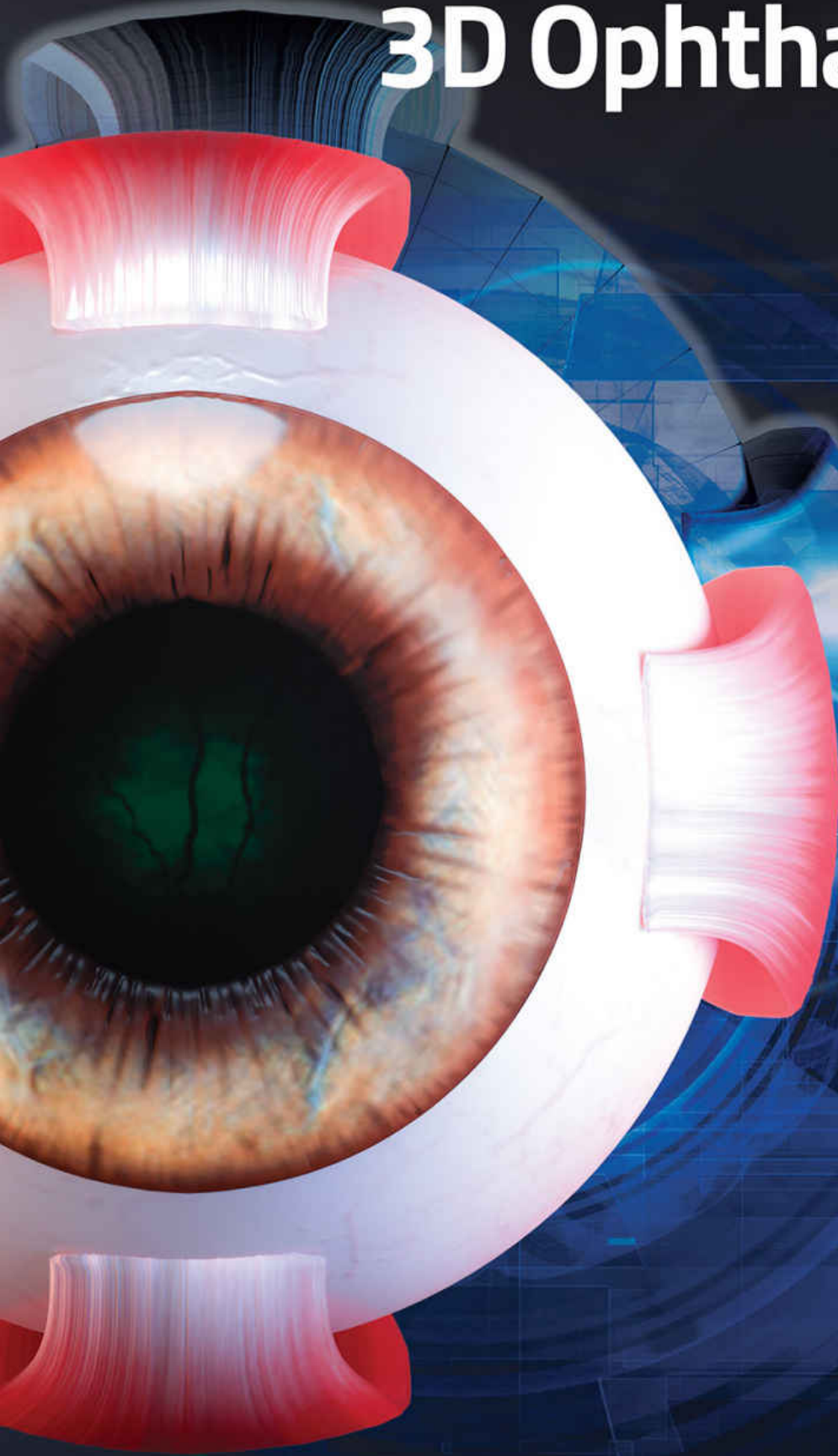


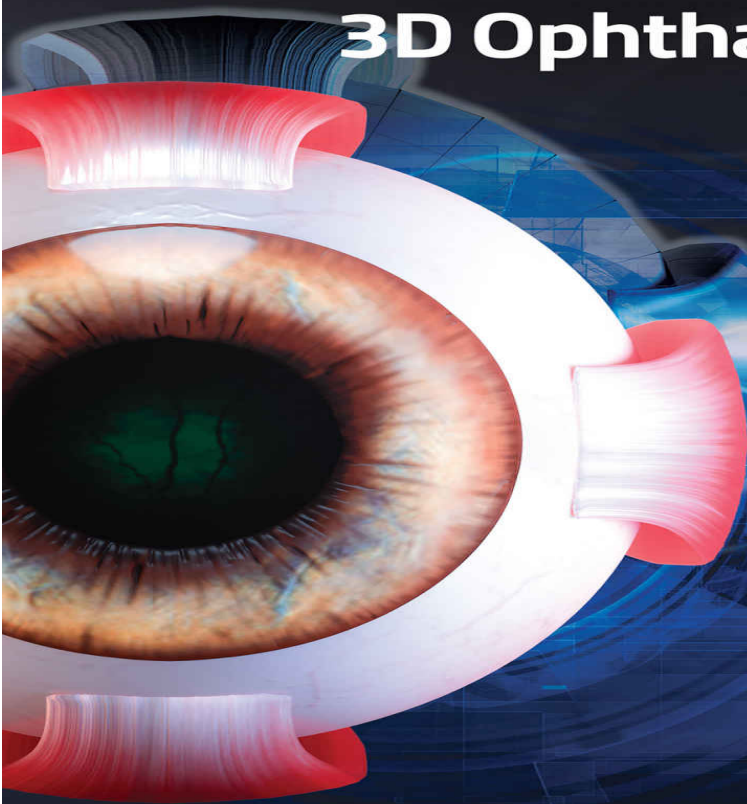
3D Ophthalmology in Dogs

FERNANDO LAGUNA SANZ
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Warning:

Veterinary science is constantly evolving, as are pharmacology and the other sciences. Inevitably, it is therefore the responsibility of the veterinary surgeon to determine and verify the dosage, the method of administration, the duration of treatment and any possible contraindications to the treatments given to each individual patient, based on his or her professional experience. Neither the publisher nor the author can be held liable for any damage or harm caused to people, animals or properties resulting from the correct or incorrect application of the information contained in this book.

Dedication

To our families, with a special thanks to our wives Paloma and Nuria, who have lavished us with endless support and patience.

To grandpa Enrique, the family's first veterinary surgeon, who taught us so much about this vocation that he held so dear.

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Fernando Laguna earned his degree in veterinary medicine at the Complutense University of Madrid in 2009. After a period in a veterinary emergency department in Paris, he served a general internship, then an ophthalmology internship at the Alfort National Veterinary School (France). Between 2012 and 2015, he completed his European College of Veterinary Ophthalmologists (ECVO) residency at the Autonomous University of Barcelona (UAB). He became a Diplomate of the ECVO in 2016.

Fernando has written several publications in journals and presented work at international conferences. At present, he works in Madrid at the Puchol Veterinary Hospital, and the Optivet ophthalmology referral centre in Havant, England.

Fernando Sanz Herrera

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He is a member of several Spanish and international ophthalmological societies and a founding member of the first executive committee for the Spanish Society of Veterinary Ophthalmology (SEOVET), over which he presided for 2 years. Fernando is also AVEPA-certified (the Spanish Small Animal Veterinary Association) and a member of the European Network in Veterinary Ophthalmology and Animal Vision (REOVVA) working group.

He has presented material in several courses, workshops, and seminars on ophthalmology. In addition, he has authored and coauthored original work published in prestigious Spanish and international journals. He cowrote the series of books *La Oftalmología en Colores* (Ophthalmology in Colour), wrote chapters on ophthalmology in the books *La leishmaniosis canina: una visión práctica* (Canine Leishmaniasis: A Practical Vision), *Inmunología clínica del perro* (Clinical Immunology of the Dog), and *Inmunología clínica del gato* (Clinical Immunology of the Cat), authored the *Manual of Ophthalmology in the Dog*, translated into English and Japanese, and *Veterinary Ophthalmology: Examination Procedures and Basic Surgical Techniques*. In April 2008 he opened the Visionvet Veterinary Ophthalmology Clinic – the first ophthalmology referral centre in southern Spain – where he exercises his profession on a full-time basis.

Foreword

“To acquire knowledge, one must study; but to acquire wisdom, one must observe”

Marilyn von Savant

There has never been a better opportunity to observe such detailed illustrations as those brought to us by the authors of this work, *3D Ophthalmology in Dogs*.

Anyone lucky enough to hold this magnificent work in their hands only has to scan through a few pages before they realise it is a real gem, a very special book that will prove an unforgettable read.

It contains a highly organised, concise, and comprehensive explanation of the anatomy and physiology of the eye, with in-depth descriptions of the most common eye diseases affecting dogs and the different medical and surgical treatments available for each condition.

Several works on veterinary ophthalmology have been published in recent years; however, the images, illustrations, and above all the 3D animations that reflect the authors' artistic quality and ingenuity set this book apart from the rest. The overall result is an outstanding manual.

It will undoubtedly serve as a reference for students, general veterinary practitioners, and veterinary ophthalmologists who wish to further their knowledge. It deserves a place in all good veterinary libraries, and I am sure it will be translated into other languages very soon.

I feel honoured to have been invited to write this foreword for the first edition of *3D Ophthalmology in Dogs* which I'm certain is destined to be a resounding success.

Thank you and congratulations.

Javier Esteban Martín

Diplomate of the Latin American College of Veterinary Ophthalmology
(CLOVE)

AVEPA-certified in Ophthalmology

Preface

Dear colleague,

3D Ophthalmology in Dogs was conceived as a practical tool that provides a clear picture, literally, of some of the most important aspects of the eye's anatomy and physiology, and of course the keys to the surgical interventions most frequently used to treat eye diseases in our canine patients.

As part of the challenges thrown up when bringing this work together, we thought that the creation of computer-enhanced videos would help readers understand what occurs inside the diseased eye and make an innovative, interesting, and above all extremely practical project.

The texts you'll find in this book, the photos, and particularly the videos will provide you with fundamental knowledge and serve as a support when explaining to owner's how you will treat their pet's eye condition using some of the surgical procedures described within.

While everything explained in the book is based on sound scientific evidence, we also wanted to share the experience we have gained over many years dedicated to the speciality of ophthalmology, including details and tips to achieve better results.

Enjoy reading the text, the photographs, and this new way of learning through the animated computer graphics we have developed for you.

Fernando Laguna and Fernando Sanz

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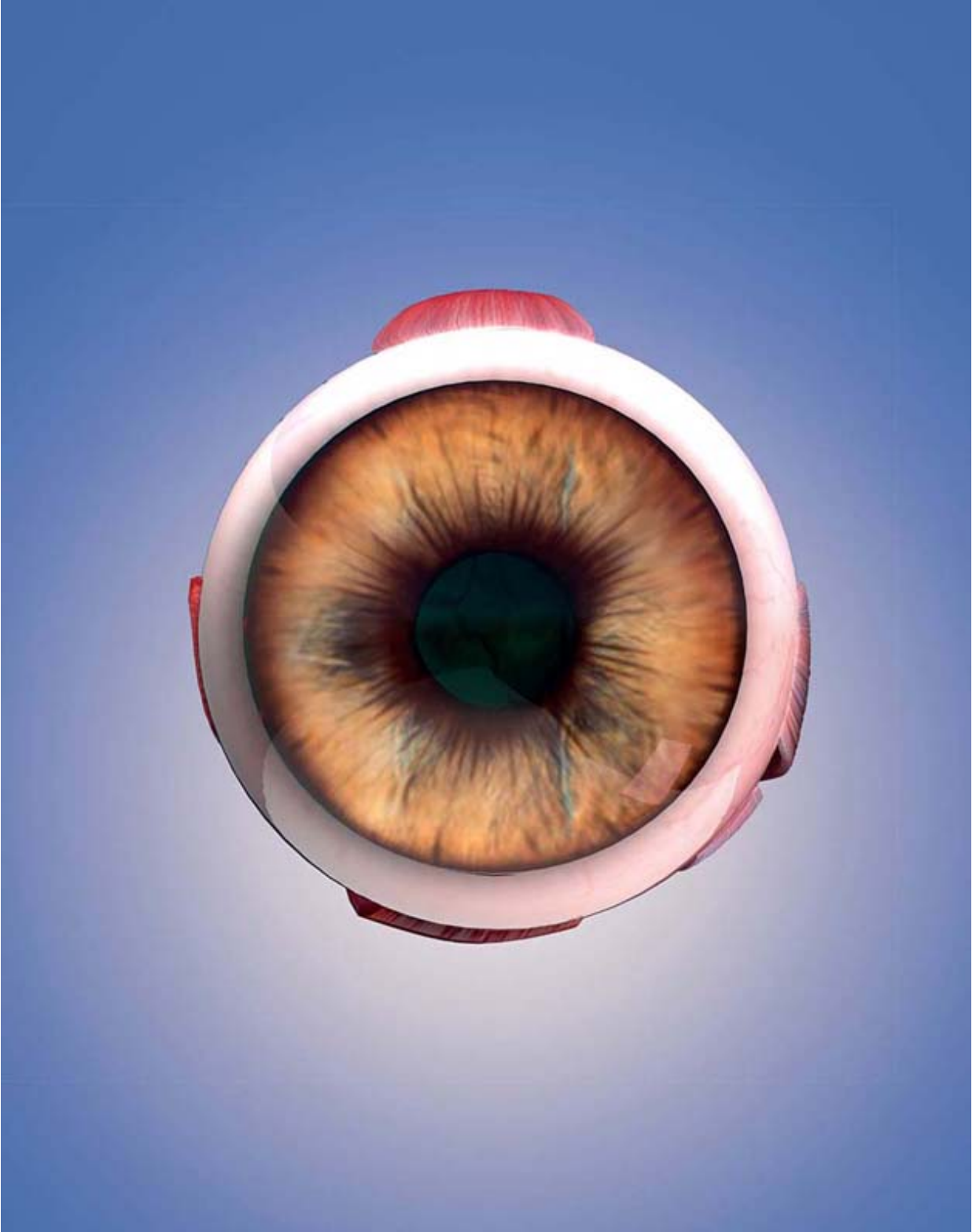
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01

Structure and function of the eye and its adnexa



Introduction

Vision is the ability of living creatures to interpret their environment through rays of light (photons).

The visual system comprises the eyes, the connecting pathways, and the visual cortex. In order for an animal to see, light photons must enter their eyes and be focused correctly on the retina. Retinal photopigments convert this light energy into electrical energy. All the properties of the image (brightness, orientation, depth, etc.) are subsequently interpreted by the visual cortex, which extracts the most relevant information.

The eye, which is almost perfectly spherical, is one of the most highly specialised organs. The main function of the eye is to focus the image and regulate the amount of light that hits the retina, thereby reducing optical aberrations and ensuring the best possible visual acuity. Any change in the transparency or the physiological arrangement of the ocular structures can diminish visual capacity.

The ocular adnexa encompass all of the eyeball's supporting structures, that is, those surrounding the eye and which contribute to its function. These include, amongst others, the eyelids, conjunctiva, nasolacrimal system, and the nictitating membrane. The eyeball is located in the orbit, where it is held in place by the extraocular muscles and orbital fasciae.

The adnexa and orbit are essential for the normal function of the eye.

Anatomy of the eye

The eye is traditionally divided into three layers:

- The fibrous tunic: formed by the **cornea** and **sclera** . This is a collagen-rich layer that lends the eye its structure, and the anterior portion is transparent to allow light to enter the eye. The optic nerve emerges at the rear of the eye and passes through a structure called the cribriform plate.
- The uvea: a highly vascularised layer, comprising the **iris** , **ciliary body** , and **choroid** .
- The retina and optic nerve: the **retina** is responsible for transduction, that is, the act of transforming light into an electrical signal. The electrical information is then transmitted to the nervous system via the **optic nerve** .

The **crystalline lens** , which helps focus the image on the retina, separates the anterior and posterior segments of the eye. The anterior segment is full of

aqueous humour , which nourishes the structures contained within and helps maintain intraocular pressure. Aqueous humour is secreted by the ciliary body and drains into the iridocorneal angle. The posterior segment contains **vitreous humour** , which is rich in collagen and helps shape the eye and hold the retina in place.

The cornea and sclera

The cornea is the most anterior part of the fibrous tunic. It is a transparent layer which in dogs has a marginally greater width than height. It is around 0.5 mm thick. It features four layers: a stratified epithelium, the collagen stroma, Descemet's membrane, and the endothelium.

The **epithelium** contains basal, intermediate, and squamous cells which are all continuously renewed. It does not contain keratin, thus promoting transparency.

The **stroma** makes up most of the cornea's thickness. It is formed from collagen fibres and keratocytes, which are special fibroblasts charged with keeping the cornea transparent, healing ulcers, and synthesising the components of the stroma. Corneal collagen is organised in lamellae that are arranged orthogonally and at a fixed separation. This special configuration allows light to pass through and differentiates the cornea from other collagen-rich structures, such as the sclera, which are opaque. Furthermore, the stroma is rich in glycosaminoglycans, and again these also contribute to transparency.

Some scarring processes can cause a loss of transparency in the cornea.

Descemet's membrane is the basement membrane for the corneal endothelium. It is an elastic layer that gets thicker as the animal ages.

In the case of glaucoma, Descemet's membrane can rupture, resulting in what are known as Haab's striae.

A corneal ulceration accompanied by a total loss of the stroma leads to exposure of Descemet's membrane (descemetocoele).

The **corneal endothelium** is the most metabolically active layer. This monocellular layer dehydrates the cornea, thus helping to maintain its transparency. Corneal endothelium cells are postmitotic, so they are unable to regenerate, and their number decreases over the course of a lifetime. The remaining cells grow larger to fill the spaces left by those which die. If the number falls below a critical value, it becomes impossible to maintain a

suitable balance between the amount of water that “enters” and “leaves” the stroma, leading to corneal decompensation and in turn oedema.

A healthy cornea should be smooth, shiny, avascular, and free from pigment. The corneal stroma is avascular and contains very few cells, so it receives oxygen and metabolites from tears, the aqueous humour, and sclera. Any alterations to these structures will cause changes in the cornea. A common example is dry eye, in which the cornea may accumulate pigment, experience neovascularisation, and lose its smooth surface.

The cornea is one of the most sensitive parts of the body. It is innervated by the long ciliary nerves, which enter the anterior corneal stroma radially, and they are subsequently attached to the ophthalmic branch of the trigeminal nerve. Corneal sensitivity is greater at the surface and in the anterior stroma; therefore, as a lesion gets deeper, sensitivity declines.

Brachycephalic dogs have less corneal sensitivity than mesocephalic or dolichocephalic breeds, which is why they show less signs of pain when affected by corneal ulcers.

The **sclera** makes up most of the fibrous tunic and is divided into three layers: the most superficial layer is called the episclera, the middle layer is the scleral stroma, and the deepest is the lamina fusca. The episclera provides the point of attachment between Tenon’s capsule and the scleral stroma. It is a highly vascularised fibrous layer. Like the cornea, the scleral stroma consists of collagen and fibroblasts; however, in this case the collagen is disorganised, so the scleral stroma is not transparent. The lamina fusca is where the most external layers of the choroid and ciliary body attach to the sclera.

The uvea

The uvea is the middle layer of the eye. It can be further divided into the anterior uvea, formed by the iris and ciliary body, and posterior uvea, comprising the choroid.

The **iris** is a diaphragm that controls how much light enters the eye. This control is achieved with two muscles: the pupillary dilator and sphincter muscles. They open or close the pupil depending on how much light enters the eye, adjusting pupil size to ambient conditions. The anterior aspect of the

iris does not have an epithelium and is composed of stromal cells. The posterior aspect has a pigmented epithelium. There are two arteries (long ciliary arteries), entering nasally and temporally, forming an arterial circle, which may or may not be complete.

The **ciliary body** is located behind the iris (Fig. 1). The anterior section features a series of folds (pars plicata), known as ciliary processes. These folds subsequently become less prominent, until they give way to a flat area (pars plana) that eventually joins the retina at the ora serrata. The ciliary body is covered with an epithelial bilayer and has a muscle at its base formed from smooth muscle fibres innervated by the parasympathetic system. These fibres attach to the base of the ciliary body and are closely related to the iridocorneal angle. Uveitis causes the ciliary muscle to contract, which is painful and increases the amount of drainage via the normal route.

The **choroid** can be found in the posterior uvea. It is a fine layer of very well vascularised tissue with a variable degree of pigmentation. Found uniquely at the back of the choroid is a triangular layer call the tapetum. Its function is to reflect light and therefore double the stimulation of the retina's photoreceptor cells. The tapetum varies in size and colour, depending on the breed of dog, and may even be physiologically absent.

One of the most important functions of the choroid is to act as the blood–ocular barrier. This separates the eye from the body's general circulation to prevent proteins and macromolecules from filtering into the aqueous humour and vitreous humour. Inflammation can alter the barrier's impermeability.

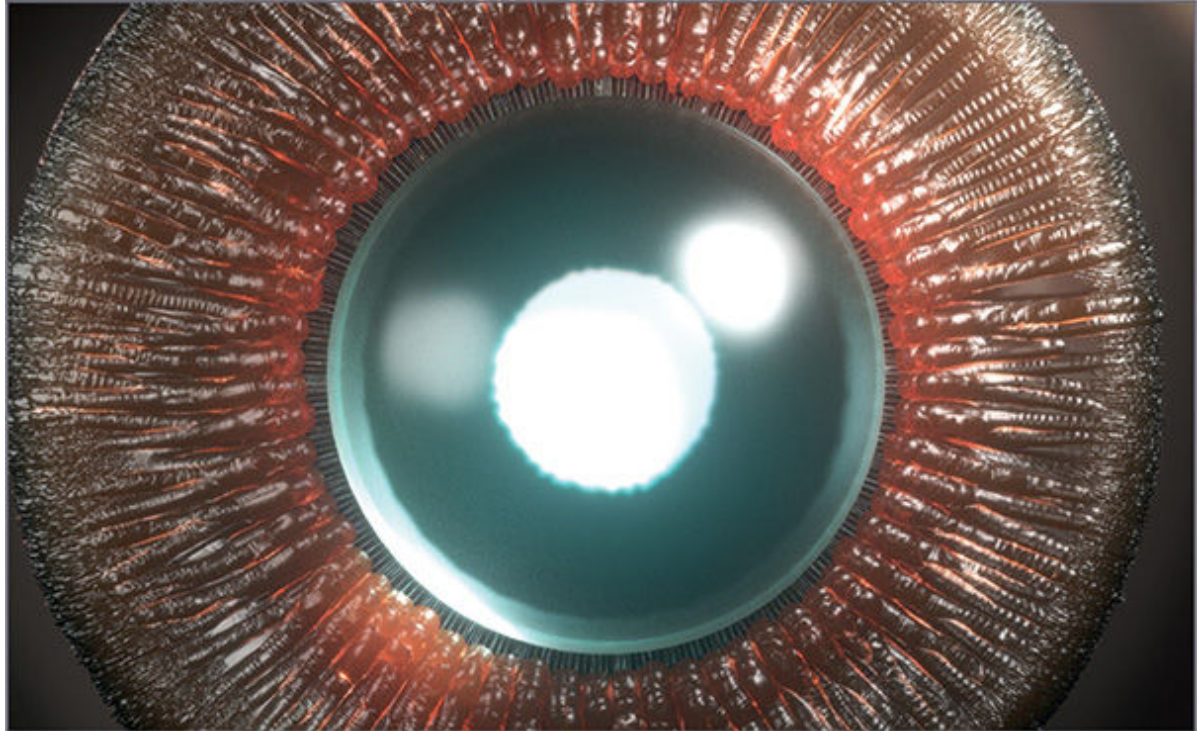


Figure 1. Crystalline lens, iris, and ciliary body as seen from the retina.

The crystalline lens

The **crystalline lens** is a biconvex, avascular lens located inside the eye and suspended by ciliary zonular fibres. The anterior face is more planar than the posterior face and the lens has an equator, which is the circumference where the two faces meet. The crystalline lens is surrounded by a capsule and has an epithelium, which is located under the anterior capsule. The lens fibres are arranged in concentric layers, running out from the central nucleus and forming the cortex. These fibres are produced throughout the animal's life, so they get pushed towards the lens nucleus.

The main purpose of the lens is to accommodate for objects at different distances and focus the image on the retina. However, dogs have a very low capacity for accommodation.

The vitreous humour

The vitreous humour is an elastic, transparent hydrogel that occupies a cavity with the same name and comprises up to 80 % of the eye's volume. It mainly consists of water, collagen fibres, hyaluronic acid, and hyalocytes.

Hyalocytes present phagocytic activity and can transform into fibroblasts and, therefore, form scar tissue.

Anatomically, this gel can be divided into the anterior, posterior, peripheral, and central vitreous, and it is worth noting that the collagen fibres are especially dense in the peripheral area, which is where these fibres attach strongly to the surface of the retina. Degeneration and liquefaction of the peripheral vitreous can reduce the force it exerts on the retina, increasing its chance of detachment.

There is another area where the vitreous fibres are firmly adhered to an intraocular structure. The crystalline lens sits in a depression in the vitreous called the hyaloid fossa, which is exactly the point where the posterior lens capsule is strongly attached to the vitreous. Shifts of the lens, which accompany its luxation, produce traction in the vitreous fibres and, consequently, in the retina.

The retina and optic nerve

The most important part of the eye is the **retina**. The ultimate objective of all the ocular structures is to focus an image on the photoreceptor cells and produce a picture with the greatest possible visual acuity. The retina is a very complex tissue composed of many different types of cell involved in generating the final electrical impulse. In simple terms, the retina contains photoreceptor cells that transmit the electrical impulse to the bipolar cells, which then transmit it to the ganglion cells. The axons of the ganglion cells form the optic nerve. Photoreceptors are comprised of cone and rod cells, both of which contain photopigments that react to light and produce an action potential.

Cone cells are sensitive to more intense light and provide colour vision, while rod cells are stimulated by less intense illumination.

The photoreceptors rely on the retinal pigment epithelium, as it performs several functions such as recycling photopigments. In the event of retinal detachment, the pigment epithelium always remains attached to the choroid.

The **optic nerve** is formed when all the ganglion cell axons merge to establish the optic nerve head. The nerve then traverses the cribriform plate, through the cone formed by the extraocular muscles, and the optic foramen

of the orbit, after which it joins with the optic nerve from the other eye at a point called the optic chiasm (Fig. 2).

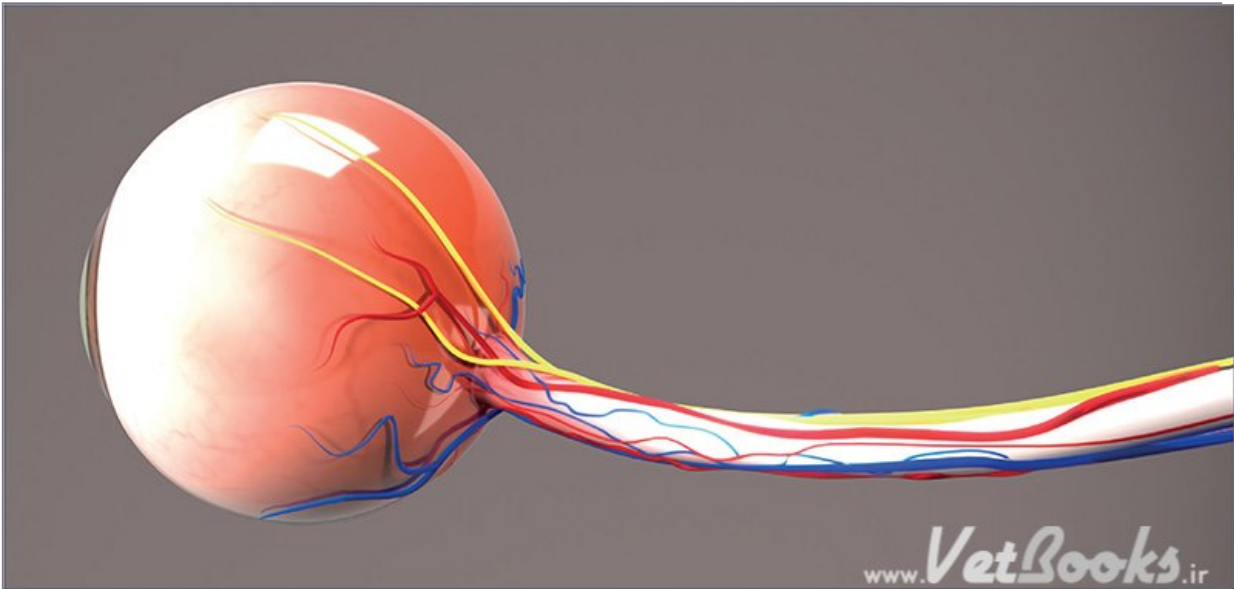


Figure 2. Eyeball with the departing optic nerve.

Anatomy of the ocular adnexa

Eyelids

The eyelids are musculocutaneous flaps vital to maintaining a healthy ocular surface. They comprise the tarsus (a fibrous plate that provides structure), muscles that control its position and contraction, elastic skin on the external surface, and palpebral conjunctiva on the internal surface. Dogs only have eyelashes on the upper eyelid (Fig. 3).

There are some glands on the eyelid margin, called meibomian or tarsal glands, that secrete a mixture of lipids. The glands' orifices can be examined with an optical magnification system. There are some other glands, e.g. the glands of Zeis and Moll, but they are clinically insignificant.

Principally, the eyelids are closed by contracting the orbicularis oculi muscle, which is situated in the anterior portion of the tarsus of both eyelids. In dogs, the upper eyelid is more mobile than the lower eyelid and innervated by the oculomotor nerve, unlike other palpebral muscles which are innervated by the facial nerve. The eyelids have a ligament in the medial canthus and a muscle that serves as a ligament in the lateral canthus, the retractor muscle of the lateral angle (retractor anguli oculi lateralis).

Sensory information from the eyelids is transmitted via the trigeminal nerve. When pain is detected, the orbicular muscle contracts strongly, resulting in blepharospasm.

The eyelids protect the eyes and help distribute and drain tears. When blinking, the eyelids start to close at the lateral canthus and the closure advances like a zip until it reaches the medial canthus. This flushes tears towards the lacrimal puncta from which point they drain towards the nose.

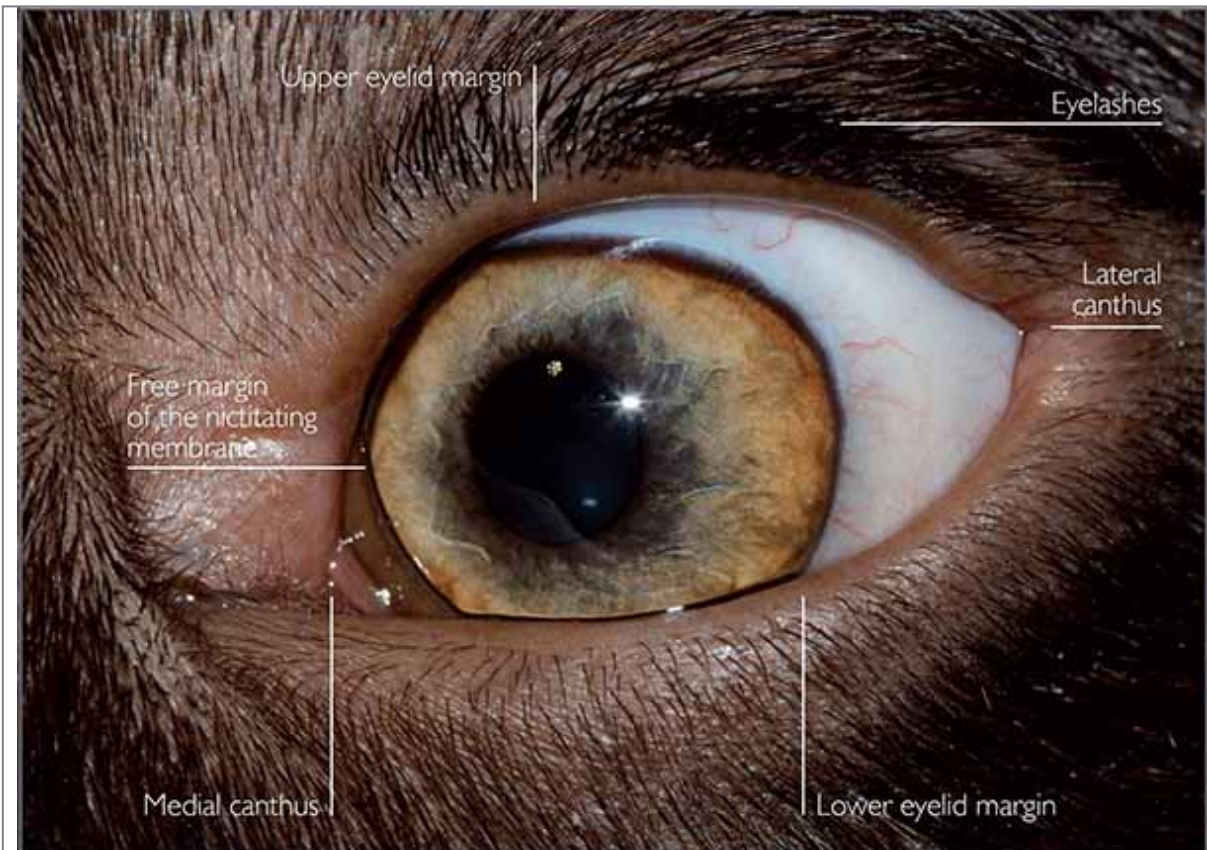


Figure 3. Structures of the eyelid in dogs.

Nictitating membrane

Many companion pets have a nictitating membrane, or third eyelid, which is a structure located in the medial area and ventral to the eye and held firm by a T-shaped cartilage. The base of the cartilage is connected to the lacrimal gland and both its bulbar and palpebral surfaces are covered with a conjunctiva. The internal surface of the conjunctiva is covered with lymphatic follicles.

The nictitating membrane in dogs has a vestigial musculature, which moves passively when the eyeball (innervated by the abducens nerve) is retracted. The lacrimal gland housed in this membrane is important, as it produces 30–50 % of the aqueous component of tears (Fig. 4).

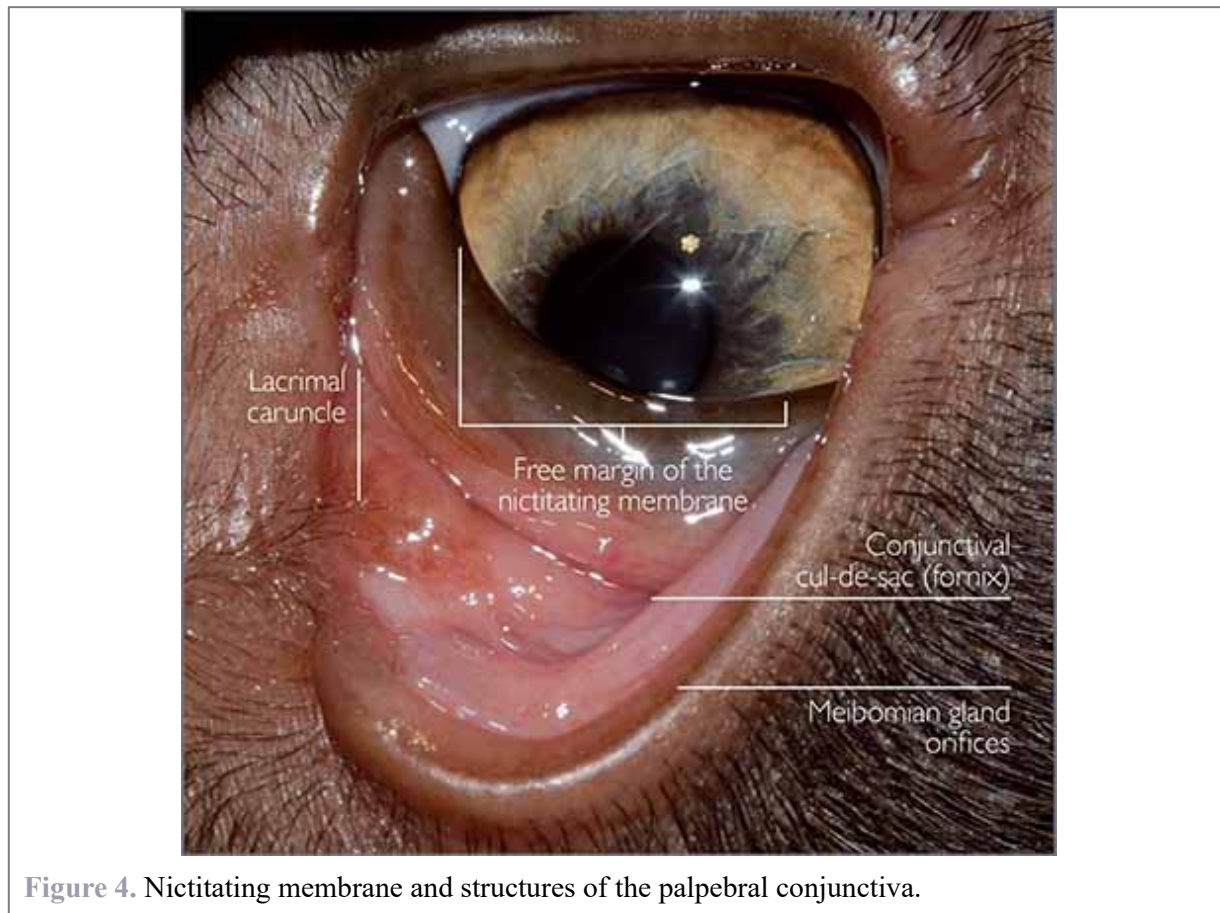


Figure 4. Nictitating membrane and structures of the palpebral conjunctiva.

Conjunctiva

The conjunctiva is a mobile, elastic mucous membrane covering the internal, or palpebral, surface of the eyelids and the bulbar surface of the nictitating membrane; it is attached to the corneoscleral limbus where it forms the bulbar conjunctiva (Fig. 5). It also has a fornix, which is a cul-de-sac-like structure that establishes the junction between the dorsal and ventral palpebral and bulbar conjunctivae.

It is composed of a cylindrical or columnar nonkeratinised epithelium that contains mucus-secreting goblet cells. This mucus corresponds to the protein phase of tears, and amongst other functions, it helps ensure tears adhere to the eye.

Any inflammatory or infectious process affecting the ocular surface can alter the conjunctiva's morphology, causing a loss of goblet cells and modifying its characteristics.

Under the epithelium there is a layer called the substantia propria formed from conjunctival tissue.

The conjunctiva plays a crucial role in the immune system and tends to develop many follicles in response to antigenic stimulation.

A notable characteristic of the conjunctiva is its significant capacity for regeneration and healing, in fact a conjunctival lesion can heal in just a few hours.

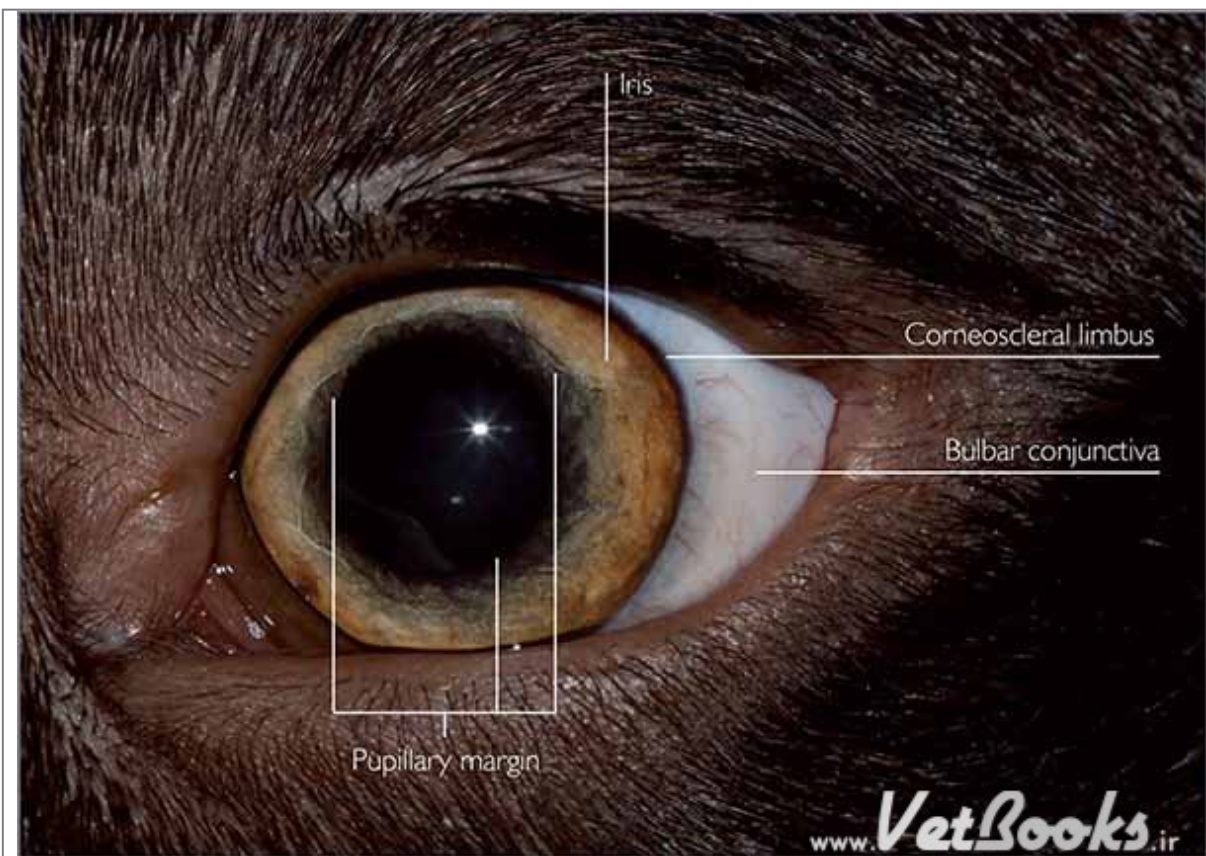


Figure 5. Structures of the anterior segment.

The nasolacrimal system

The nasolacrimal system secretes, distributes, and drains tears from the ocular surface. It has a main gland in the superotemporal region, the nictitating membrane gland, and some accessory glands. The lacrimal gland secretes tears through some small ducts in the dorsal fornix. The nictitating

membrane gland has several secretion ducts on the bulbar surface of the membrane's conjunctiva.

The ocular surface (cornea and conjunctiva), the main and accessory (nictitating membrane) lacrimal glands, and the tarsal glands form the lacrimal functional unit. Any alterations in one of these components will compromise the function of the lacrimal system and may have very severe consequences.

