolacrimal drainage
- **(c)** tear overflow due to both increased lacrimation and reduced drainage
- **(d)** tear overflow due to a painful ocular complaint
- **(e)** tear overflow due to both c and d.

18. The treatment of choice for dacryocystitis in dogs is:
- **(a)** topical antibiotics only
- **(b)** systemic antibiotics only
- **(c)** both topical and systemic antibiotics
- **(d)** repeated nasolacrimal duct flushing and topical antibiotics
- **(e)** repeated nasolacrimal duct flushing with both topical and systemic antibiotics.

THE CORNEA

19. The thickest layer of the cornea is the:
- **(a)** epithelium
- **(b)** stroma
- **(c)** Descemet's membrane
- **(d)** endothelium
- **(e)** both the stroma and endothelium are equally thick.

20. Corneal healing of a superficial ulcer occurs by:
- **(a)** epithelial cell migration and mitosis
- **(b)** stromal collagen fibre replication
- **(c)** mitosis of endothelial cells
- **(d)** regeneration of Descemet's membrane
- **(e)** fibroblast and blood vessel infiltration of the epithelium.

21. Superficial recurrent corneal erosions are characterized by:
- **(a)** a regular circular outline
- **(b)** marked superficial corneal vascularization
- **(c)** severe corneal oedema
- **(d)** an irregular shape with loose margins
- **(e)** any of the above presentations.

22. A grid keratotomy is a suitable treatment option for:
- **(a)** any corneal ulcer
- **(b)** infected superficial corneal ulcers
- **(c)** non-healing superficial corneal ulcers
- **(d)** ulceration secondary to keratoconjunctivitis sicca
- **(e)** ulceration in cats associated with feline herpes virus.

23. If presented with a puppy which has been scratched in the eye it is important to:
- **(a)** check to see if a full thickness laceration has occurred
- **(b)** examine the depth of the anterior chamber
- **(c)** ascertain if the injury has damaged the lens
- **(d)** consider early referral for full evaluation
- **(e)** all of the above.

24. The treatment of a corneal sequestrum in a Persian cat:
- **(a)** always requires surgery with a superficial keratectomy
- **(b)** frequently requires surgery but some cases can be managed conservatively
- **(c)** can always be managed medically
- **(d)** always requires surgery with a conjunctival graft
- **(e)** frequently requires surgery with enucleation.

THE UVEAL TRACT

25. The typical ophthalmic signs of acute uveitis include:
- **(a)** corneal oedema, pain, mydriasis and episcleral congestion
- **(b)** corneal oedema, no pain, mydriasis and episcleral congestion
- **(c)** corneal oedema, pain, miosis and episcleral congestion
- **(d)** corneal oedema, no pain, miosis and episcleral congestion
- **(e)** corneal oedema, pain, miosis but no episcleral congestion.

26. Bilateral cases of uveitis require:
- **(a)** full clinical examination to look for other systemic signs
- **(b)** minimal haematology, biochemistry and urinalysis screening
- **(c)** careful history taking regarding vaccination status and travel history
- **(d)** ocular ultrasonography to look for lens and retinal involvement
- **(e)** all of the above.

27. Feline uveitis is frequently:
- **(a)** acute and easily treated
- **(b)** acute but requires long-term treatment
- **(c)** chronic and requires long-term treatment
- **(d)** both a and c
- **(e)** both b and c.

28. Hyphaema is defined as:
- **(a)** corneal haemorrhage
- **(b)** haemorrhage in the retina
- **(c)** haemorrhage in the vitreous
- **(d)** haemorrhage in the anterior chamber
- **(e)** subconjunctival haemorrhage.

THE LENS

29. Posterior polar subcapsular cataracts are:
- **(a)** inherited in several breeds of dog – most commonly the Labrador breeds
- **(b)** most frequently seen in dogs with diabetes mellitus
- **(c)** an early indication of lens instability
- **(d)** commonly found in dogs with progressive retinal atrophy
- **(e)** found in dogs with traumatic uveitis.

30. Dogs with cataracts associated with diabetes mellitus:
- **(a)** usually have sudden onset total cataracts
- **(b)** frequently develop lens-induced uveitis
- **(c)** are not suitable for cataract surgery
- **(d)** all of the above (a, b and c)
- **(e)** a and b only but are suitable for surgery.

31. Clinical signs of lens luxation in dogs include:
- **(a)** sudden onset blindness with corneal oedema and pain
- **(b)** gradual reddening of the conjunctiva but no pain
- **(c)** sudden onset blindness due to retinal detachment but no pain
- **(d)** gradual onset blindness due to retinal detachment but with pain
- **(e)** sudden onset blindness with no other signs.

32. Lens luxations in cats:
- **(a)** have a similar aetiology and presentation as in dogs
- **(b)** normally occur secondary to chronic uveitis
- **(c)** will cause acute glaucoma similar to dogs
- **(d)** require urgent surgery to prevent permanent blindness
- **(e)** are seen in conjunction with multiple ocular abnormalities in cats.

GLAUCOMA

33. Primary glaucoma in dogs occurs due to:
- **(a)** the overproduction of aqueous humour
- **(b)** the impaired drainage of aqueous humour
- **(c)** both increased production and decreased drainage of aqueous humour
- **(d)** vitreous blocking the drainage angle
- **(e)** lens luxation.

34. Gonioscopy is defined as:
- **(a)** measurement of intraocular pressure using a goniolens
- **(b)** measurement of aqueous drainage using a goniolens
- **(c)** measurement of aqueous pressure using a tonometer
- **(d)** examination of the drainage angle using a goniolens
- **(e)** examination of the drainage angle using a tonometer.

35. The medical treatment of glaucoma in dogs:
- **(a)** should treat any underlying pathology if possible
- **(b)** should use drugs to lower intraocular pressure
- **(c)** should use drugs to improve aqueous drainage
- **(d)** b and c only
- **(e)** a, b and c.

36. Which of the following is not a cause of secondary glaucoma in dogs:
- **(a)** uveal neoplasia
- **(b)** cataract
- **(c)** anterior uveitis
- **(d)** corneal ulceration
- **(e)** lens luxation.

THE FUNDUS

37. The fundus of dogs and cats has a:
- **(a)** paurangiotic distribution
- **(b)** merangiotic distribution
- **(c)** holangiotic distribution
- **(d)** anangiotic distribution
- **(e)** none of the above.

38. The optic disc in cats is normally:
- **(a)** myelinated with a white fluffy appearance
- **(b)** myelinated and small and dark
- **(c)** non-myelinated with a white fluffy appearance
- **(d)** non-myelinated and small and dark
- **(e)** non-myelinated and large and dark.

39. Tapetal hyper-reflectivity is:
- **(a)** a manifestation of thinning of the retina
- **(b)** a manifestation of swelling of the retina
- **(c)** due to hypertrophy of the tapetal cells
- **(d)** due to degeneration of the tapetal cells
- **(e)** both a and c.

40. Which of the following is not true of generalized progressive retinal atrophy:
- **(a)** It is an acute onset inherited disease in many canine breeds
- **(b)** It is a chronic onset inherited disease in many canine breeds
- **(c)** It manifests as night blindness which progresses to total blindness
- **(d)** Cataracts commonly occur in affected animals
- **(e)** Owners might notice increased 'eye shine' in affected dogs.

41. Ophthalmic signs of generalized progressive retinal atrophy can include:
- **(a)** poor pupillary light reflexes, tapetal hyper-reflectivity and lens luxation
- **(b)** poor pupillary light reflexes, tapetal hypo-reflectivity and lens luxation
- **(c)** normal pupillary light reflexes, tapetal hyper-reflectivity and cataract
- **(d)** poor pupillary light reflexes, tapetal hypo-reflectivity and lens cataract
- **(e)** poor pupillary light reflexes, tapetal hyper-reflectivity and cataract.

42. Retinal detachments due to hypertension are usually:
- **(a)** rhegmatogenous in nature
- **(b)** bullous in nature
- **(c)** traction detachments
- **(d)** a combination of all three types above
- **(e)** retinal detachments are not seen in hypertensive patients.

THE ORBIT AND GLOBE

43. Exophthalmos is defined as:
- **(a)** enlargement of the globe
- **(b)** shrinkage of the globe
- **(c)** a sunken globe
- **(d)** a protruding globe
- **(e)** a squint.

44. Which of the following is not a possible cause for exophthalmos in cats:
- **(a)** retrobulbar cellulitis
- **(b)** retrobulbar neoplasia
- **(c)** retrobulbar loss of the fat pad
- **(d)** retrobulbar haemorrhage
- **(e)** retrobulbar foreign body.

45. Horner's syndrome in dogs will manifest with:
- **(a)** enophthalmos, ptosis, miosis and elevation of the nictitans membrane
- **(b)** exophthalmos, ptosis, miosis and elevation of the nictitans membrane
- **(c)** enophthalmos, ptosis, mydriasis and elevation of the nictitans membrane
- **(d)** enophthalmos, ptosis, miosis and with normal positioning of the nictitans membrane
- **(e)** enophthalmos, ptosis, mydriasis and normal positioning of the nictitans membrane.

MCQs – Answers

1. **(d)** The movement is detected by the retinal photoreceptors and is transmitted centrally via the optic nerve. Blinking is caused by stimulation from the facial nerve.
2. **(c)** Strips should be placed in the lower conjunctival fornix away from the nictitans membrane. Readings in cats are variable and often low.
3. **(e)** A tapetal reflex will be clearly visible if the lens clouding is due to nuclear sclerosis alone, while the presence of cataract formation will cause areas of opacification of this tapetal reflex.
4. **(c)** The drainage angle can be of normal width or narrowed while variable amounts of pectinate ligament dysplasia are present. Such abnormal angles are predisposed to the development of primary glaucoma.
5. **(e)** There are many potential causes for blepharitis and a thorough general clinical examination is required as well as the ophthalmic examination to try to reduce the differential list. Cases can also be primary or secondary.
6. **(b)** If the entropion is causing discomfort, then it should be treated and surgery is the only option. However, in young animals owners should be warned that further procedures might be necessary as the patient matures. Secondary cases, such as those due to chronic corneal ulceration, might still require surgery (if the spastic component is not abolished with topical anaesthesia, which suggests that the blepharospasm has become more permanent). Although many Shar Peis do require surgery, the occasional one has good eyelid anatomy!
7. **(d)** In many cases this is true, and histology should always be performed in cats, and should be considered in dogs if the mass is ulcerated, large, unusual in appearance or has regrown.
8. **(a)** Lashes that originate in the meibomian gland but penetrate through the palpebral conjunctiva to rub on the cornea are called ectopic cilia. Distichia originate in the same place but emerge along the lid margin.
9. **(c)** Two commonly employed techniques are appropriate – the pocket and the anchoring techniques. The gland produces part of the aqueous tear film and its removal is not recommended. The third eyelid should only be removed if proven neoplasia is present. Although anti-inflammatory drops will reduce some swelling they are not sufficient to correct the anatomical abnormality and by leaving the gland untreated the risk of infection is present, nictitans membrane function is compromised and tear production can also be affected.
10. **(a)** It is the area of conjunctiva which extends from the limbus to the fornix and provides a loose covering to the globe.
11. **(c)** Corneal or intraocular involvement is not a feature of simple conjunctivitis and, if present, then there is a potentially more serious ocular problem which requires accurate diagnosis.
12. **(e)** Primary bacterial infections are rare in cats. Feline herpes virus and *Chlamydophila* are the most frequently implicated infectious causes of conjunctivitis in this species.
13. **(d)** Acute forms of the infection are bilateral with respiratory signs but chronic cases normally affect only one eye and other systemic signs are absent.
14. **(c)** The meibomian glands produce the lipid layer while the goblet cells produce mucin.
15. **(e)** Tear production is improved by a combination of increased normal tear production and

supplementation while any bacterial infections are controlled with topical antibiotics. If excessive keratitis is present, topical anti-inflammatory agents can be used providing no corneal ulceration is present.