Although it is usually considered as a complete unit, the tear film can be subdivided into three parts:

1. The **lipid phase** , secreted by the tarsal glands, prevents excessive tear evaporation and reduces tear surface tension, thus stabilising them.
2. The **aqueous phase** , secreted by the main and nictitating membrane tear glands, is the thickest phase and transports nutrients, oxygen, etc. In addition, it plays a significant role in the fight against microorganisms by flushing them away from the ocular surface and destroying them thanks to antibacterial substances such as lactoferrin and lysozyme.
3. The **protein phase** , secreted by goblet cells, is lipophilic and therefore helps tears adhere to the corneal epithelium. Any alterations in this phase will increase the tear break-up time.

Tears are dispersed across the ocular surface by the eyelids and nictitating membrane, and drain via the lacrimal puncta, which are situated in the palpebral conjunctiva, approximately 5 mm from the medial canthus. The dorsal and ventral ducts join and form the lacrimal sac, which is relatively small in pets. The nasolacrimal duct carries tears to the nasal cavity where they eventually drain. In some dogs the nasolacrimal duct can lead to the mouth.

Anatomy of the orbit

The orbit is the conical cavity surrounding the eye, and it has an open form in domestic carnivores. It comprises the bony orbital rim and lateral orbital ligament, which runs from the zygomatic process of the temporal bone to the temporal process of the zygomatic bone. This configuration allows the animal to open its mouth wider.

The orientation of the orbit determines the animal's visual field. The bone forming the orbit has various foramina through which blood vessels and nerves pass to reach the eye and its adnexa. This bone is covered by the periorbital, which is where extraocular muscles are inserted and the orbital fat pad is housed (Fig. 6). Tenon's capsule is a fascia that joins with the episclera.

Domestic carnivores have four rectus muscles (dorsal, ventral, lateral, medial), two oblique muscles, and the retractor bulbi muscle. The rectus (except the lateral) and the ventral oblique muscles are innervated by the oculomotor nerve. The lateral rectus and retractor bulbi are innervated by the abducens nerve. Finally, the dorsal oblique muscle is stimulated by the trochlear nerve.

The optic nerve lies in the most interior part of the orbit, surrounded by the extraocular muscles. It is enveloped by the meninges and therefore considered part of the central nervous system.

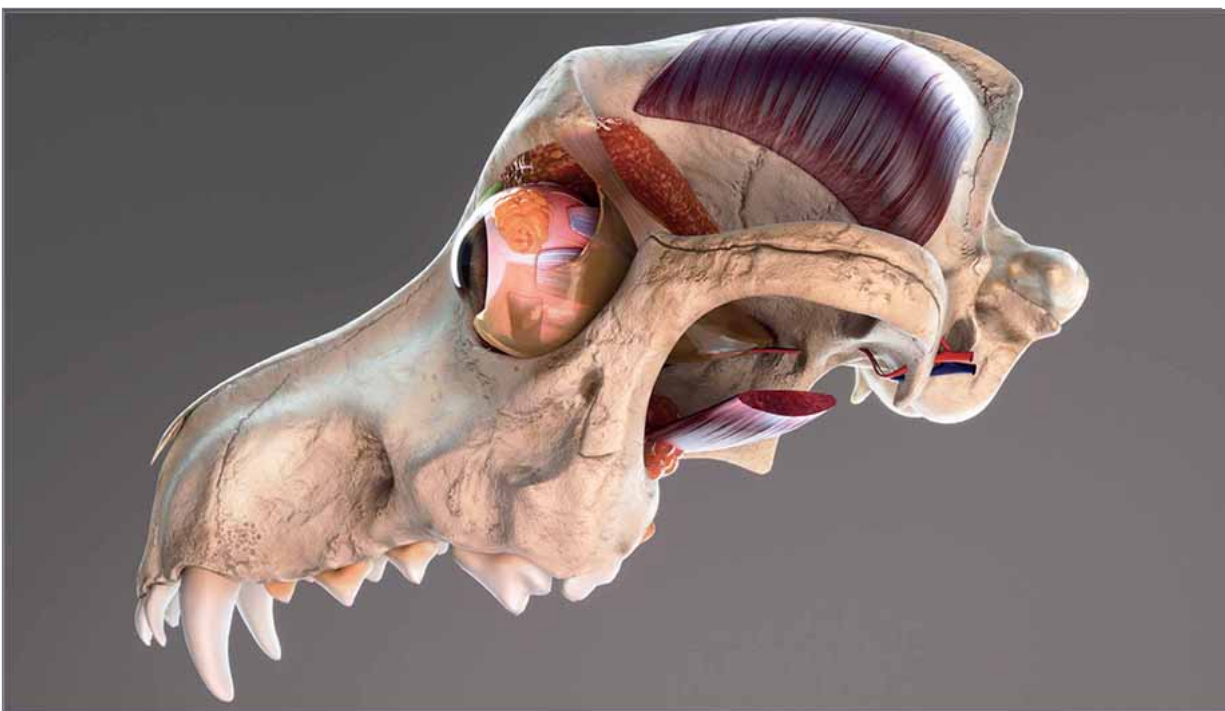


Figure 6. Eyeball and periorbital structures.





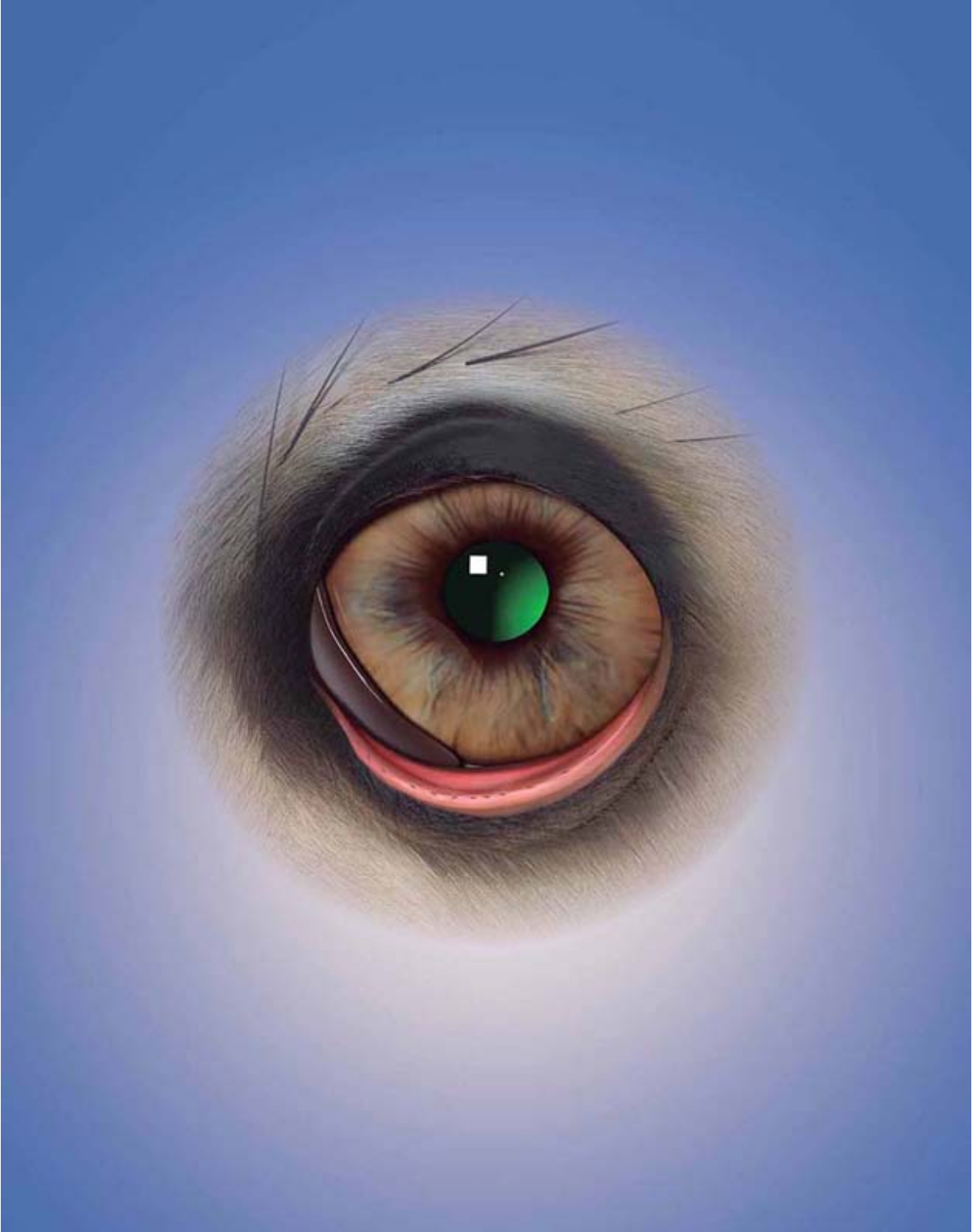
Structures of the eyeball



Extraocular structures

02

**Eyelids.
How to treat the
most common
diseases**



Introduction

The eyelids form the boundary between the skin and the eye; consequently, they are affected by the same pathogenic mechanisms. Unlike “normal” skin, the eyelids have several associated structures, including the eyelashes, glands, tarsi, etc. They are also highly vascular.

The position of the eyelids is crucial for the health of the ocular surface and for maintaining an unobstructed visual axis. This means that some eyelid conditions can cause blindness if they are not treated correctly.

This chapter discusses different eyelid abnormalities and their treatment.

Eyelid opening anomalies in newborns

Puppies and kittens are both born with their eyelids closed, and they usually open after approximately 14 days. This period of closed eyelids is called physiologic ankyloblepharon.

Early eyelid opening is uncommon, and puppies tend to suffer from dry eye, keratitis, and conjunctivitis because tear production is not yet at physiological levels due to the immaturity of the excretory system. The recommended treatment for this problem is ocular lubricants to improve hydration of the ocular surface and topical antibiotics to restrict bacterial overgrowth. Other options include the nictitating membrane flap procedure or a temporary tarsorrhaphy until the lacrimal system is sufficiently mature.

It is much more typical, however, to come across animals with delayed opening of the palpebral fissure (beyond 14 days of life), a situation called pathologic ankyloblepharon. These patients develop a significant accumulation of mucous secreted under the eyelids, which can be complicated by bacterial overgrowth and pus (ophthalmia neonatorum). What is more, cats also tend to suffer from chlamydia and feline herpesvirus.

The application of warm, moist compresses is indicated to treat pathologic ankyloblepharon as it will encourage eyelid opening and help eliminate secretions. If this does not work, you can try to open the palpebral fissure with gentle digital pressure or by introducing a blunt instrument in the small aperture of the medial canthus. The use of a sharp instrument is ill-advised, as it would alter the tarsal glands and result in a lifelong qualitative tear production deficit. The use of topical antiseptics, e.g. 2 % iodopovidone, and an inspection of the cornea are also recommended.

Eyelid colobomata

A coloboma is the underdevelopment of part of an eyelid, resulting in a defect which, depending on its location, will cause complications such as overexposed corneas and conjunctivae or surrounding hairs brushing against the ocular surface.

The most common presentation of this abnormality tends to affect cats, in particular, the most lateral portion of the upper eyelid. Animals with an eyelid coloboma often display signs of discomfort as hairs close to the defect brush against the eye, which can occasionally cause corneal ulcers and chronic keratitis.

The application of moisturising gels and, if necessary, antibiotic ointments helps prevent complications until a blepharoplasty can be performed to correct the problem. Depending on the size of the defect, the operation may require the use of skin flaps.

Distichiasis

Distichiasis is the presence of extra hairs or cilia emerging from the meibomian gland orifices (Fig. 1). These hairs, which should not be found in this location, emerge from ectopic hair follicles in the tarsi. They are a relatively common anatomical anomaly in dogs, particularly in certain breeds such as Cocker Spaniel, English Bulldog, Poodle, and Golden Retriever. While they may course without any clinical consequences, the extra eyelashes can irritate the ocular surface and cause blepharospasm, conjunctival hyperaemia, tearing, and corneal ulcers. As a general rule, longer, softer cilia tend to be less irritating and produce fewer clinical signs than shorter, harder ones. Therefore, epilation with forceps is contraindicated as the cilia will only grow back stronger. With distichiasis, the treatment objective is to destroy the follicles so the hairs will stop growing back. Some of the techniques include:

- **Cryotherapy:** this is the most used method. It consists of freezing the palpebral conjunctiva close to the eyelid margin, in the area near the glands (Fig. 2). Typically, two freeze/thaw cycles are applied to ensure treatment efficacy. Despite this, recurrences are still possible. Marked blepharoeidema and necrosis of the eyelid margin can develop after the application of cryotherapy. As such, we recommend the use of oral and topical steroidal or nonsteroidal anti-inflammatories. In the long-term, the

eyelid margin could present depigmentation and alopecia. It is important to examine the four eyelids carefully to make sure you treat all of the glands with ectopic cilia. Sometimes the cilia will grow back, but with very little vigour. If they are removed with epilation forceps, they may grow back later.

- **Electrolysis:** or electroepilation, involves the use of a very fine electrode inserted in the gland opening in order to apply an electric current and destroy the follicle. The electric current must be low enough to avoid undue tissue necrosis and to ensure the orbicularis oculi muscle does not contract.
- **En-bloc resection of the follicles:** after using a magnification system to identify the effected follicles, they are surgically removed approaching from the conjunctival side of the eyelid or via a blunt dissection of the eyelid's full thickness.

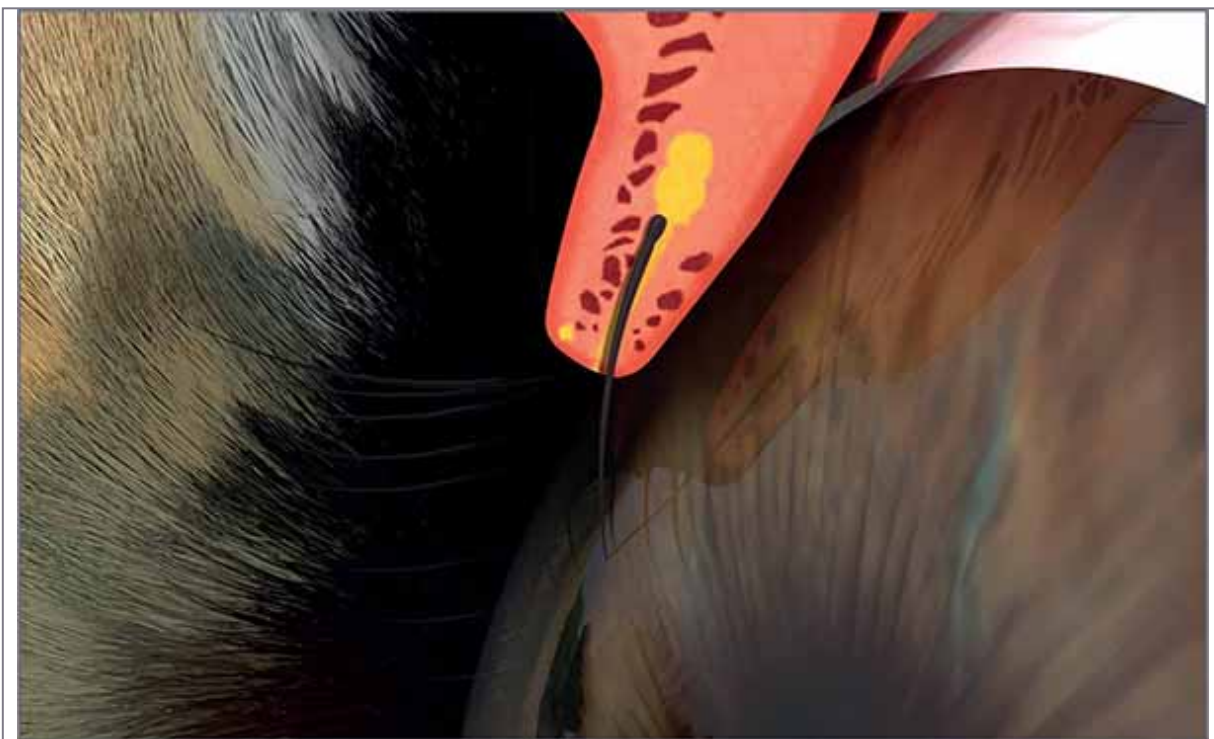


Figure 1. Diagram of distichiasis.



Figure 2. Eyelid eversion using chalazion forceps and cryotherapy.

Ectopic cilia

Ectopic cilia are hairs that develop in the tarsus and emerge from the palpebral conjunctiva towards the ocular surface (Figs. 3 and 4). Unlike distichiae, they always have adverse clinical consequences. Ectopic cilia are not always considered congenital, but they generally appear before adulthood. They constantly brush against the cornea and cause ulcers in most cases. There may also be signs of chronic trauma as well as corneal granulomas. The ulcers are located in the region of the ectopic cilia, which is usually the upper eyelid. Ectopic cilia may or may not be pigmented, so they can be very hard to find. Whenever an animal has one ectopic cilium, it is very important to examine the palpebral conjunctiva of both eyes very carefully in search of any other cilia.

Ectopic cilia are always treated surgically, namely by resecting the cilia and their associated follicles. They can be removed via simple resection or using a small-gauge skin biopsy punch. Often, multiple hairs emerge from each ectopic cilium follicle, and it is important to excise all of them.



Figure 3. Diagram of an ectopic cilium.



Figure 4. Ectopic cilium.



Hair and eyelash alterations: trichiasis, distichiasis, ectopic cilium



Surgical resolution of an ectopic cilium

Trichiasis

Trichiasis is when hairs that originate in a physiologically normal site grow in the wrong direction (Fig. 5). The situation may be primary or secondary to eyelid coloboma, nasal folds, or entropion. Caruncular trichiasis is a subtype in which hairs originating from the caruncle in the medial angle of the eye cause irritation, potentially resulting in corneal pigmentation, ulcers, and epiphora because the hairs attract tears by capillary action.

The treatment of trichiasis varies widely depending on the origin of the hairs. Nasal fold trichiasis can be treated by means of a partial or total resection of the fold. Caruncular trichiasis is resolved by performing a medial canthoplasty (see later in this chapter).



Figure 5. Diagram of trichiasis.

Entropion and ectropion

Entropion is when the eyelid turns inward. This means the hairs on the skin side of the eyelid will brush against the eye (trichiasis). This constant trauma at the ocular surface may induce conjunctival hyperaemia, tearing, and keratitis. The keratitis could be ulcerative in acute cases or feature corneal pigmentation and fibrosis in chronic cases (Fig. 6). In addition, medial

entropion may ‘conceal’ the lacrimal punctum causing epiphora. This usually occurs in the inferior punctum, but it can also affect the superior one.

Entropion can be due to various causes:

- **Anatomical or conformational entropion:** frequently observed in many different breeds, it typically impacts on the lower eyelid. Conformational entropion of the upper eyelid is normally associated with facial skin folds (pseudoptosis) and the weight they exert on the eyelids. Even though entropion is an anatomical abnormality it might not manifest until the dog reaches adulthood, as there is a very close relationship between skull shape, eyelid size and opening, and eyeball size.
- **Spastic entropion:** this is the result of an intense contraction of the orbicularis oculi muscle that causes the eyelid to turn inward. When assessing entropion, it is vitally important to take this spastic component into account to avoid overcorrection which could lead to secondary ectropion.

Chronic spastic entropion can produce changes in eyelid anatomy such that the situation transforms into a case of anatomic entropion. This is the main cause of entropion in cats.

In the case of entropion, it is essential to examine the ocular surface by conducting a Schirmer’s test and slit lamp examination of the cornea, conjunctivae, and eyelids. The use of fluorescein and rose bengal is highly recommended, as is a topical anaesthetic to alleviate eye pain.

Ectropion is when the eyelid turns outward and occurs almost exclusively in the lower eyelid. The two main causes of ectropion are:

- **Conformational ectropion:** affects breeds such as San Bernardo, Cocker Spaniel, and Dogo Argentino.
- **Cicatricial ectropion:** this occurs following traumatic eyelid surgery or can be due to scars or burns.

Both entropion and ectropion are resolved exclusively through surgical procedures.



Figure 6. Entropion giving rise to pigmentary keratitis in a Shar Pei.

3D



Entropion and its surgical resolution

3D



Ectropion and its surgical resolution

Lacerations

Eyelid lacerations are one of the most frequent ophthalmic emergencies; they may warrant an urgent intervention if they are transfixing (affect the full thickness of the eyelid) or involve the margin. Given that they are highly vascularised, and because of the tension produced by the various muscles and ligaments, the eyelids become swollen and inflamed and the lacerations may retract.

Before making a surgical decision, the veterinary surgeon must assess the animal's condition and perform a complete ophthalmological examination to detect damage to the conjunctiva, cornea, sclera, extraocular muscles, and so on. If the laceration is close to the medial canthus, it may have affected the nasolacrimal system. In this case reconstruction is required, especially if it affects the lower lacrimal duct.

Handle the ocular structures with the utmost care during the intervention and always remove as little devitalised tissue as possible. The margins of the laceration can be revived by scraping them carefully with a scalpel blade. As with all full-thickness eyelid surgeries, it is a good idea to apply two planes of suture using a resorbable material in the deepest plane while taking great care to avoid piercing the palpebral conjunctiva. We recommend using a figure-of-eight stitch at the eyelid margin. If the nasolacrimal system needs reconstructing, inject air into the lacrimal puncta and note where bubbles appear in the laceration. The bubbles mark the point where the ends should be probed to reconstruct the duct.

Besides topical antibiotics (you could use a triple antibiotic or an aminoglycoside), patients should also be given systemic antibiotics and systemic nonsteroidal anti-inflammatories.

Blepharitis

Blepharitis, or inflammation of the eyelids, may be a primary (no association with a systemic disease) or secondary condition (Fig. 7). The markedly

vascularised character of the eyelids means the inflammation often has severe consequences for eyelid anatomy, such as a loss of gland structure or cicatricial ectropion.



Figure 7. Blepharitis in a dog with leishmaniasis.

Chalazion

Chalazion is the accumulation of lipids secreted by the tarsal glands. This mass of fat is usually due to poor drainage, which is frequently related to the presence of a tumour on the eyelid margin. The result is a granulomatous foreign-body reaction. In many cases, the “mass effect” is much more attributable to the lipids and associated inflammation than the actual tumour. Tumour excision and scraping the chalazion using a conjunctival approach tend to resolve the problem.

Bacterial blepharitis

Also known as staphylococcal blepharitis or canine juvenile cellulitis. It is a common disease among puppies but less frequently encountered in adult dogs. It produces small abscesses on the skin of the head, mainly on the eyelids, and submandibular lymphadenopathy. Labradors and Golden Retrievers are particularly predisposed to bacterial blepharitis.

The bacteria involved are typically from the *Staphylococcus* genus. They produce necrotising toxins that trigger the rapid destruction of the eyelid margin accompanied by tarsal gland distortion and fibrosis. Therefore, in addition to topical and systemic antibiotic therapy, patients should also be given systemic corticosteroids (prednisolone: 0.5 mg/kg every 12 hours) as soon as possible.

Medial canthal ulcerative blepharitis

Commonly observed in German Shepherd Dogs, medial canthal blepharitis may be the expression of other autoimmune diseases, such as pannus or chronic superficial keratitis. It features a lymphoplasmacytic infiltrate with sebaceous hyperplasia and not only affects the medial canthus, but also the lateral canthus. It has also been reported in other breeds including long-haired Dachshund and toy and miniature Poodle.

Correct treatment controls the disease very effectively, with the first choice being topical corticosteroids or cyclosporine.

Acute allergic blepharoconjunctivitis

This is a hypersensitivity reaction to allergens (insect bites, pollen, etc.) which causes urticaria. There is no breed-specific predisposition, but there is a higher incidence among young dogs. It is important to extract information about possible causes during the anamnesis, such as a walk in the countryside or through freshly cut grass. The most common clinical signs are eyelid oedema and erythema, and conjunctival hyperaemia and chemosis. These are accompanied by other cutaneous lesions, such as papules or angio-oedema (Fig. 8).

The condition is a self-limiting process. In severe cases, the ocular surface should be irrigated and patients given systemic corticosteroids or antihistamines, for example cetirizine (1 mg/kg, every 24 hours).



Figure 8. German Shepherd Dog with acute allergic blepharitis. The patient also has papules on the pinna.

Chronic allergic blepharoconjunctivitis

Although this condition is well known in human medicine, allergic conjunctivitis is uncommon in veterinary medicine. It often requires diagnosis by exclusion after ruling out all other possible causes.

Allergic blepharitis and conjunctivitis fall within the spectrum of atopic dermatitis and may be triggered by environmental allergens or a food hypersensitivity. Therefore, intradermal skin testing and elimination diets with novel or hydrolysed proteins are important in the diagnosis. Blepharitis is more likely to be the result of an environmental allergen than a food allergy, which tend to course with more gastrointestinal clinical signs.

Moreover, food allergies do not vary with the seasons and have a poor response to systemic glucocorticoid treatment.

The key step in the treatment of allergic blepharoconjunctivitis is the identification and desensitisation of the allergen involved. Unfortunately, in some cases the causal allergen cannot be identified, so treatment should focus on controlling any inflammation, pruritus, or secondary infections. The use of topical corticosteroids and nonsteroidal anti-inflammatories is not very effective. Vasoconstrictors, such as phenylephrine or tetryzoline, can be used as symptomatic treatment to significantly reduce any redness, but they will not eliminate the cause. There are only a handful of studies into the use of antihistamines in dogs.

Eyelid tumours

Eyelid tumours are common in both cats and dogs, but each species presents very different types of tumour. In dogs, the most regularly encountered examples are meibomian gland adenomas, papillomas, histiocytomas, and melanomas. They are usually benign tumours that can be treated surgically and without any adjuvant therapies. As for cats, eyelid tumours have a worse prognosis and the neoplasms with the highest incidence are squamous cell carcinomas and mast cell tumours.

The aim of surgery on eyelid tumours is to remove the entire mass and restore eyelid anatomy. This can be hard to achieve for large tumours that affect more than a third of the eyelid's length as their resolution necessitates a blepharoplasty.

The H-plasty technique

A H-plasty involves using a skin advancement flap to fill a rectangular eyelid defect (Fig. 9). The height of the rectangle must match the size of the excised Burow's discharge triangles and the pedicle must be freed from the deepest layers to ensure there is no tension.

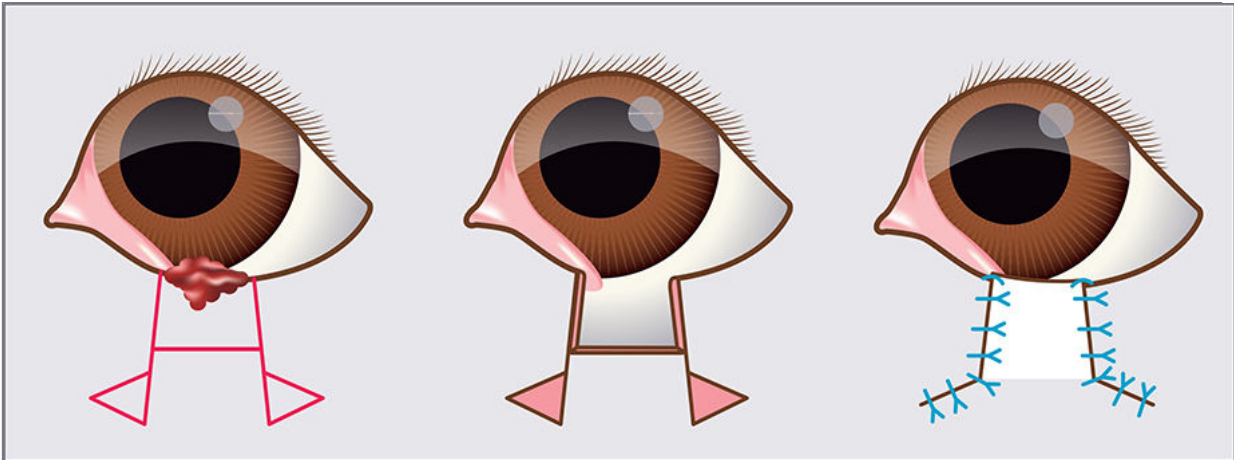
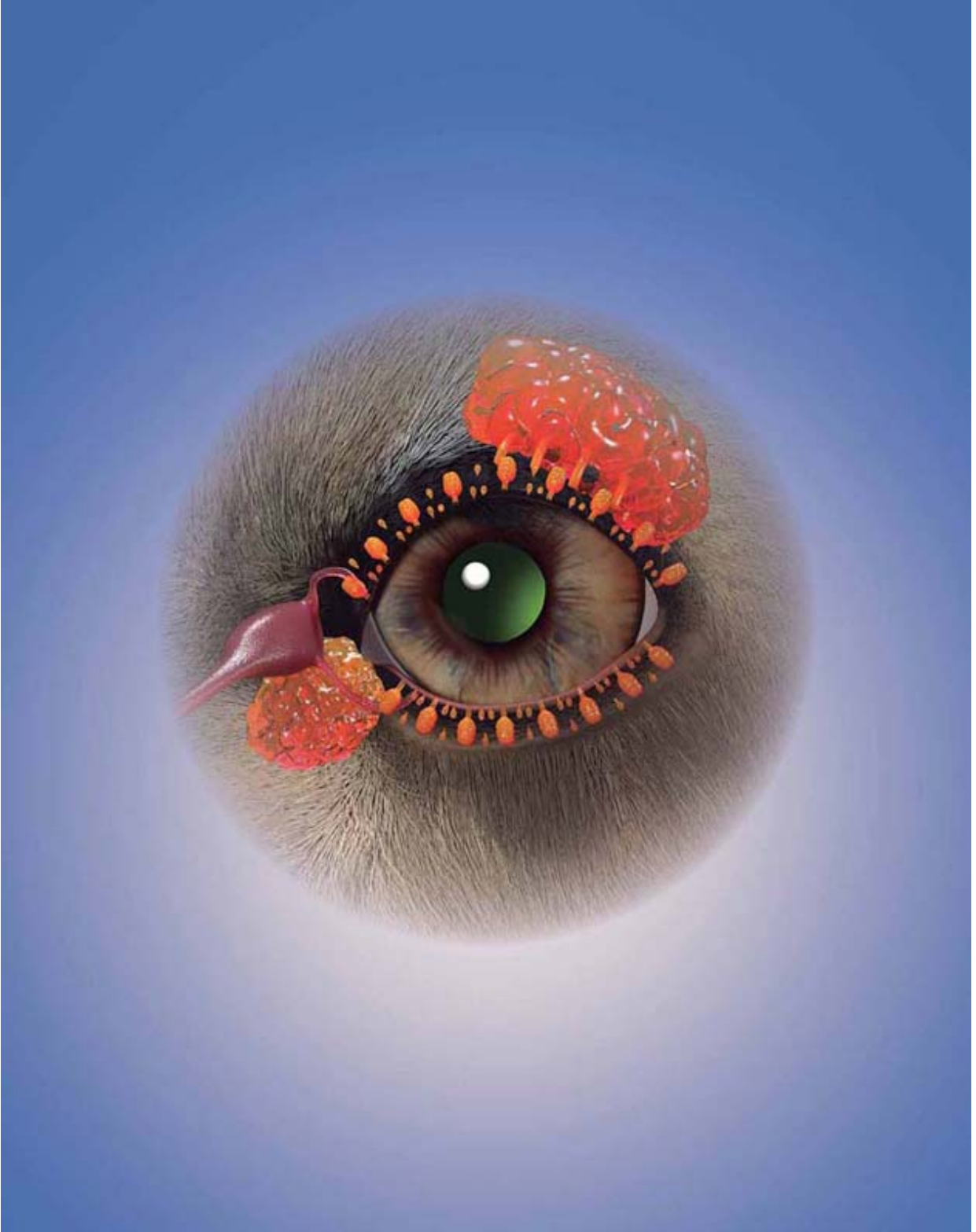


Figure 9. H-plasty.

03

The lacrimal system



Tear production and drainage

The lacrimal or tear film is vital in the physiological maintenance of the structures of the ocular surface. The cornea, conjunctivae, and even the eyelid margins are highly dependent on the “health” of the tears, to such an extent that any condition that affects tear production, composition, distribution, or their subsequent elimination will also result in palpebral, conjunctival, and corneal compromise.

Traditionally, the tear film has always been described as a trilayer structure, and while it is now believed to be a complex formed from two well-differentiated fractions, there is a general consensus that it includes: a lipid-rich surface which helps reduce evaporation; an intermediate aqueous portion involved in trophism, lubrication, and supplying leukocytes and substances that contribute to immunity; and lastly, the innermost layer which is rich in mucins and plays a vital role in helping tears attach and adhere to the surface of the cornea.

Both the main and accessory lacrimal glands are involved in tear production. The main glands are planar, almond-shaped structures located in the orbit, just above the superotemporal portion of the eyeball, and their secretions drain into the bottom of the upper conjunctival sac through 3–5 ducts. The accessory lacrimal glands are located in the thickness of the nictitating membrane and drain into multiple ducts that lead to the bulbar aspect of said membrane. These glands are involved in the production of the serous portion of tears.

The tarsal or meibomian glands, along with the glands of Zeis, which are sebaceous glands associated with the eyelashes, are responsible for producing the lipid layer of the tear film and finally the goblet cells distributed across the conjunctiva produce the mucin fraction that makes up the film’s deepest layer.

The collective structure of all the components involved in the ocular surface is designed so that the movement of the eyelids and conjunctivae directs tears towards the medial canthus where the superior and inferior lacrimal puncta are located (Figs. 1 and 2). The puncta form the “escape hatch” by draining tears through two narrow ducts. The upper and lower lacrimal ducts converge into a small lacrimal sac from which emerges the duct that ultimately drains the tears into the nasal cavity (Fig. 3).

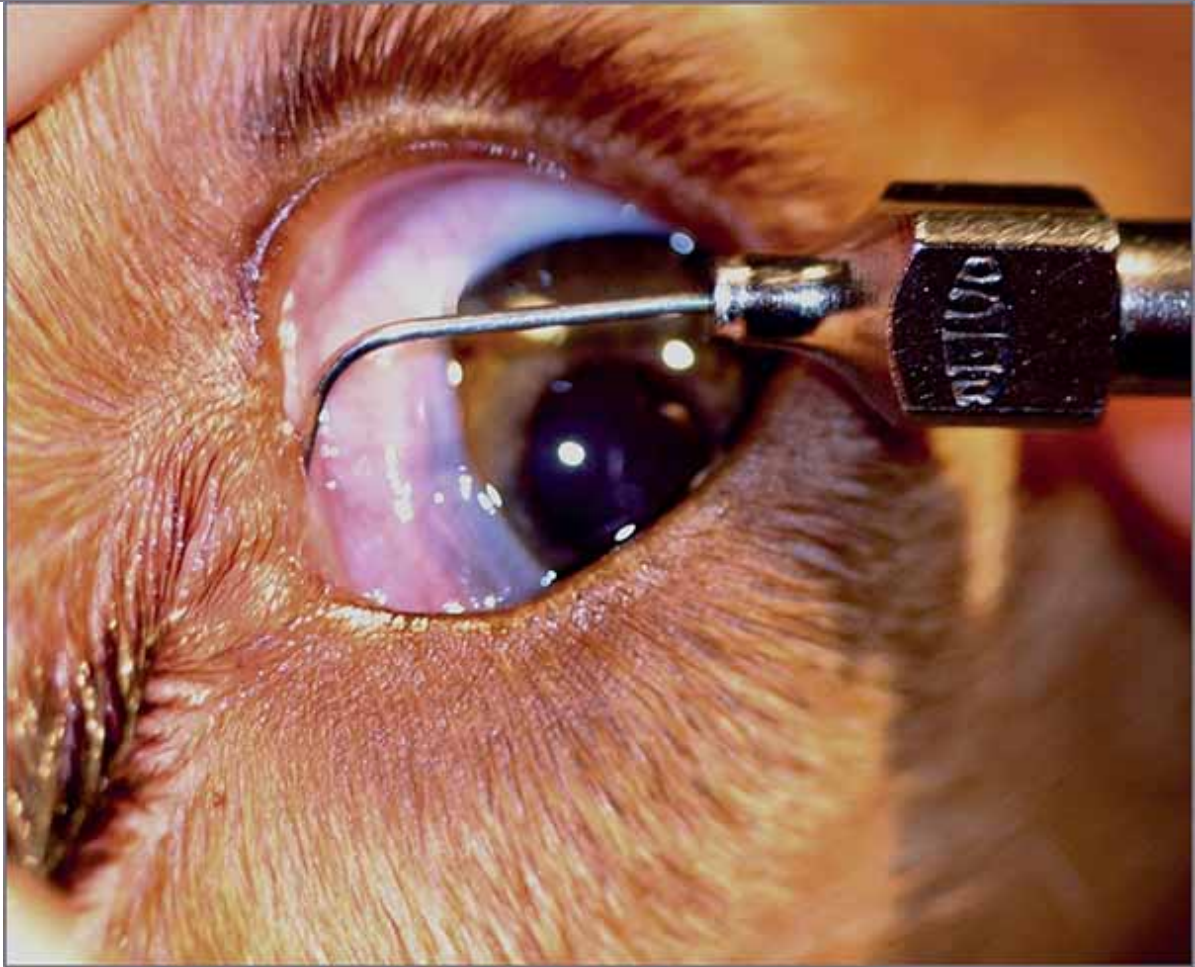


Figure 1. Catheterisation of the superior lacrimal punctum.

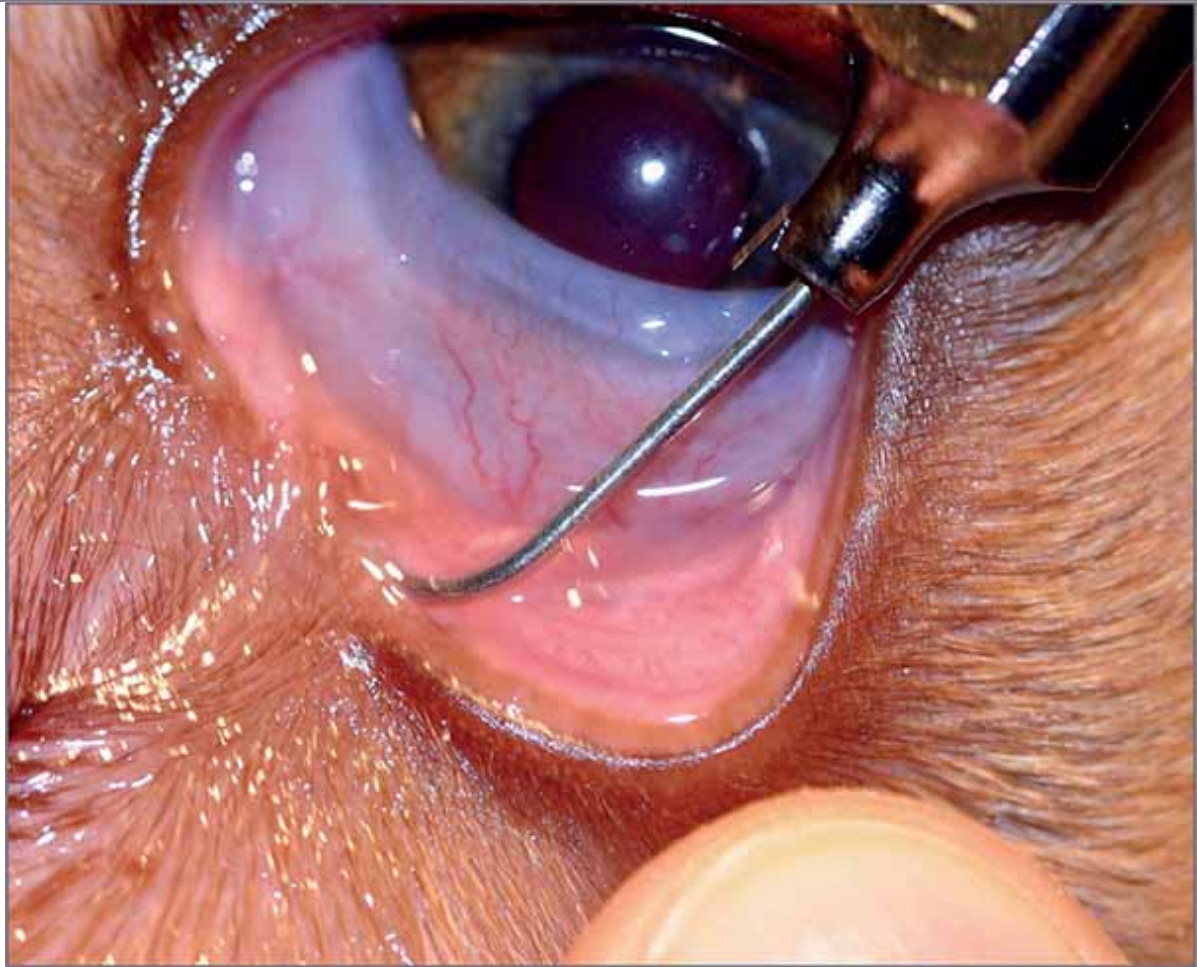


Figure 2. Catheterisation of the inferior lacrimal punctum.



Figure 3. Catheterisation of the nasolacrimal duct from the superior lacrimal punctum.



Tear production and drainage process

Imperforate lacrimal puncta and lacrimal duct stenosis

It is not uncommon to come across patients with chronic epiphora due to imperforate or very small lacrimal puncta, and it may be accompanied by

blepharitis and dermatitis of the medial canthus depending on the duration of the process.

When presented with such a case, it is very important to determine whether the defect affects one or both puncta. If only one lacrimal punctum is imperforated, it should be opened using a “pigtail” lacrimal probe (Fig. 4). If both puncta are compromised, identify where they should be located and open them with the aid of some Westcott or tenotomy scissors.

A narrow lacrimal punctum can also be broadened by inserting the blade of some scissors into the duct or a dilator (a metal probe with a gradually increasing circumference) serves to widen its entrance. Dilators can also be used for the surgical treatment of lacrimal duct stenosis.

After this intervention, patients must be given a topical antibiotic that includes a corticosteroid for at least 10 days postoperatively to make sure the new lacrimal punctum does not heal closed. The use of a Silastic tube reduces the likelihood of stenosis.



Figure 4. Pigtail nasolacrimal probe.

Dacryocystitis and nasolacrimal duct obstruction

Dacryocystitis is the term given to inflammation of the lacrimal sac. The process may be due to purely infectious causes or sometimes because of a foreign body that reaches the sac via one of the lacrimal puncta; the foreign body is usually plant matter, such as awns, and will need to be removed surgically.

The main clinical signs of dacryocystitis are epiphora, discharge (which may even be mucopurulent), and swelling and bulging in the projection area of the lacrimal sac. Applying gentle, yet constant pressure to the sac tends to produce a purulent discharge from one or both lacrimal puncta (Fig. 5).

Treatment often requires a surgical intervention given the difficulty in performing effective lavage from either the lacrimal puncta located in the eyelids or from the end of the ducts' pathway in the nose.