16. **(a)** Any dog presenting with a deep corneal ulcer should have Schirmer tear test readings taken in both eyes. Acute ulceration in cases of keratoconjunctivitis sicca should be treated urgently.

17. **(b)** Epiphora is a passive overflow of tears and cases will have normal Schirmer tear test readings.

18. **(e)** Treatment for dacryocystitis needs to be aggressive, with a combination of repeated flushings plus both topical and systemic antibiosis for several weeks. Culture and sensitivity testing should always be considered.

19. **(b)** The stroma is by far the largest layer of the cornea, and provides shape and support, while its parallel arrangement of collagen fibrils maintains transparency.

20. **(a)** The epithelial cells adjacent to the injury will flatten and slide into the damaged area while multiplication of these cells starts. Deeper ulcers will heal by this method, along with stromal multiplication, but this is a much slower process. Endothelial cells have poor regenerative capacity while Descemet's membrane cannot regenerate. Fibroblast and blood vessel infiltration occurs in deep or chronic ulcers.

21. **(d)** These ulcers are typically irregular and a loose lip of non-adherent corneal epithelium surrounds the actual ulcer.

22. **(c)** Superficial epithelial erosions are the only indication for performing a grid keratotomy. They should not be used in deep or infected ulcers, and in cats with feline herpes virus the use of a grid keratotomy can increase the likelihood of sequestrum formation.

23. **(e)** The early diagnosis and treatment for scratch injuries is essential if a successful outcome is to be achieved. Since it can be very difficult to determine whether the lens is damaged in some patients, especially if marked corneal oedema is present, then early referral should always be considered.

24. **(b)** Most cases do require surgery, with a superficial keratectomy ± conjunctival graft. However, if the lesion is not ulcerated and therefore not painful, then conservative management can be an option. Enucleation is only required in cases which have been neglected or where facilities for microsurgery are not available or affordable.

25. **(c)** The miosis and pain are associated with a prostaglandin-mediated spasm in the iris sphincter muscle and are typical in acute uveitis.

26. **(e)** A thorough examination and work-up is required for all bilateral cases of uveitis – the clinical appearance is totally non-specific and therefore careful evaluation of each case is required to reach a definitive diagnosis and therefore treat appropriately.

27. **(d)** Acute cases are frequently traumatic and respond well to appropriate medication. Chronic cases are often more difficult to cure and can be recurrent.

28. **(d)** Bleeding into the anterior chamber is quite common and a full work-up is required to determine the aetiology.

29. **(a)** The typical upside down 'Y' (Mercedes sign) is seen as an inherited form of cataract in several breeds of dog and can be progressive, leading to blindness.

30. **(e)** Providing the diabetes and lens-induced uveitis are both well controlled, such patients are good candidates for cataract surgery.

31. **(a)** The sudden dislocation of the lens causes corneal oedema and marked pain. Vision is impaired or absent. Although retinal detachments can occur with lens luxations, they are not usually part of the initial presentation. Conjunctival hyperaemia will also be present but this is not the main presenting sign.

32. **(b)** Unlike in dogs there is normally a history of ongoing ocular pathology, namely uveitis, prior to lens luxation. Glaucoma is not always a feature, and again can be chronic. Surgery is not always required and multiple ocular abnormalities are very rare in cats.

33. **(b)** Goniodysgenesis is the most common cause of primary glaucoma although open-angle glaucoma does also occur.

34. **(d)** A special contact lens is placed on the anaesthetized cornea such that the drainage angle can be observed and assessed.

35. **(e)** If any underlying pathology such as uveitis is present, then this needs addressing along with specific medications to both improve drainage and decrease aqueous production. A multiple approach is likely to be more successful than using one treatment alone.

36. **(d)** Surface and adnexal disease do not disrupt the intraocular production and drainage of aqueous in the same way as the other intraocular conditions can do.

37. **(c)** An optic disc is present with three to four pairs of branching arterioles and venules.

38. **(d)** The feline optic disc is normally roughly circular and smaller and darker than in the dog due to the absence of myelin.

39. **(a)** Thinning of the retina allows more light to be reflected in the underlying tapetum, thus increasing the reflectivity seen on ophthalmoscopy. The tapetal cells themselves are not directly involved.

40. **(a)** The condition is not acute, but owners might suddenly notice the dog's visual problems when they take it somewhere unfamiliar (e.g. a relative's house).

41. **(e)** Lens luxation can sometimes occur very late in the course of the disease, when the cataract has been hypermature for some time but is not usually a typical feature. Some patients do have normal light reflexes but this is less common.

42. **(b)** Typically fluid leaks from the retinal vessels and accumulates between the neurosensory retina and underlying retinal pigment epithelium, leading to a bullous detachment. Traction detachments can occur following extensive vitreal haemorrhage but are much less common. Similarly, rhegmatogenous detachments with peripheral retinal tears can occur but with far less frequency than bullous detachments (which can be partial or total).

43. **(d)** The globe is of normal size but positioned further anterior than normal.

44. **(c)** Loss of the fat pad, for example in severe dehydration or following sudden weight loss, will result in enophthalmos or a sunken eye.

45. **(a)** The symptoms can all be attributed to disruption in the sympathetic supply to the globe.

1 APPENDIX Equipment and instruments required for ophthalmic surgery in general practice

For requirements for the ophthalmic examination, see Chapter 1.

For all ophthalmic surgery good illumination is required and a vacuum positioning pillow is invaluable to stabilize the head during surgery (Figure A1.1). Magnification is required for all corneal surgery and is also helpful for some eyelid procedures, particularly those on the nictitans membrane (e.g. replacement of prolapsed nictitans glands). Magnifying loupes are appropriate in general practice (see Figure 1.2, see p. 5). All ophthalmic instruments are delicate and require careful handling and cleaning to prevent damage. Ideally they should be stored in a special instrument box, which has small silicone fingers to keep the instruments separate from each other and prevent them from moving (Figure A1.2).

Table A1.1 Instruments for ophthalmic surgery

Instrument	Use
Basic general ophthalmic kit	
Straight scissors	Cutting skin and canthotomy
Fine rat-tooth forceps	Holding skin and conjunctiva
Steven's tenotomy scissors	Cutting conjunctiva
St Martin's tying forceps	Holding conjunctiva and sutures
Needle holders (e.g. Castroviejo – without catch)	Suturing
Fine mosquito haemostats (curved and straight; 3 pairs)	Stabilizing the globe and for haemostasis
Eyelid speculum	Provide globe exposure
Chalazion clamp	Stabilize lid margins
Blade handle (Bard Parker)	Sharp incision
Towel clamps (2–4)	Stabilize drapes
For corneal surgery	
Beaver handle and blade	Corneal incision
Eyelid speculum	Globe exposure
Small straight scissors	Canthotomy
Tenotomy scissors (Steven's or Westcott's)	Conjunctival cutting
Corneal micro forceps (Colibri)	Handling cornea
Tying forceps (St Martin's)	For handling delicate suture material
Delicate needle holders	To handle tiny needles
Mosquito forceps (curved and straight; 3 pairs)	Stabilize the globe and provide haemostasis
Towel clamps/serrafines (4)	Secure drapes (although sticky drapes are preferred for corneal surgery)



Figure A1.1 Vacuum positioning pillow greatly assists in stabilizing the head during surgery while ensuring that the eye remains in the correct position.

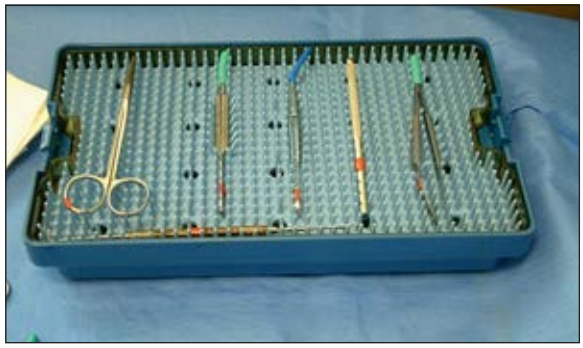


Figure A1.2 Storage box for microsurgical instruments.

Suggested instruments for an enucleation kit include:

- Allis tissue forceps
- Adson-Brown forceps
- eyelid speculum
- curved and straight haemostatic forceps
- curved and straight mosquito haemostatic forceps
- Metzenbaum scissors
- enucleation scissors
- tenotomy scissors
- Bard Parker scalpel handle
- towel clamps
- needle holders.

Table A1.2 Suture material for ophthalmic surgery

Suture type	Size	Use
Non-absorbable		
Nylon (monofilament)	3/0–4/0	Stay sutures Occasionally skin
	8/0–10/0	Cornea
Silk (braided)	4/0–7/0	Eyelid skin Conjunctiva
Absorbable		
Polyglactin 910 (Vicryl, Ethicon)	4/0–6/0	Subcutaneous tissue Skin
	8/0–10/0	Conjunctiva Cornea
Polydioxanone (PDS, Ethicon)	2/0–5/0	Subcutaneous tissue Subconjunctival tissue

Table A1.3 Basic steps in surgical preparation for ophthalmic surgery

Step	Agent used	Method
1	Fine clippers	Apply K-Y Jelly (Johnson & Johnson) to clipper blades so hairs stick to them Ensure that the clipper blades are clean and sharp to avoid any clipper rash which could encourage postoperative self-trauma
2	Soft gauze-free swabs moistened with sterile saline	Gently remove any hairs and debris from the surgical field
3	1:50 dilution povidone–iodine solution	Use 5–10 ml syringe to flush conjunctival sac
4	Sterile saline	Use 5–10 ml syringe to flush conjunctival sac again
5	1:10 povidone–iodine solution	On gauze-free swabs (e.g. Topper; Johnson & Johnson) to surgically prepare skin routinely but ensure that none of this stronger solution enters the conjunctival sac

In addition to the correct instruments, it is important to consider the suture material used for ophthalmic surgery, and various examples are shown in Table A1.2.

Prior to actually performing ocular surgery it is necessary to ensure that the surgical preparation is adequate. Disinfecting solutions which contain detergents should be avoided as they will cause corneal damage. Thus scrubs such as chlorhexidine and povidone–iodine scrub are not appropriate. Instead, diluted solutions of povidone–iodine are suggested. The basic steps in surgical preparation are outlined in Table A1.3.

2 APPENDIX Ocular pharmacology

It should be remembered that many agents used in veterinary ophthalmology are not licensed for use in our species. Clearly, according to UK legislation, if a licensed product is available it must be used first, but under the Cascade system it is acceptable to use a licensed human product should there be no veterinary alternative. Owners must sign appropriate forms to this effect.

ANTIVIRAL AGENTS

No antiviral agents are currently licensed in the UK for use in animals. Topical preparations of trifluorothymidine are useful for primary feline herpes virus (FHV-1) infections and can be obtained from Moorfields Eye Hospital. Aciclovir is available as a topical preparation but its efficacy in feline cases appears limited – certainly trifluorothymidine has been shown to be more efficacious in both in vitro and in vivo studies. Systemic agents such as famciclovir have been used by specialists but their use in general practice cannot be advocated until further efficacy and safety studies have been undertaken. L-Lysine is an amino acid which seems to reduce viral replication of FHV-1 and it can be used orally at doses of 250–500 mg once or twice daily. It is available from health food shops.