The technique of choice was first described by Allgoewer and uses two metal probes (Fig. 6) to locate the exact point from where the sac should be accessed, with the entire intervention carried out through the conjunctiva close to the medial canthus. This procedure also provides access to the canalicular portions running from the lacrimal puncta to the sac.

Obstructions situated in more distal regions of the nasolacrimal duct are typically resolved by means of forced irrigation performed from the puncta and with the aid of a catheter introduced in the duct. A dacryocystography is often indicated in order to diagnose these problems, locate the affected area, and rule out extraluminal causes; it can be done with the same iodine derivatives used as contrast agents in other radiological techniques.



Figure 5. Dacryocystitis with discharge from the medial canthus and protrusion in the projection area of the lacrimal sac.



Figure 6. Metal probes used to dilate lacrimal puncta.

Quantitative keratoconjunctivitis sicca

Quantitative dry eye is one of the most frequent causes of eye disease in veterinary patients. It is caused by deficient tear production, more specifically the aqueous portion of the film, so it is actually the result of a problem with the main lacrimal glands, the accessory glands, or both.

The main clinical signs observed upon examination are strands or accumulations of mucus (Fig. 7), as well as signs of conjunctival inflammation, such as hyperaemia, and a mild loss of corneal lustre and even transparency (Figs. 8 and 9). As the disease advances, the signs progressively worsen with abundant secretions across the ocular surface, in the conjunctival sacs, and even dry secretions in the eyelid margins. Corneal inflammation, which courses with oedema, neovascularisation, and even ulcers, becomes apparent as the clinical picture deteriorates (Fig. 10).

In parallel to the above, the use of tests such as rose bengal staining, Schirmer's test (Fig. 11), and observing for changes in the normal corneal

epithelium to something more reminiscent of the conjunctival epithelium are all indicative of the extent of the disease.

Neurogenic keratoconjunctivitis sicca is a type of quantitative tear deficiency caused by a lack of innervation in the glands. It may be associated with other processes, such as Horner's syndrome or facial paralysis. It is usually unilateral and may be accompanied by ipsilateral nasal dryness (xeromycteria). In most cases, the cause of the deficient innervation is idiopathic. Neurogenic keratoconjunctivitis sicca should be suspected in any patient with unilateral dry eye that is unresponsive to conventional treatment.

The treatment of quantitative keratoconjunctivitis sicca is based on the correct application of ocular moisturisers, an appropriate choice of carbomers and artificial tears, the use of serum and plasma blood products, and the administration of immunomodulators to recover gland activity – some of which are available as subconjunctival inserts. For neurogenic keratoconjunctivitis sicca, the recommended treatment is topical or oral pilocarpine.

Surgical interventions are generally reserved for pharmacologically refractory cases and the options include reducing the palpebral fissure in animals with euryblepharon, an oral mucosa graft, or a parotid duct (Stensen's duct) transposition (Fig. 12).

Aetiologies of keratoconjunctivitis sicca

- Immune mediated
- Neurogenic
- Infectious causes:
 - Herpesvirus
 - Distemper virus
 - *Leishmania*
- Iatrogenic:
 - Atropine
 - Barbiturates
 - Sulphamides
 - Orbital/ocular surgery
- Congenital

- Endocrine disorders:
 - Diabetes mellitus
 - Hypothyroidism
 - Cushing's syndrome

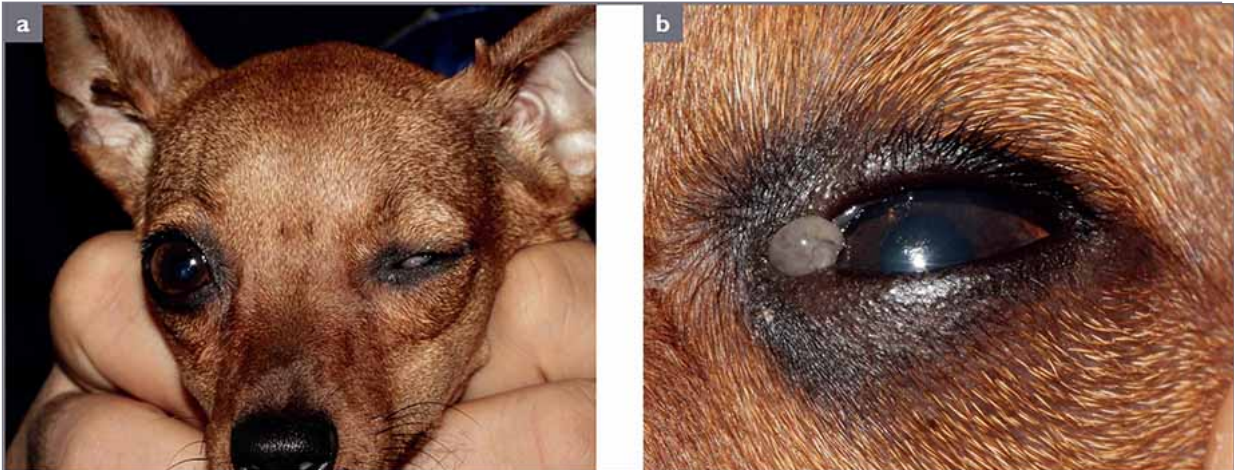


Figure 7. Signs of discomfort in the left eye of a dog with dry eye (a). Close up of the left eye showing an accumulation of mucus in the medial canthus (b).



Figure 8. Loss of lustre, particularly notable when taking a photograph with flash, in a dog with incipient dry eye.



Figure 9. Acute manifestation of dry eye. The cornea is completely without moisture and ulcers have already formed on the surface.



Figure 10. Severe dry eye. Dry secretions at the eyelid margins, strands of mucus, and chronic lesions of the cornea.

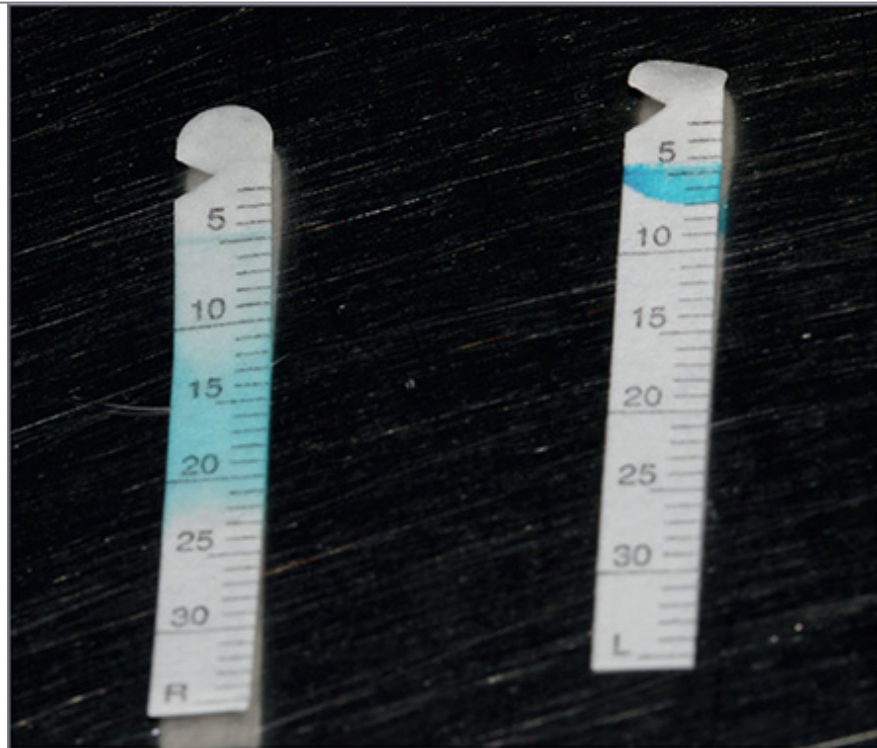


Figure 11. Results of Schirmer's test.

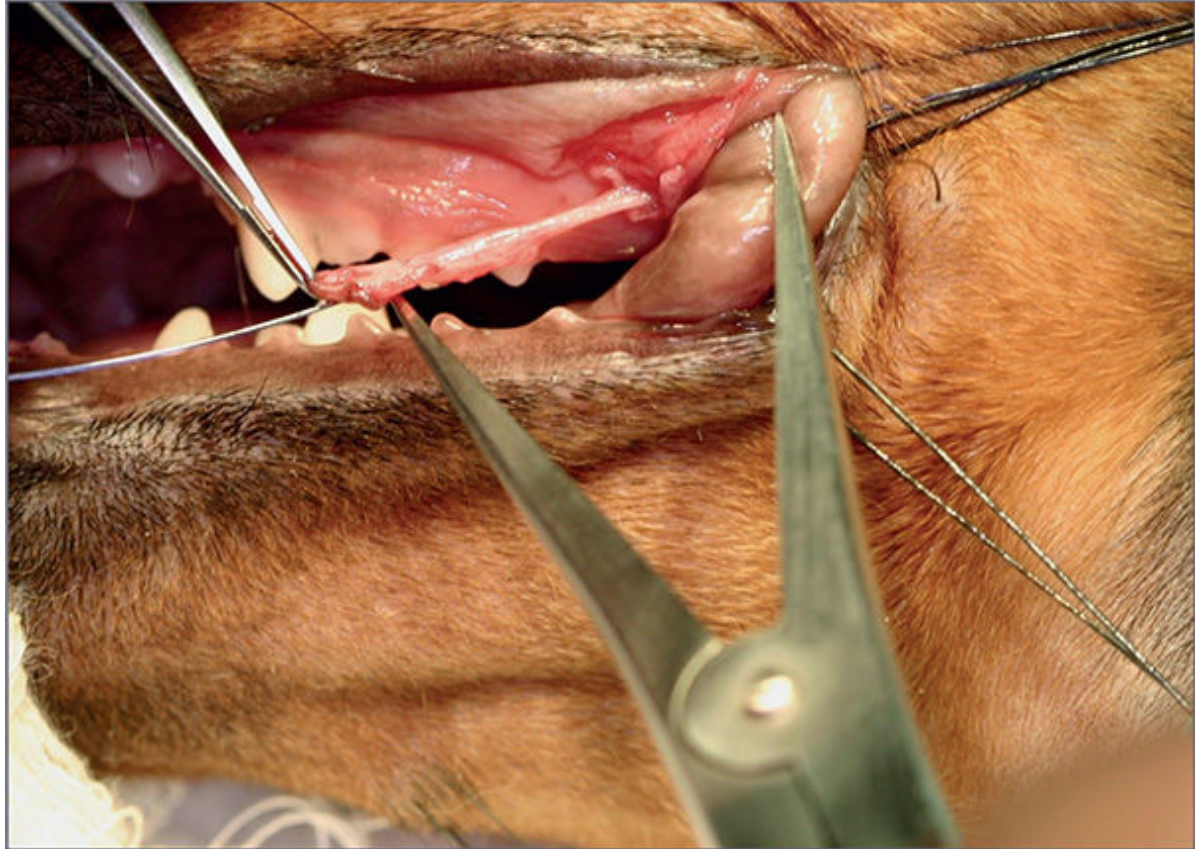


Figure 12. Dissection and measurement of the parotid duct before its transposition.

Qualitative keratoconjunctivitis sicca

Also known as **qualitative dry eye**, this condition is a deficit in tear production caused by a loss of lipid or mucin components in the tear film.

It is usually secondary to other underlying diseases (e.g. blepharitis), particularly those which affect the eyelid margin and therefore eventually alter the tarsal glands. In such cases, the lipid component will be altered.

The clinical presentation may be different in animals that have had chronic conjunctivitis. In this situation the goblet cells eventually suffer a modification and therefore produce less of the mucopolysaccharides normally found in the tear film. One of the main causes of a goblet cell deficit in cats is feline herpesvirus.

Irrespective of whether the eye examination in animals with qualitative dry eye syndrome reveals indications of conjunctival and corneal inflammation, it is notable that most cases feature marked epiphora and normal or even high Schirmer's test scores (Fig. 13).

The aetiological diagnosis is completed with findings from tests such as the precorneal tear film break-up time (BUT), which can be used to determine how the deficiency in tear film components leads to tears with a very low surface tension.

The treatment of qualitative keratoconjunctivitis sicca must consider the primary, causal disease, if any, and compensate for the components missing from the tear film by selecting a corresponding artificial tear product and even contemplate supplementing the patient's diet with an appropriate nutraceutical.

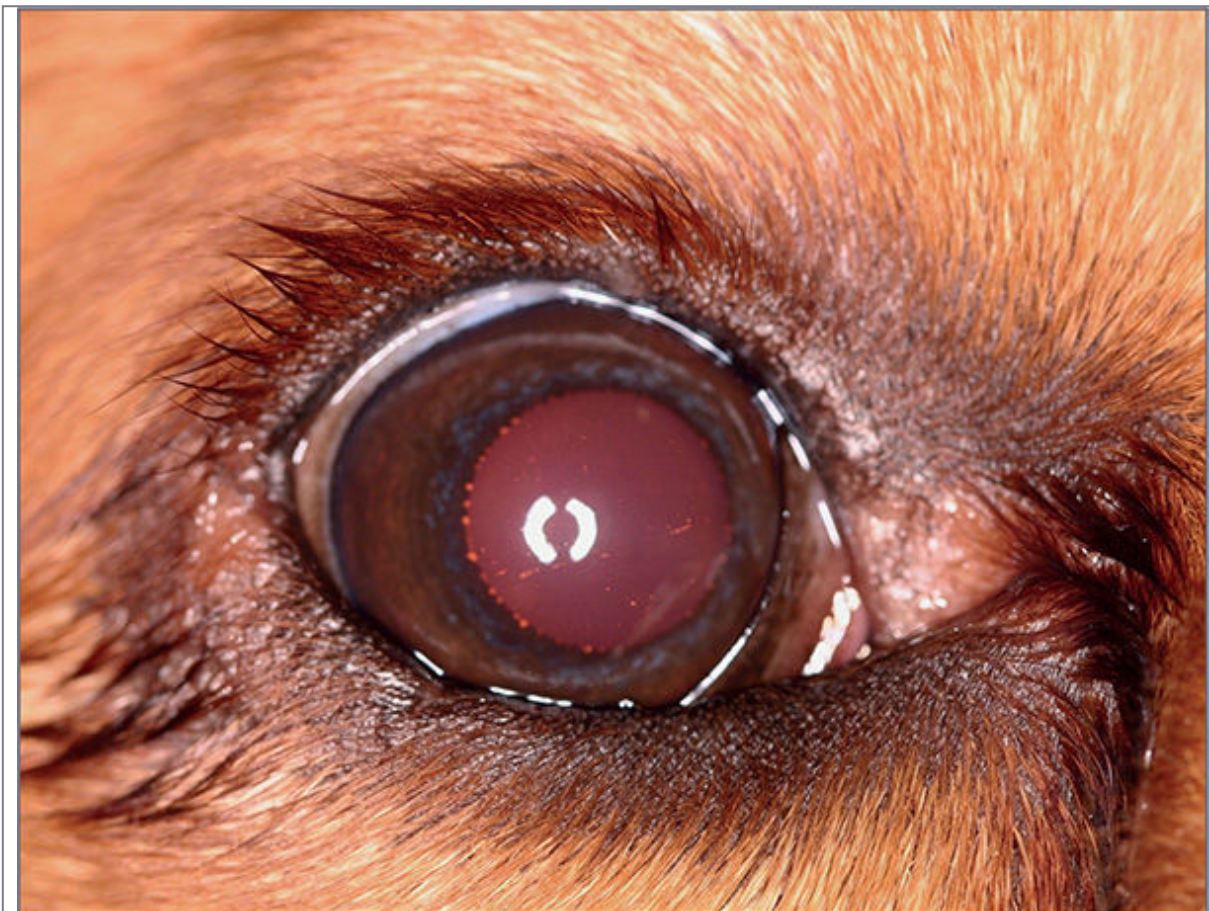


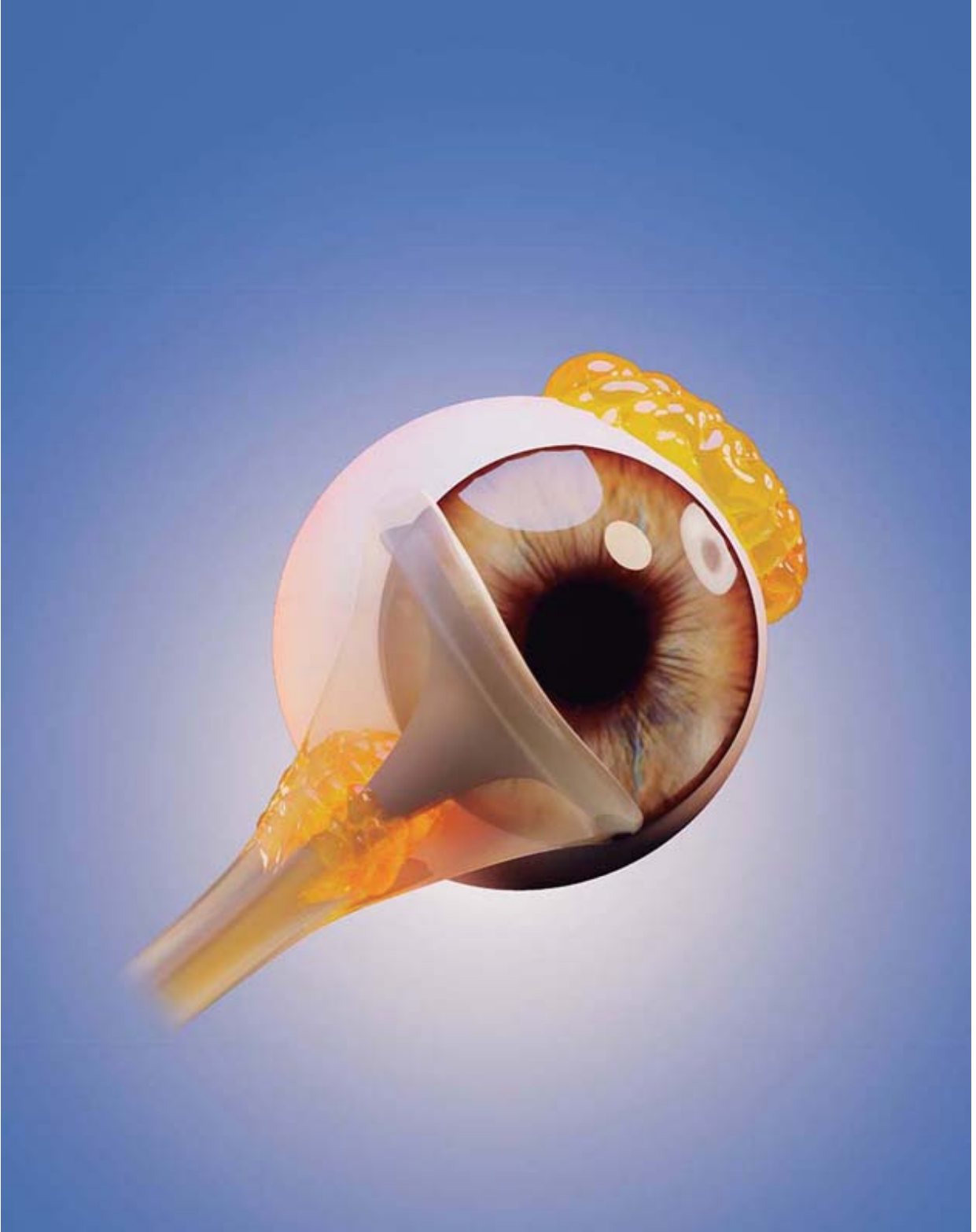
Figure 13. Marked epiphora in an eye affected by qualitative keratoconjunctivitis sicca.



Aetiopathogenesis of keratoconjunctivitis sicca

04

The conjunctivae and nictitating membrane



Anatomy and physiology of the conjunctiva

The conjunctiva is a mucous membrane covering the inner surface of the eyelids, the bulbar and palpebral surfaces of the nictitating membrane, and the anterior portion of the sclera (Fig. 1). It is a vital tissue for eye functionality, it facilitates ocular movements, and it contains goblet cells, which produce the mucin component of the tear film.

The conjunctiva also has important immunological properties, and this is significant if we consider that it is one of the most exposed mucous membranes in the body; but despite this, primary infectious conjunctivitis is rare in dogs, aside from cases of distemper and leishmaniasis. Thanks to these properties, most minor wounds to the conjunctiva regenerate and heal by themselves within just 24 hours.

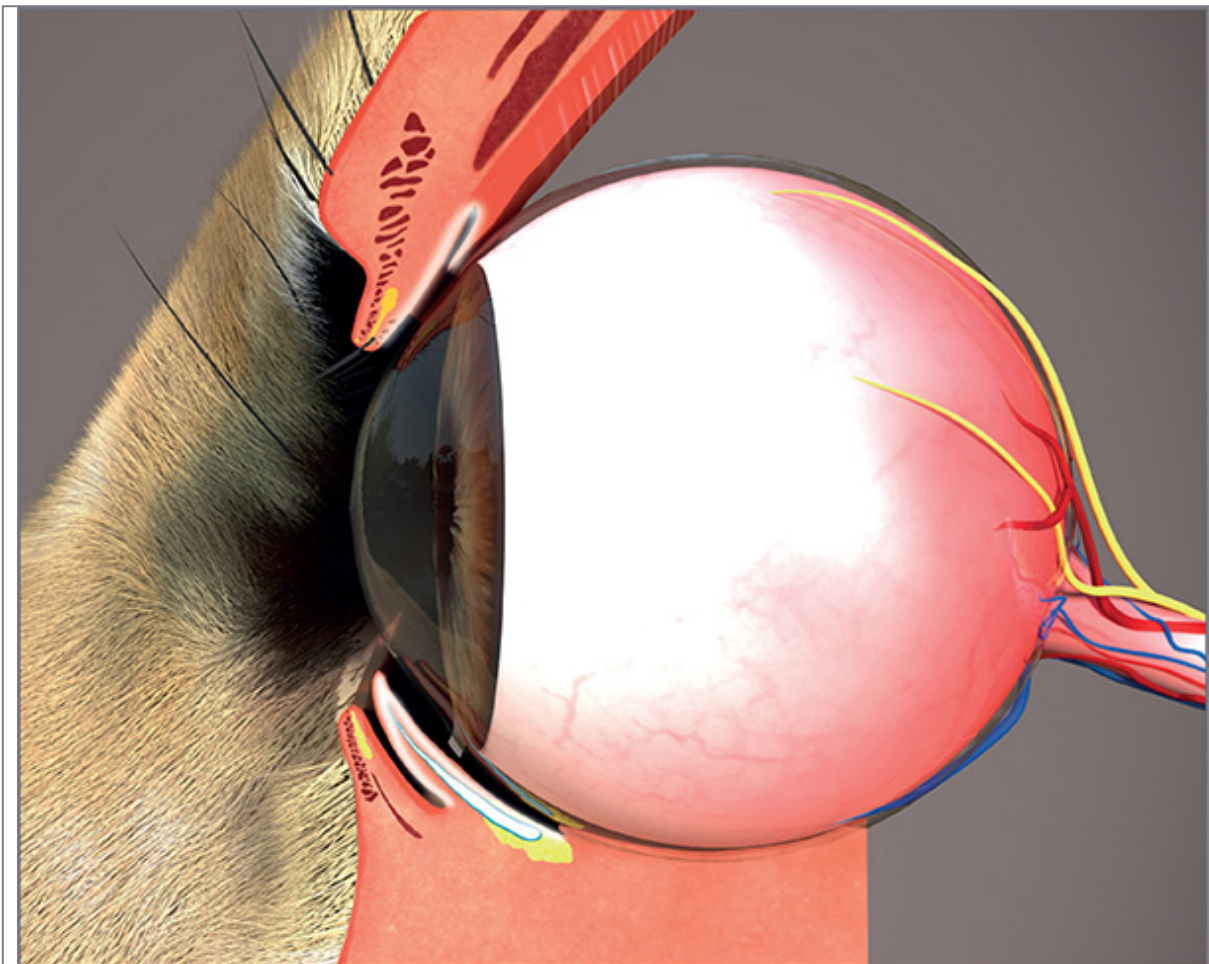


Figure 1. Ocular conjunctiva.

Diseases of the conjunctiva

Follicular conjunctivitis

Follicular conjunctivitis occurs when lymphoid follicles grow in abnormal locations such as the palpebral conjunctiva, the eyeball, or the anterior face of the nictitating membrane. It is usually associated with hyperaemia and an aqueous or seromucoid discharge because of the irritation it causes (Fig. 2).

Follicular conjunctivitis is a very common condition in dogs under 18 months and is triggered by antigenic stimulation.

The clinical signs, including discomfort, conjunctival hyperaemia, and seromucoid discharge in the medial canthus of the eyelids, are always observed in young animals and are highly suggestive of the disease, although lymphocytes must be observed during conjunctival cytology before a definitive diagnosis can be made.

This disease usually responds very well to topical corticosteroid therapy, but bear in mind that the dosage frequency should be reduced gradually in order to prevent recurrences.

Follicle debridement using a topical anaesthetic and a cotton swab is recommended for patients who do not respond to this treatment.

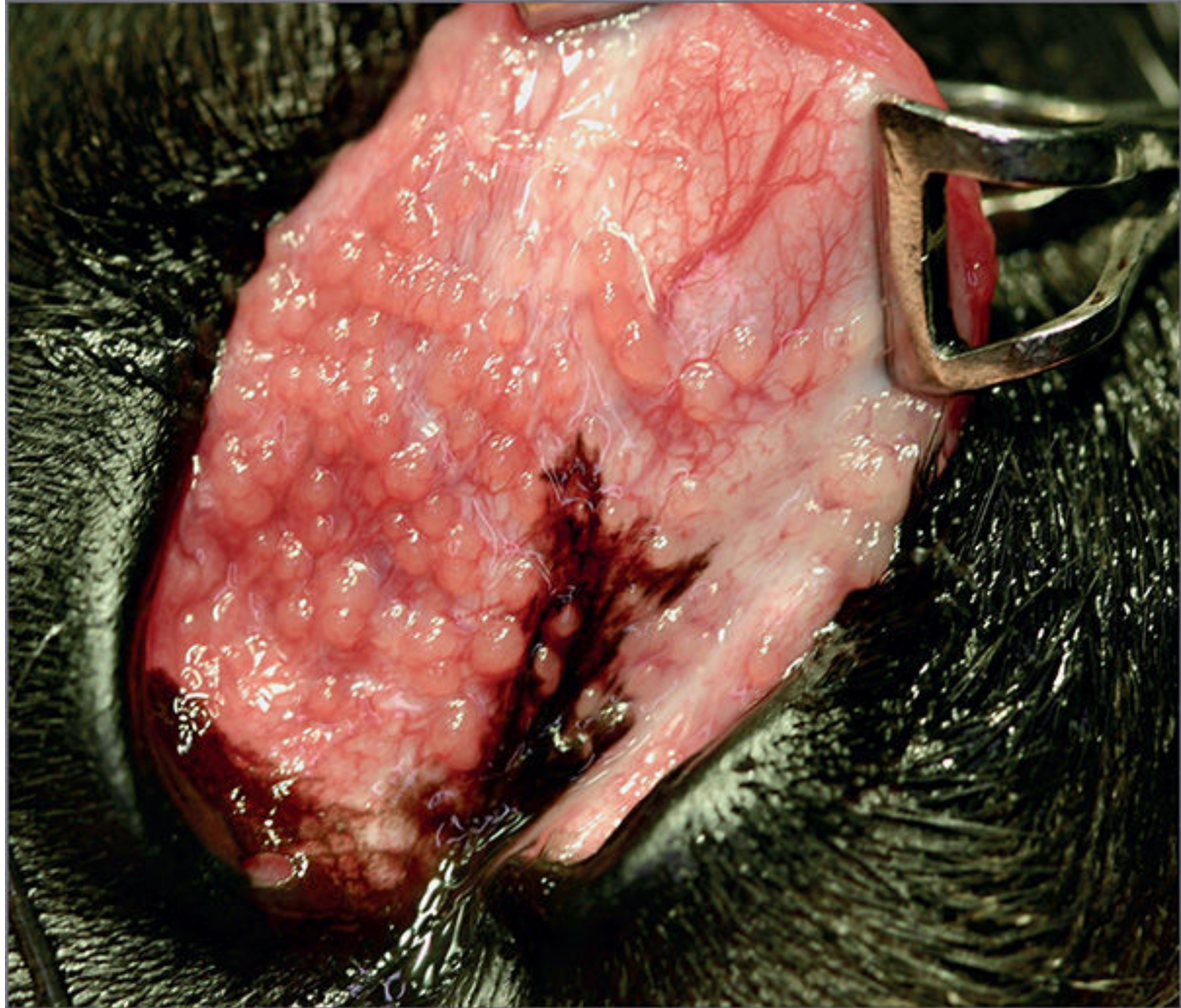


Figure 2. Follicular conjunctivitis in a Great Dane puppy.

Conjunctival folliculitis

Patients develop multiple small, reddish nodular formations, namely hyperplastic lymphoid follicles, which are found particularly concentrated in one area – the bulbar surface of the nictitating membrane (Fig. 3).

Antigenic stimulation and the subsequent increase in the size of the follicles may be down to various causes, but they are often associated with hypersensitivity reactions and infectious processes, such as parasitisation by worms of the genus *Thelazia* .

Elimination of the triggering factor and, if necessary, the administration of eye drops with corticosteroids are normally enough to resolve the problem.



Figure 3. Conjunctival folliculitis.

Chronic allergic blepharoconjunctivitis

Although this condition is well known in human medicine, allergic conjunctivitis is a rare diagnosis in veterinary medicine. It often requires diagnosis by exclusion, a process that involves ruling out all other possible causes.

Allergic blepharitis and conjunctivitis fall within the spectrum of atopic dermatitis and may be triggered by environmental allergens or a food hypersensitivity, and so the diagnosis relies on intradermal skin testing and elimination diets with novel or hydrolysed proteins (Fig. 4).

Environmental allergens are generally responsible for blepharitis, whereas food allergies tend to produce more gastrointestinal clinical signs. What is more, food allergies do not vary with the seasons and respond poorly to systemic treatment with glucocorticoids.

The key step in the treatment of allergic blepharoconjunctivitis is the identification and desensitisation of the allergen involved. Unfortunately, in some cases the causal allergen cannot be identified, so treatment should focus on controlling any inflammation, pruritus, or secondary infections.

The use of topical corticosteroids and nonsteroidal anti-inflammatories is not very effective and very few studies have examined the efficacy of antihistamines.

Vasoconstrictors, such as phenylephrine or tetrahydrozoline, can be used as symptomatic treatment to significantly reduce any redness, but they will not eliminate the cause.

It is important to emphasise that some patients are very intolerant of practically any type of topical treatment. Given the conjunctival reaction to medications instilled in the eyes, we recommend flushing the eyes with a very mild, preservative- and detergent-free product and the instillation of artificial tears.



Figure 4. Atopic dog with allergic blepharoconjunctivitis.

Clinical signs of conjunctival disease

- **Chemosis:** oedema of the conjunctival tissue, with the formation of pockets of fluid (Fig. 5).
- **Conjunctival hyperaemia:** congestion of the conjunctival blood vessels causing redness (Fig. 6).
- **Conjunctival follicles:** in the form of small nodules due to lymphoid hyperplasia.
- **Subconjunctival haemorrhage:** as the tiny blood vessels in the conjunctiva are quite fragile (Fig. 7).
- **Conjunctival ulcers:** often caused by the animal rubbing its eye.

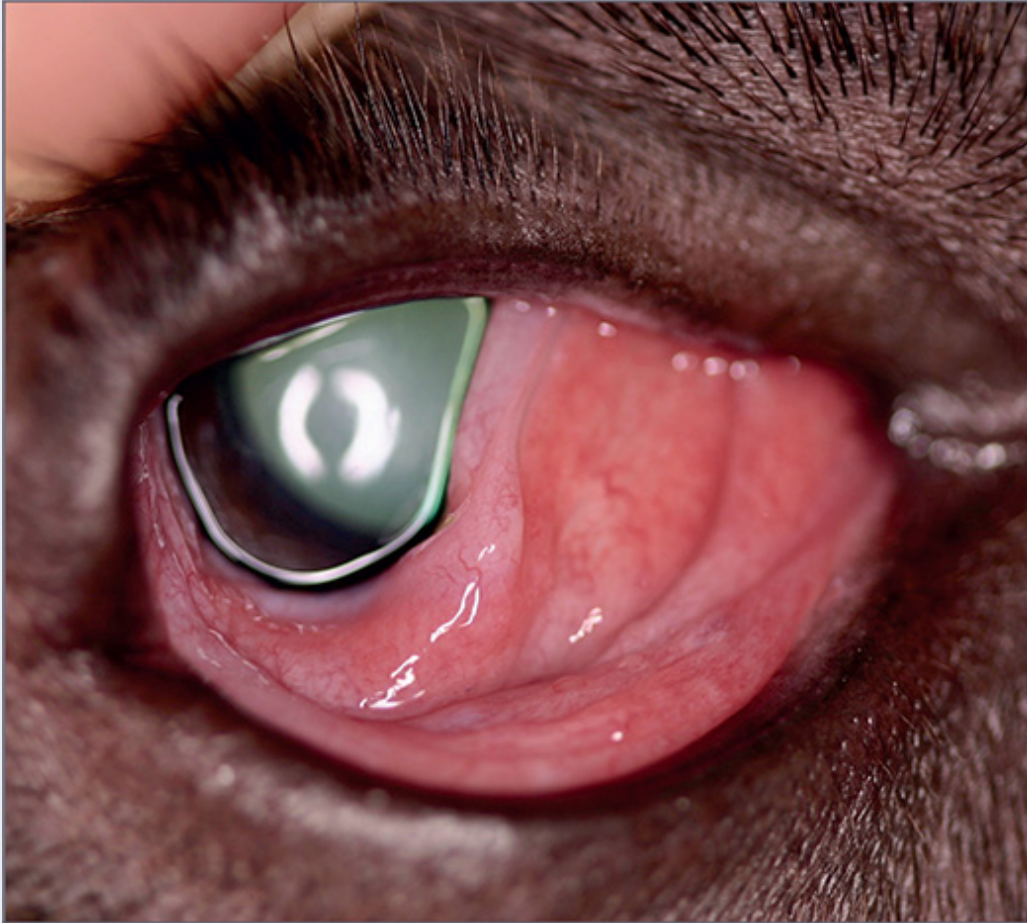


Figure 5. Conjunctiva with severe hyperaemia and chemosis.



Figure 6. Very intense conjunctival hyperaemia.

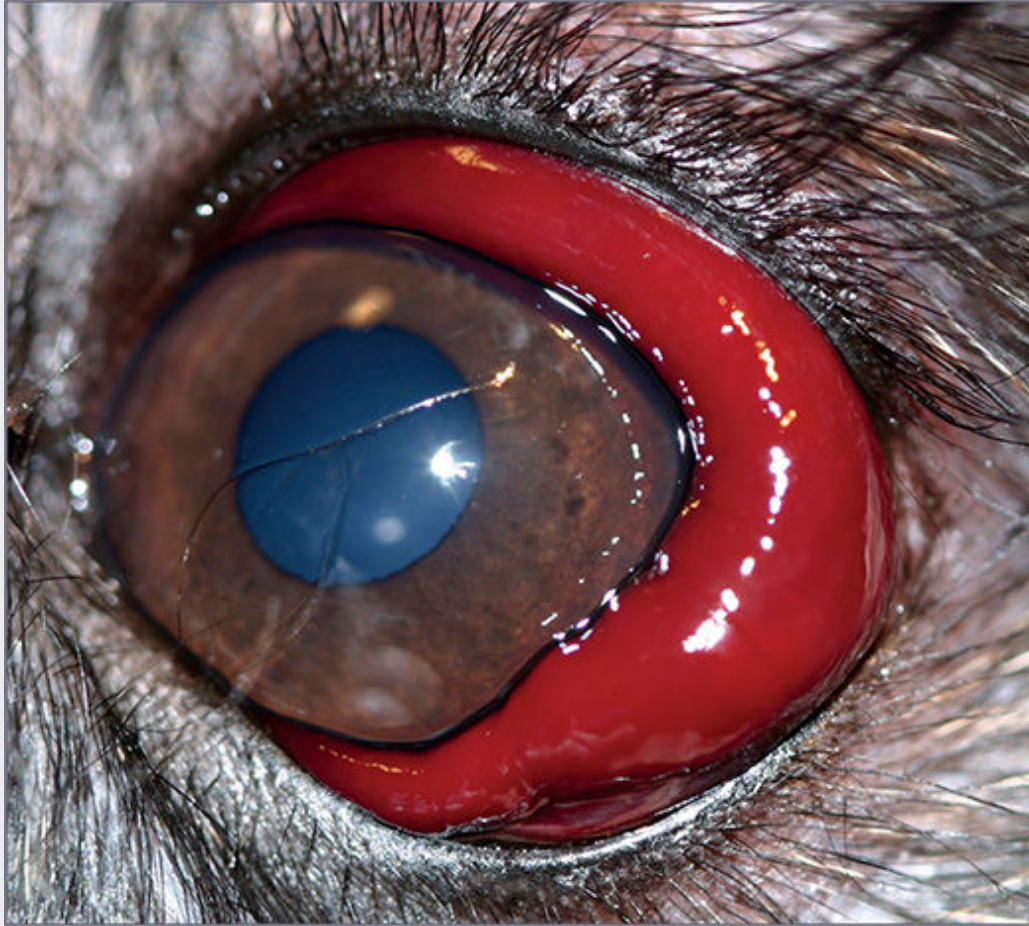


Figure 7. Subconjunctival haemorrhage secondary to blunt trauma.

Parasitic conjunctivitis

While it is true that conjunctivitis of a parasitic aetiology is fairly rare, the increase in emergent diseases, such as thelaziasis, means that vets must include parasites as part of their differential diagnosis.

Thelazia callipaeda is a nematode that can be found in the conjunctival sacs of dogs. It has a serrated cuticle that causes intense irritation when the nematode moves across the ocular surface. Some animals remain asymptomatic, while others show signs of conjunctival hyperaemia, with or without follicles, and a mucopurulent discharge. These worms can even provoke epiphora due to a blocked nasolacrimal drainage system (Fig. 8).

Besides the signs of inflammation, the examiner may observe numerous worms in the conjunctival sacs.

Treatment is based on removing the nematodes after instilling anaesthetic eye drops to facilitate an in-depth examination of the conjunctival sacs, and it is always a good idea to finish with forced irrigation using physiological saline. Complete the treatment by applying a nematocide with milbemycin oxime, moxidectin, or ivermectin.

Another parasite that can produce conjunctivitis is *Onchocerca lupi* , which mainly affects dogs and to a lesser degree cats. It is considered an emergent disease in southern Spain.

It triggers variable ocular signs, but the most common observation is subconjunctival nodules surrounding the parasite (Fig. 9), although in severe cases patients can develop corneal oedema, chemosis, glaucoma, and even retinal detachment.

Surgical excision of the nodules is curative, but if surgery is contraindicated, medical treatment may also prove effective. Treatment involves a combination of melarsomine and ivermectin coadministered with systemic doxycycline.

Lastly, we must not overlook *Leishmania* as an infectious agent in dogs that can cause conjunctivitis, which is frequently accompanied by blepharitis and keratitis. *Leishmania* should be included in the differential diagnosis whenever there are general signs of conjunctival inflammation, coursing with eyelid and corneal inflammation, or even dry eye, in patients who live or who have spent time in endemic areas (Fig. 10).

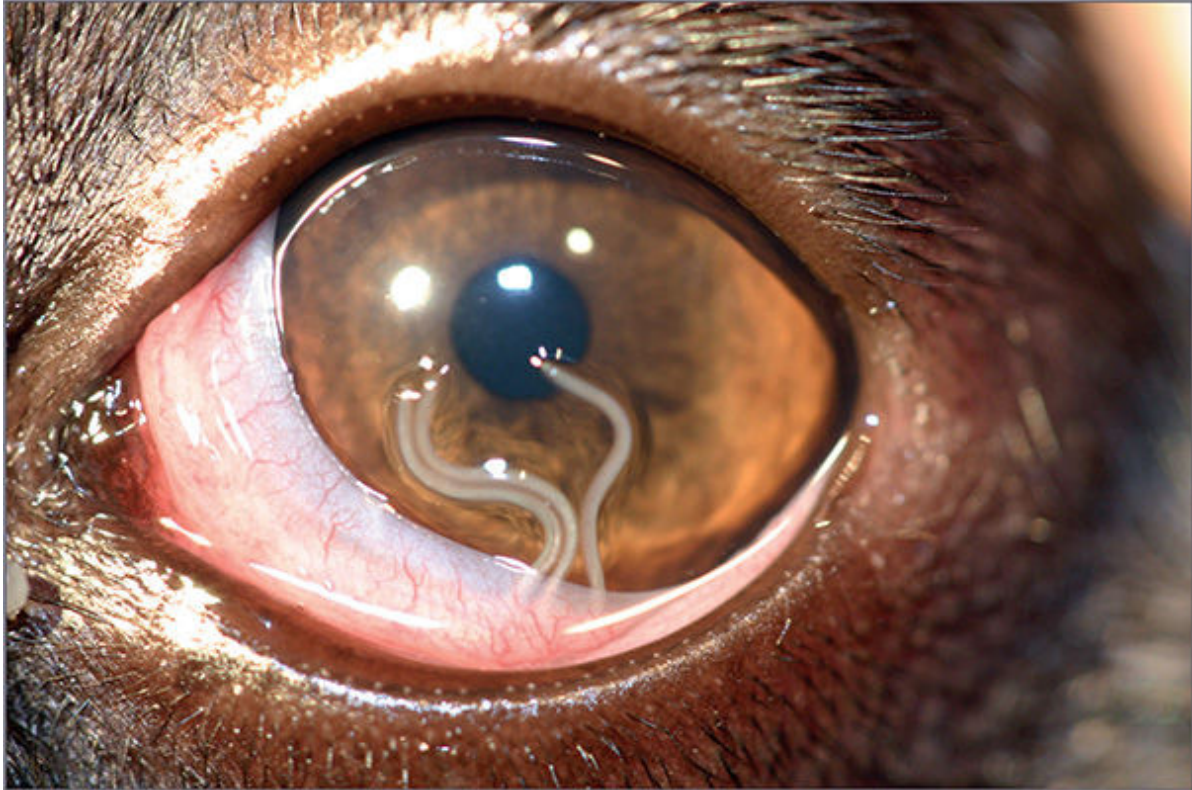


Figure 8. Conjunctivitis due to *Thelazia callipaeda* infection.

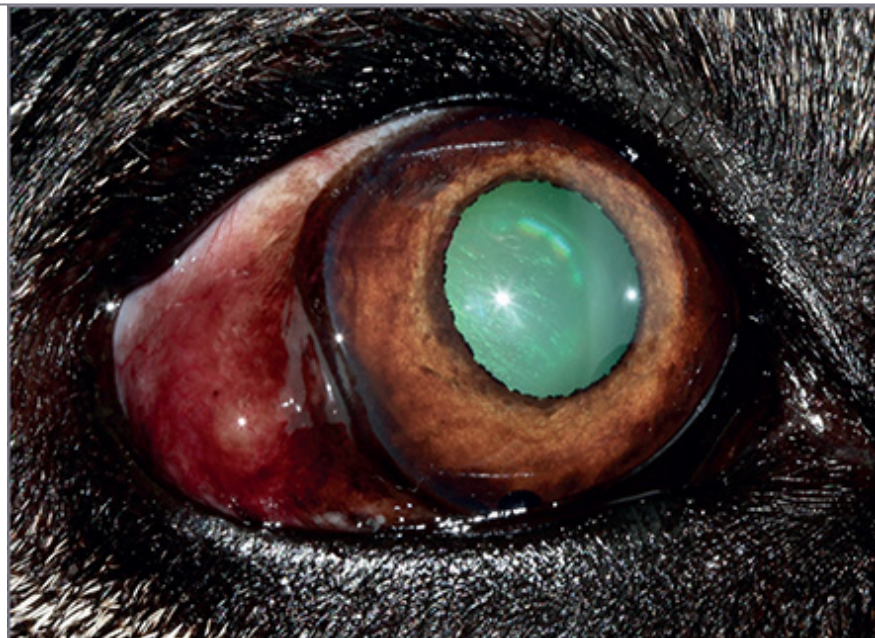


Figure 9. Conjunctival nodule caused by a worm in the genus *Onchocerca* .



Figure 10. Conjunctivitis associated with moist blepharitis and a corneal ulcer in a Boxer with leishmaniasis.

Conjunctival tumours

Conjunctival tumours are not very common and tend to be primary. The most frequent types are papillomas, haemangiomas and haemangiosarcomas, melanomas, and mast cell tumours. Cytology and biopsy are essential tests in their diagnosis, as the tumours may manifest as nonneoplastic diseases (Figs. 11 and 12).



Figure 11. Nodular tumour located in the lateral bulbar conjunctiva.



Figure 12. Pedunculated, friable tumour located in the conjunctiva.

Conjunctival dermoids

Dermoids or choristomas are remains of ectodermal tissue found in an abnormal location, in this case, in the conjunctiva. They can affect the conjunctiva in isolation, or the eyelids and cornea may also be compromised.

These defects, which are clearly congenital and of proven heritability in some breeds of dog, will cause complications depending on their extension and exact location. Of the associated complications, the main concern is the formation of ulcers due to abrasive hairs which, after the first few weeks of life, will start to grow across the abnormal dermoid tissue and often towards the cornea (Figs. 13 and 14).

They always require surgical resolution, and the complexity of the intervention depends on their size and location.



Figure 13. Conjunctival dermoid, with the hairs causing severe keratitis in the nearby cornea.



Figure 14. Conjunctival dermoid with slight compromise of the cornea and lateral canthus of the eyelids.

Conjunctival burns

It is a relatively common reason for consultation and is undoubtedly a genuine medical emergency.

Contact between the ocular conjunctivae and any acidic or alkaline substances triggers an inflammatory process resulting in significant cicatricial retraction of the conjunctivae. In turn, this situation leads to phimosis which, if left uncorrected, will pull on the eyelid margins and disfigure the eyelid fissure. Only the centre of the cornea is visible and the conjunctivae are exposed, further contributing to the clinical picture of dry eye, which in itself may already be very severe if the burn affects the outlet of the lacrimal ducts (Figs. 15 and 16).

Besides treatment to correct the tear deficiency, conjunctival burns may also require debridement of the retracted tissues and eyelid release.



Figure 15. Chemical burn caused by cement dust.

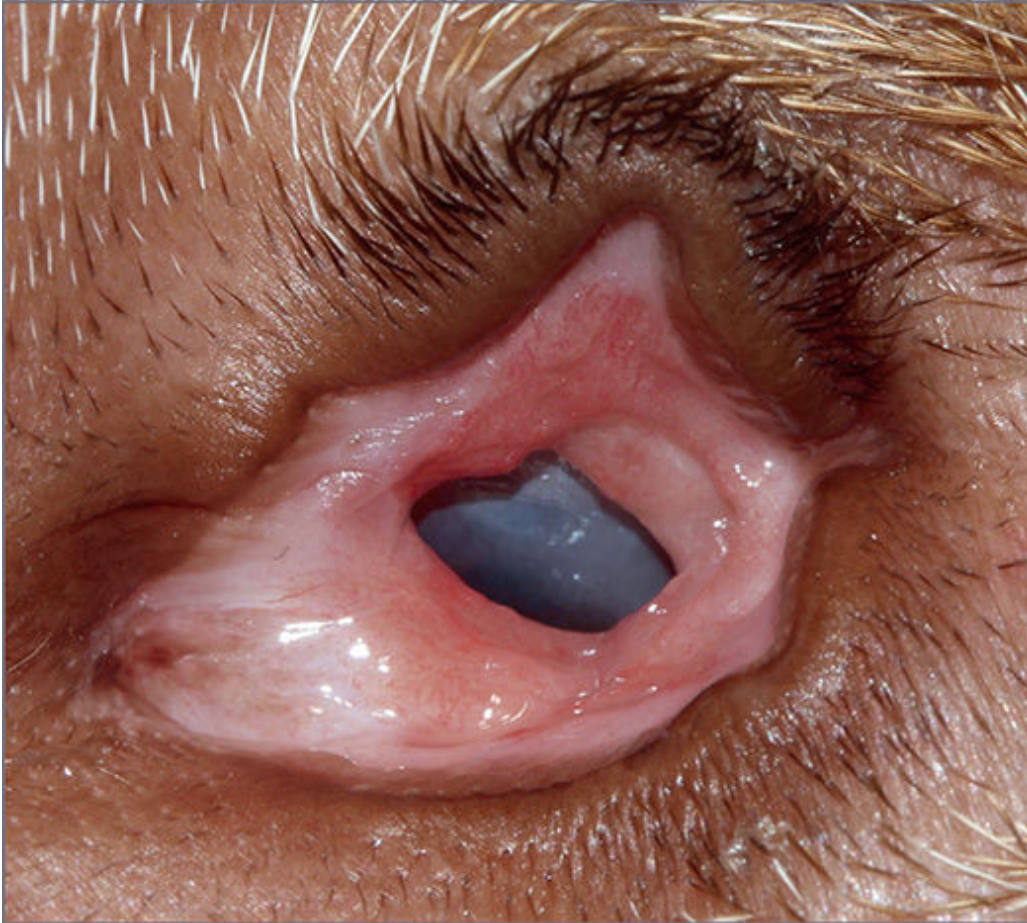


Figure 16. Rapid deterioration of the previous case towards conjunctival phimosis.

Anatomy and physiology of the nictitating membrane

The nictitating membrane or third eyelid is an anatomical feature found in nonprimate mammals, some reptiles, and birds. It consists of a T-shaped cartilage with vertical and horizontal branches (the latter running parallel and adjacent to the margin); a gland at its base; conjunctivae on the bulbar and palpebral surfaces; lymphoid tissue on the bulbar surface; and connective tissue (Fig. 17). In dogs, the nictitating membrane moves passively across the globe when this is retracted by the retractor bulbi muscle.

Its functions are:

- To distribute tears across the ocular surface.
- To protect the cornea.

- To produce part of the tear film's aqueous component.

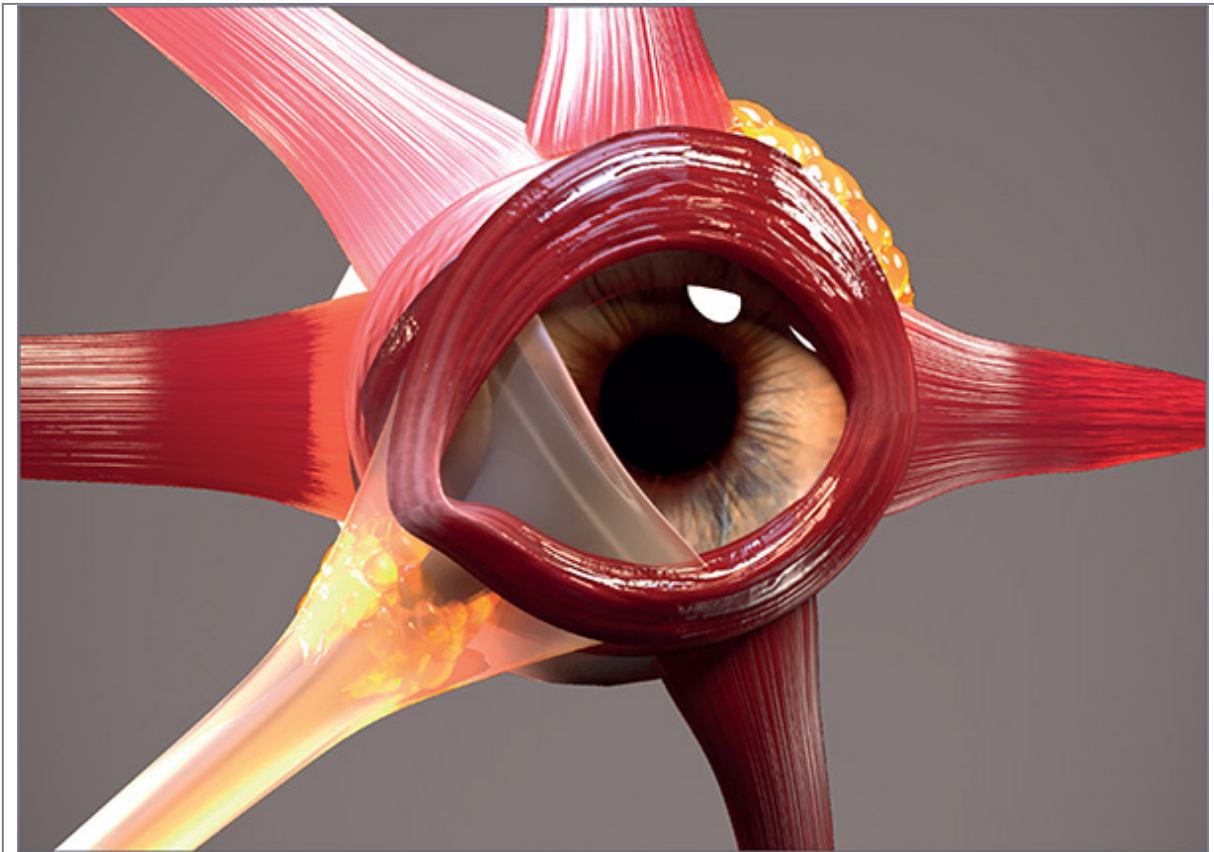


Figure 17. Nictitating membrane and its associated cartilage.

Diseases of the nictitating membrane

The nictitating membrane is covered by the conjunctiva, so it develops similar clinical signs as those described above, namely hyperaemia, chemosis, and mucous secretion.

A prolapsed, or protruding, nictitating membrane is the product of a diseased membrane, but it may also be a clinical sign of other eye diseases. The nictitating membrane can also suffer structural alterations, such as cartilage eversion, prolapse of the membrane's lacrimal gland, adenitis, or lymphoid hyperplasia.

Prolapsed gland of the nictitating membrane

This is the most common affliction of the nictitating membrane and occurs when the gland moves out of its normal position due to hyperplasia and

laxity of the ligament attaching it to the periorbita. As a result, the gland bulges behind the nictitating membrane and gives the eye a very distinctive appearance.

It occurs in young animals and particularly in brachycephalic and molosser breeds. It can develop unilaterally or bilaterally, although in the latter scenario it does not necessarily affect both eyes at the same time, with the second eye often prolapsing 3 months after the first in most patients (Fig. 18).

Treatment is almost always surgical, as returning the gland to its normal position usually requires the use of forceps.

There are several techniques, and they fall into two main types – pocket and anchoring techniques. In general terms, pocket techniques are considered more physiological because they permit normal membrane movement; however, and based on the authors' experience, the rate of recurrence and number of complications, such as lacrimal cysts, are slightly higher.

The emergence of new techniques, for example attaching the gland to the ventral rectus muscle, means surgeons can choose from even more options with good outcomes.

In our opinion, techniques that involve attaching the gland to the ventral rectus muscle are particularly recommendable when dealing with large glands or patients from a molosser breed.

Broadly speaking, and regardless of which technique is chosen, it is very important to bury the suture threads and stitches so they do not brush against the corneal surface and produce an ulcer.

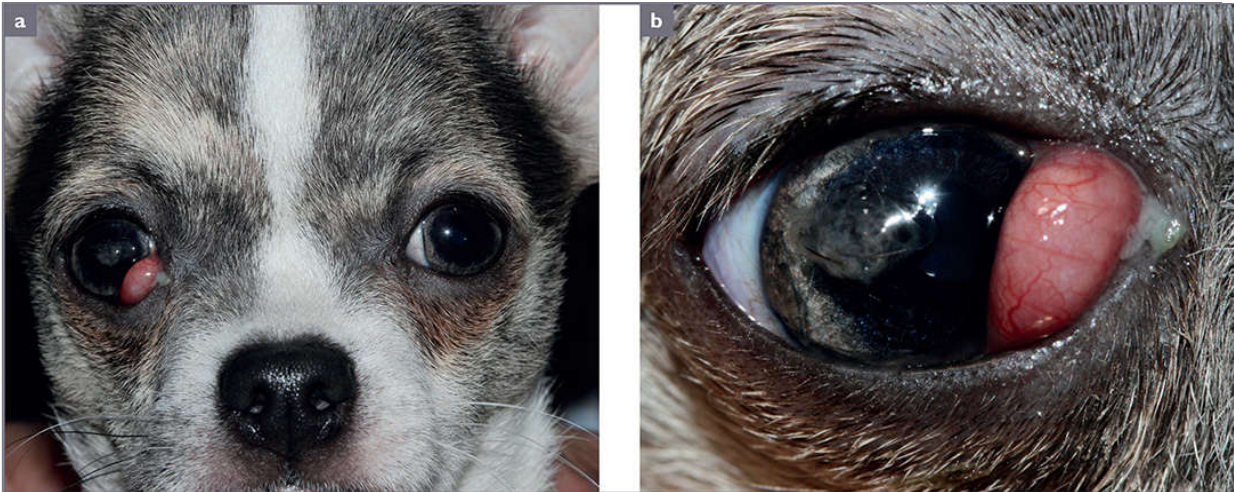


Figure 18. Prolapsed gland of the nictitating membrane (a). Closer view of the right eye (b).



Prolapsed gland of the nictitating membrane

Everted cartilage of the nictitating membrane

This anomaly occurs when the vertical branch of the membrane cartilage bends outwards or inwards. It affects young and middle-aged dogs, with a greater incidence among large breeds including San Bernardo, Weimaraner, or Great Dane (Fig. 19).

An everted cartilage may also be secondary to a prolapsed gland or surgery on the nictitating membrane.

As eversion of the nictitating membrane cartilage can cause secondary keratitis, it should be corrected promptly through surgery. The intervention consists of resecting the section of “defective” cartilage, which is usually positioned close to the junction of the vertical and horizontal branches.

It has recently been reported that thermocauterisation generates cicatricial retraction and “straightens” the cartilage, but the results obtained with the technique are inconsistent.

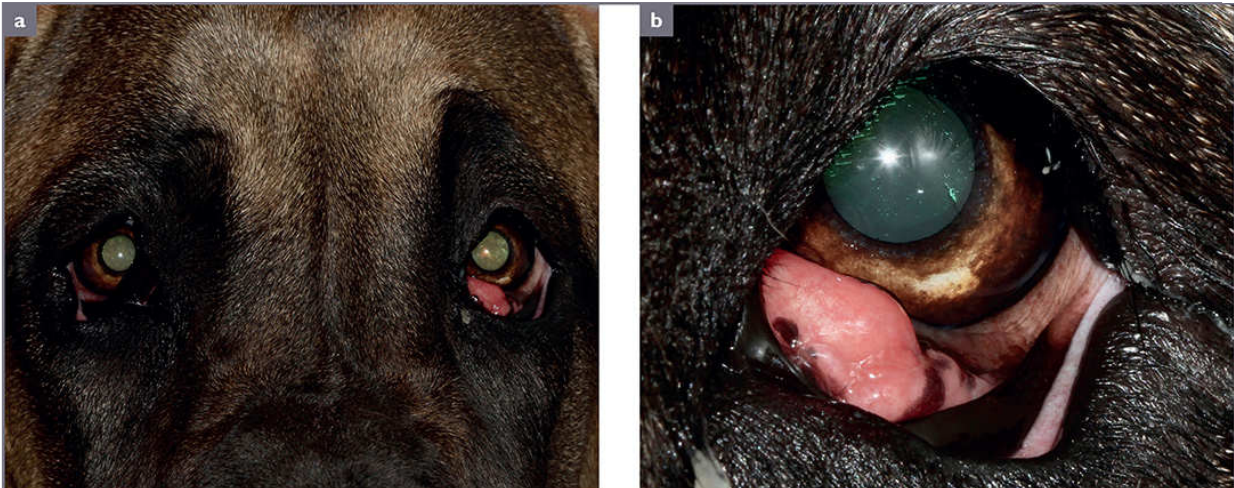


Figure 19. Great Dane with everted cartilage of the nictitating membrane (a). Closer view of the left eye (b).



Surgical resolution of a prolapsed gland of the nictitating membrane



Eversion of the cartilage and its surgical resolution

Plasmacytoma

A plasmacytoma is a lymphoplasmacytic (plasma cells) infiltration of the nictitating membrane. It tends to be associated with chronic superficial keratitis or pannus, which is an immune-mediated disease, and is more frequently encountered in pastoral dogs, although it has been reported in many breeds (Fig. 20).

Medical treatment comprises the topical administration of cyclosporine A and/or corticosteroids. Since it is a chronic, immune-mediated disorder, vets

should examine patients before and after periods with the highest levels of solar radiation and adjust the medication as each case evolves. As solar radiation is one of the contributing factors, dogs who tolerate them should wear glasses with UV protection.



Figure 20. Plasmacytoma associated with chronic superficial keratitis in a White Swiss Shepherd Dog.

Lacerations of the nictitating membrane

The nictitating membrane may be lacerated in the same act as the eyelids, so it is essential to perform a complete examination to identify and assess any damage to the membrane or globe.

If the lesion only affects the conjunctiva, it will not even require any stitches. However, if the cartilage is exposed or has lost a lot of tissue, the damage will need surgical attention.

The intervention focuses on suturing the cartilage, re-covering the conjunctiva, and finishing up with the smoothest, straightest margin possible.

Nictitating membrane tumours

Although rare, the most prevalent nictitating membrane tumours in cats and dogs are adenocarcinomas, followed by adenomas, and, for cats, squamous cell carcinomas (Fig. 21).

These patients require full tumour staging, with the application of advanced imaging techniques (CT, MRI) to the orbit and a study of the draining lymph nodes, chest, and abdomen to rule out metastasis.

If necessary, the nictitating membrane can be completely resected, but efforts must be made to prevent secondary dry eye syndrome.



Figure 21. Neoplastic mass growing on the free margin of the nictitating membrane.

Nictitating membrane flap

This is a simple surgical procedure that can be used to protect the ocular surface, but under no circumstances should it replace corneal surgery. It can be performed as an emergency measure to reduce exophthalmos or to protect an eye until it can be examined by a specialist and consists of suturing the nictitating membrane to the upper eyelid or bulbar conjunctiva.

The procedure requires the administration of general anaesthesia and, if attaching the membrane to the upper eyelid, we recommend the use of soft silicone tubes to disperse suture tension and prevent skin necrosis. The first stitch must be placed near the membrane's vertical section of cartilage and with a partial-thickness suture to ensure it does not pierce the bulbar surface.

If the chosen approach is to attach the membrane to the superior bulbar conjunctiva, always remember that the stitch in the conjunctiva should include the underlying Tenon's capsule to harvest a flap with sufficient tissue. We tend to apply at least four stitches and leave very long ends so that they do not prickle and irritate the conjunctiva when the patient blinks.

Following a nictitating membrane flap procedure, patients must wear an Elizabethan collar and attend weekly follow-ups to check suture tension. The intervention does not contraindicate topical treatments.



Nictitating membrane flap procedure, attaching the membrane to the upper eyelid



Nictitating membrane flap procedure, attaching the membrane to the bulbar conjunctiva

05

The cornea and sclera



Introduction

The cornea and sclera are the external, fibrous layers that make up the sheath of the eyeball.

The cornea is transparent and corresponds to the window through which light from an object passes into the eye and is therefore located at the front of the globe, while the rest of the circumference consists of the white, totally opaque sclera.

The sclera, which extends over a much larger surface than the cornea, is very rich in collagen and comprises three well-differentiated layers: the episclera, which provides the external connection with Tenon's capsule; the stroma; and the lamina fusca, which is in contact with the uveal structures (the choroid and ciliary body).

The optic nerve enters the eye at the rear of the sclera, specifically the portion lying inferonasally to the ocular axis, after passing through a structure called the cribriform plate which, in histological terms, is formed like a colander. Visible on the outside of this structure are the orifices through which the short posterior ciliary arteries and nerves pass, while the anterior ciliary arteries and vortex veins enter the sclera at the ciliary body.

Similarly, the cornea consists of four well-differentiated layers: the stratified epithelium located on the anterior aspect, which is accompanied by the thin basement membrane; the stroma arranged in around 200 layers of organised, low-cellularity collagen formed almost exclusively by keratocytes; Descemet's membrane which continues to grow thicker throughout the animal's lifetime, has a certain degree of elasticity, and forms the basement for the last layer; which is the endothelium (Fig. 1).

The area of transition between the cornea and sclera is called the limbus. The corneoscleral limbus is very important because it forms the starting point for most of the mechanisms that repair any corneal lesions and its integrity is fundamental to prevent the conjunctiva from invading the corneal surface, as occurs in the case of symblepharon.

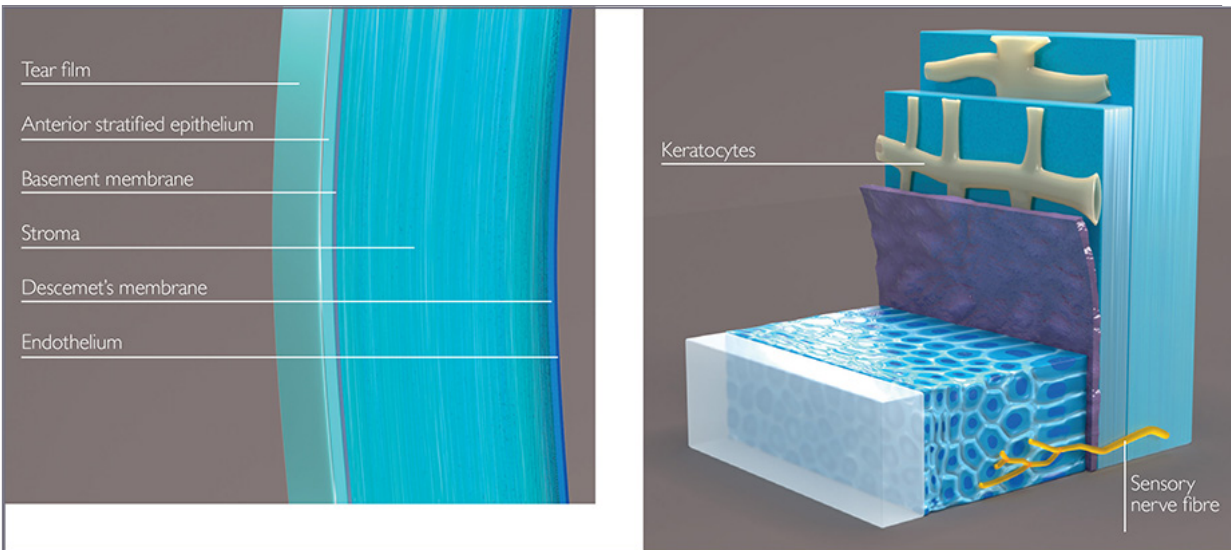


Figure 1. Layers and components of the cornea.

Keys to the pathophysiology of the cornea and sclera

- The appearance of spots in the sclera and the dilation of its vessels may be indicative of a disease process affecting the sclera or the inside of the eyeball.
- The sclera has a certain capacity to tolerate increases in intraocular pressure, but it will eventually yield if the pressure remains elevated for some time, thus increasing the eye's volume (buphthalmos) (Fig. 2). Similarly, sustained low pressures tend to cause shrinkage of these layers and, consequently, reduce the size of the eyeball (phthisis bulbi).
- The corneal epithelium has a high capacity for regeneration that depends directly on normal limbic activity. Blood vessels (i.e. neovascularisation) will also emanate from the limbus if needed to help repair any deep lesions (Fig. 3).
- The correct arrangement of the collagen fibres in the different layers of the corneal stroma and their proper hydration are two of the most influential parameters with respect to the transparency of the cornea. Stromal lesions, particularly deep ones, may imply disorganised collagen and fibrosis, which results in the development of scar tissue (cicatricial leukomas) (Fig. 4).
- The cornea is prone to suffer problems due to calcium and cholesterol deposits. These accumulations provoke the loss of corneal transparency and may be accompanied by inflammation and ulceration. The cornea

may also form the foundation for pigment deposits, for example superficial melanin or uveal pigment following the rupture of uveal cysts in the posterior chamber, which may break up and adhere to the endothelium upon coming into contact with it.

- The high density of sensory nerve fibres in the cornea, above all in the most superficial third, is directly proportional to the pain that tends to accompany corneal disorders.
- The endothelium plays a fundamental role in nourishing, through the aqueous humour, and actively dehydrating the cornea, which is why any diseases that affect the endothelium usually produce stromal oedema (Fig. 5).



Figure 2. Dog with buphthalmos in the left eye caused by absolute glaucoma.



Figure 3. A scarring reaction indicated by neovascularisation and granulation in a corneal ulcer.



Figure 4. Vessels associated with a cicatricial leukoma due to a severe lesion.



Figure 5. Stromal oedema of endothelial origin. Small bullae can be seen in the inferotemporal quadrant.

Examination of the cornea and sclera

When examining the cornea, always bear in mind that it has four main characteristics; it should be smooth, shiny, transparent, and avascular.

The sclera should also be smooth, off-white in colour, and uniform.

The cornea should be examined using equipment fitted with magnification lenses and a light source, so the eye can be observed in detail. Most ophthalmologists use a slit lamp which can be used to change the angle of the light source with respect to the observer and filter the light into a thin sheet. The clinician can use these two features to examine the shape and depth of any lesions to the anterior segment (Figs. 6 and 7). Damage to the cornea can therefore be classified as epithelial, anterior, mid, or posterior stromal, and descemetic corneal lesions based on their depth.

Vital stains are especially important when assessing the cornea. Rose bengal is a stain with a high affinity for necrotic or degenerated cells in the

conjunctival and corneal epithelia, so it is often used to detect alterations that manifest in the early stages of conditions such as dry eye syndrome. It is also used to identify dendritic ulcers, although this complaint is more common among cats. Some patients find rose bengal irritating.

Fluorescein staining is essential when examining eyes suspected of corneal compromise. The test's utility derives from the fact that fluorescein readily impregnates the very hydrophilic stroma but does not stain the hydrophobic corneal epithelium (Fig. 8). Finally, always remember that Descemet's membrane, the last layer before piercing the eye, is also very hydrophobic and therefore is not stained by fluorescein. Consequently, whenever fluorescein binds to the cornea, we can be certain that there is an ulcer and a lack of, at the very least, epithelium. If the ulcer is deep enough and Descemet's membrane is exposed, the deepest part of the lesion will not be stained with fluorescein.

Complementary techniques for examining the cornea, such as high-frequency ultrasound, ultrabiomicroscopy (UBM), which is also used to inspect the sclera, or optical coherence tomography (OCT), can provide information about the histology of these structures (Fig. 9).

The study should also include a corneal sensitivity test which can be carried out with a cotton swab or in a more controlled manner using an aesthesiometer.



Figure 6. Examination using a handheld slit lamp.



Figure 7. Examination with a tabletop slit lamp.

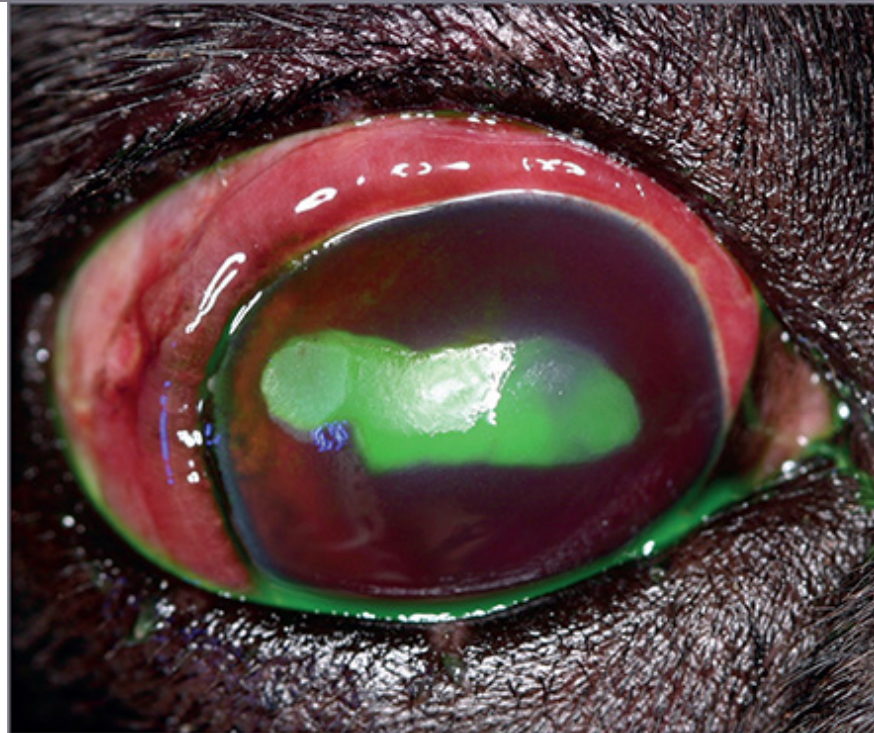


Figure 8. Ulcer stained with fluorescein.

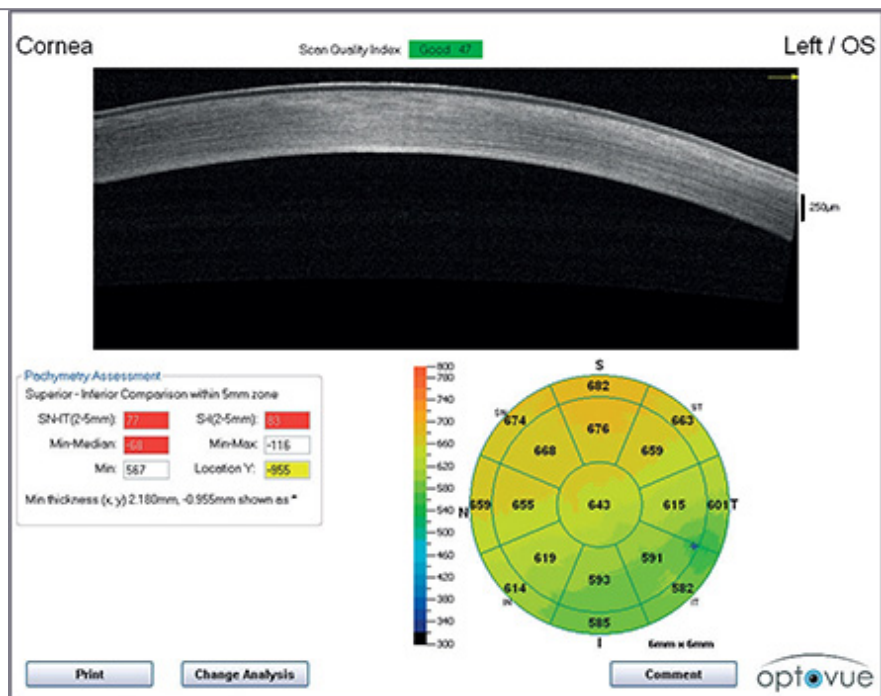


Figure 9. Optical coherence tomography (OCT) cross-sectional scan of a cornea and pachymetry map.

Signs of disease in the cornea and sclera

- **Loss of their smooth, uniform character:** for the cornea, this is usually related to alterations in the endothelium, granulation, and regions of reduced thickness (Fig. 10). A staphyloma is a debilitated and thinned area of cornea or sclera with protrusion of underlying tissue (usually the uvea). Some diseases of the sclera course with nodules and granulomas, which may be accompanied by vascularisation (Fig. 11).
- **Loss of corneal lustre:** generally associated with alterations to the quality and quantity of tear production.
- **Oedema:** due to lesions affecting either the endothelium or the corneal surface.
- **Corneal neovascularisation:** indication of a process' chronicity and severity (Fig. 12).
- **Pigment deposition:** this is also related to the chronicity of the inflammation. It is caused by the migration of melanocytes to the ocular surface, and, if the pigment is sufficiently widespread and dense, it can result in blindness (Fig. 13).
- **Signs of pain:** such as enophthalmos, epiphora, blepharospasm, photophobia, and miosis.

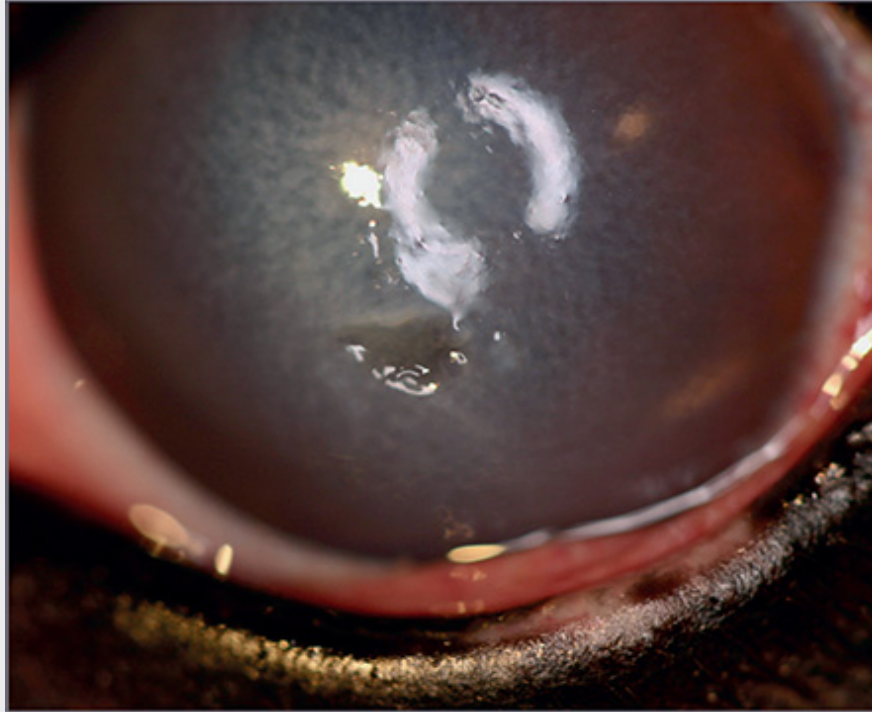


Figure 10. Loss of smoothness and ulcers in a case of corneal oedema. Note how the reflection of the camera's flash on the corneal surface is granulated.

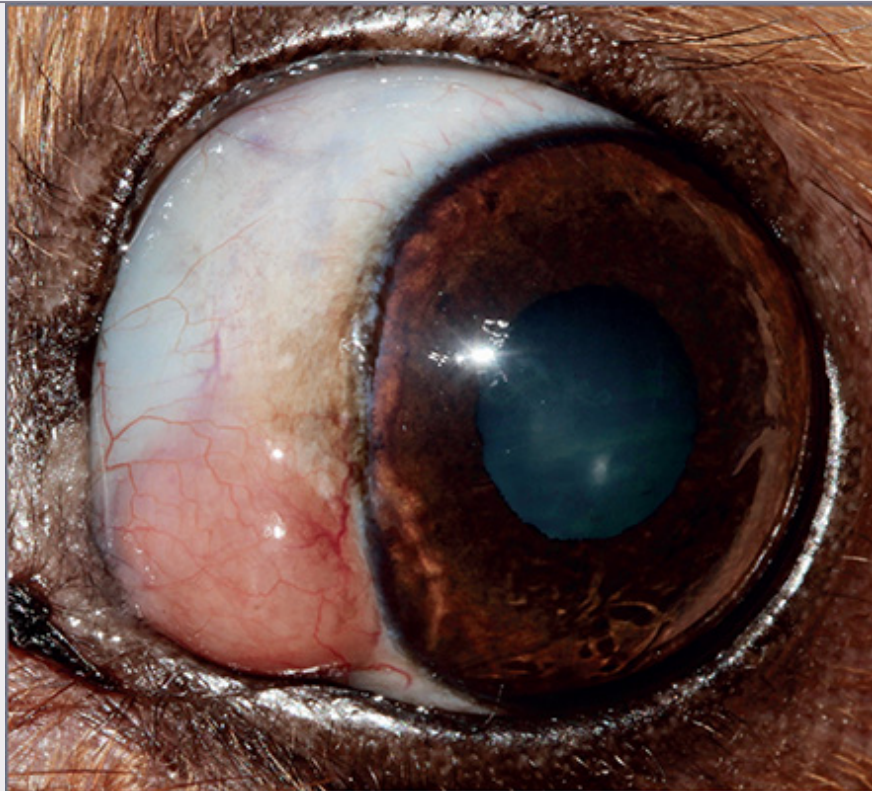


Figure 11. Scleral nodule with vascularisation.

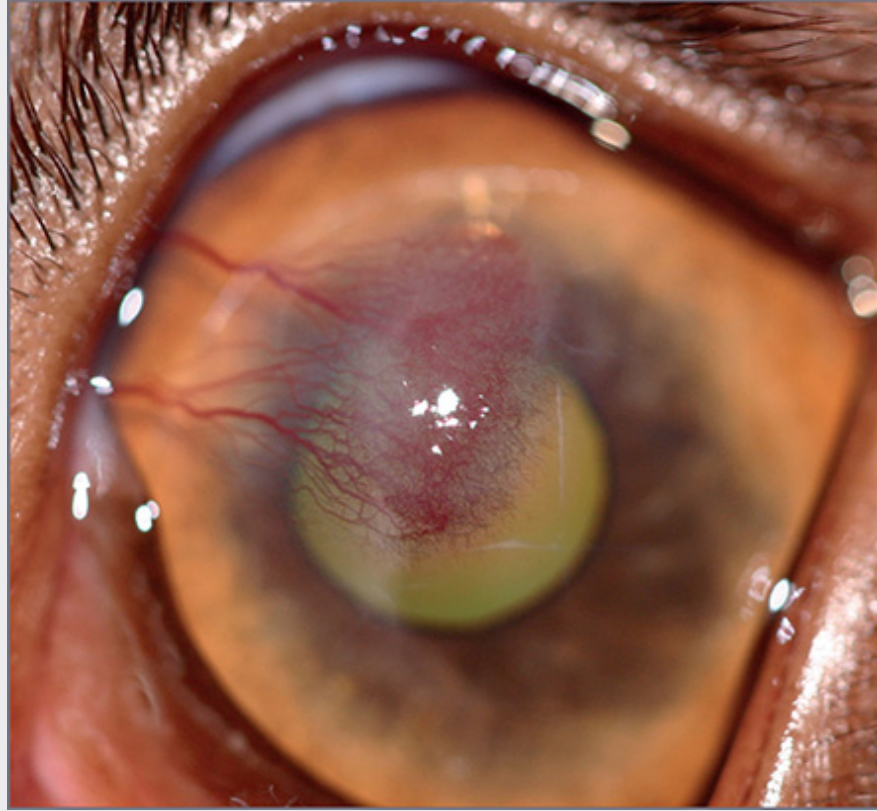


Figure 12. Neovascularisation and granulation are indicative of the process' chronicity.

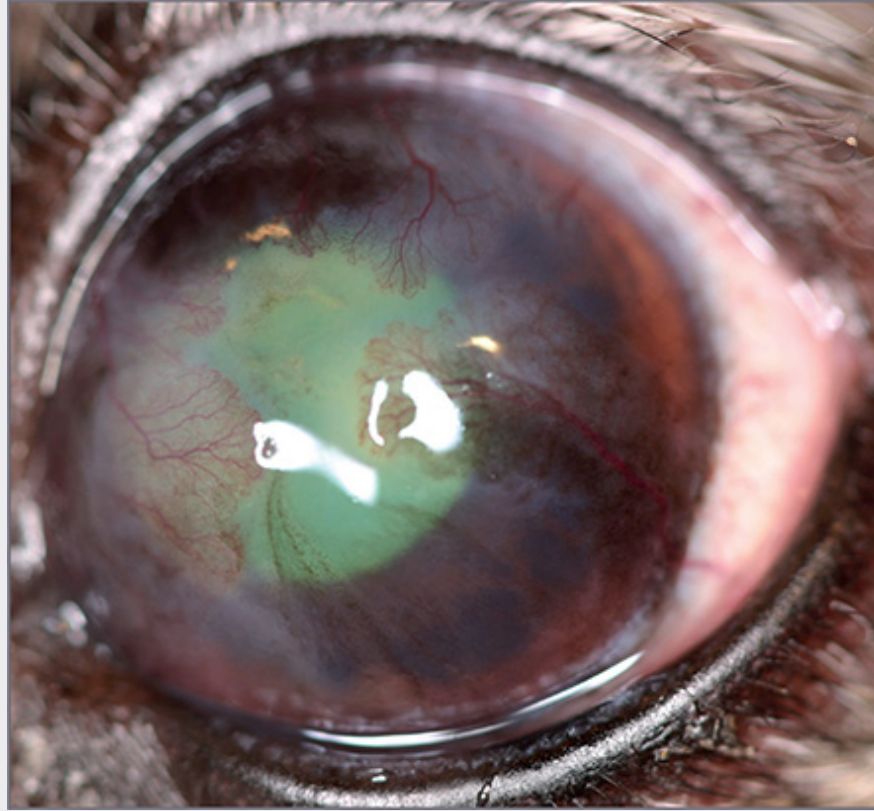


Figure 13. Penetration of melanin and neovascularisation in a case of chronic keratitis.

Diseases of the cornea and sclera

Hereditary diseases of the cornea

Persistent pupillary membrane

This refers to remnants of the uveal tract that do not atrophy in the eye's final stages of embryonic development forming strands that arc from the iris collarette to the corneal endothelium.

Bear in mind that they produce opacity at any point where they attach to the cornea and that this will affect the animal's vision to a varying extent depending on the site of attachment.

Dermoids or choristomas

As explained in other chapters, these remains of dermal tissue can affect the eyelids, conjunctivae, cornea, or a combination thereof. If there is corneal

compromise, these growths will logically cause a loss of transparency and irritation when the eyelids brush against them (Fig. 14).

It is crucial to remember that a dog's eye may tolerate these remains of skin covering the cornea relatively well for the first few weeks of life, but the first hairs will emerge shortly afterwards resulting in pain and often a corneal ulcer.