ANTIBACTERIAL AGENTS

See Table A2.1 for details.

ANTIFUNGAL AGENTS

Both topical and systemic antifungal agents can be obtained for ophthalmic use, but since fungal infections are rare in the UK the reader is advised to consult appropriate texts for further information.

ANTI-INFLAMMATORY AGENTS

None of the agents listed in Tables A2.2 and A2.3 is licensed for use in our patients.

Other anti-inflammatory agents such as mast cell stabilizers are available and are used to treat allergic conjunctivitis in humans. Their use in animals can be beneficial in some patients but specialist advice should be sought.

TOPICAL TEAR STIMULANTS AND OCULAR LUBRICANTS

For cases of keratoconjunctivitis sicca, ciclosporin will stimulate aqueous tear production. A 0.2% ointment is available (Optimmune, Schering-Plough) which is licensed for use in dogs. In addition to simulating tearing, lubrication is usually required. There are many forms of topical lubricants, in drop, gel and ointment formulation, and the choice of product will depend upon owner preference and the frequency of application. Generally a gel preparation such as polyacrylic acid (carbomer gel) is used two to four times during the day with a soft white paraffin ointment at night. Drop formulations do not last in the eye and most owners are unable to use these frequently enough (e.g. hourly) for their use to be worthwhile.

MYDRIATICS AND CYCLOPLEGICS

Mydriatics for diagnostic purposes are limited to 1% tropicamide (Mydriacyl, Alcon), which has a rapid onset (20 minutes) and lasts for 3–4 hours. Atropine should not be used for diagnostic purposes, but solely for treatment. Uveitis – both reflex and primary – is the main indication for topical atropine, which is used to effect to maintain a dilated pupil (from just one application in

Table A2.1 Commonly used antibacterial agents (**topical** and systemic)

Drug type	Properties	Uses	Commercial products
β-Lactam antibiotics – penicillins and cephalosporins	Bactericidal Excreted via kidneys Poor ocular penetration when given systemically unless BOB disrupted Topical agents will penetrate anterior chamber if cornea ulcerated Mostly Gram-positive activity	Aerobic infections of the orbit, eyelids and globe Cephalosporins safe for use in rabbits	Amoxicillin/clavulanic acid (Synulox, Pfizer, plus many others) Cefalexin (Ceporex, Schering-Plough; Cefaseptin, Vetoquinol; plus many others)
Tetracyclines	Broad spectrum Bacteriostatic Doxycycline has good ocular penetration unlike other tetracyclines Excreted via gastrointestinal, biliary and urinary systems Good therapeutic levels in anterior chamber following topical administration	*Chlamydophila* *Mycoplasma* Rickettsia	Oxytetracycline – multiple systemic preparations Doxycycline (Ronaxan, Merial) **Chlortetracycline hydrochloride** (Aureomycin ointment, Fort Dodge)
Aminoglycosides	Bactericidal Excreted in urine Systemic agents do not cross BOB Gram-negative activity mainly Synergistic with penicillins	*Pseudomonas* spp. Melting corneal ulcers	**Gentamicin** (Tiacil drops#, Virbac; Clinagel Vet, Janssen) **Tobramycin** (Tobrex, Alcon) **Neomycin** (often in combination with polymyxin B and bacitracin (e.g. Neosporin drops, PLIVA))
Macrolides and lincosamides	Usually bacteriostatic Mainly Gram-positive activity Metabolized in the liver Good tissue penetration	*Toxoplasma gondii* *Chlamydophila* infections (especially azithromycin) *Pseudomonas* *Pasteurella multocida*	Clindamycin (Antirobe, Pfizer) Azithromycin (Zithromax, Pfizer)
Fluoroquinolones	Bactericidal Broad spectrum Some metabolism in liver before excretion via kidneys	*Pseudomonas* and other Gram-negative infections	Enrofloxacin (Baytril, Bayer) (care in cats due to retinal degeneration) Marbofloxacin (Marbocyl, Vetoquinol) **Ciprofloxacin** eye drops (Ciloxan, Alcon) **Ofloxacin** eye drops (Exocin, Allergan)
Fusidic acid	Mainly Gram-positive with some Gram-negative activity Good corneal penetration	Gram-positive ocular surface disease, especially canine bacterial conjunctivitis	**Fusidic acid** gel (Fucithalmic #, VetXX)
Chloramphenicol	Bacteriostatic Broad spectrum Good corneal penetration and ocular penetration if used systemically	*Pasteurella multocida* *Chlamydophila* Prophylactically for ocular surgery	Many products available – in systemic forms as well as **drops and ointments**
Metronidazole	Bactericidal Good tissue penetration following systemic use	Anaerobic infections, e.g. retrobulbar or orbital disease	Several systemic preparations available
Polypeptide antibiotics	Bactericidal Do not penetrate cornea so restricted to surface disease Triple antibiotic provides broad spectrum activity	Mixed ocular surface disease	Combined preparations with **polymyxin B, bacitracin** and **neomycin** (Neosporin drops, PLIVA)

BOB, blood–ocular barrier; #, licensed for use in rabbits.
Agents in bold are **topical** medications.