The treatment of both dermoids and choristomas requires the surgical resection of all of the abnormal tissue by means of a keratectomy.



Figure 14. Corneal dermoid.

Noninflammatory keratopathies

Corneal dystrophy

Dystrophies are slowly progressive and typically bilateral diseases that cause a loss of corneal transparency. They are highly variable and can develop in dogs of different ages and breeds.

In infantile corneal dystrophy, young puppies can have subepithelial spots that often disappear spontaneously by the age of 8–10 weeks as the eye develops.

Corneal dystrophies can be classed as subepithelial (also known as geographic), stromal, and even endothelial.

Stromal dystrophies are common to several breeds, including the Siberian Husky and Beagle. They tend to evolve very slowly and rarely require any treatment.

Endothelial dystrophies, which are frequently encountered in Chihuahuas, for example, can cause severe stromal oedema requiring treatment in an attempt to correct the corneal hydration (Figs. 15 and 16).

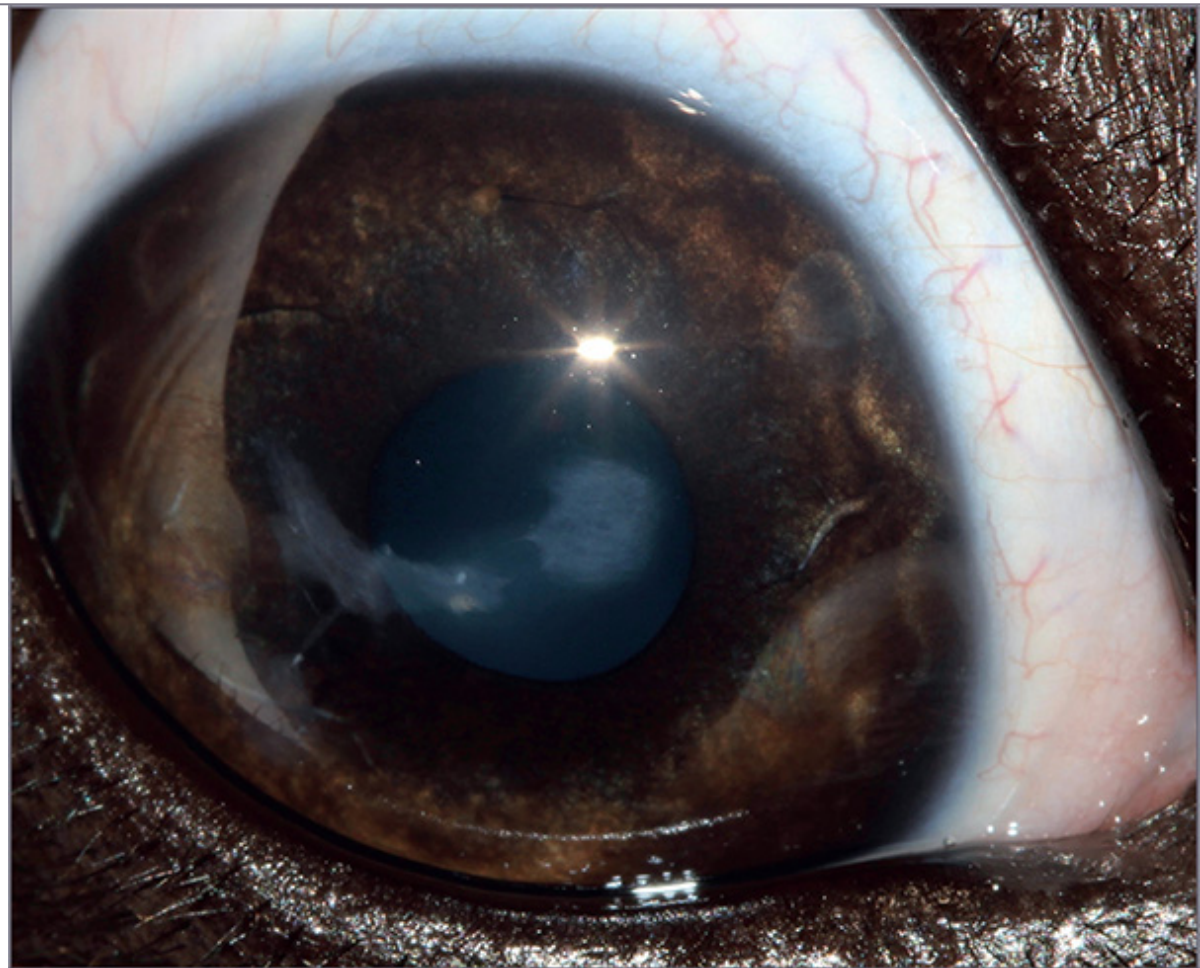


Figure 15. Mild case of endothelial dystrophy.

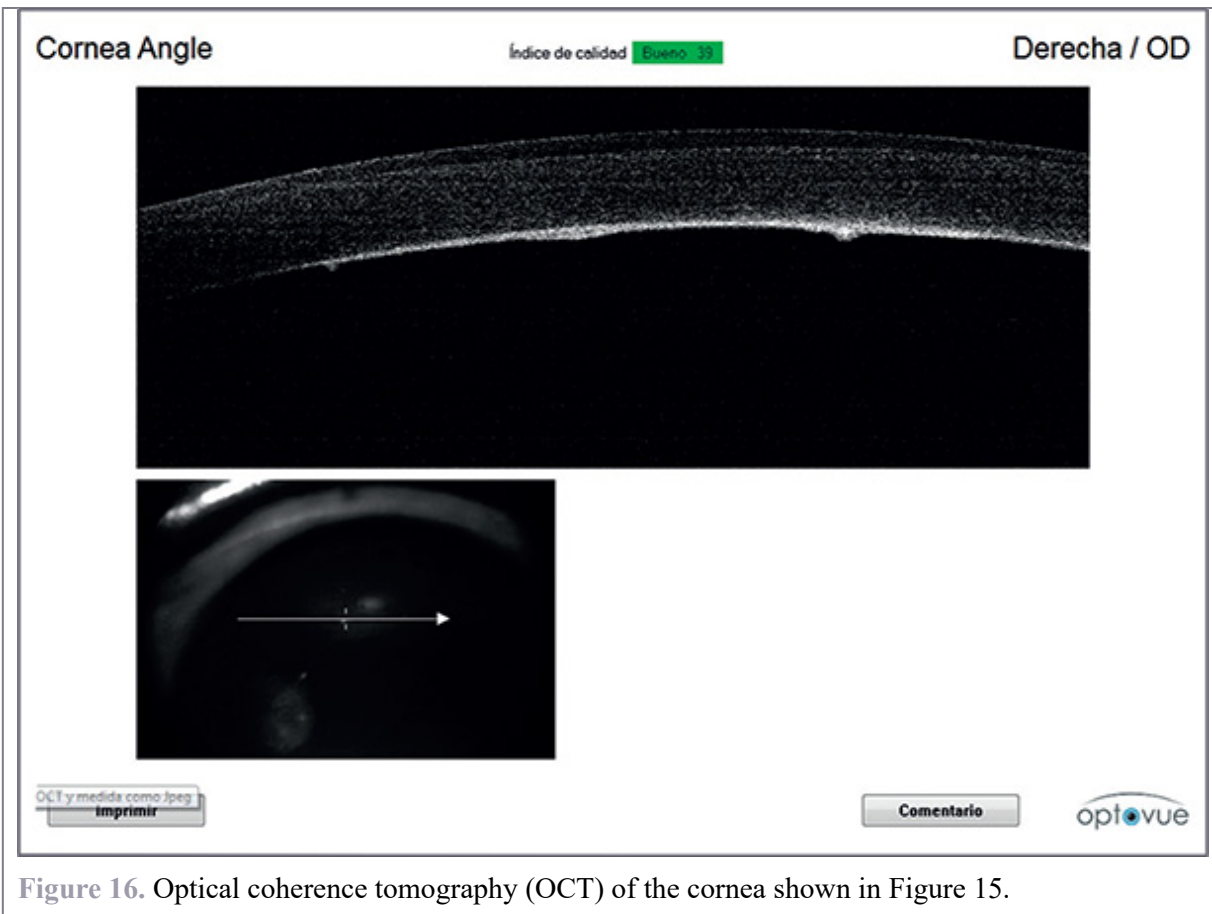


Figure 16. Optical coherence tomography (OCT) of the cornea shown in Figure 15.

Inflammatory keratopathies

Ulcerative keratitis

Corneal ulcers are almost certainly one of the leading reasons for ophthalmological consultations at the veterinary clinic.

The most logical classification of corneal ulcers categorises them as simple or complex ulcers. Simple ulcers are superficial, of known origin, and devoid of any signs of infection. Complex ulcers include spontaneous chronic corneal epithelial defects (SCCED), which are also known as indolent ulcers, underrun ulcers, or recurrent corneal erosion, and stromal ulcers.

Vets need to bear this classification in mind when assessing a corneal ulcer for the first time; if the process does not resolve within 5–7 days, a complex ulcer should be suspected and may require the attention of a specialist.

The treatment of corneal ulcers hinges heavily on finding the cause, because if the cause persists, the ulcer may recur, or the treatment could fail.

Ulcers can also be classified according to their depth of penetration into the cornea.



Physiological structure of the cornea and different types of ulceration

Superficial ulcers

These only affect the epithelial layers of the cornea. They are generally simple processes that may be related to known causes such as trauma, abrasion from hairs and eyelashes, and so on.

The diagnosis is based on the clinical signs and confirmed by staining with dyes, such as fluorescein, which stain the underlying stroma and indicate a lack of epithelium. Superficial ulcers have a favourable prognosis.

Treatment usually follows a topical medical approach and should always include pain relief with cycloplegic agents and first-intention antibiotic therapy. Just as for all corneal ulcers, the use of an Elizabethan collar is an essential part of treatment.

Spontaneous chronic corneal epithelial defects (SCCED)

SCCED are superficial ulcers that prove refractory to conventional treatments. They have an unattached epithelial lip and, when they become chronic, are accompanied by clinical signs such as neovascularisation and granulation (Fig. 17).

They are the result of a genetic defect in the corneal epithelium's capacity to adhere to its underlying base, hence owners should be advised that there is a high probability of SCCED developing in the other eye in the future.

Treatment involves eliminating the raised epithelium from the entire surface of the cornea by debridement, then introducing a therapeutic lens which allows for the growth of a healthy new epithelium (Fig. 18).

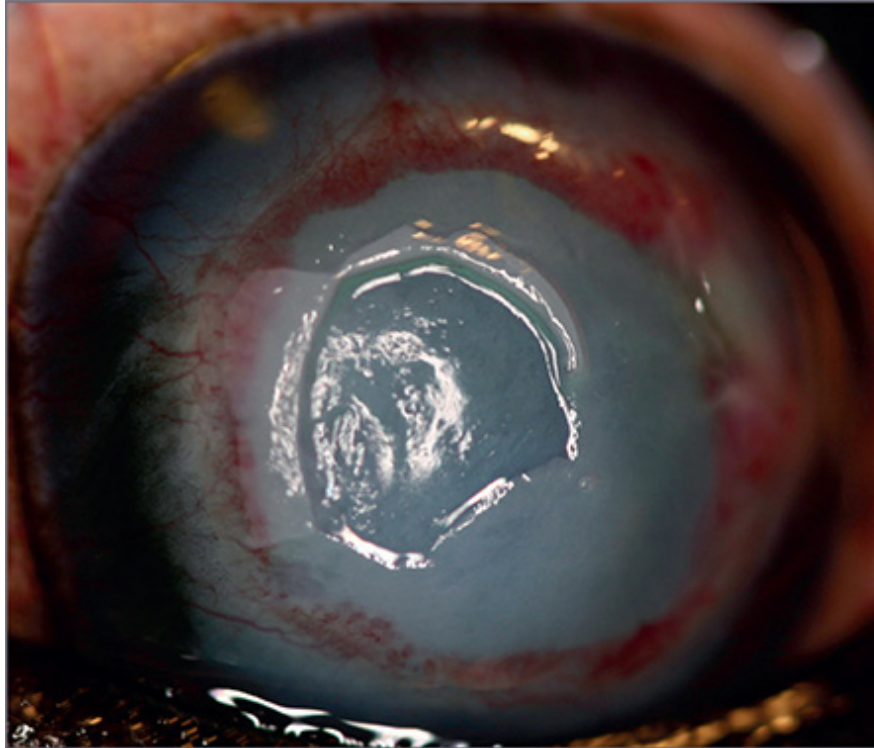


Figure 17. Spontaneous chronic corneal epithelial defect (SCCED) with signs of chronicity. There are also indications of hypertrophic and nonadherent epithelial folds.

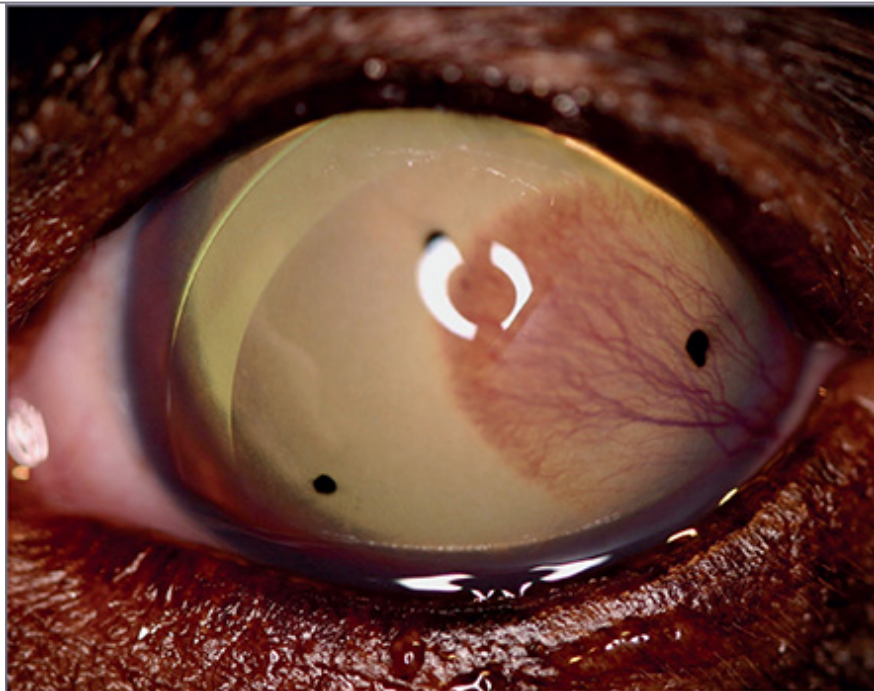


Figure 18. Signs of scarring in a spontaneous chronic corneal epithelial defect (SCCED) under a therapeutic lens.

Stromal ulcers

These ulcers develop to varying depths and the stromal defect (crater) is visible to the naked eye.

They usually course with clinical signs of intense pain including blepharospasms, tearing, prolapsed nictitating membrane, miosis, and pawing at the eye. Pawing can aggravate the lesion significantly.

Besides attempting to eliminate the cause, treatment tends to include systemic and topical therapy and, depending on the depth of the lesion, often requires surgical actions such as the application a biomaterial graft. These procedures are designed to restore the tissue's tectonic properties (an architectural concept that combines structural characteristics with the optimal arrangement of the components in order to support external attacks and pressure) and facilitate healing of the damaged cornea by minimising scarring (Fig. 19).

All of these ulcers tend to course with a varying degree of associated uveitis which should be considered during treatment.

The prognosis depends on the depth of the ulcer and whether the patient can avoid fibrosis and, therefore, the development of scarring, which would affect their vision and, in many cases, eventually become pigmented. The extent of vision compromise is also determined by the impact on the visual axis, with central ulcers being far more incapacitating.

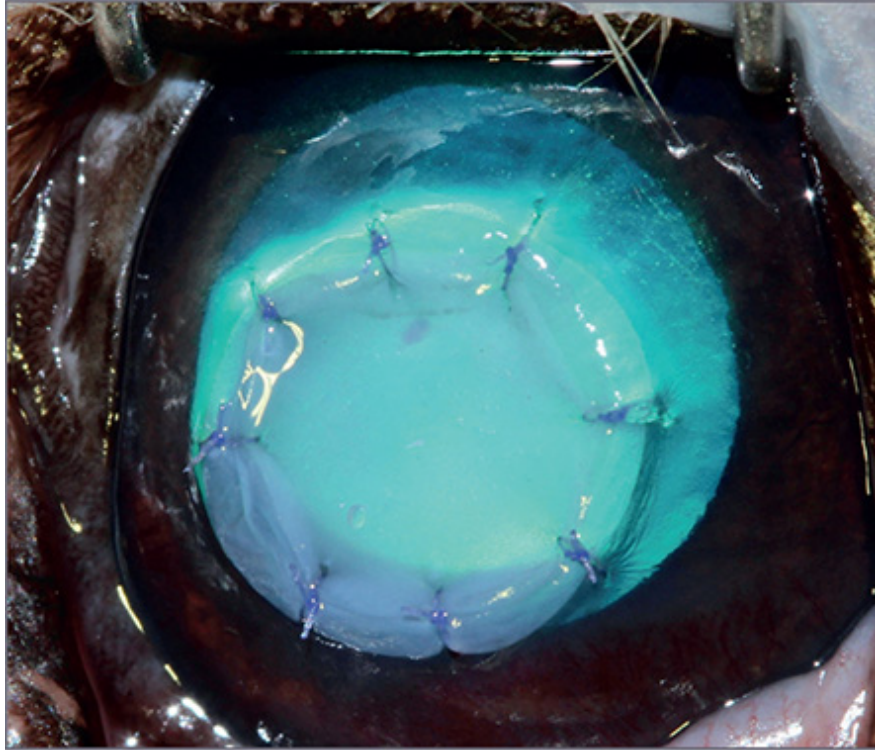


Figure 19. Amniotic membrane graft in a deep ulcer.



Surgical treatment of an ulcer with detached margins

Collagenase ulcers

These deserve their own section because of their severity. Collagenase ulcers normally evolve very quickly and are accompanied by intense uveal inflammation that will require treatment.

While their aetiology can include fungi, the most common cause is contamination of a primary lesion by germs such as streptococci, staphylococci, and *Pseudomonas*.

Patients will have a “dirty eye” with a mucopurulent discharge, corneal oedema, and areas of corneal deterioration where the tissue is starting to gel due to the action of collagenolytic enzymes (Fig. 20).

This problem requires medical and surgical treatment with the use of drugs that inhibit the enzymatic activity and the elimination of the degraded areas which in themselves generate collagenolysis. These ulcers require the frequent instillation of eye drops, so patients often need to be hospitalised during treatment.

The prognosis is guarded, because the process will result in a perforated globe if it is not brought under control.

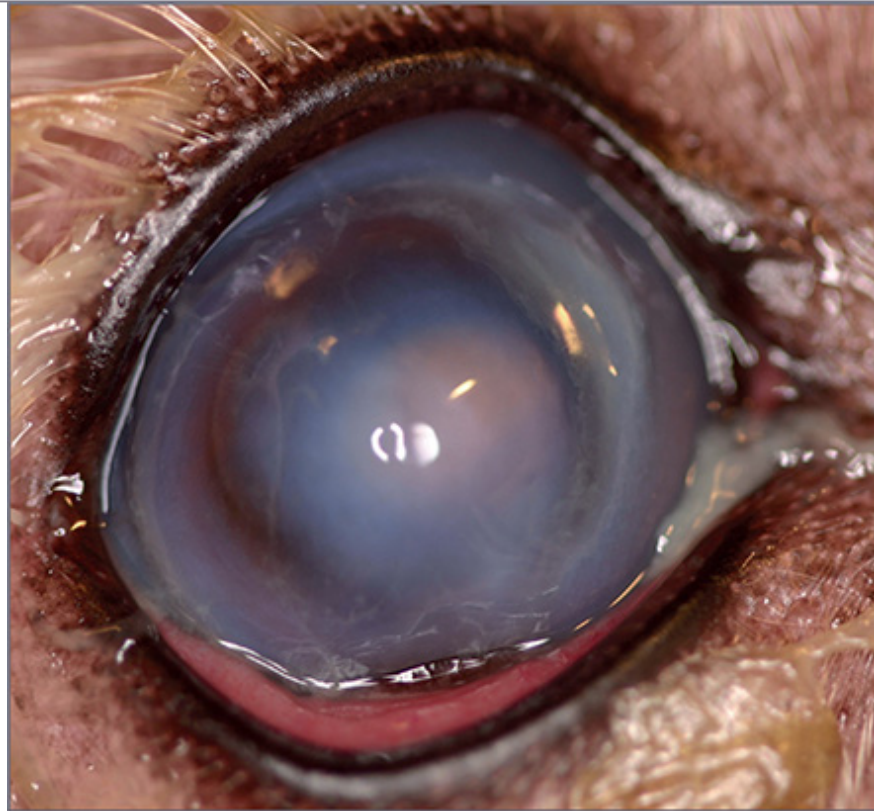


Figure 20. Severe collagenase ulcer. The transparent areas correspond to an exposed Descemet's membrane.

Descemet's ulcers

Ulcers that are deep enough to expose Descemet's membrane. They are considered the last step before globe perforation and their examination will reveal a lack of staining in this final layer of the cornea; this produces a doughnut-shaped fluorescein pattern with stained edges and an unstained centre.

In some cases, the intraocular pressure makes the relatively elastic Descemet's membrane bulge outwards, giving rise to a descemetocoele (Fig.

21).

Descemetocelles are medical emergencies requiring surgical intervention to recover corneal thickness and avoid perforation.



Figure 21. Lateral view of a severe descemetocoele.

Perforated ulcers

Contrary to popular belief, these ulcers do not necessarily result in vision loss, but they do require prompt surgical treatment to recover the eyeball's and cornea's seal (Fig. 22).

Their gravity is also linked to the possibility of patients developing an intraocular infection, called ophthalmitis, with very severe consequences.

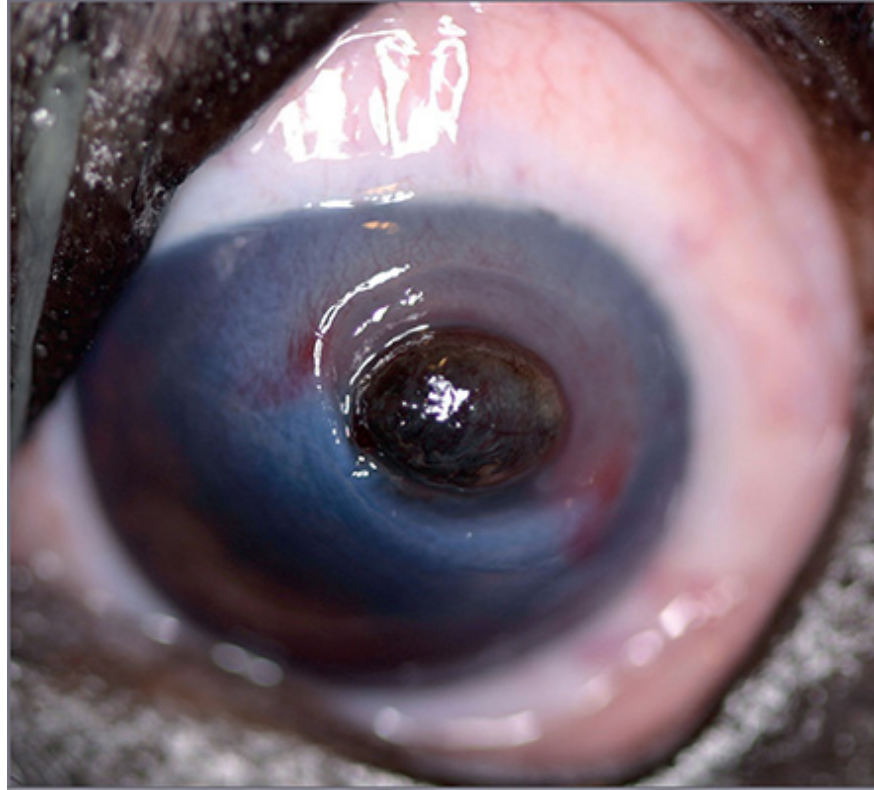


Figure 22. Perforated ulcer with iris herniation.

Chemical ulcers

These are caused when acidic or alkaline substances come into contact with the ocular surface. The chemicals trigger a sterile collagen degradation process, and the conjunctivae also tend to be affected (Fig. 23).

Profuse irrigation of the entire ocular surface, including the fundus of the conjunctival sacs, is absolutely crucial. Treatment should also include antibiotics, cycloplegic mydriatic agents to alleviate the pain, and, in the case of deep ulcers, the application of a biomaterial graft to act as an amniotic membrane.

The prognosis is guarded because patients may suffer corneal fibrosis and retraction of the conjunctivae, which will often culminate in phimosis and anatomical alterations of the eyelid margins. Another common complication is tear deficiency due to compromise of the glandular ducts' drainage points.



Figure 23. Chemical burn from cement dust. There is complete destruction of the corneoscleral limbus.

Nonulcerative keratitis

Corneal abscesses

These are accumulations of pus in the heart of the corneal stroma accompanied by, although to a lesser degree, an inflammatory uveal reaction and a lot of pain.

They are quite common in the Spanish clinical setting and in our experience are one of the most typical manifestations of contact with the hairs of the processionary caterpillar which contain the toxin thaumetopoein (Fig. 24).

Treatment includes keratotomy to drain the abscess and to facilitate irrigation and the application of antibiotic solutions.

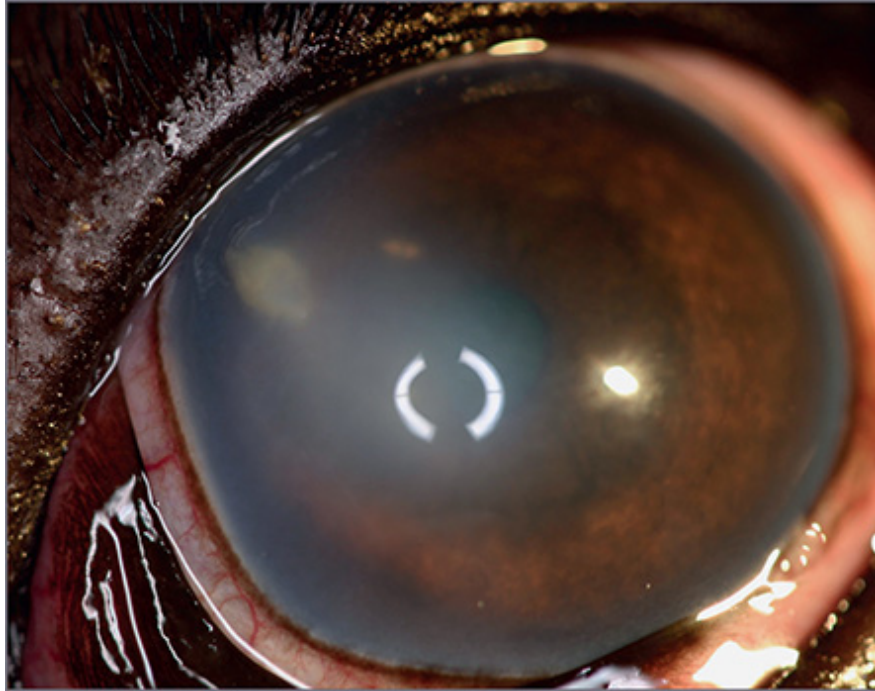


Figure 24. Stromal abscess following contact with the hairs of the processionary caterpillar (*Thaumetopoea pityocampa*).

Pigmentary keratitis

This is an expression of chronic irritation of the corneal surface which may be due to various causes, such as abrasion from hairs, distichiasis, processes affecting the quality and quantity of tear production, etc.

It results from the tendency of the dog's melanocytes to migrate towards inflamed areas of the eye's surface; animals with intense pigmentation and brachycephalic breeds are particularly predisposed, and eventually develop corneal opacity and neovascularisation (Fig. 25).

The use of topical immunomodulators, such as cyclosporine and tacrolimus, and resolution of the triggering cause are key measures in preventing total pigmentation of the cornea. Any associated eyelid abnormalities (euryblepharon, inferomedial entropion, etc.) should also be corrected.



Figure 25. Pigmentary keratitis in the nasal quadrant of the eye of a Pug.

Chronic superficial keratitis (pannus)

This is an immune-mediated process resulting in the expression of corneal surface antigens followed by the corresponding immune response. It is a bilateral condition, and often associated with plasmacytomas and lymphoplasmacytic conjunctivitis.

During the early stages of the disease, cellular infiltrates, comprised of lymphocytes and plasma cells, can be observed in the most superficial layers of the cornea. The infiltrates course concurrently with the formation of blood vessels and gradually give way to melanin, although this may occur to a lesser degree or not at all in patients from scantily pigmented breeds.

Areas of conjunctiva lying adjacent to the affected areas of cornea may develop a cellular infiltrate on the free margin of the nictitating membrane, which is visible as a thickened, greyish region, giving rise to what is known as a plasmacytoma (Fig. 26).

Although it can affect any breed, German and Belgian Shepherd Dogs are more susceptible, and exposure to sunlight appears to be one of the triggers, which is why it is more prevalent in animals that live outdoors.

Treatment is based on topical therapy with corticosteroids, and this can be combined with immunomodulators and patient management that seeks to avoid too much exposure to the sun.



Figure 26. Pannus and plasmacytoma in a German Shepherd Dog. There are small grey spots in the free margin of the nictitating membrane.

Neurogenic keratitis

This condition is due to neurological damage to the cornea's sensory innervation (trigeminal nerve) and possibly the motor innervation (facial nerve) as well.

The patient's clinical record may include previous disorders affecting the central or peripheral nervous system, such as otitis media, or even orbital lesions, and owners often report having noted their pet sleep with its eyes half open.

The clinical signs associated with neurogenic keratitis are a reduced rate of blinking, enophthalmos, lagophthalmos, and dry eye due to poor tear distribution or even a decline in tear production.

An eye examination will reveal corneal hypoaesthesia with impaired palpebral reflex, loss of sensitivity in the periocular skin, and signs of overexposure, such as stromal oedema and neovascularisation, in central

portions of the cornea. In advanced stages, the cornea will suffer desiccation and it may finally result in perforation of the globe.

Besides addressing the cause of the neurological deficit, appropriate therapeutic measures include very frequent use of moisturisers and the placement of stitches in the medial and lateral canthi in order to reduce the palpebral fissure and protect the ocular surface against dehydration.

Lipid keratopathy

Lipid keratopathy is associated with systemic diseases that course with elevated blood lipid levels, such as diabetes mellitus, hypothyroidism, Cushing's syndrome, and pancreatitis.

The fat deposits may accumulate in one or both corneas, and while initially there may be no clinical signs of inflammation, when the process becomes chronic, there will be evidence of neovascularisation and even inflammatory infiltrates (Fig. 27).

Treatment includes low-fat diets, and surgery may be required if the lesions end up affecting the centre of the cornea.



Figure 27. Cholesterol crystals deposited in the centre of the cornea.

Corneal degeneration

This process corresponds to the final stage of other keratopathies that produce an accumulation of lipid or calcium crystals. The presence of blood

vessels running towards the lesion is a sign that the process is accompanied by inflammation.

The affected area will acquire a whitish-grey colour with spicular mineral deposits that coalesce to form hard plaques, ultimately resulting in a brittle cornea. If these lesions are rubbed with a swab, portions of affected cornea may flake off, leaving ulcers of varying depths that do not heal easily and may expose Descemet's membrane (Fig. 28).

Blood tests should be used to rule out diseases such as hyperadrenocorticism and processes that course with hypercalcaemia, hyperphosphataemia, uraemia, and hypervitaminosis D.

These patients often require surgery to avoid perforation of the globe (Fig. 29).



Figure 28. Descemet's membrane ulcer associated with corneal degeneration.

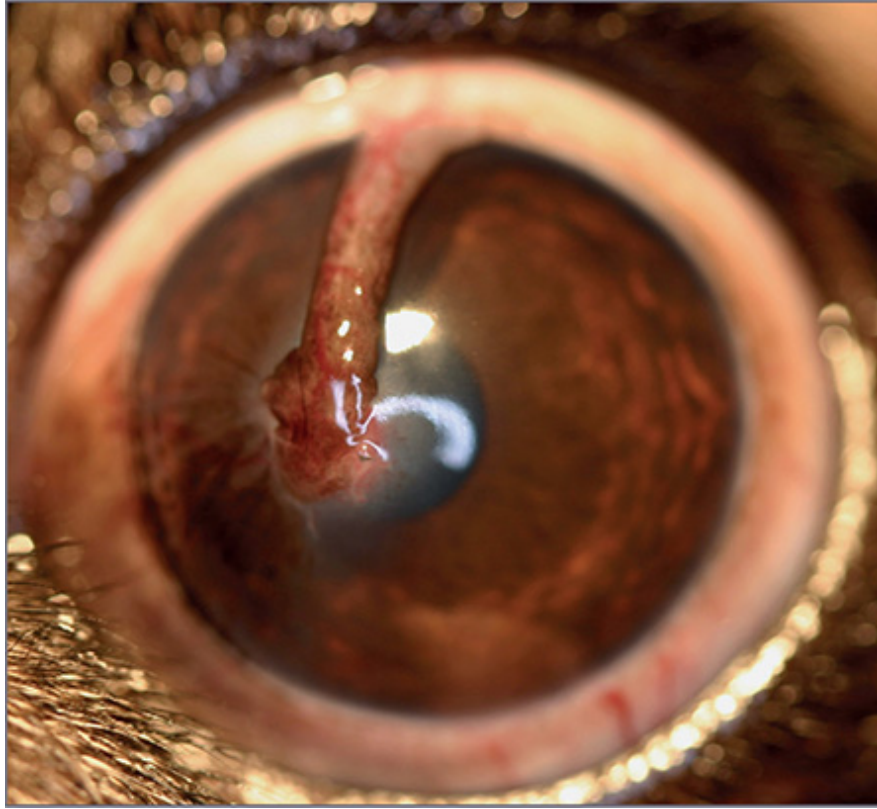


Figure 29. Pedunculated conjunctival flap as treatment for a deep ulcer associated with corneal degeneration.

Corneal and scleral tumours

Although uncommon, the cornea and sclera can develop tumours, above all in animals who have received chronic treatment with a topical immunosuppressant.

Epibulbar melanocytoma is a tumour that emanates in the corneoscleral limbus and produces slowly advancing pigmented masses, usually in the dorsal arc. They are most frequently encountered in German Shepherd Dogs, Golden Retrievers, and Labrador Retrievers. These tumours may invade the cornea, iridocorneal angle, and anterior uvea (Fig. 30). Treatment is normally surgical involving a sclerokeratoplasty with a graft or laser surgery following debridement. The prognosis is usually good.



Figure 30. Melanocytic tumour affecting the sclera and cornea.

Corneal and scleral lacerations

These lesions are caused by sharp force traumas and tend to provoke herniation of some of the underlying tissue. Frequently, a fibrin and blood clot develops which, in the case of small lesions, may block the outflow of humour and help preserve the seal.

They are emergencies that need immediate treatment, for which the aim should be to restore the eyeball's seal and its normal anatomical form. To do this, the surgeon will require appropriate magnification, instruments, and suture materials, for example threads made especially for corneas and always using spatulated needles (Fig. 31).

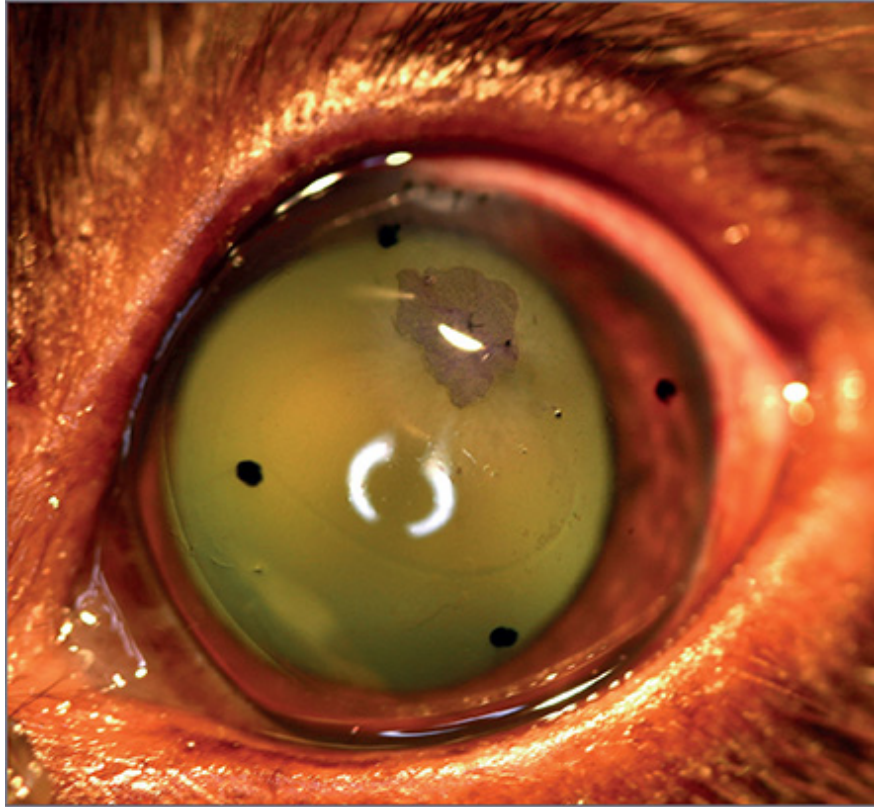


Figure 31. Penetrating wound in the cornea treated with sutures, corneal glue, and a therapeutic lens.

Foreign bodies in the cornea

Animals are commonly brought to the clinic with foreign bodies impacting the cornea. Patients demonstrate obvious signs of pain, and the eye tends to produce abundant epiphora and either a mucous or mucopurulent discharge, depending on the nature of the cause (Fig. 32).

Most foreign bodies are plant matter and may affect the cornea when they become lodged in the conjunctival sacs (e.g. grass awns), situated at the surface (e.g. small pieces of vegetable matter that adhere to the cornea and which are held in place by the blinking action of the eyelids), or embedded in the thickness of the cornea and possibly even emerge in the anterior chamber (e.g. thorns).

Manual extraction, aided by forced irrigation, functions well enough in most cases (Fig. 33).

When dealing with piercing elements lodged in the thickness of the cornea or protruding into the anterior chamber, such as splinters or thorns, it is a

good idea to assess the situation and determine whether the case should be referred to a specialist.

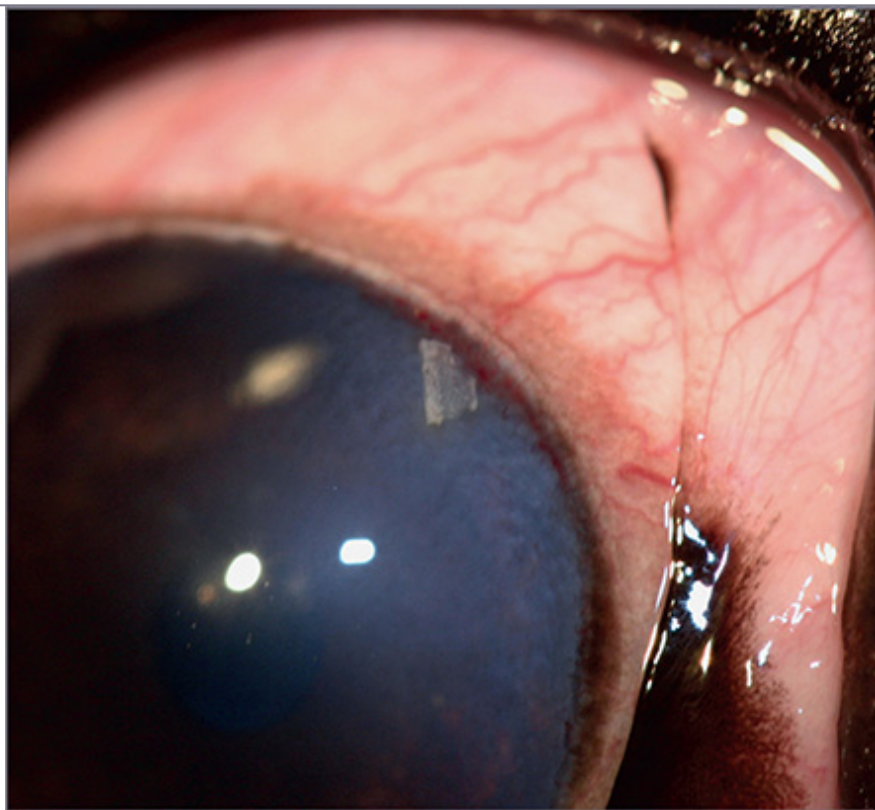


Figure 32. Foreign body of plant origin adhering to the cornea.



Figure 33. Millet husk being removed from the cornea. Note the residual ulcerated lesion left in the epithelium.

06

The uvea: uveitis



Anatomy and physiology of the uvea

The uvea is the collection of intraocular tissues formed by the iris, ciliary body, and choroid (Fig. 1). These structures provide vascularisation and nourishment to the eye, while also helping to maintain homeostasis.

The ciliary body is responsible for the production and elimination of aqueous humour, which is why any alterations to the uvea can hinder the correct function of these mechanisms.

The iris, which consists of the sphincter and dilator muscles, controls how much light enters the eye (Fig. 2), while the ciliary body regulates the accommodation of the crystalline lens in order to focus the image correctly. Household pets have a low power of accommodation, and it is particularly weak in dogs.

The uvea's dark colour is due to its high melanin content, which is necessary to absorb scattered light and produce more accurate vision.

With respect to immunity, the uvea plays a fundamental role in the eye as it corresponds to the immune barrier that isolates the intraocular space from systemic circulation and the immune system. Isolation is provided by the blood–aqueous barrier located in the anterior uvea and the blood–retinal barrier in the posterior segment. Dogs have a delicate blood–aqueous barrier, while all species have a more stable blood–retinal barrier.