Table A2.2 Topical steroids

Generic name	Trade name	Indications
Betamethasone sodium phosphate 0.1%	Betnesol (Celltech) Betsolan (Schering-Plough)	Superficial ocular inflammation
Dexamethasone acetate 0.1% (in hypromellose) Also available in mixed preparation with antibiotics	Maxidex (Alcon) Maxitrol (Alcon) – with neomycin and polymyxin B	Anterior uveitis
Fluorometholone acetate 1.4% (in polyvinyl alcohol)	FML (Allergan)	Anterior uveitis and superficial ocular inflammation
Prednisolone acetate 1%	Pred Forte (Allergan)	Anterior uveitis
Prednisolone sodium phosphate 0.5%	Predsol (Celltech)	Superficial ocular inflammation

Table A2.3 Topical NSAIDs

Generic name	Trade name	Indications
Diclofenac sodium 0.1%	Voltarol Ophtha (Novartis)	Cataract surgery
Flurbiprofen sodium 0.03%	Ocufen (Allergan)	Cataract surgery
Ketorolac trometamol 0.5%	Acular (Allergan)	Anterior uveitis

Table A2.4 Anti-glaucoma preparations

Drug type	Action	Generic name	Trade name
Carbonic anhydrase inhibitors	Reduce aqueous production Usually administered 2–4 times daily	Dorzolamide Brinzolamide	Trusopt (MSD) Azopt (Alcon)
Prostaglandin analogues	Increase uveoscleral outflow Used once daily at night, sometimes twice Avoid if uveitis present	Latanoprost Travoprost	Xalatan (Pharmacia) Travatan (Alcon)
Miotics	Direct acting parasympathomimetics which increase aqueous outflow Limited efficacy in dogs	Pilocarpine	Non-proprietary products available in 0.5–4% strengths
Beta-blockers	Reduce aqueous production Used in conjunction with topical carbonic anhydrase inhibitors but limited efficacy	Timolol Betaxolol	Non-proprietary Betoptic (Alcon)

Agents in bold are drugs of choice for most cases of glaucoma in dogs.

mild cases to three to four times daily in severe uveitis). Cats tolerate the ointment formulation better than the drops (which drain down the nasolacrimal duct and cause profuse salivation due to their bitter taste).

ANTIGLAUCOMA AGENTS

See Table A2.4 for details.

TOPICAL ANAESTHETICS

Proxymetacaine is the topical anaesthetic of choice and is available in 0.5% single use vials (Minims, Chauvin). It can be used for diagnostic and minor surgical procedures but should not be used to provide symptomatic pain relief to patients. Tetracaine (Minims Amethocaine, Chauvin) is also available.

OPHTHALMIC DYES

Fluorescein sodium is available as impregnated paper strips and as a 1% solution in single use vials. It is an orange dye which turns green in alkaline conditions and is used to delineate corneal ulcers, to detect aqueous leakage (Seidel test) and to check nasolacrimal duct patency.

Rose Bengal 1% eye drops can be used to detect subtle corneal surface disease such as the fine dendritic ulcers associated with FHV-1. However, it stings and is irritating if not flushed very thoroughly and is normally only available in specialist practices.

HOW TO APPLY TOPICAL MEDICATION

See Figure A2.1.

- Clean any discharge
- Gentle restraint
- Nose slightly elevated
- Come from behind the head with medication
- Hold eyelids open (using palm of hand holding medication bottle)
- Apply one drop onto cornea or conjunctiva
- Apply ointment into fornix or onto the eye itself
- Avoid touching bottle/tube on animal fur (results in contamination)
- Gently occlude nasolacrimal punctae
- Prevent rubbing in immediate post-medication period
- Apply drops before ointments
- Leave 10 minutes between subsequent medications



Figure A2.1 How to apply eye drops to a cat.

3 APPENDIX Ocular emergencies and what to refer

OCULAR EMERGENCIES

The following conditions are genuine ocular emergencies:

- Proptosis
- Corneal laceration/iris prolapse
- Corneal foreign body
- Deep corneal ulcer (e.g. descemetocoele)
- Melting corneal ulcer
- Chemical injuries
- Blunt trauma
- Acute uveitis
- Acute glaucoma
- Acute lens luxation
- Acute exophthalmos
- Acute hyphaema
- Sudden onset blindness.

Cases for which referral should be considered (*italic indicates urgent referral advised*):

- Cataracts (if surgery is considered)
- *Deep corneal ulcers*
- *Penetrating or perforating corneal foreign bodies*
- *Melting ulcers*
- Complicated eyelid problems
- Feline sequestrum
- *Glaucoma*
- *Uveitis (especially where the cause is not obvious)*
- *Sudden blindness*
- *Panophthalmitis or endophthalmitis*
- Orbital neoplasia
- Parotid duct transposition
- Unexplained ocular pain
- Refractory conjunctivitis.

4 APPENDIX Congenital ocular problems – what to check for at the first vaccination

Several congenital ocular conditions occur and they can be inherited in some breeds of dog, although inherited congenital problems are much less common in cats. They are listed below.

CONGENITAL OCULAR PROBLEMS IN DOGS

- Ophthalmia neonatorum
- Microphthalmos
- Imperforate or micropunctum
- Retinal dysplasia (multifocal, geographic and total)
- Collie eye anomaly
- Dermoid
- Persistent pupillary membrane (PPM) remnants
- Persistent hyaloid vessels
- Persistent hyperplastic primary vitreous
- Congenital cataract
- Glaucoma
- Multiple ocular abnormalities (microphthalmia, nystagmus, PPM, cataract, retinal dysplasia in any combination)
- Optic nerve hypoplasia

CONGENITAL OCULAR PROBLEMS IN CATS

- Ophthalmia neonatorum
- Eyelid agenesis
- Dermoid
- In utero viral infections and subsequent ocular abnormalities
- Bilateral esotropia
- Persistent pupillary membrane remnants
- Retinal dysplasia (usually virally induced)
- Glaucoma

Table A4.1 Inherited congenital ocular problems in dogs (per BVA/KC/ISDS Eye Testing Scheme)