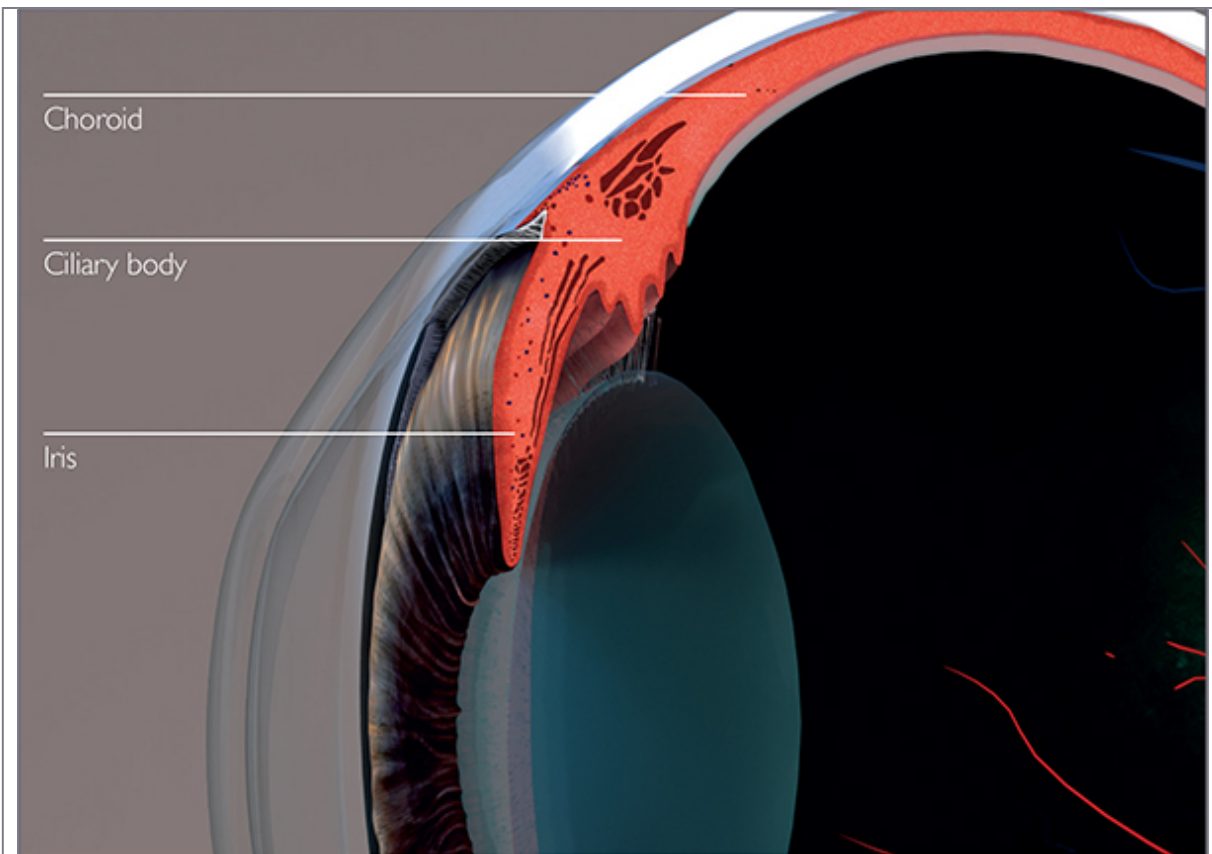


Figure 1. Components of the uvea.

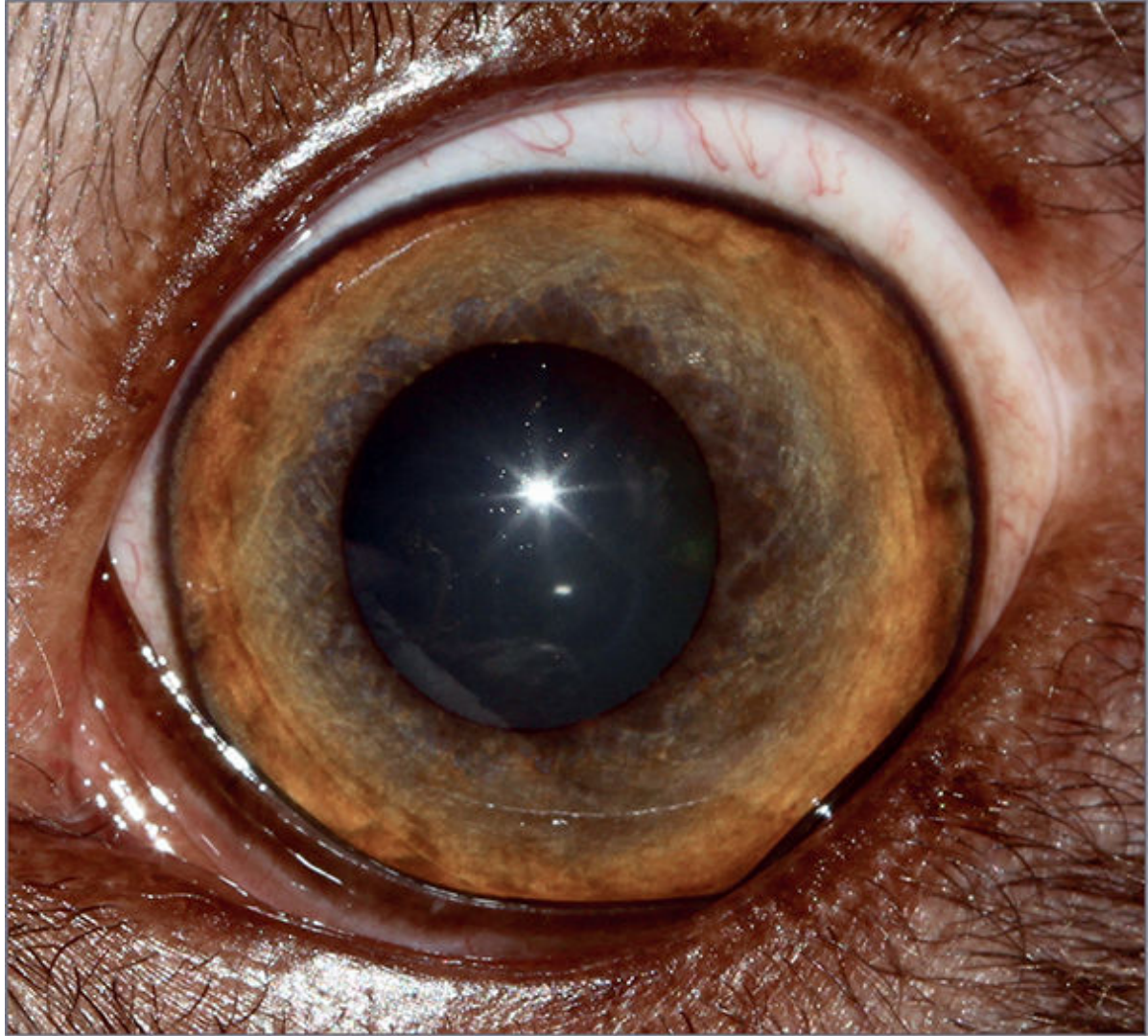


Figure 2. Appearance of a normal, healthy, light-coloured iris.

Clinical signs of uveitis

The clinical signs of uveitis are always very similar irrespective of the underlying cause.

In the case of acute uveitis, patients often have:

- Signs associated with pain: blepharospasm, photophobia, tearing.
- Signs associated with inflammation: conjunctival congestion, circumcorneal ring, corneal oedema (Fig. 3), miosis (Fig. 4), Tyndall effect (Fig. 5), hypopyon or hyphema (Fig. 6), resistance to mydriatics, inflammation of the iris (rubeosis iridis), and low intraocular pressure.

Miosis is the most frequent sign of anterior uveitis observed in dogs with corneal ulcers.

Signs associated with the chronicity of the process include:

- Cells adhered to the corneal endothelium (keratic precipitates) (Figs. 7 and 8).
- Anterior and posterior synechiae of the iris (Fig. 9).
- Hyperpigmentation, atrophy of the iris (Fig. 10).
- Appearance of preiridal fibrovascular membranes and cyclitic membranes.
- Development of cataracts.
- Secondary glaucoma.
- Phthisis bulbi.



Figure 3. Conjunctival hyperaemia, circumcorneal ring, and corneal oedema in an eye affected by keratouveitis.



Figure 4. Miosis, in the left eye, is an indication of uveitis.

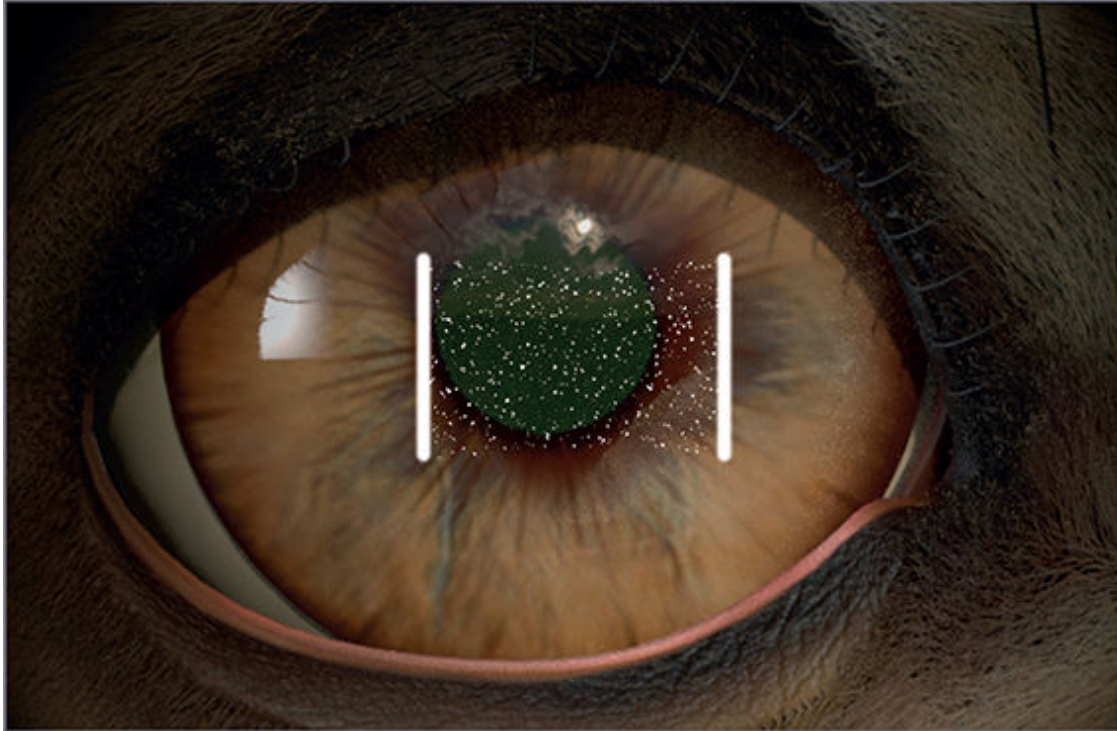


Figure 5. Illustration of the Tyndall effect caused by turbidity in the aqueous humour.



Figure 6. Hyphema affecting over half of the anterior chamber.

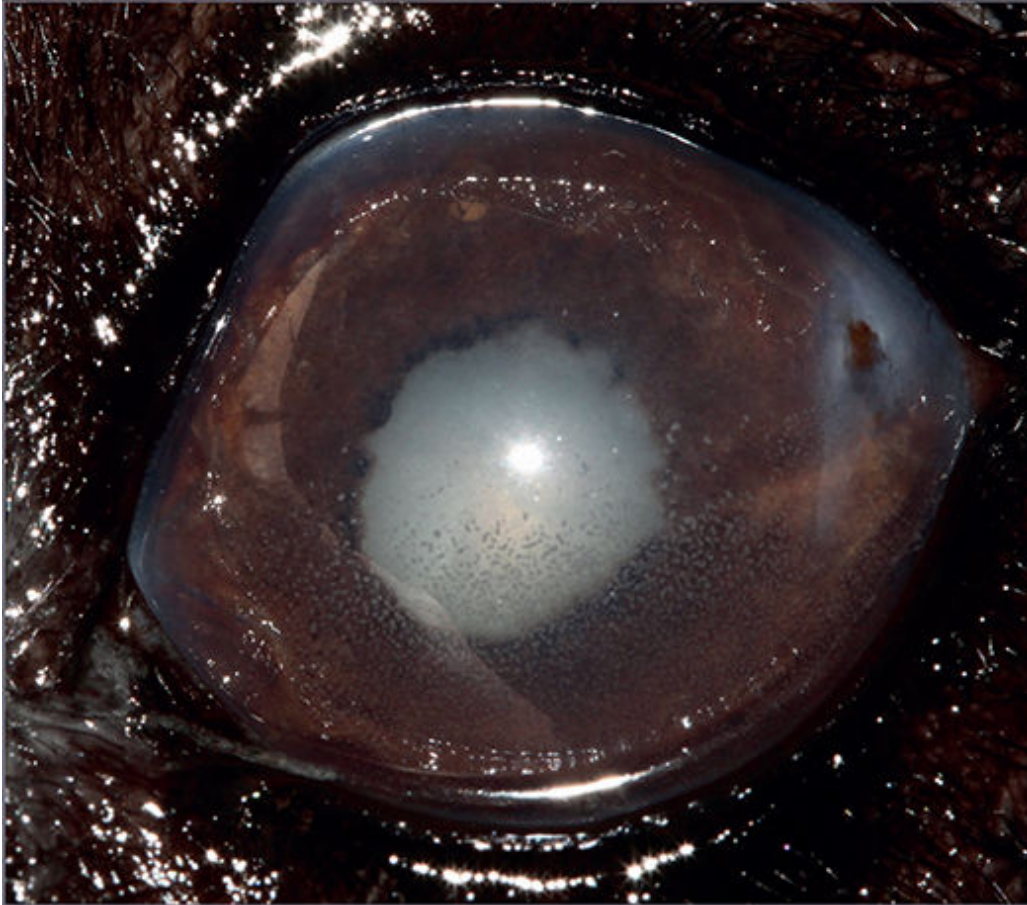


Figure 7. Keratic precipitates in a patient with lens-induced uveitis.

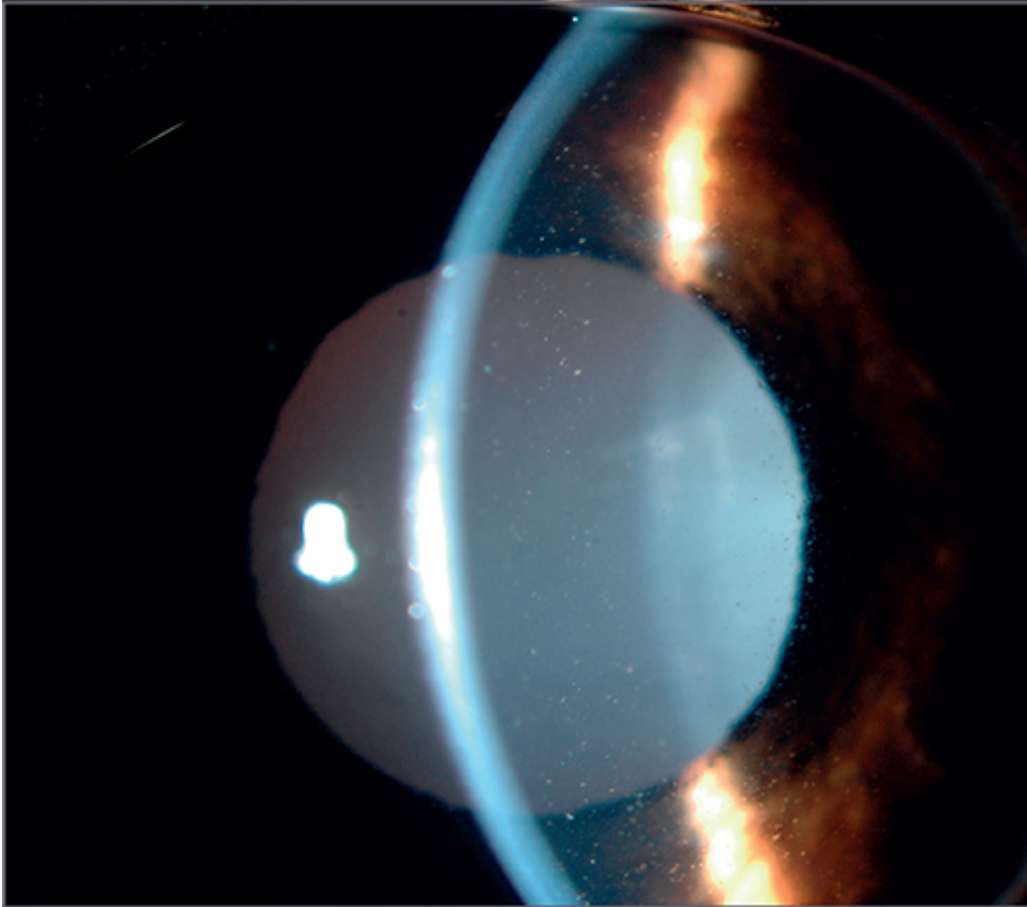


Figure 8. Backlit keratic precipitates observed during a slit lamp examination.



Uveitis

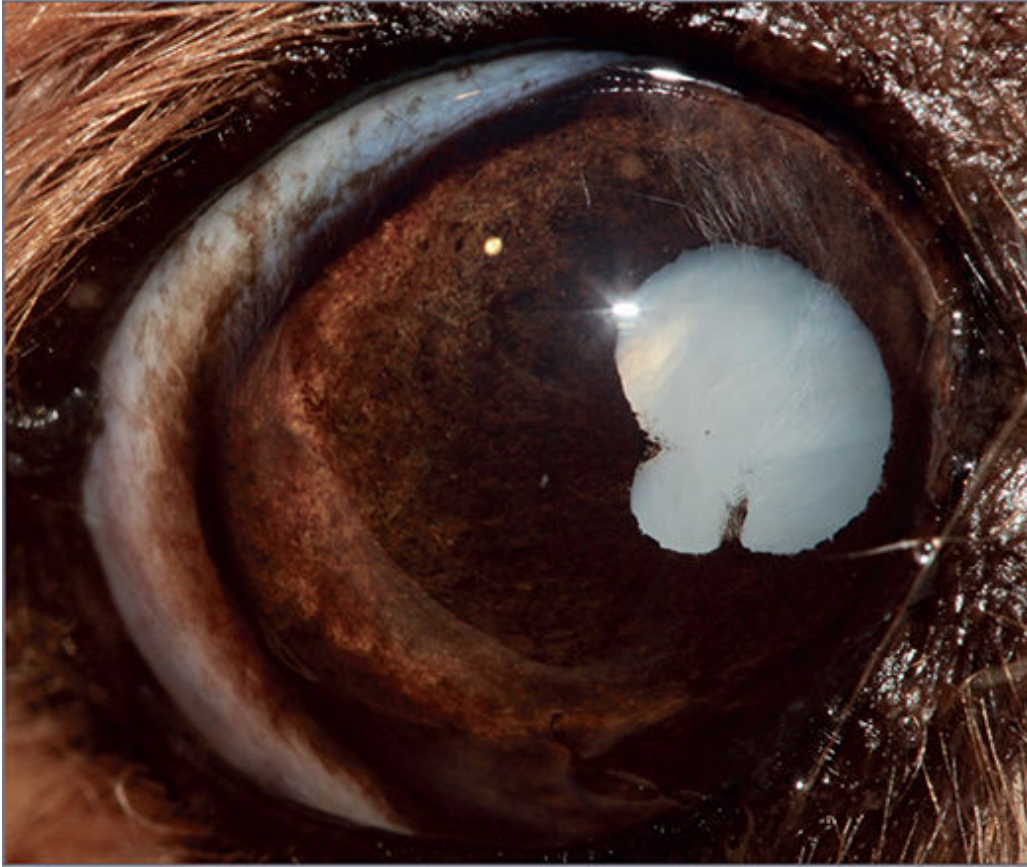


Figure 9. Synechia of the iris to the crystalline lens in a dog with lens-induced uveitis.



Figure 10. Iris atrophy.

Causes of uveitis

Ocular causes

The uvea is affected by various ocular processes:

- Reflex or neurogenic uveitis: this often develops in association with corneal ulcers and is due to irritation of the superficial corneal nerves which triggers the release of substance P in the iris and ciliary body. Substance P induces vascular dilatation, with a subsequent increase in local permeability, and the release of neutrophils and chemotactic factors. The severity of this type of uveitis usually correlates with that of the associated ulcer.
- Cataract- or lens-induced uveitis: the lens capsule isolates its contents from the exterior even before the animal's immune system has matured. If there is filtration of crystalline proteins or lens capsule rupture, a frequent occurrence during the evolution of a cataract, then it will result in acute or

chronic immune-mediated uveitis. The liquefaction of the crystalline lens proteins induced by the cataract leads to their solubilisation and filtration through the capsule. This situation is known as phacolytic uveitis and is commonly observed in advanced cataracts. Good control of this inflammation before the cataract surgery will result in fewer complications and better outcomes. Another possibility is that the capsule is no longer intact, whether due to trauma or because it is ruptured by the cataract itself, as often occurs in the case of diabetic cataract. This triggers acute and intense inflammation, which gives rise to phacoclastic uveitis that requires emergency surgery to save the eye.

- Pigmentary uveitis: reported in Golden Retrievers and Great Danes. Pigment dispersion in the anterior chamber of the eye is accompanied by a darker, thicker iris. Upon examination there are signs of uveal pigment deposited on the corneal endothelium and anterior lens capsule. The Tyndall effect, synechiae, cataracts, and glaucoma may also be detected. It seems the underlying cause of pigmentary uveitis may be immune mediated.
- Intraocular tumours.
- Blunt or penetrating traumas: it is important to pay special attention to blunt force traumas as the damage may initially appear less severe than it really is (Fig. 11).
- Uveitis secondary to substances applied to the ocular surface, such as pilocarpine, carbachol, or prostaglandins.
- Radiotherapy is another possible cause of uveitis.

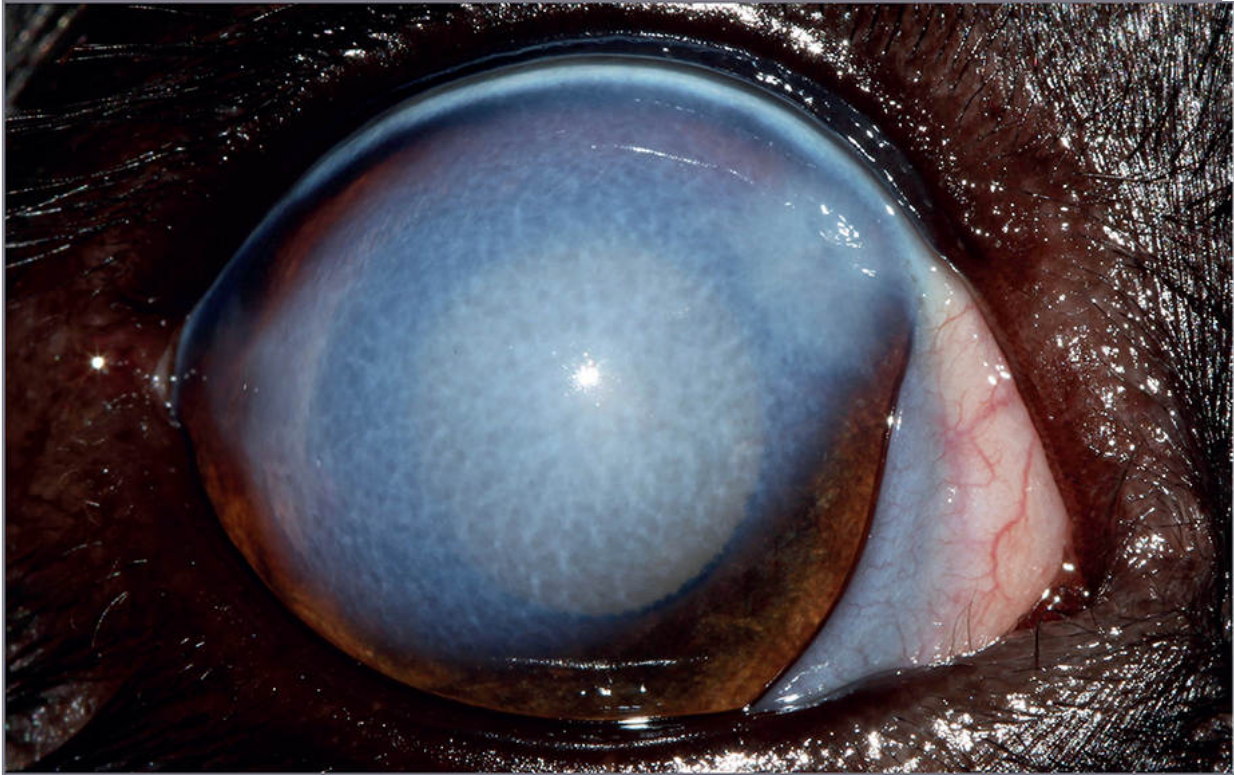


Figure 11. Traumatic uveitis secondary to a gunshot. Note the severe stromal oedema and projectile entry wound.

Systemic causes

Similarly, there are various systemic aetiologies:

- Infectious origin: in many cases of infectious uveitis, it is important to remember that the infectious agent is not necessarily located in the eye, rather the uveitis may develop in response to bacterial toxins generated either inside or outside the eye. These infectious agents may include:
 - Bacteria: septicaemia (pyometra, mastitis, pneumonia, etc.), *Brucella canis*, *Bartonella vinsonii* subsp. *berkhoffii*, *Leptospira* spp., and *Borrelia burgdorferi*. These bacteria have an inclination for the uvea.
 - Rickettsias: *Ehrlichia canis*, *Rickettsia rickettsii* or Rocky Mountain spotted fever, and *Anaplasma platys*. It should be noted that these microorganisms have a greater propensity for the posterior segment.
 - Fungi: *Blastomyces* (blastomycosis) *Cryptococcus* (cryptococcosis), *Histoplasma* (histoplasmosis), *Coccidioides* (coccidioidomycosis). All these fungi have a greater tendency to affect the posterior segment.
 - Algae: *Prototheca* (protothecosis). *Prototheca* is a genus of achlorophyllic alga with a round or oval shape and a thin wall that tends

to cause profuse haemorrhagic diarrhoea.

- Viruses: canine adenovirus-1 (CAV-1), the cause of infectious canine hepatitis. Natural infection tends to occur in unvaccinated dogs under 1 year old. The eye will develop severe corneal oedema, which is why this disease was known as “blue eye”, and anterior uveitis, which may be the only clinical signs. The uveitis is nongranulomatous and the pathogenesis is related to a type III hypersensitivity (or immune complex) reaction or Arthus reaction. This type of uveitis may also be due to a postvaccination reaction, particularly following CAV-1 vaccination.
- Protozoa: *Leishmania infantum*, *Toxoplasma gondii*, and *Neospora caninum*. *Leishmania* is a flagellate organism that may course exclusively with ocular signs including blepharitis, periocular alopecia, keratoconjunctivitis sicca, anterior uveitis, hyphema, synechiae, cataracts, chorioretinitis, retinal detachment, orbital cellulitis, extraocular myositis, and granulomatous nodular episcleritis. It has a more severe impact on the anterior segment of the eye and histopathology will reveal vasculitis and areas of granulomatous inflammation.
- Parasites: *Dirofilaria immitis*, *Angiostrongylus vasorum*, *Toxocara canis*, *Diptera* spp.

Uveitis secondary to ehrlichiosis

Ehrlichia canis is an obligate intracellular parasite transmitted to dogs by the tick *Rhipicephalus sanguineus*. Infection causes fever, lymphadenopathy, anaemia, leukopaenia, monoclonal thrombocytopaenia or polyclonal gammopathy, neurological signs, and bleeding. Ocular manifestations are common and may be the only clinical signs. Presentation can be uni- or bilateral and there are both acute and chronic forms of the disease. Ocular bleeding secondary to ehrlichiosis has been associated with thrombocytopaenia, platelet alterations, and/or hyperviscosity. The ocular signs are conjunctivitis, petechiae in the conjunctiva or iris, anterior uveitis, hyphema (Fig. 12), retinal haemorrhage, papilloedema, optic neuritis, and panuveitis. Histopathology may reveal monocyte infiltration.



Figure 12. Hyphema in a Yorkshire Terrier with *Ehrlichia canis* .

Metabolic causes

Some of the most common ones are:

- Hyperlipidaemia: systemic increase in triglycerides and cholesterol associated with rupture of the blood–aqueous barrier can allow lipids to enter the aqueous humour (Fig. 13). It is important to emphasise that under normal conditions the barrier would block the passage of these substances, but when it is compromised, they can pass through.
- Hyperviscosity syndrome: this is due to the development of monoclonal gammopathy associated with lymphoproliferative alterations. It originates clinical signs in various organs and is accompanied by increased IgG, IgM, and IgA levels.
- Systemic hypertension.

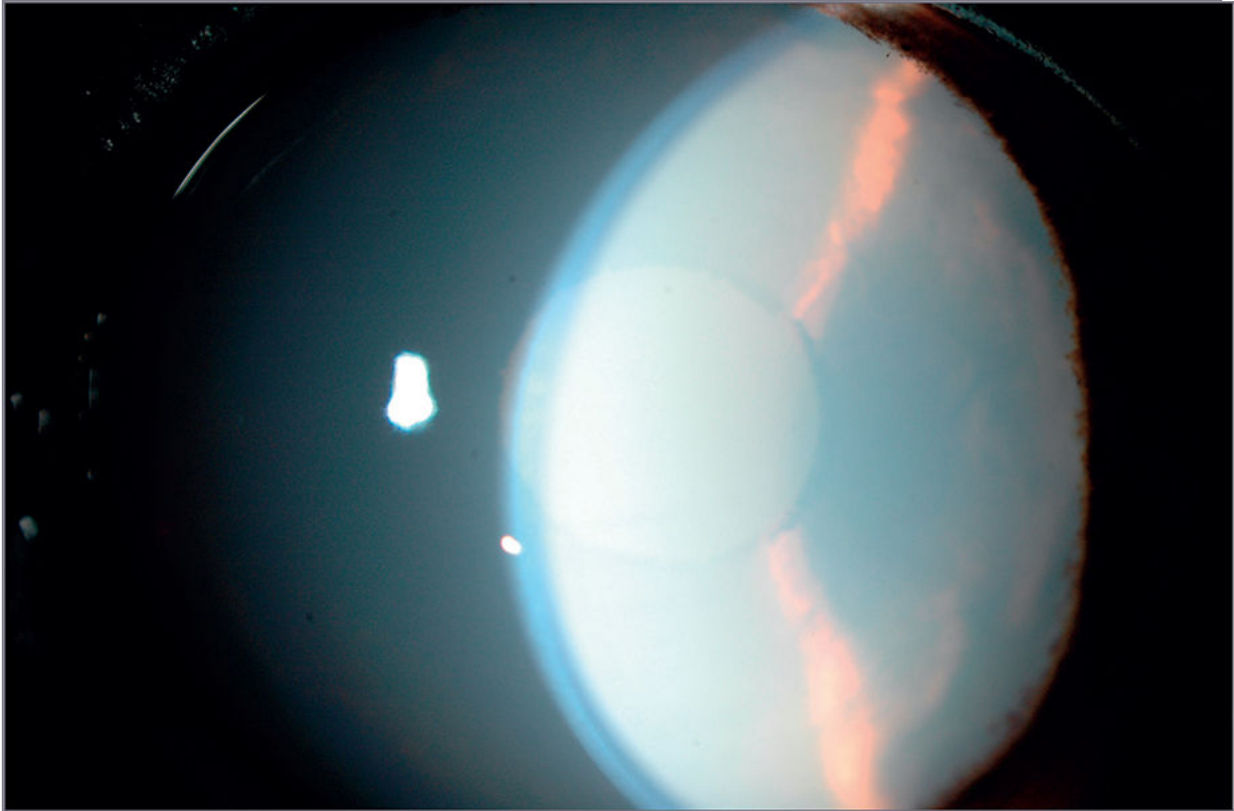


Figure 13. Hyperlipidaemia in the aqueous humour as seen with a slit lamp.



Hypertensive uveitis

Immune-mediated causes

The most relevant immune-mediated processes are:

- Uveodermatologic syndrome (Vogt–Koyanagi–Harada syndrome). This particularly seems to affect Nordic breeds such as Akita, Husky, and Samoyed, although in recent years it has been reported in many other breeds and always in young animals. Patients will present depigmentation of the mucocutaneous junctions, poliosis (white hair), and vitiligo (depigmented skin). This occurs because it is a spontaneous autoimmune disease that targets melanocytes, including those located in the uvea, and

which involves an element of cellular response. Diagnosis is made through a skin biopsy which will reveal the presence of histiocytes, plasma cells, melanophages, and mononuclear cells. Ocular histopathology findings may include granulomatous panuveitis with perivascular lymphoid aggregates and melanophages. The anterior chamber contains lymphocytes and plasma cells. Immunohistochemistry shows that skin alterations are mediated by T cells and macrophages (Th1 immunity), whereas ocular lesions are mediated by B cells and macrophages (Th2 immunity). Glaucoma and blindness are common sequelae.

- Immune-mediated thrombocytopaenia.
- Immune-mediated vasculitis.

Neoplastic causes

Various tumours can contribute to uveitis:

- Lymphoma (Fig. 14).
- Haemangiosarcoma.
- Melanoma.
- Adenocarcinoma.
- Malignant histiocytoma.
- Transmissible venereal tumour.

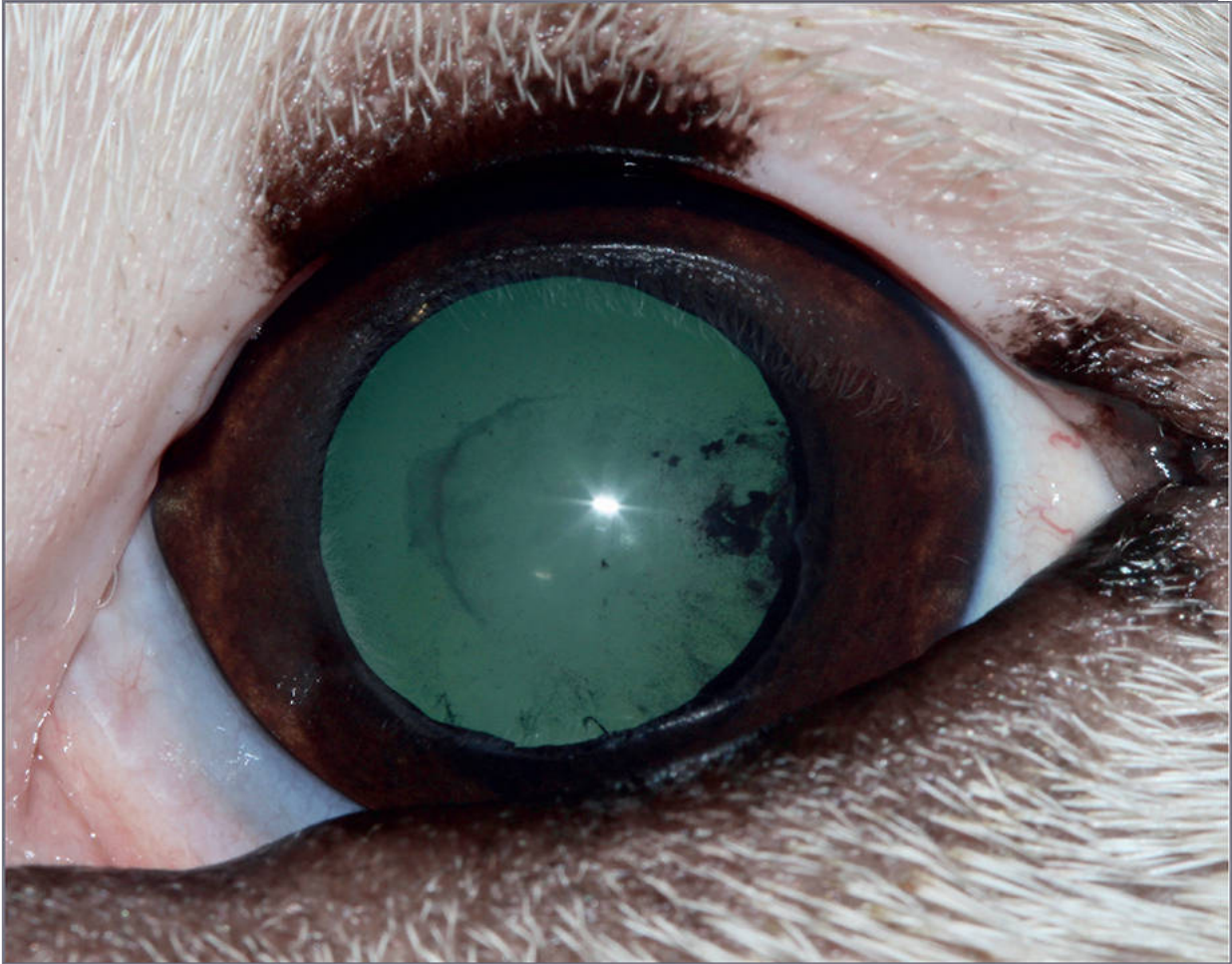


Figure 14. Uveitis in a dog with a lymphoma.

Treatment of uveitis

Controlling the inflammation is important in the treatment of uveitis, but the associated pain must also be addressed. The pain is caused by ciliary muscle spasm, so treating the spasm will help patients feel more comfortable and minimise any complications, such as glaucoma or synechiae, which improves the prognosis in terms of preserving vision. Uveitis should be managed with topical and systemic treatments.

Topical treatments

The following should be administered locally:

- **Corticosteroids:** these have traditionally been used to manage ocular inflammation. Their effect largely depends on their ability to cross the

corneal epithelial barrier (lipophilic). Acetate and alcohol salts of corticosteroids penetrate the barrier better than phosphate salts. Corticosteroids have an array of undesirable side effects, for instance they can delay corneal healing, promote collagenolysis, and reduce local immunity, which increases the risk of viral, bacterial, and fungal infections.

- **Nonsteroidal anti-inflammatory drugs (NSAIDs):** there is a growing arsenal of clinically very useful topical ophthalmic formulations, particularly with respect to their penetration. NSAIDs produce fewer side effects than corticosteroids, although they can also delay healing and have been linked to collagenolysis by some authors. NSAIDs and corticosteroids can be coadministered, but always with caution.
- **Cycloplegic agents:** as mentioned earlier, relaxation of the ciliary muscle helps soothe eyes affected by uveitis. This relaxation may stabilise the blood–ocular barrier, but it will never have the same reach as anti-inflammatories. Their mydriatic effect can also reduce the formation of synechiae, which is helpful in preventing secondary glaucoma, above all when using short-duration mydriatic agents (tropicamide or phenylephrine). Atropine and cyclopentolate have a very marked cycloplegic effect, but also of long-duration, so the pupil will remain immobile for a considerable period.

All mydriatics, apart from phenylephrine, are contraindicated in cases of hypertensive uveitis.

- **Antiglaucoma preparations:** these should be used in animals with hypertensive uveitis. Those prescribed most often are carbonic anhydrase inhibitors, whereas prostaglandin analogues and mannitol are contraindicated for uveitis.

Systemic treatments

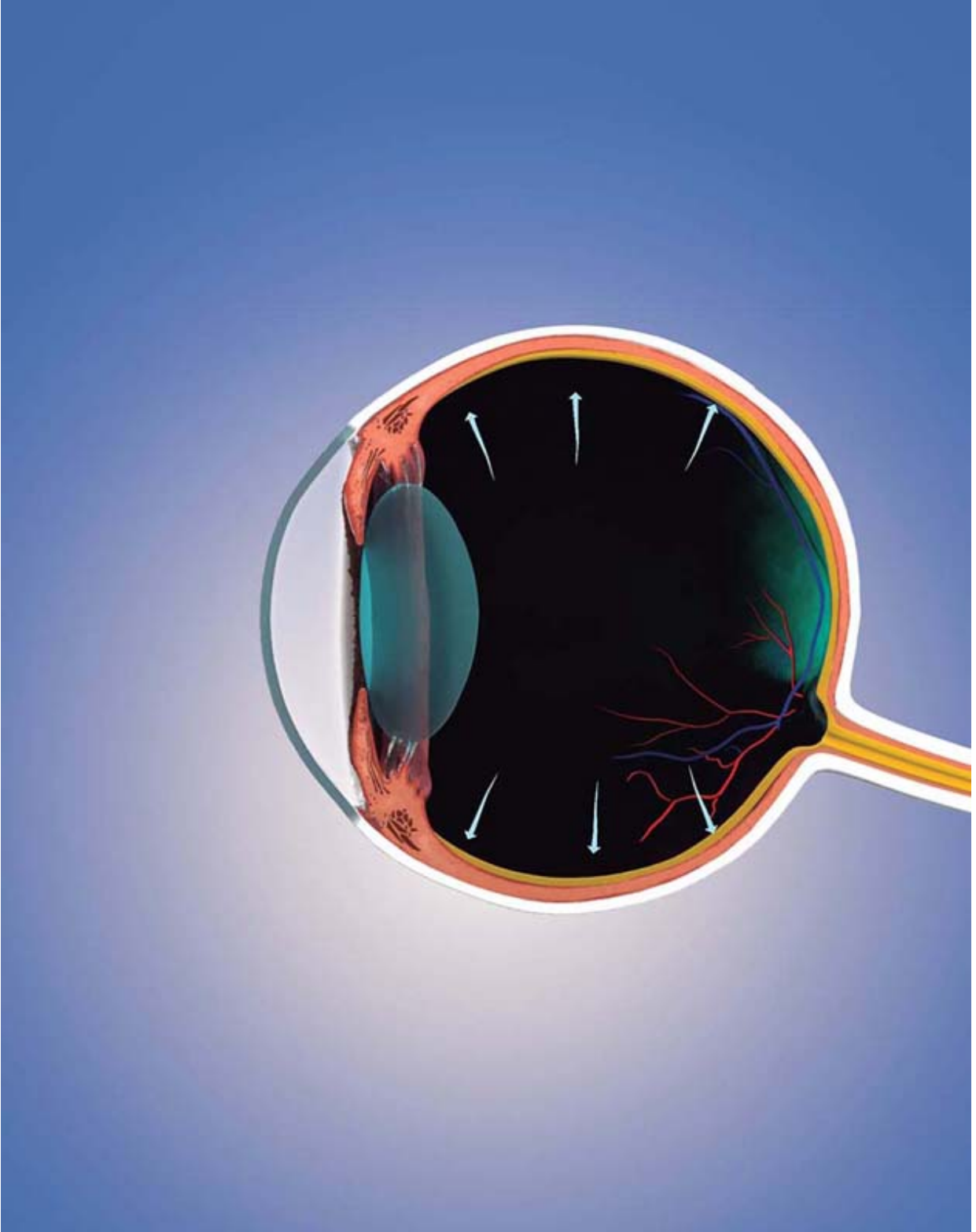
The following can be prescribed:

- **Anti-inflammatories:** corticosteroids and NSAIDs can be used, although as a general rule corticosteroids are more effective at controlling inflammation.

- **Immunosuppressants:** only administered for cases of autoimmune uveitis, but always bear in mind that many of them have significant contraindications, such as myelosuppression.
- **Aetiological treatments:** these are essential and should target the primary cause of the uveitis.

07

Glaucoma



Aetiopathogenesis of glaucoma

In veterinary practice glaucoma courses with a pathological increase in intraocular pressure (IOP) and has severe consequences for each of the eyeball's structures, but above all it impacts on the optic nerve. This increase in IOP may be due to several factors:

- Changes in the iridocorneal angle, whether in the anterior (pectinate ligament) or posterior portion (ciliary fissure).
- Any obstacles to the free circulation of the aqueous humour, such as crystalline lens luxation and posterior or anterior synechiae.
- Changes to the composition of the aqueous humour, which becomes harder to filter if the viscosity or cellularity increase. This occurs in the case of uveitis, during which there may be increases in the number of proteins, inflammatory cells, or haemorrhages. Ocular tumours that release cells may also alter its composition.
- The mass effect in primary or secondary intraocular tumours.

Figure 1 shows the structures involved in the production and drainage of aqueous humour; the impaired function of any of these components can have repercussions for the IOP.

In human medicine, glaucoma is considered an optic neuropathy that can occur even without an increase in the IOP. In veterinary medicine, however, all glaucomas are accompanied by an elevated IOP irrespective of whether any changes can be detected using electrophysiological tests. By contrast, sometimes the results of the tests are suggestive of glaucoma even before the pressure increases (Fig. 2), a point which has been confirmed for primary glaucoma in experimental animals. This is why an elevated IOP is considered a risk factor for canine glaucoma.

Besides increased IOP, there are other risk factors including age, sex, race, family history, and the presence of goniodysgenesis, which is a developmental abnormality in the aqueous humour drainage pathways.

Age is a significant risk factor. Over the course of a lifetime, the eye undergoes anatomical changes that affect the normal flow of aqueous humour as it circulates towards its drainage points. The scaling of pigmented cells from the posterior epithelium of the iris, pigment dispersion in the iridocorneal angle, phagocytosis of the iridocorneal angle due to the macrophages in the trabecular meshwork, accumulation of collagen at the

base of the ciliary muscle, and so on, will all result in increased resistance to drainage and therefore contribute to an elevated IOP.

With regard to sex as a predisposing factor, it is worth noting that primary angle-closure glaucoma affects twice as many females as males. The smaller aperture of the iridocorneal angle and greater predisposition to autoimmune diseases could be two factors that explain this difference.

The influence of family history, bearing in mind that glaucoma is associated with certain genetic mutations and has been strongly linked to goniodysgenesis, is really quite evident.

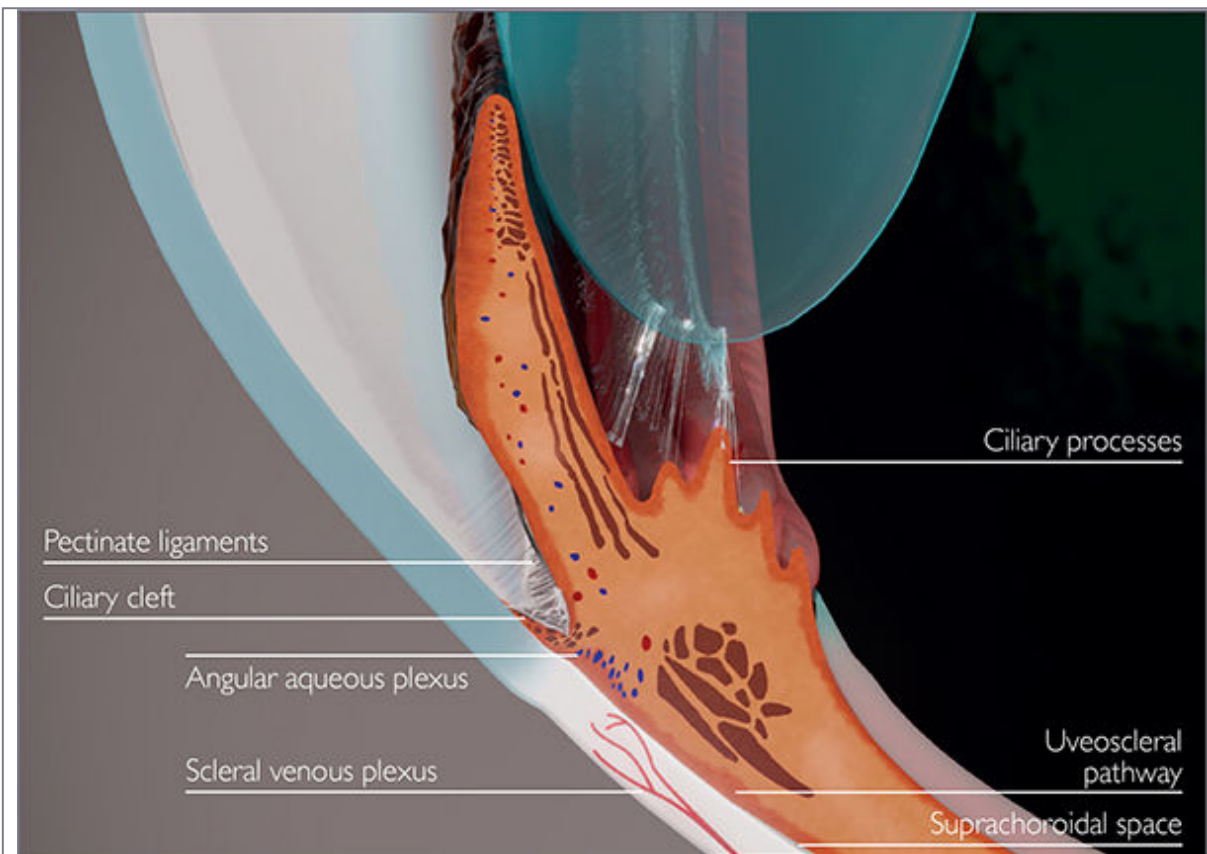


Figure 1. Structures involved in the production and drainage of aqueous humour.



Figure 2. Measuring intraocular pressure using an applanation tonometer.

Goniodysgenesis

Goniodysgenesis is an abnormality of the pectinate ligament characterised by the formation of sheets of tissue which, depending on their width, can obstruct the passage of aqueous humour flowing into the ciliary cleft. Patients with over 75 % involvement have a high risk of developing glaucoma. The severity of goniodysgenesis, which by itself is not enough to occasion glaucoma, increases with age.





Formation and drainage of aqueous humour

Classification of glaucoma

Through a simple classification we can delineate three main types of glaucoma, namely congenital, primary, and secondary glaucoma.

Congenital glaucoma

This starts to develop from birth and possibly even in the animal's first few weeks or months of life, and it might affect just one eye. It stems from a severe alteration during the embryonic development of the eye or because of a genetic mutation.

An important point to take into account for congenital glaucoma is that puppies suffering from a significant increase in the size of their eyeball may still maintain their vision for a while. This is because a puppy's sclera still conserves some elasticity, which means it can bear the pressure increase without transferring the effects to the optic nerve head.

Primary glaucoma

Primary glaucoma occurs spontaneously due to alterations in the mechanisms controlling the circulation of aqueous humour. As such, these processes are devoid of any preceding trauma, tumours, lens luxation, or haemorrhages which might induce the ocular hypertension.

Focusing on the appearance of the iridocorneal angle, this type of glaucoma can be divided into two subclasses:

- 1. Primary open-angle glaucoma:** this is one of the most frequent presentations in human medicine, but the rarest in the case of canine patients. It has been reported in a colony of Beagles and, more recently, in Basset Hounds, Basset Fauve de Bretagne, and Shar Pei. In the last three breeds, the gene responsible for primary lens luxation (*ADAMTS 17*) also appears to play a part in the glaucoma process. The angle will look normal until the disease progresses to an advanced stage, at which point alterations will become evident.

2. Primary angle-closure glaucoma: this subtype has the highest incidence and two of the most affected breeds are Basset Hounds and Cocker Spaniels. The unaffected eye should be used to gather data about the gonioscopic appearance of its angle, as the equivalent structure in the compromised eye will have suffered a significant anatomical change due to the constant hypertension. Additionally, in many cases the presence of corneal oedema prevents a clear view inside the eye. During the gonioscopic examination of the healthy eye, the clinician should consider the distance between the pigmented scleral line and the base of the peripheral iris, while also examining the entire circumference of the pectinate ligament (Figs. 3 and 4).

Another important factor to be assessed through the gonioscopy is any variation in the fibres forming the pectinate ligament (Fig. 5) with respect to their thickness, the degree to which they are joined together, and their insertion in the ligament. The alterations progressively evolve from grade 1 to grade 5 (Table 1), which translates into a gradual reduction in the free space between the fibres and in turn a growing impediment to the passage of aqueous humour.

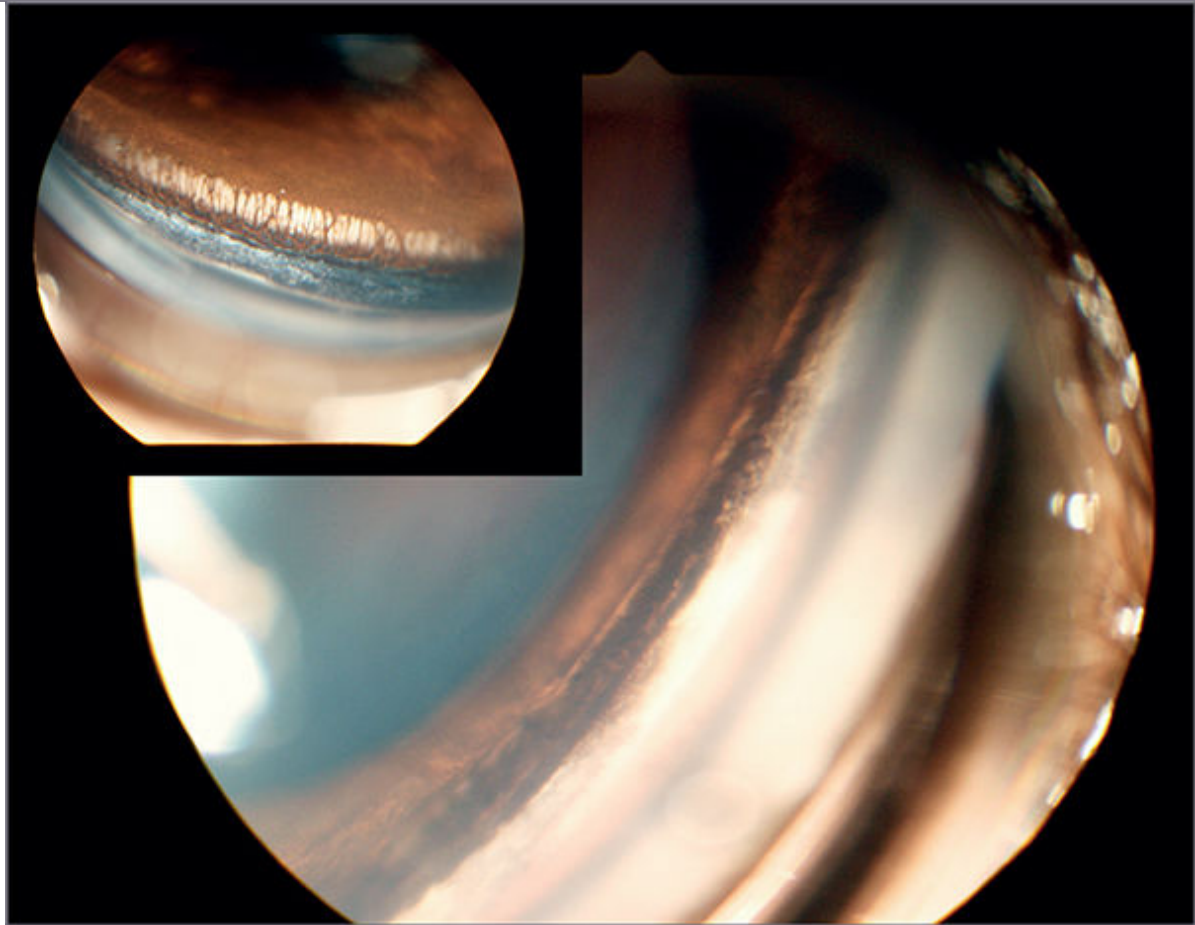


Figure 3. Normal appearance of the pectinate ligaments (inset) and an iridocorneal angle affected by severe goniodysgenesis.

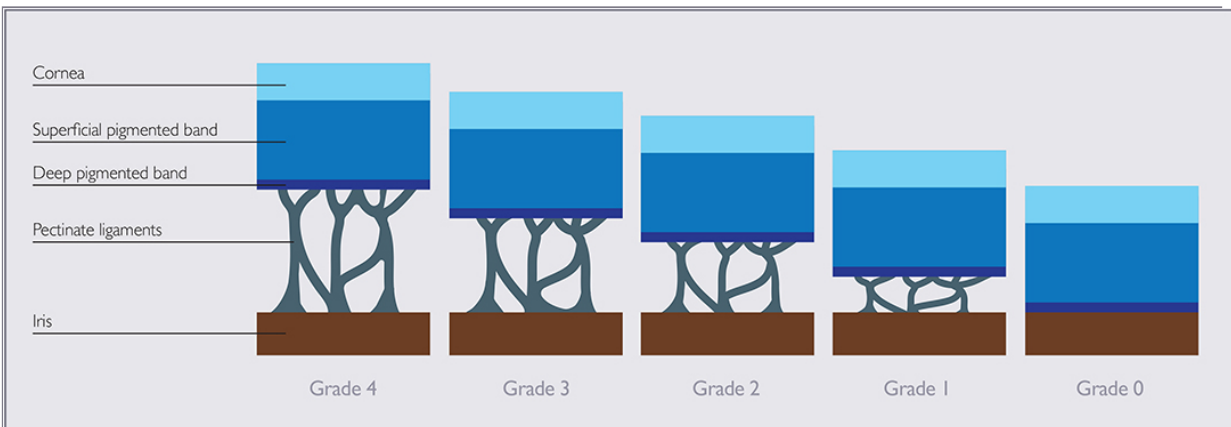


Figure 4. Shaffer iridocorneal angle grading scale modified by Ekesten. From Grade 4 (wide open) to Grade 0 (closed).

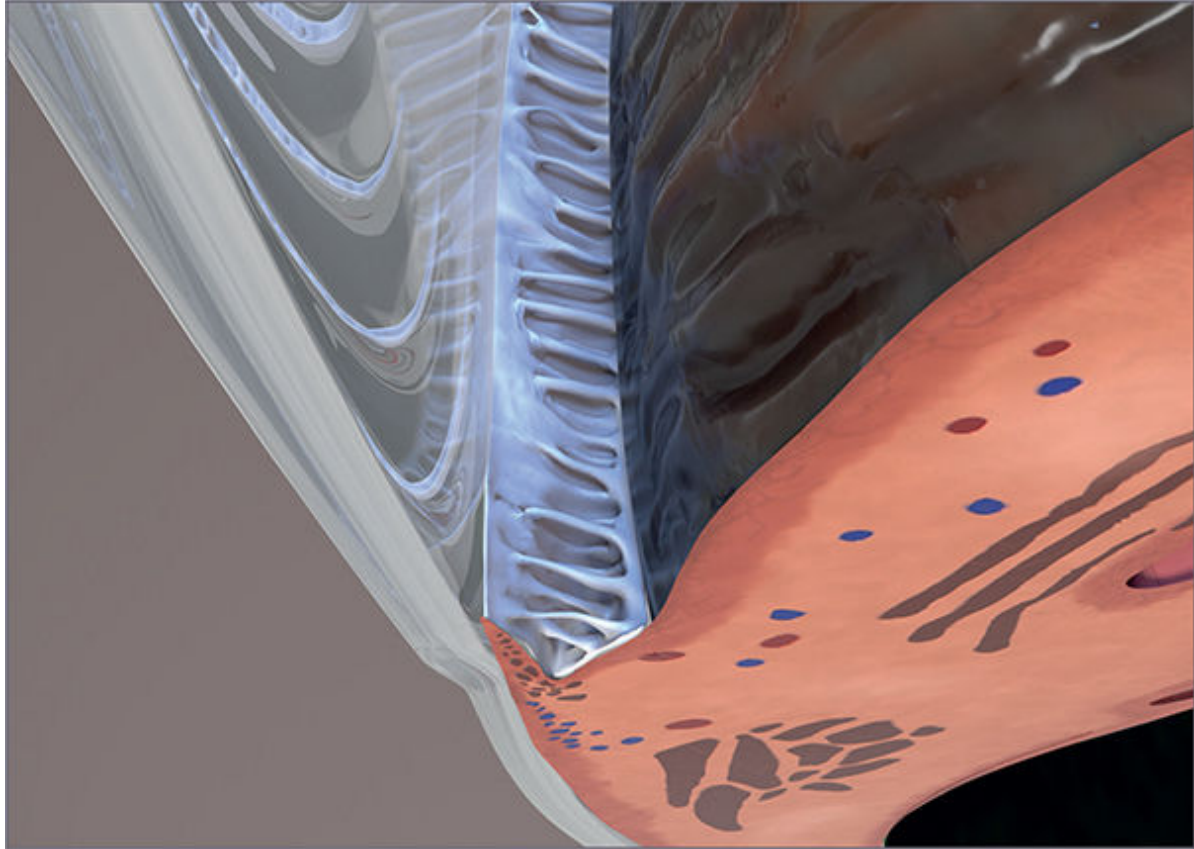


Figure 5. Pectinate ligaments at the iridocorneal angle.

TABLE 1. Grades of pectinate ligament dysplasia.	
Grade	Description
1 (normal)	Individual strands of the pectinate ligament with a single insertion on the corneal side. Two to four strands may emerge from the same point on the iris. There may be some connections between adjacent strands. The thickness of the strands may vary slightly from one dog to another.
2	Four to 10 strands of pectinate ligament merge to form thick strands.
3	Ten or more strands of ligament combine to create small sheets of tissue. The iridocorneal angle is narrow in approximately 30 % of normal eyes.
4	Broad, solid sheets of iris tissue with or without flow holes.
5	Broad sheets with very short strands on the corneal side. There are no normal pectinate ligaments.

Secondary glaucoma

In this case the increase in IOP is due to obstruction of the drainage of aqueous humour, which may be blocked in the pupil or the structures of the

iridocorneal angle.

Some of the most common causes of secondary glaucoma in dogs include:

- Uveitis (Fig. 6).
- Crystalline lens luxation (Figs. 7 and 8).
- Cataracts.
- Intraocular surgery.
- Retinal detachment.
- Primary or secondary intraocular tumours.

Only 20 % of cases of secondary glaucoma are bilateral and, logically, a thorough general physical examination is crucial to detect any systemic causes that might be associated with the ocular disorder.

Uveitis is the leading cause of secondary glaucoma and accounts for 45 % of cases.

Up to 25 % of dogs with a cataract go on to develop glaucoma. This complication may be due to various underlying mechanisms ranging from phacolytic uveitis to phacomorphic glaucoma. Eyes with a cataract, particularly intumescent cataracts, have a shallow anterior chamber, which means the iridocorneal angle narrows and obstructs the passage of the aqueous humour flowing through it.

Cataract surgery can also provoke secondary glaucoma, reported as a complication in 5–15 % of patients operated on for a cataract.

Retinal detachment is another potential cause of secondary glaucoma. The release of photoreceptor outer segments, which eventually settle in the angle, can lead to obstruction.

Similarly, secondary glaucoma is a potential complication of vitreoretinal surgery due to the migration of silicone oil into the anterior chamber.



Figure 6. Glaucoma secondary to chronic uveitis in the left eye.

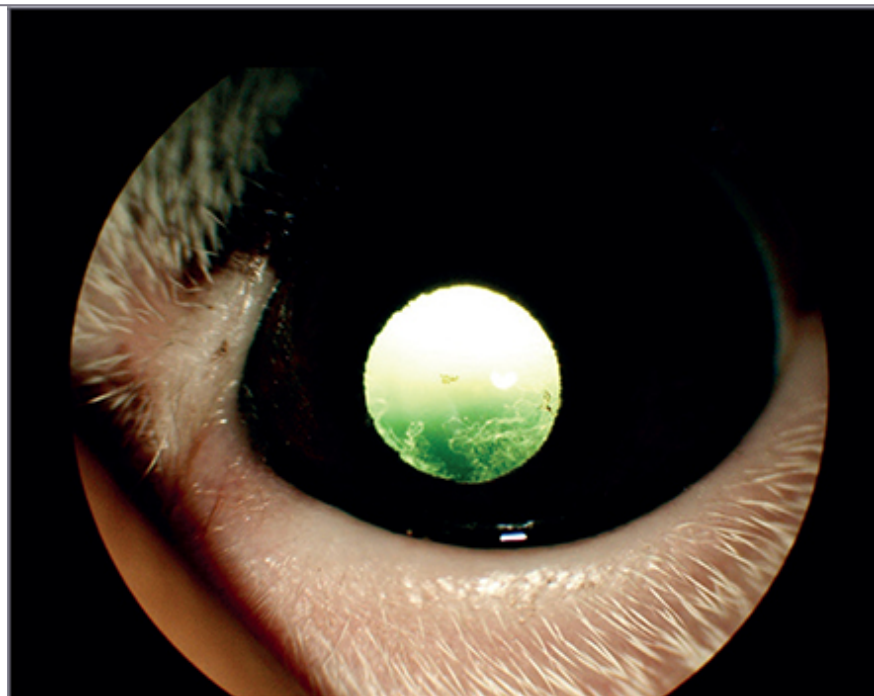


Figure 7. Vitreous herniation and crystalline lens instability in an eye with glaucoma.

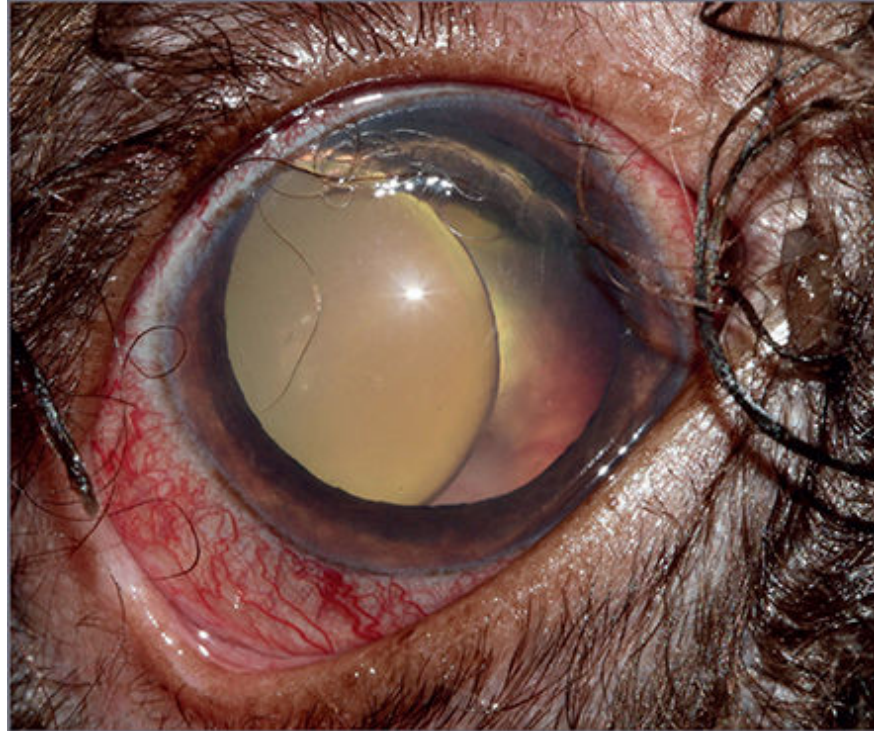


Figure 8. Glaucoma secondary to crystalline lens luxation.



Primary and secondary glaucoma

Pathophysiological mechanisms of glaucoma

As glaucoma progresses, it precipitates various phenomena, some of which are connected to the increase in IOP.

Ganglion cells in the scleral cribriform plate suffer progressive cell death. Neurotransmitters, such as glutamate, are released and these favour the degeneration.

The death of retinal ganglion cells is one of the most significant consequences. An elevated intraocular pressure interrupts axonal transport and an IOP of 30–35 mmHg is estimated to inhibit as much as 80 % of the flow.

In addition to the above, the hypertension will cause vascular alterations in both retinal and choroidal vessels.

Diagnosis of glaucoma

The clinical signs observed in animals with glaucoma include (Fig. 9):

- Blepharospasm.
- Epiphora (tearing).
- Conjunctival congestion.
- Engorgement of the episcleral vessels.
- Corneal oedema.
- Mydriasis.
- Papilloedema at the start of the process.
- Cupping or “hollowing out” of the optic nerve head as a direct result of the pressure and apoptosis.

The vet needs to determine whether they are dealing with primary or secondary glaucoma. Only then can they plan the appropriate treatment and make a prognosis in line with the process. In secondary glaucoma, it is vital to treat the underlying cause.

A comprehensive eye exam should always include gonioscopy in the healthy eye to rule out the presence of any anomalies in the structures comprising the iridocorneal angle.

Ultrabiomicroscopy (UBM) is an ultrasound technique that is becoming increasingly relevant in veterinary ophthalmology. The procedure is usually performed under general anaesthesia or sedation and permits a detailed examination of the anatomy of the iridocorneal angle. It can also be used to study the appearance of the ciliary cleft and thus obtain an idea about the aperture in the trabecular meshwork.

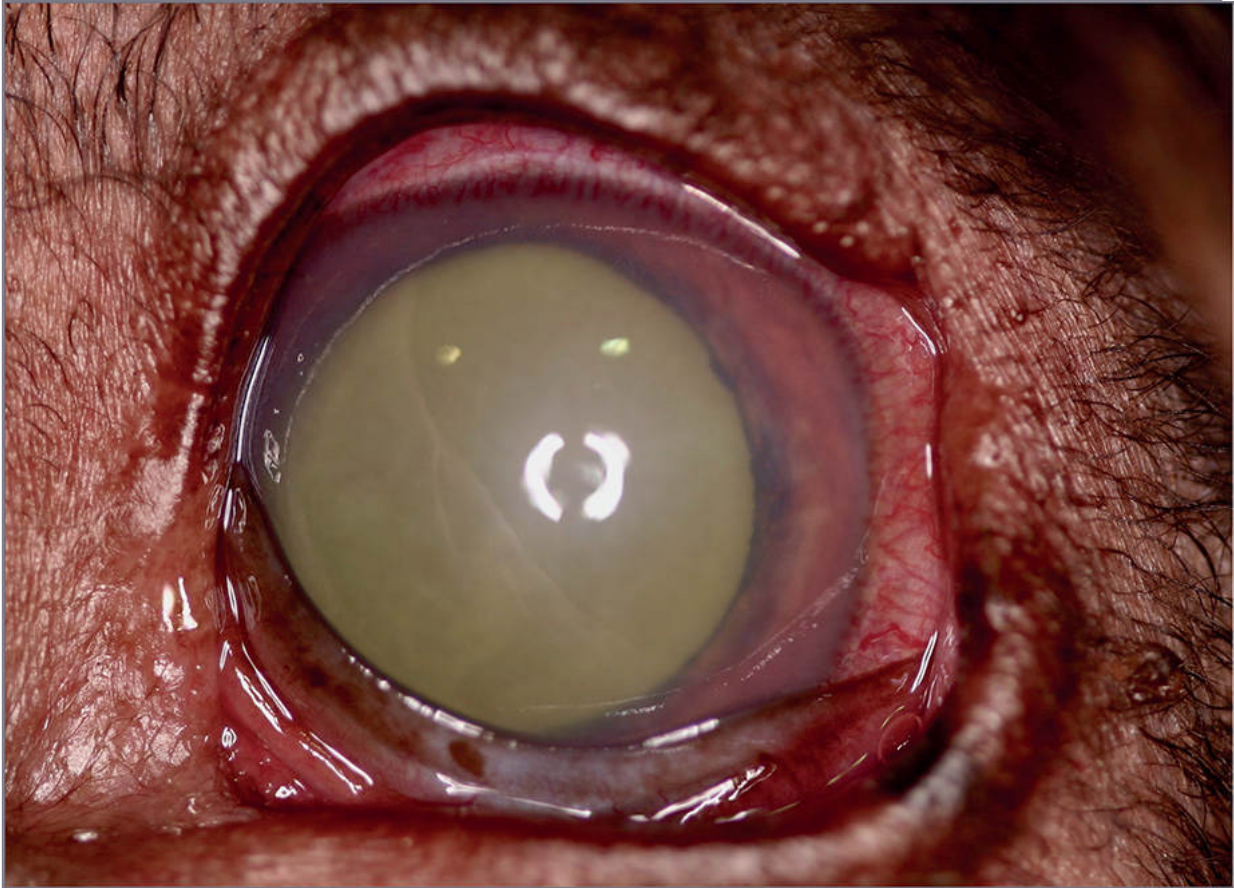


Figure 9. Primary glaucoma crisis accompanied by severe conjunctival congestion, corneal oedema, and mydriasis.

Treatment of glaucoma

Medical treatment

The medical treatment of glaucoma is far from simple and requires sound knowledge of the physiology of the aqueous humour's circulation in order to prescribe the correct drugs from the different pharmacological groups available:

- **Cholinergic agents:** for instance, carbachol, pilocarpine, and demecarium bromide. Cholinergics cause miosis (contraction of the ciliary muscle) and temporary disruption of the blood–aqueous barrier. Because of its acidic pH, pilocarpine causes intense discomfort when instilled; this, together with the fact that its main indication is open-angle glaucoma in human medicine, makes it impractical for clinical use in animals. Demecarium bromide is only available under a master formulation record in Europe.

- **Sympathomimetics:** phenylephrine, whose mechanism of action remains unclear, is hardly used in the treatment of glaucoma. It is thought to reduce the production of aqueous humour and increase its normal drainage. Its use is almost confined to the treatment of hypertensive uveitis because it induces mydriasis and reduces the IOP. Apraclonidine, another drug in this group, is an α_2 agonist with a moderate impact on the intraocular pressure.
- **Beta-blockers:** widely used in human medicine. Their ability to reduce the production of aqueous humour in dogs remains a point of contention, yet they are still used on a regular basis, either alone or in combination with carbonic anhydrase inhibitors or prostaglandins. Following the frequent use of beta-blockers in small animals, their systemic absorption can lead to bradycardia, hypotension, bronchospasm, and dyspnoea, hence they are contraindicated in patients with severe cardiac or pulmonary diseases, such as asthma, or cardiorespiratory failure.
- **Carbonic anhydrase inhibitors (CAIs):** CAIs are routinely used in both human and veterinary medicine. They inhibit the enzyme responsible for the transfer of bicarbonate into the aqueous humour, which reduces the production of humour. They can be administered in both systemic (acetazolamide, methazolamide) and topical formulations (dorzolamide, brinzolamide). No differences have been reported between the topical and systemic effect, hence topical use is preferred as CAIs can cause metabolic acidosis. They reduce the production of aqueous humour very effectively, while the main advantage of brinzolamide over dorzolamide is that its pH is closer to a physiological value and so it is better tolerated locally. Carbonic anhydrase inhibitors can produce keratitis and blepharitis after long-term administration.
- **Prostaglandins:** this group has acquired an increasingly important role in the treatment of both human and veterinary glaucoma. They manage to increase drainage via both the conventional and alternative outflow pathways. They should be administered twice a day. Prostaglandins cause marked miosis and with time may darken the colour of the iris. As they affect the blood–aqueous barrier, prostaglandins should be used with caution in dogs with secondary glaucoma.

Surgical treatment

Primary glaucoma is considered to require combined medical/surgical treatment, as even when there is a good response to the initial

pharmacological treatment, in all likelihood the animal will still need surgery in the short-to-medium term.

A key consideration before reaching a surgical decision is to categorise patients into one of two groups:

- Animals with eyes that still retain vision but are unresponsive to medication or who have had terminal glaucoma in the contralateral eye: the aim is to preserve vision in the operated eye.
- Animals with terminal glaucoma, blind eyes, and a high IOP over prolonged periods: the aim of surgery is to alleviate the associated pain.

The surgical interventions carried out to preserve the vision of seeing eyes include gonioimplants, laser endocyclophotocoagulation, and transscleral laser cyclophotocoagulation (Figs. 10 , 11 , and 12).

If treatment is necessary to relieve pain, we recommend enucleation and, to a lesser extent, evisceration and placement of an intrascleral glaucoma implant. There is growing debate concerning the use of evisceration, since it is only performed for aesthetic reasons, even though it increases the patient's chances of developing dry eye and corneal ulcers due to the decline in corneal sensitivity.

In dogs with terminal glaucoma who are not ideal candidates for general anaesthesia, cyclodestructive therapy is recommended with an intravitreal injection of gentamicin.



Figure 10. An endolaser unit.

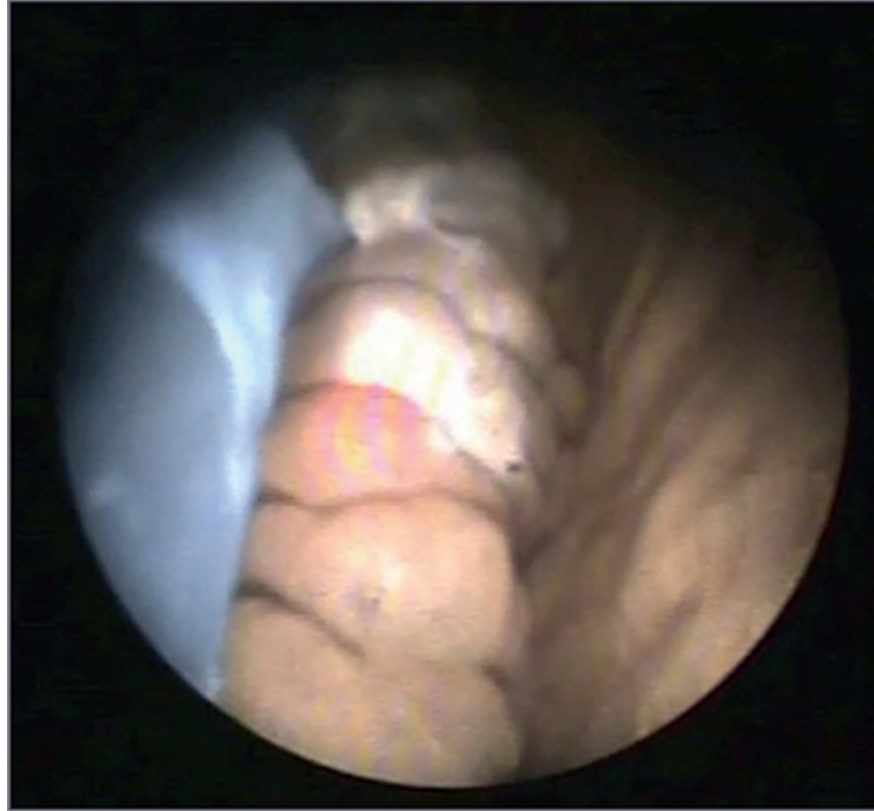


Figure 11. Intraoperative image of an endocyclophotocoagulation laser procedure.

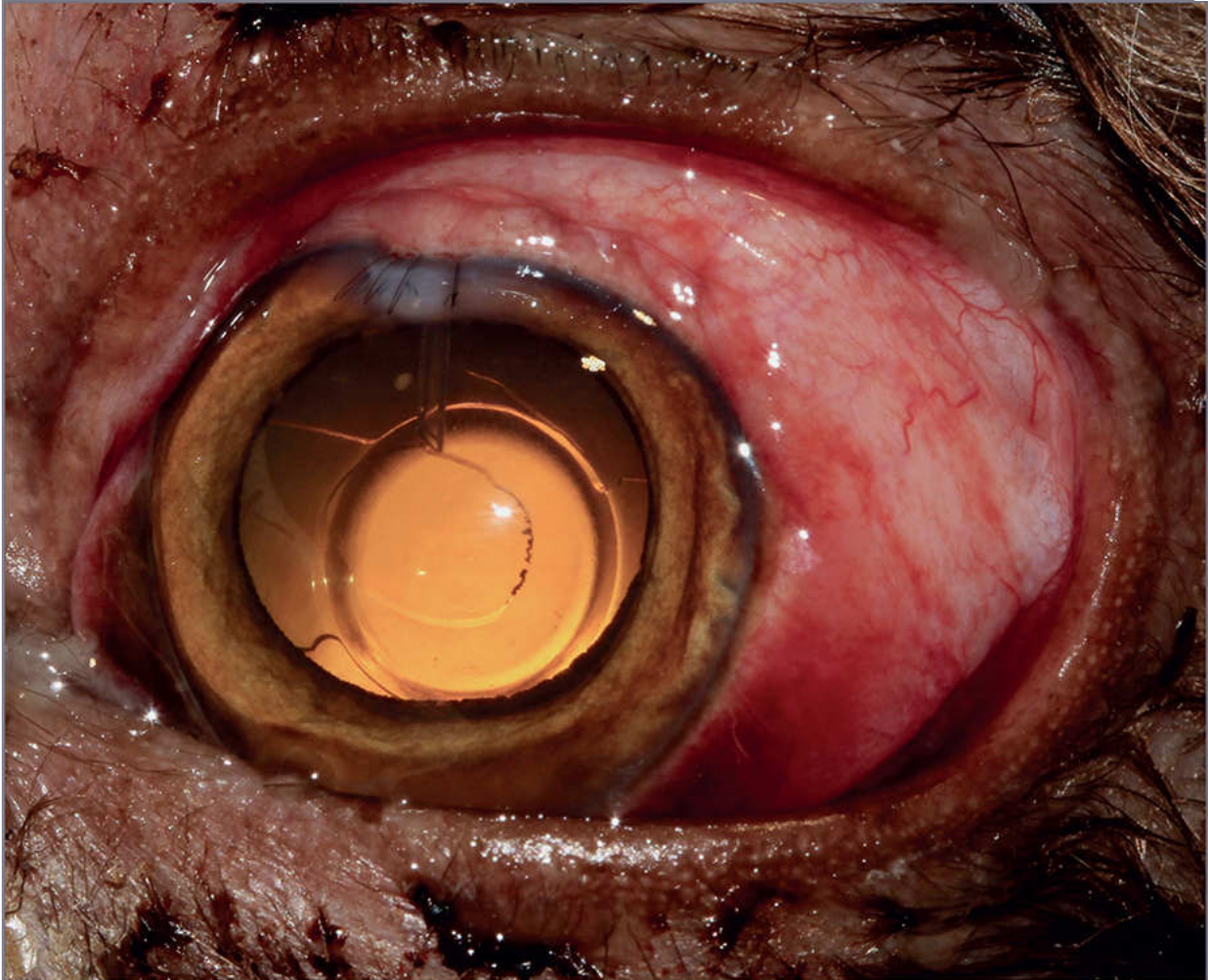
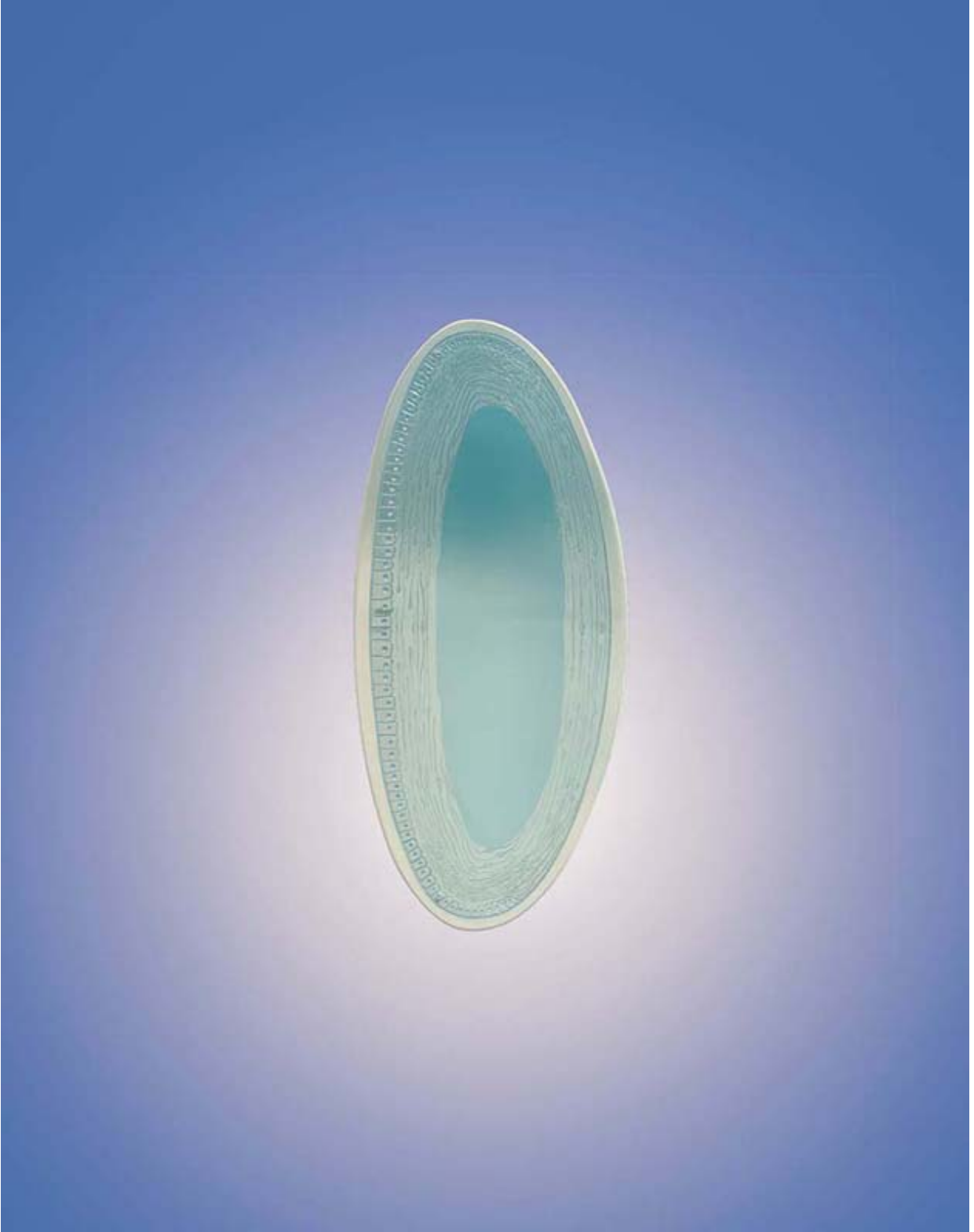


Figure 12. Patient operated on for primary glaucoma, note the intraocular lens and the tube of the implanted shunt.

08

The crystalline lens



Introduction

The crystalline lens is a biconvex lens responsible for focusing objects. It lies behind the iris where it is anchored by zonular fibres, forming a connection to the ciliary processes, and seated in the patellar fossa of the vitreous body.

There are a few anatomical details worth noting; namely, the anterior curvature of the lens is flatter than the posterior one, the lens' circumference is called the equator, the zonular fibres are inserted at the equator, and when these fibres are tensioned by the ciliary muscle through the ciliary processes, they accommodate the crystalline lens and alter its refractive power.

As for its histology, the architecture of the lens is designed to provide a unique transparency and is free from blood vessels and nerve fibres (Fig. 1).

The anterior capsule is thicker than the posterior capsule, and the lens epithelium is located on the internal aspect. The epithelium spreads towards the equator from which point emerge the fibres that comprise the lens parenchyma. The crystalline lens fibres are organised in two well-defined regions: the cortex and nucleus. As the animal grows, new layers of fibre develop from the equatorial regions and spread across the existing fibres, this causes "layering" which is clearly visible in elderly animals.