Condition	Breeds affected	
	Schedule A – known inherited	**Schedule B – suspected inherited**
Collie eye anomaly	Border collie Collie (rough) Collie (smooth) Lancashire heeler Shetland sheepdog	
Retinal dysplasia: T – total M – multifocal	Bedlington terrier (T) Cavalier King Charles spaniel (M) Hungarian Puli (M) Retriever (golden) (M) Retriever (Labrador) (M, T) Rottweiler (M) Sealyham terrier (T) Spaniel (American cocker) (M) Spaniel (English springer) (M)	Beagle (M) Collie (rough) German shepherd dog Giant schnauzer Norwegian elkhound Spaniel (field) Spaniel (Sussex)
Persistent pupillary membrane	Basenji	Bull mastiff Dachshund (miniature wire haired) Lancashire heeler Petit Basset Griffon Vendeen Rottweiler Siberian husky Spaniel (cocker) West Highland white terrier
Congenital hereditary cataract	Miniature schnauzer (may also have other ocular abnormalities)	Old English sheepdog Retriever (golden) West Highland white terrier
Persistent hyperplastic primary vitreous	Dobermann Staffordshire bull terrier	
Multiple ocular defects		Bloodhound Cavalier King Charles spaniel Collie (rough) Dobermann Old English sheepdog Poodle (standard) Retriever (golden) Rottweiler Spaniel (cocker) West Highland white terrier
Coloboma		Australian shepherd
Optic nerve hypoplasia		Dachshund (miniature long haired) Poodle (miniature) Poodle (toy)

BVA/KC/ISDS, British Veterinary Association/Kennel Club/International Sheep Dog Society.

5 APPENDIX Differential diagnosis according to the presenting signs

ABNORMAL APPEARANCE – COLOUR CHANGE, RED

	Chapter
Blepharitis	4, 38
Chronic superficial keratitis	29
Conjunctivitis	10, 11, 38
Corneal ulcer	21, 22, 23
Enophthalmos	54
Episcleritis	57
Eyelash abnormalities	7
Feline sequestrum	30
Hypertensive retinopathy	51
Hyphaema	36
Intraocular neoplasia (dog)	37
Keratoconjunctivitis sicca	16
Prolapsed nictitans gland	8
Proliferative (eosinophilic) keratoconjunctivitis	27
Rabbit aberrant conjunctival overgrowth	14
Uveitis (canine)	33, 38
Uveitis (feline)	34
Uveitis (rabbit)	35

ABNORMAL APPEARANCE – COLOUR CHANGE, CLOUDY/BLUE

	Chapter
Cataract in young dog	41
Corneal endothelial dystrophy	26
Diabetic cataract	40
Feline keratitis	5, 11, 31
Glaucoma	44, 45, 46
Lens luxation	42
Limbal melanoma	56
Pigmentary keratitis	28
Progressive retinal atrophy	48
Proliferative (eosinophilic) keratoconjunctivitis	27
Uveitis (canine)	33, 38
Uveitis (feline)	34
Uveitis (rabbit)	35

ABNORMAL APPEARANCE – SWOLLEN

	Chapter
Blepharitis	4, 38
Exophthalmos	53
Eyelid mass	6, 38
Glaucoma	44, 45, 46
Proptosis	55

DISCHARGE

	Chapter
Blepharitis	4, 38
Conjunctivitis	10, 11, 12
Corneal ulcer	21, 22, 23
Dacryocystitis	18, 19
Entropion	5
Epiphora	17
Eyelash abnormalities	7
Feline keratitis	31
Feline sequestrum	30
Keratoconjunctivitis sicca	16
Puppy scratched by cat	24

PAIN

	Chapter
Corneal foreign body	25
Corneal ulcer	21, 22, 23
Entropion	5
Exophthalmos	53
Feline keratitis	31
Feline sequestrum	30
Glaucoma	44, 45
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Puppy scratched by cat	24
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BLINDNESS

	Chapter
Diabetic cataract	40
Glaucoma	44, 45, 46
Hypertensive retinopathy	51
Pigmentary keratitis	28
Progressive retinal atrophy	48
Retinal detachment	49
Sudden acquired retinal degeneration	50
Uveitis (canine)	34, 38

Further reading

The ophthalmic examination

Gelatt K N 1999 Veterinary ophthalmology, 3rd edn. Lippincott Williams & Wilkins, Philadelphia

Petersen-Jones S, Crispin S (eds) 2002 BSAVA manual of small animal ophthalmology, 2nd edn. BSAVA, Quedgeley

Slatter D 2001 Fundamentals of veterinary ophthalmology, 3rd edn. Saunders, Philadelphia

Turner S M 2005 Veterinary ophthalmology: a manual for nurses and technicians. Butterworth-Heinemann, Oxford

The eyelids

Gelatt K N, Gelatt K P 1994 Handbook of small animal ophthalmic surgery, Vol 1: extraocular procedures. Pergamon, New York

Grahn B L, Cullen C, Peiffer R 2004 Veterinary ophthalmology essentials. Butterworth-Heinemann, Oxford

Martin C L 2005 Ophthalmic disease in veterinary medicine. Manson, London

Petersen-Jones S, Crispin S (eds) 2002 BSAVA manual of small animal ophthalmology, 2nd edn. BSAVA, Quedgeley

Slatter D 2001 Fundamentals of veterinary ophthalmology, 3rd edn. Saunders, Philadelphia

The conjunctiva

Barnett K C, Crispin S M 1998 Feline ophthalmology: an atlas and text. Saunders, London

Barnett K C, Sansom J, Heinrich C 2002 Canine ophthalmology: an atlas and text. Saunders, London

Crispin S M 2005 Notes on veterinary ophthalmology. Blackwell, Oxford

Gelatt K N 1999 Veterinary ophthalmology, 3rd edn. Lippincott Williams & Wilkins, Philadelphia

Grahn B L, Cullen C, Peiffer R 2004 Veterinary ophthalmology essentials. Butterworth-Heinemann, Oxford

Martin C L 2005 Ophthalmic disease in veterinary medicine. Manson, London

Roze M, Ridings B, Lagadic M 2001 Comparative morphology of epicorneal conjunctival membranes in rabbits and human pterygium. Veterinary Ophthalmology 4(3):171–174

The nasolacrimal system

Burling K, Murphy C J, Curiel J et al 1991 Anatomy of the rabbit nasolacrimal duct and its clinical implications. Progress in Veterinary and Comparative Ophthalmology 1(1): 33–40

Gelatt K N 1999 Veterinary ophthalmology, 3rd edn. Lippincott Williams & Wilkins, Philadelphia

Grahn B L, Cullen C, Peiffer R 2004 Veterinary ophthalmology essentials. Butterworth-Heinemann, Oxford

Martin C L 2005 Ophthalmic disease in veterinary medicine. Manson, London

Petersen-Jones S, Crispin S (eds) 2002 BSAVA manual of small animal ophthalmology, 2nd edn. BSAVA, Quedgeley

Slatter D 2001 Fundamentals of veterinary ophthalmology, 3rd edn. Saunders, Philadelphia

The cornea

Barnett K C, Crispin S M 1998 Feline ophthalmology: an atlas and text. Saunders, London

Featherstone H, Sansom J 2004 Feline corneal sequestra: a review of 64 cases (80 eyes) from 1993 to 2000. Veterinary Ophthalmology 7(4):213–227

Gelatt K N 1999 Veterinary ophthalmology, 3rd edn. Lippincott Williams & Wilkins, Philadelphia

Grahn B L, Cullen C, Peiffer R 2004 Veterinary ophthalmology essentials. Butterworth-Heinemann, Oxford

Martin C L 2005 Ophthalmic disease in veterinary medicine. Manson, London

Petersen-Jones S, Crispin S (eds) 2002 BSAVA manual of small animal ophthalmology, 2nd edn. BSAVA, Quedgeley

Slatter D 2001 Fundamentals of veterinary ophthalmology, 3rd edn. Saunders, Philadelphia

The uveal tract

Barnett K C, Crispin S M 1998 Feline ophthalmology: an atlas and text. Saunders, London

Barnett K C, Sansom J, Heinrich C 2002 Canine ophthalmology: an atlas and text. Saunders, London

Gelatt K N 1999 Veterinary ophthalmology, 3rd edn. Lippincott Williams & Wilkins, Philadelphia

Harcourt-Brown F M, Holloway H 2003 Encephalitozoon cuniculi in pet rabbits. Veterinary Record 152:427–431

Martin C L 2005 Ophthalmic disease in veterinary medicine. Manson, London

Pena T, Roura X, Davidson M G 2000 Ocular and periocular manifestations of leishmaniasis in dogs: 105 cases (1993–1998). Veterinary Ophthalmology 3(1):35–41

Slatter D 2001 Fundamentals of veterinary ophthalmology, 3rd edn. Saunders, Philadelphia

Stiles J, Didier E, Ritchie B 1997 Encephalitozoon cuniculi in the lens of a rabbit with phacoclastic uveitis: confirmation and

treatment. Veterinary and Comparative Ophthalmology 7(4):233–238
Wolfer J, Grahn B, Wilcock B et al 1993 Phacoclastic uveitis in the rabbit. Progress in Veterinary and Comparative Ophthalmology 3(3):92–97

The lens

Barnett K C, Sansom J, Heinrich C 2002 Canine ophthalmology: an atlas and text. Saunders, London
Gelatt K N 1999 Veterinary ophthalmology, 3rd edn. Lippincott Williams & Wilkins, Philadelphia
Martin C L 2005 Ophthalmic disease in veterinary medicine. Manson, London

Glaucoma

Barnett K C, Crispin S M 1998 Feline ophthalmology: an atlas and text. Saunders, London
Barnett K C, Sansom J, Heinrich C 2002 Canine ophthalmology: an atlas and text. Saunders, London
Gelatt K N 1999 Veterinary ophthalmology, 3rd edn. Lippincott Williams & Wilkins, Philadelphia
Slatter D 2001 Fundamentals of veterinary ophthalmology, 3rd edn. Saunders, Philadelphia

The fundus

Barnett K C, Crispin S M 1998 Feline ophthalmology: an atlas and text. Saunders, London
Barnett K C, Sansom J, Heinrich C 2002 Canine ophthalmology: an atlas and text. Saunders, London
Gelatt K N 1999 Veterinary ophthalmology, 3rd edn. Lippincott Williams & Wilkins, Philadelphia
Martin C L 2005 Ophthalmic disease in veterinary medicine. Manson, London
Petersen-Jones S, Crispin S (eds) 2002 BSAVA manual of small animal ophthalmology, 2nd edn. BSAVA, Quedgeley
Slatter D 2001 Fundamentals of veterinary ophthalmology, 3rd edn. Saunders, Philadelphia

The orbit and globe

Barnett K C, Sansom J, Heinrich C 2002 Canine ophthalmology: an atlas and text. Saunders, London
Gelatt K N 1999 Veterinary ophthalmology, 3rd edn. Lippincott Williams & Wilkins, Philadelphia
Grahn B L, Cullen C, Peiffer R 2004 Veterinary ophthalmology essentials. Butterworth-Heinemann, Oxford
Martin C L 1991 Canine epibulbar melanomas and their management. Journal of the American Animal Hospital Association 17:83–90
Sullivan T C, Nasisse M P, Davidson M G et al 1996 Photocoagulation of limbal melanoma in dogs and cats: 15 cases (1989–1993). Journal of the American Veterinary Medical Association 208:891–894

Canine intraocular neoplasia

Barnett K C, Sansom J, Heinrich C 2002 Canine ophthalmology: an atlas and text. Saunders, London
Gelatt K N 1999 Veterinary ophthalmology, 3rd edn. Lippincott Williams & Wilkins, Philadelphia

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