With respect to the metabolism of the crystalline lens, it is important to remember that it is avascular and therefore it must receive all its nutrition directly from the substances flowing through the aqueous and vitreous humours. Consequently, any variations in the composition of the ocular humours will have a direct influence on the metabolism and in turn the transparency of the crystalline lens. For example, patients with diabetes may have high glucose levels in the aqueous humour, which leads to the formation of sorbitol in the crystalline lens and eventually causes cataracts.

Another significant point is that the lens contains proteins which are not recognised by the eye's immune system, hence the impermeability of the capsule prevents them from coming into contact with the exterior. During the development of cataracts, these proteins start to migrate outside the lens envelope and trigger an inflammatory process called lens-induced uveitis. Similarly, the traumatic rupture of the lens capsule will release the same proteins, giving rise to phacoclastic uveitis.

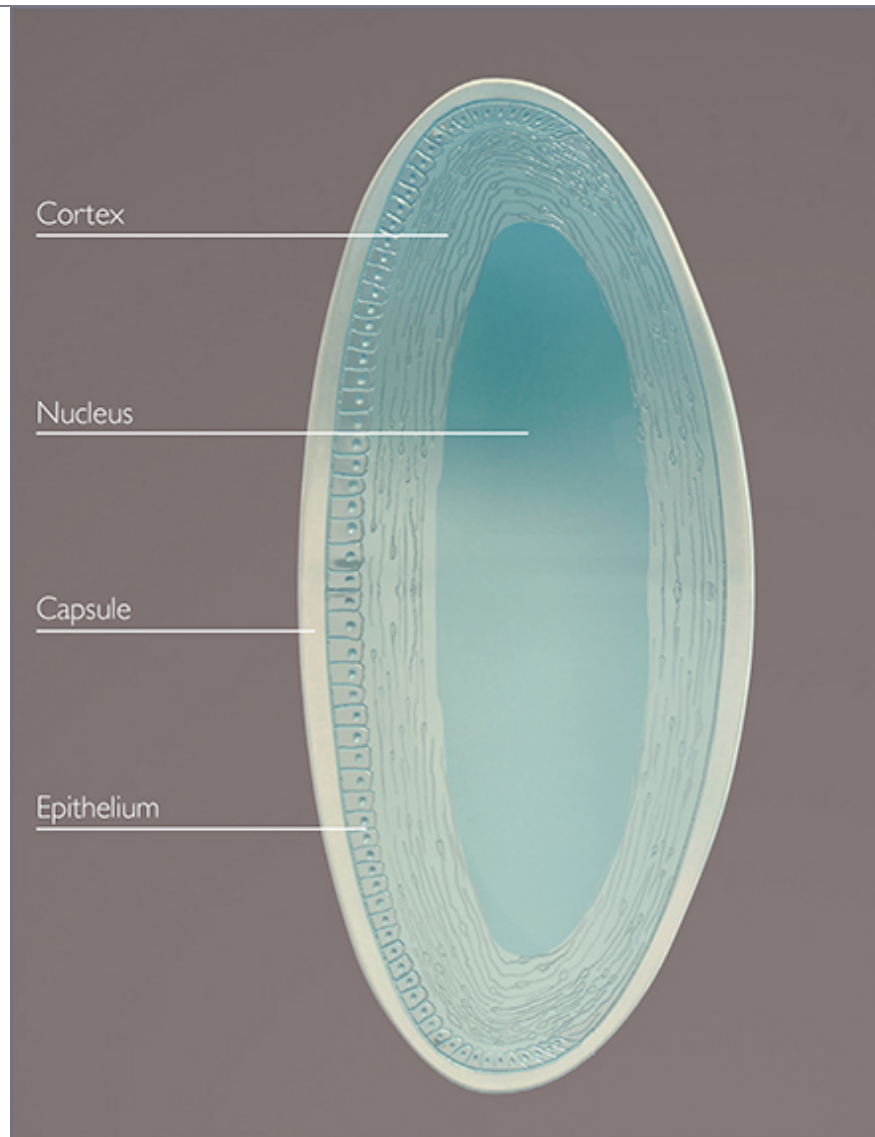


Figure 1. Histology of the crystalline lens.

Examination of the crystalline lens and signs of disease

The lens is visible through the pupil, which is why equipment fitted with an adjustable light source and magnification, such as a direct ophthalmoscope or slit lamp, are ideal for the examination of this intraocular structure.

As the lens is transparent and located behind the iris, a mydriatic agent should be instilled in the eye to allow a complete exploration of the lens.

It is important to bear in mind that the iris, particularly in conditions of more or less intense miosis, “embraces” the anterior lens capsule, which is why it is normally slightly convex in the areas closest to the pupil. Therefore, in diseases in which the lens shifts out of its normal position, such as with luxations, the iris loses its convexity and takes on a flatter aspect than usual, thus increasing the depth of the anterior chamber. In a stage before luxation occurs, when some of the zonular fibres have broken, the lens and iris vibrate with the eye’s normal movements in phenomena called, respectively, phacodonesis and iridodonesis. As such, both are signs of crystalline lens instability.

If the lens displaces anteriorly, that is, through the pupil, then the pupil will not react to light as it is trapped behind the lens. In these cases, the animal often shows signs of pain in the affected eye triggered by the lens brushing against the corneal endothelium. This abrasion alters endothelial functionality, and an oedema will develop in the affected area, which is usually the centre of the cornea.

Another common alteration associated with crystalline lens compromise is that of a colour change or, more precisely, a loss of transparency. These cases translate into a pupil of varying opacity, generally of a grey to white colour, which is responsible for the corresponding visual deficit or even complete blindness.

Diseases of the crystalline lens

Congenital abnormalities

The range of congenital defects includes a total absence of the crystalline lens (aphakia), animals with a smaller lens than usual (microphakia) (Fig. 2), or the absence of its lenticular shape because it is abnormally spherical (spherophakia).

Examination may also reveal anomalies in the poles of the crystalline lens, called lenticonus, and a lack of a regular equator which is also missing some tissue, called a coloboma.

All of these will be discussed in the chapter on congenital diseases.

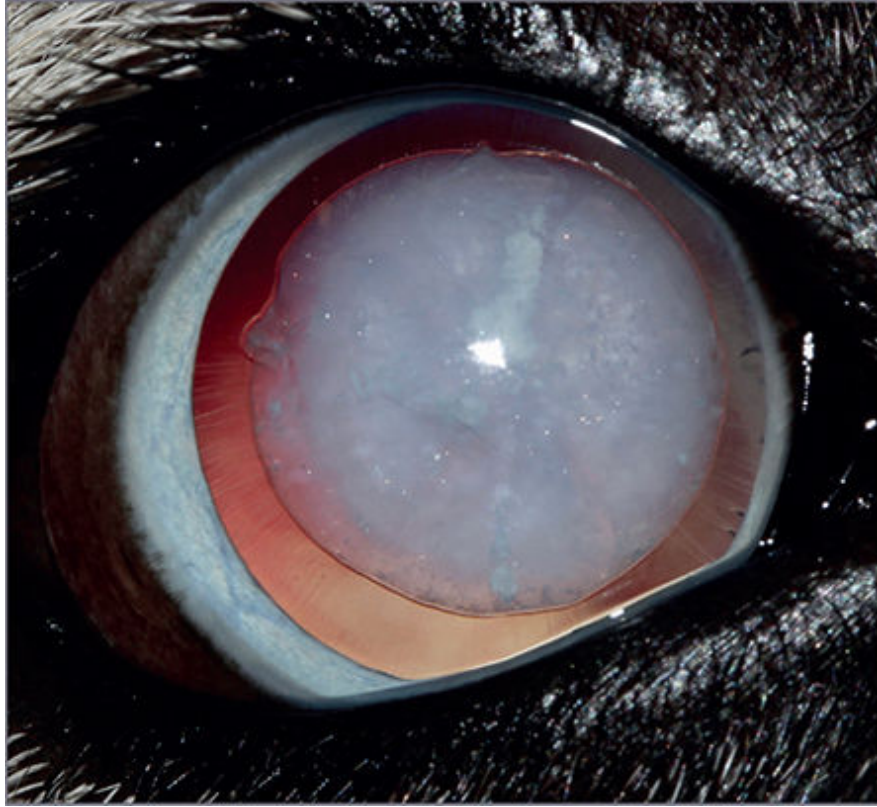


Figure 2. Microphakia and cataract in a Siberian Husky. The zonular fibres are visible around the entire circumference and so are some of the ciliary processes.

Cataracts

A cataract is the presence of opacity that affects the crystalline lens capsule or its stroma. The consequences of developing a cataract tend to be severe and irreversible, to such an extent that it is one of the primary causes of human blindness worldwide.

Although the definition of cataract appears simple enough, there are a lot of types with very different appearances and origins, so they can be classified according to different parameters.

Based on the **age of presentation** :

- Congenital (Fig. 3).
- Acquired.
- Senile.

Based on the **aetiology** :

- Primary cataracts: these occur spontaneously, for example hereditary cataracts.
- Secondary cataracts: these occur as a consequence of other conditions, such as uveitis, traumas, systemic diseases, toxins, tumours, etc.

Based on the stage of opacification :

- Incipient (Fig. 4).
- Early immature.
- Late immature (Fig. 5).
- Mature (Fig. 6).
- Hypermature (Figs. 7 and 8).

During the evolution of the disease, cataracts pass through various stages, from incipient to immature, followed by mature, and finally hypermature (Fig. 9). In the final stage, the lens capsule appears wrinkled due to the loss of substances through the envelopes, which can result in a notably shrunken crystalline lens with a small, hard nucleus that has sedimented towards the bottom and which is accompanied by a more or less transparent fluid content. This is known as a Morgagnian cataract (Fig. 10).

When dealing with a disease that can involve so many variables, a thorough examination of the patient is essential to identify the cause of the cataract, its stage, the consequences of any associated inflammation (lens-induced uveitis), and to collect all the data needed to make a prognosis regarding restoring the patient's vision if the case is to be treated.

Gathering data, including the patient's menace response, dazzle reflex, white and chromatic pupil light reflexes, and from complementary tests such as ultrasound (Fig. 11), electroretinography, high-frequency ultrasound of the ciliary cleft, and gonioscopy will provide enough information to determine whether or not surgery is indicated.

Cataracts often need topical anti-inflammatory treatment before the surgical intervention in order to reduce any uveitis and guarantee better results (Fig. 12).

Traumatic rupture of a crystalline lens capsule, with the subsequent development of an acute cataract and severe intraocular inflammation (phacoclastic uveitis), is considered a medical emergency and any delay in surgery will only increase the patient's risk of losing their vision and the eye itself.

The veterinary surgeon must also remember that diabetic cataracts, which rapidly progress to the mature stage and in which the crystalline lens becomes considerably hydrated and swollen (intumescent cataract) (Fig. 13), are often accompanied by a ruptured capsule. These cataracts are also deemed to be a situation that requires emergency treatment.

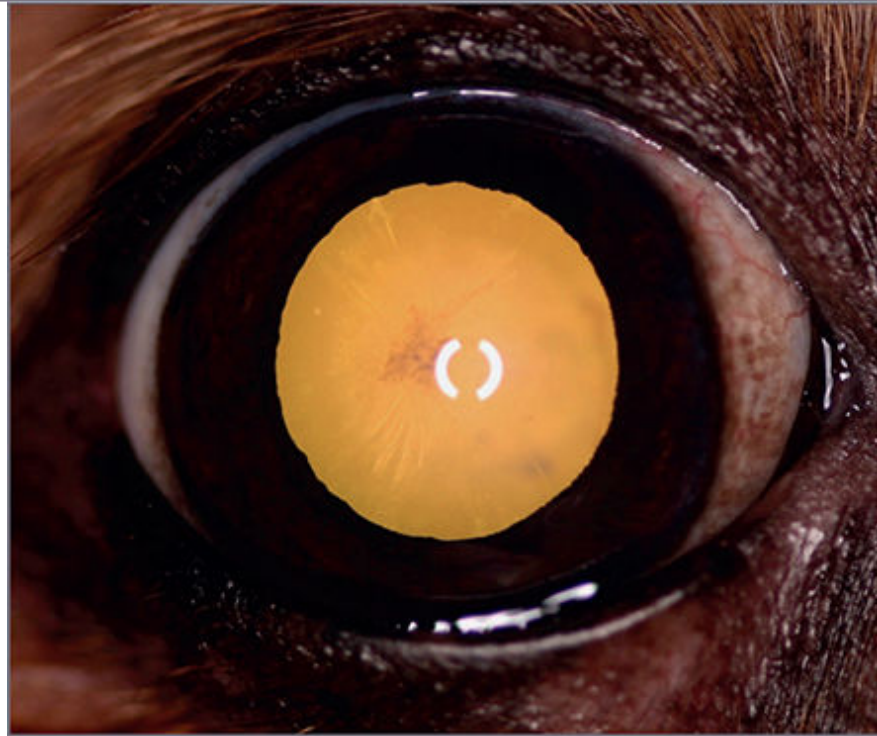


Figure 3. Congenital posterior subcapsular cataract.

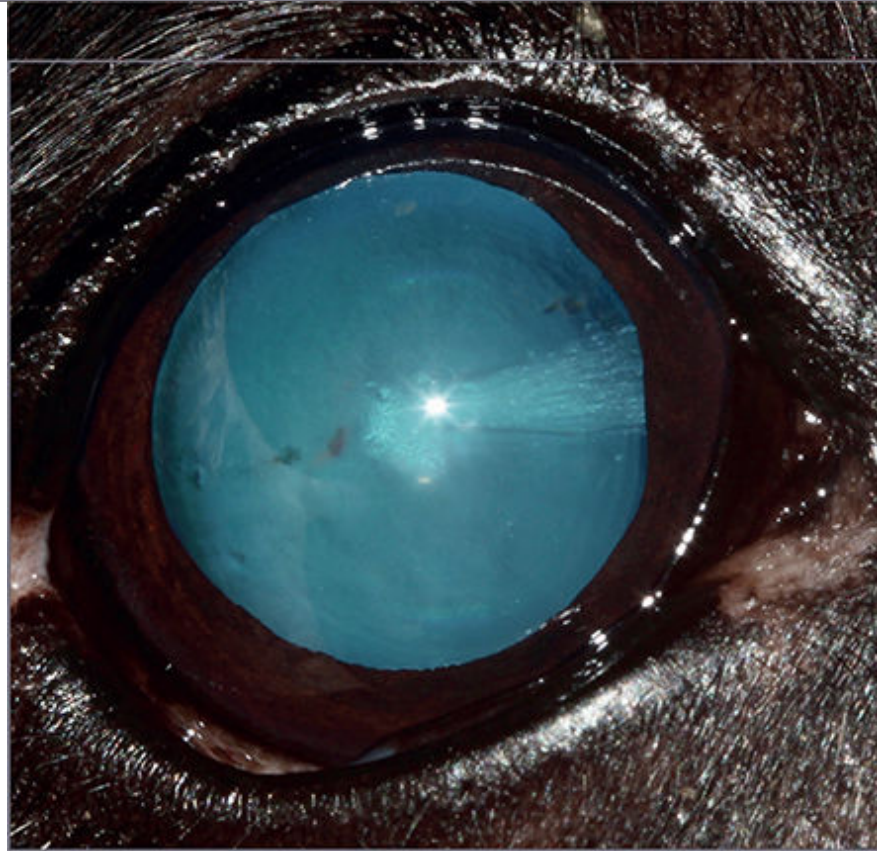


Figure 4. Radial incipient cataract.



Figure 5. Immature cataract.

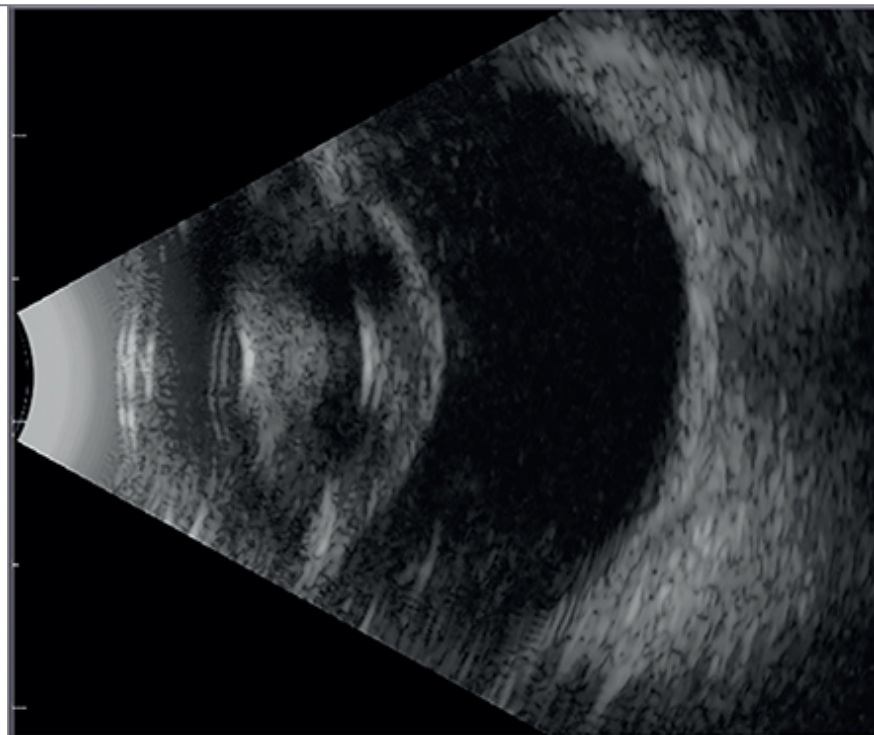


Figure 6. Ultrasound of a mature cataract.



Figure 7. Hypermature cataract accompanied by severe uveitis.

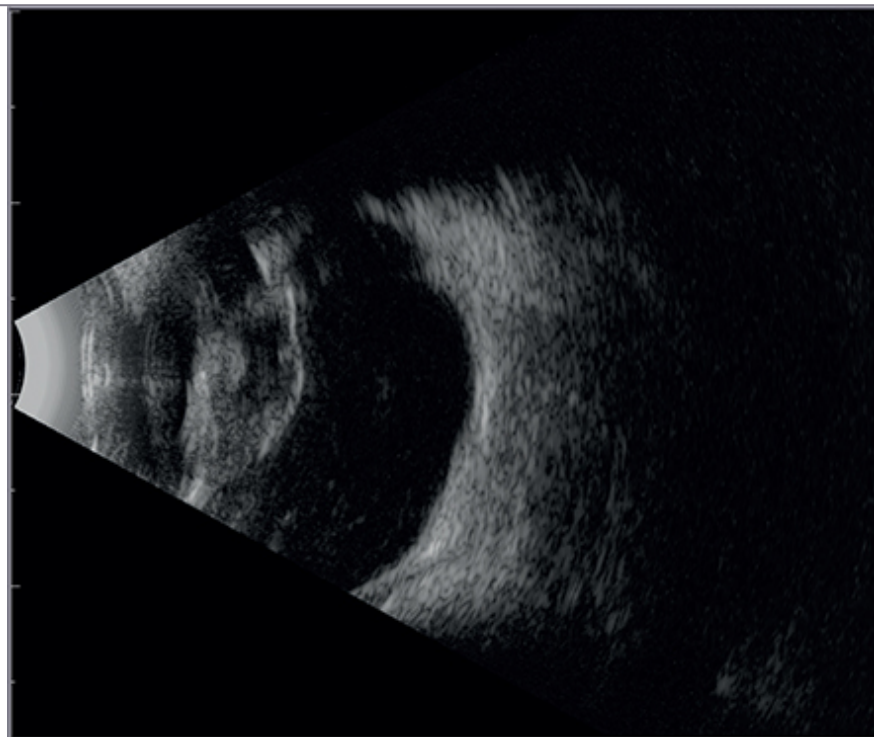


Figure 8. Ultrasound of a hypermature cataract.



Figure 9. Patient affected by cataracts in different stages of development.

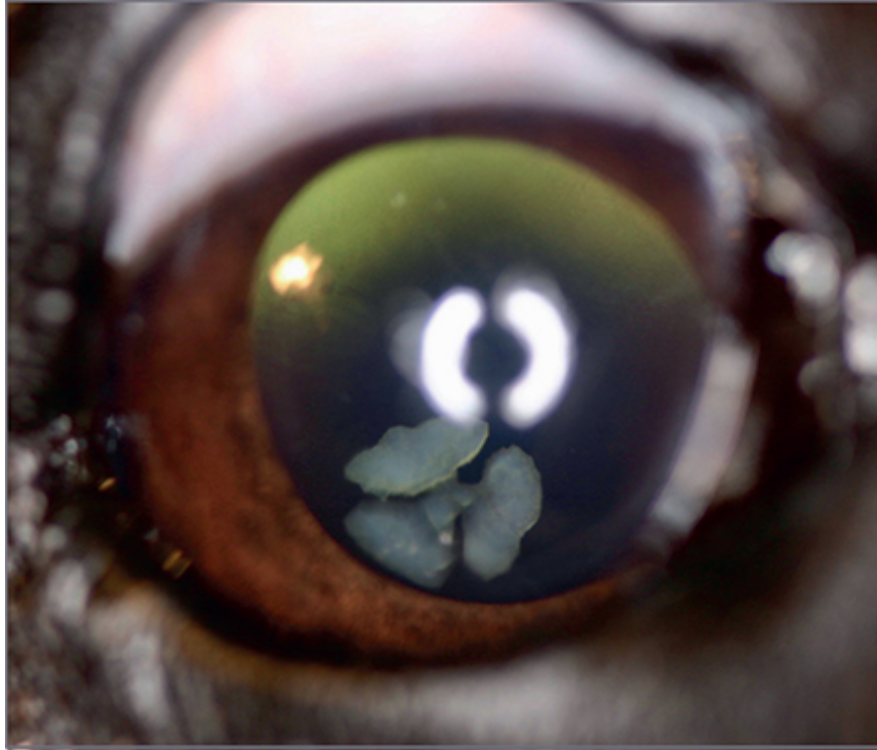


Figure 10. Morgagnian cataract, only remains of the nucleus deposited at the bottom of the crystalline lens sac are visible.



Figure 11. Cataract associated with partial retinal detachment.



Figure 12. Intumescent mature cataract accompanied by synechiae due to lens-induced uveitis.

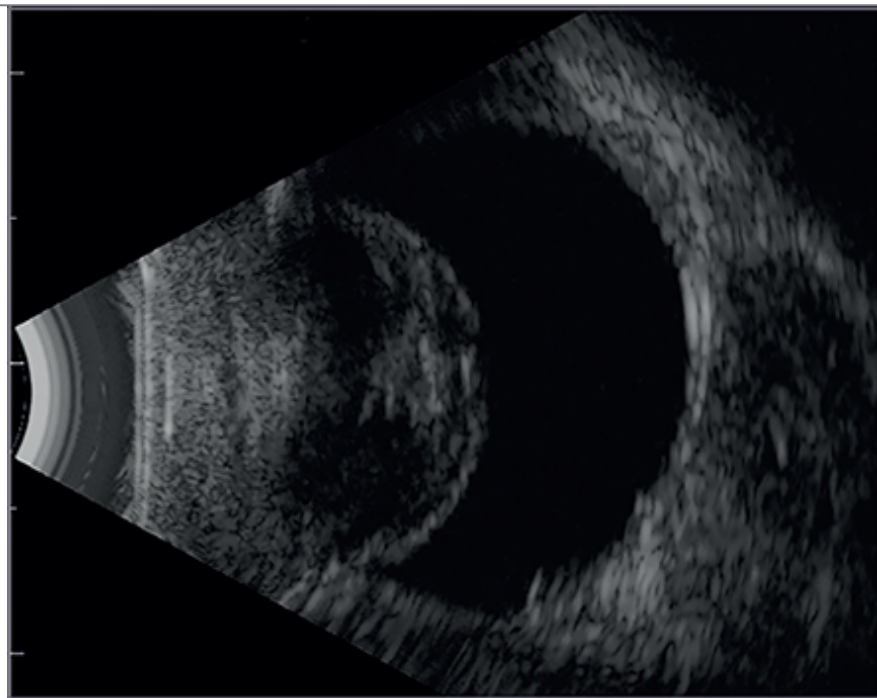


Figure 13. Ultrasound of an intumescent diabetic cataract. Note the size and sphericity of the crystalline lens.



Cataracts

Treatment of cataracts

Cataracts can only be treated by surgical means. Any delay in operating on the eye only gives the uveitis more time to provoke irreversible deterioration of structures such as the retina, which would make it impossible to recover the patient's vision.

Do not wait until a cataract matures before intervening. As with any disease, therapy in the early stages will always yield better outcomes and reduce the rate of postoperative complications.

Phacoemulsification, which is the name given to the intervention to eliminate the cataractous material, is completed by implanting an intraocular lens within the crystalline sac, whenever this is possible, thereby recovering normal vision (emmetropia) (Fig. 14). The intervention has a very high success rate, and it means patients can avoid the complications that all cataracts produce if left untreated, while also preserving or restoring their vision.

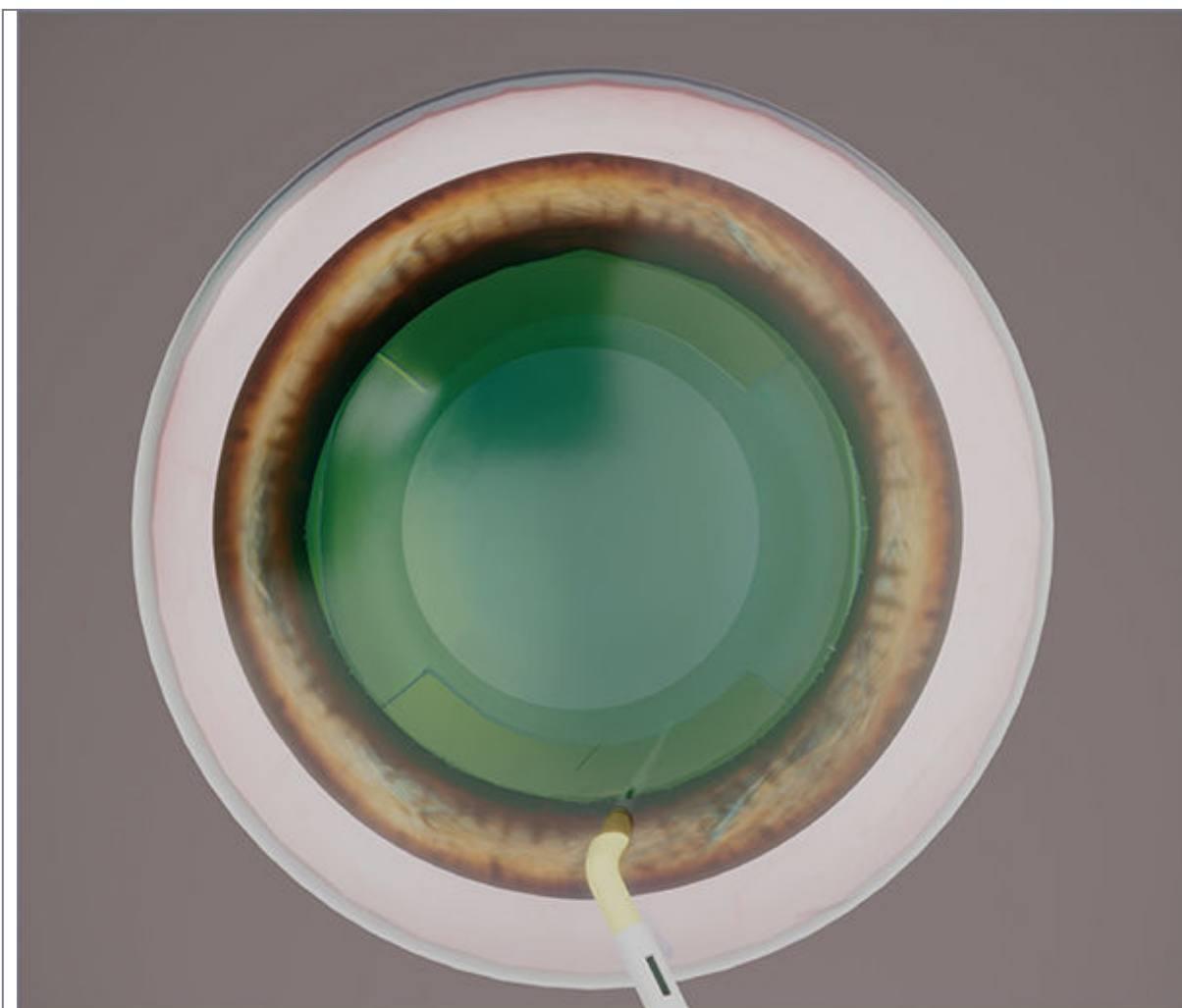


Figure 14. Intraocular lens implanted after phacoemulsification.

Crystalline lens luxation

The rupture of the zonular fibres anchoring the crystalline lens in position causes lens instability (phacodonesis) and eventually, if the fibres break completely, the lens will shift forwards or backwards.

Incipient signs of this process are iridodonesis and the presence of strands of vitreous herniated in the anterior chamber.

With regard to the aetiology of lens luxation, the causes can be classified as:

- **Primary lens luxation** : this occurs due to a weakness in the zonular fibres, which ultimately suffer spontaneous rupture and cause the lens to fall back. It is a process of proven heritability in breeds including various types of Terrier and Shar Pei (Fig. 15).

- **Secondary lens luxation** : in this case an underlying cause weakens or ruptures the zonules and the lens ultimately undergoes displacement. Some of the possible underlying causes are:
 - Intraocular tumours which can press against the crystalline lens and break the zonules.
 - Trauma, either directly to the eyeball or affecting the orbit.
 - Intraocular inflammation (uveitis), which over time will finally weaken and break the zonular fibres.
 - Intumescent cataracts that swell and displace the crystalline lens and which usually course with severe uveitis. They are very typical in patients with diabetes.
 - The development of glaucoma. The eyeball increases in volume due to the increase in intraocular pressure, which results in narrowing and rupture of the zonular junctions and a slipped lens (Fig. 16).

Vets need to be aware that following lens luxation, and even in the early stages of lens instability, vitreous humour will pass into the anterior chamber. This substance will move towards and obstruct the aqueous humour drainage points, leading to the development of a hypertensive clinical picture.

What is more, if the lens suffers a forward dislocation towards the anterior chamber, then we can also conclude that the lens itself will become a physical impediment to the passage of aqueous humour and the process will evolve into a very acute case of ocular hypertension.

Finally, the crystalline lens may also fall backwards into the vitreous cavity. This will cause inflammation and syneresis of the vitreous humour, so this gel becomes less dense and can now flow completely unobstructed through the pupil and into the anterior segment of the eye (Fig. 17). An elevated intraocular pressure is another common complication in patients with a cataract.

Whenever the crystalline lens luxates and falls into the vitreous humour, examination will reveal a crescent-shaped portion of retina that is directly visible through the pupil in the absence of an interceding lens. This is called an aphakic crescent and it is a pathognomonic sign of this condition (Fig. 18).

It should be noted that a luxated crystalline lens will undergo cataractogenesis and quickly lose its transparency and develop the characteristic white colour.

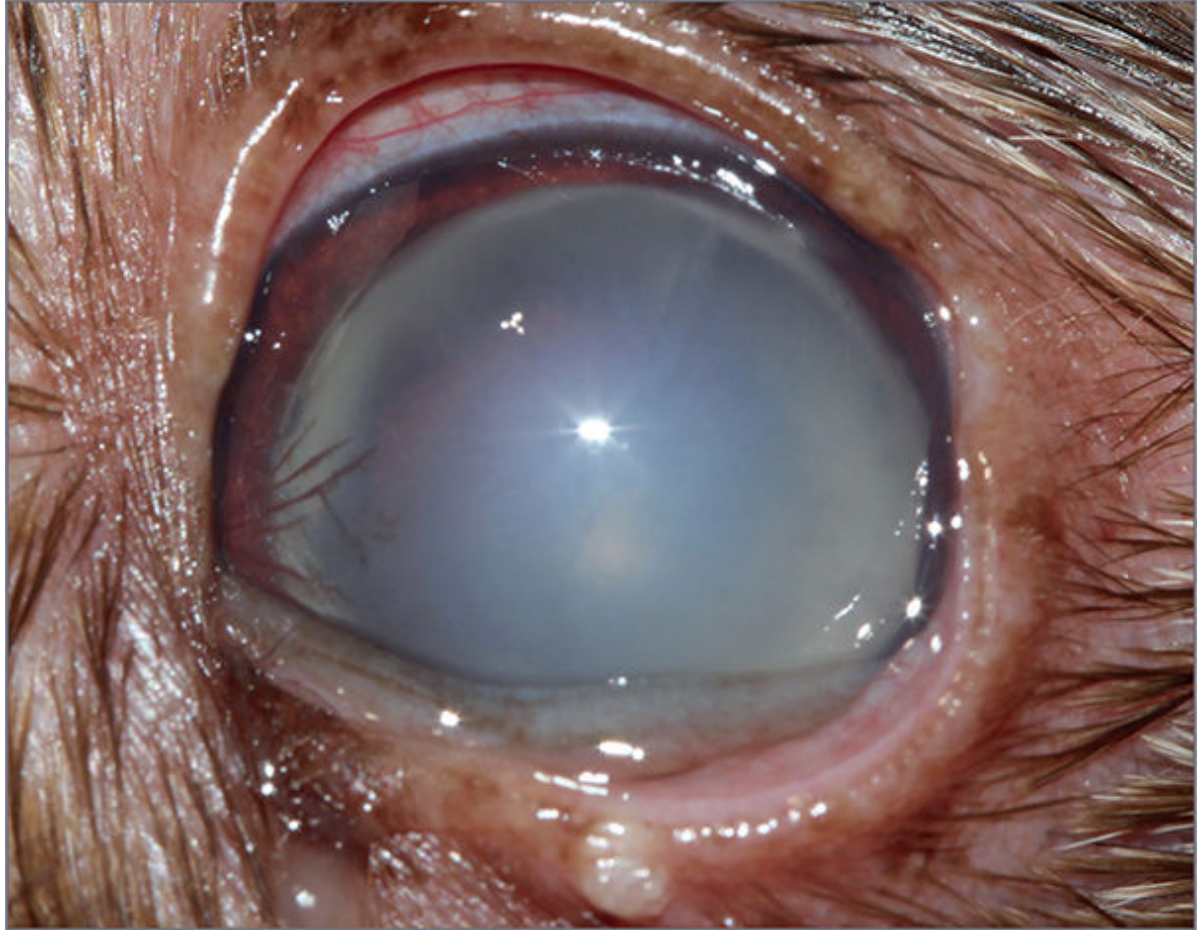


Figure 15. Primary lens luxation towards the anterior chamber.

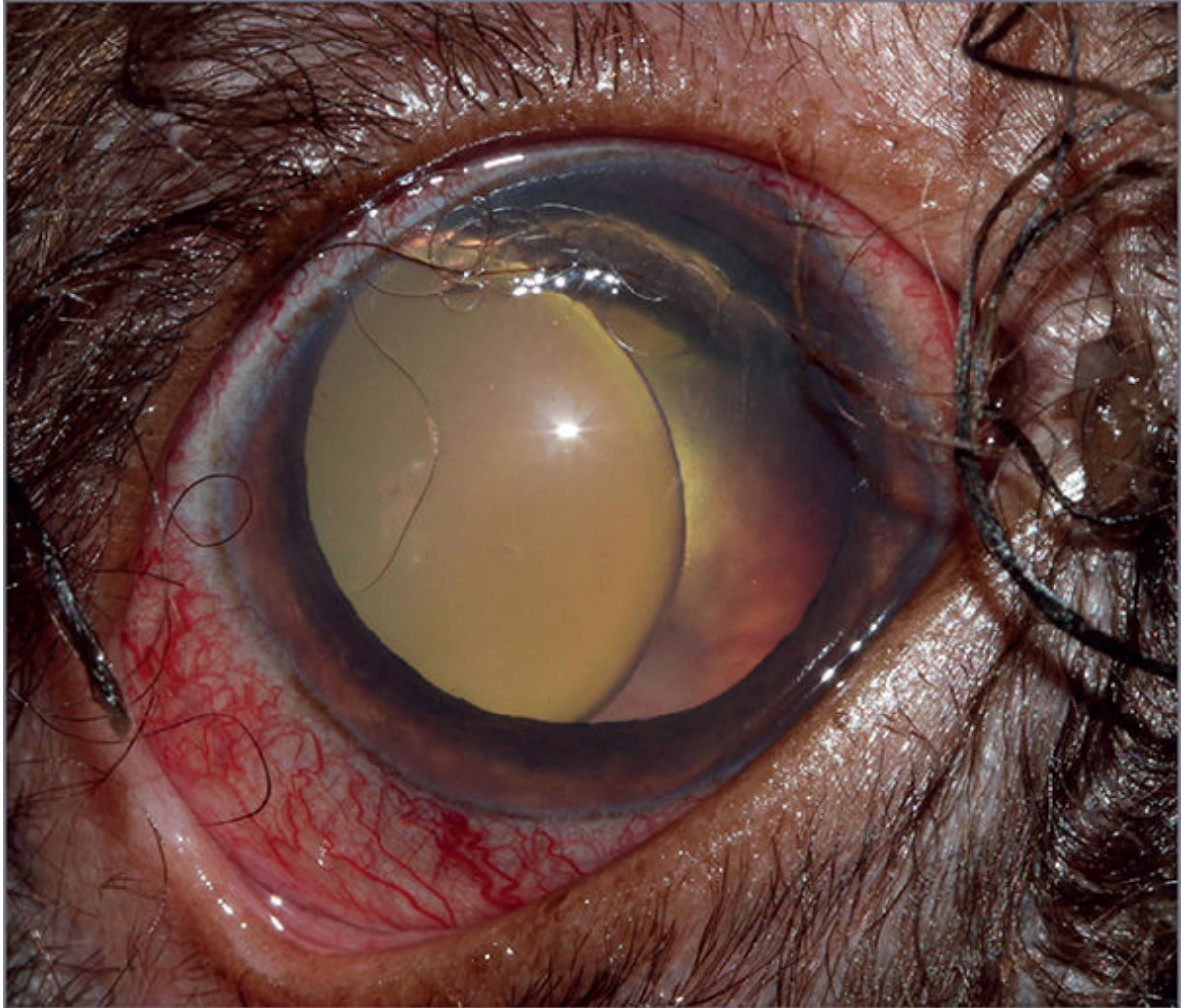


Figure 16. Lens luxation secondary to glaucoma in a Spanish Water Dog.

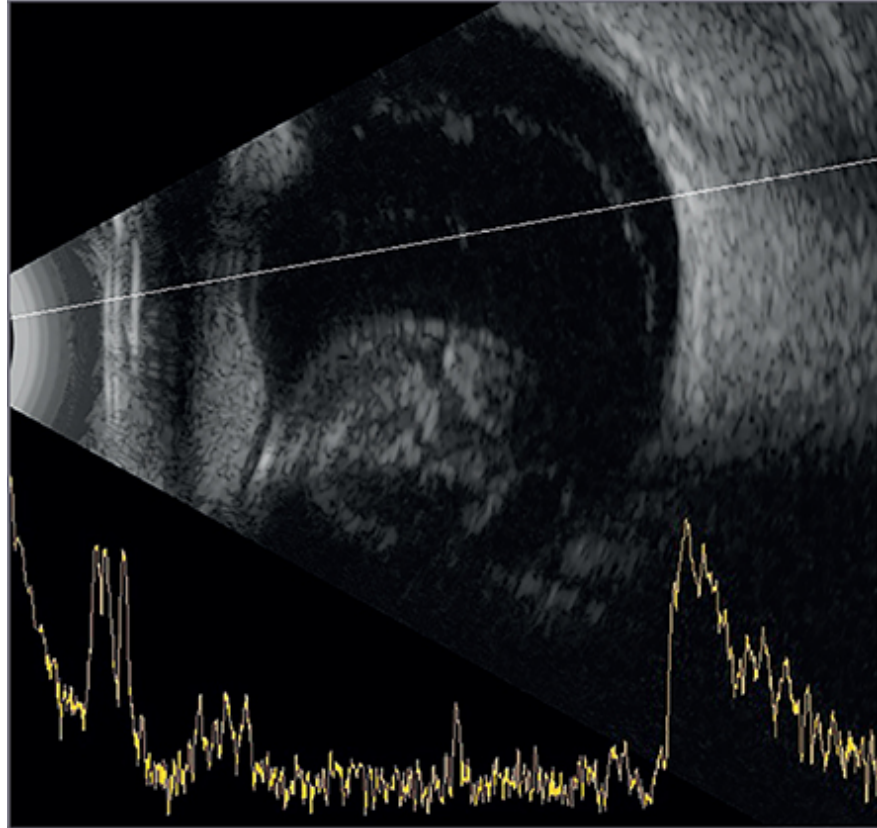


Figure 17. Ultrasound of an eye with a luxated crystalline lens. The vitreous detachment can be seen in the dorsal region.



Figure 18. Aphakic crescent formed by a luxated lens.



Phacoemulsification and implantation of an intraocular lens





Crystalline lens luxation

Treatment of crystalline lens luxation

We must not forget that lens luxation is a process that will always culminate in vision loss and most likely secondary glaucoma unless it is treated surgically.

In cases of displacement towards the anterior chamber, and provided that the eye still preserves vision, the correct treatment is an intracapsular lens extraction (ICLE). The procedure involves the complete extraction of the crystalline lens with its corresponding capsules and is usually complemented with an anterior vitrectomy to remove the portion of vitreous displaced towards the anterior segment, thus preventing the development of a glaucomatous disease pattern.

If the displacement is towards the vitreous, an ICLE is again the indicated course of action, although in this case the first step is to recover the crystalline lens and bring it forwards so that it can be removed through the incision in the cornea. A vitrectomy is also necessary in this situation and prophylactic laser retinopexy can often be considered to prevent retinal detachment due to a lack of vitreous support.

If, for whatever reason, surgery is contraindicated to treat a posterior luxation of the crystalline lens, we recommend the instillation of miotic eye drops, such as prostaglandin analogues, to encourage the pupil to close and prevent the crystalline lens from passing through the pupillary aperture. The animal will maintain its vision for some time, but the short-to medium-term prognosis is blindness and the onset of ocular hypertension.

Crystalline lens capsule rupture

This occurs quite often and is caused by two factors:

- The development of cataracts accompanied by intense intumescence, as occurs with diabetic cataracts. The lens swells and eventually ruptures the capsule, with its contents then spilling into the aqueous humour or the vitreous.
- Direct trauma to the crystalline lens which involves rupture of a capsule, generally the anterior one. This often occurs when thorns or other sharp

objects penetrate the eye, although the leading cause is probably being scratched by a cat (Fig. 19). These patients require good pharmacological mydriasis so the vet can view the full contour of the lens, even up to the equator region, in search of ruptured capsules that initially course without any inflammation, but which in a few days will give rise to very severe phacoclastic uveitis.

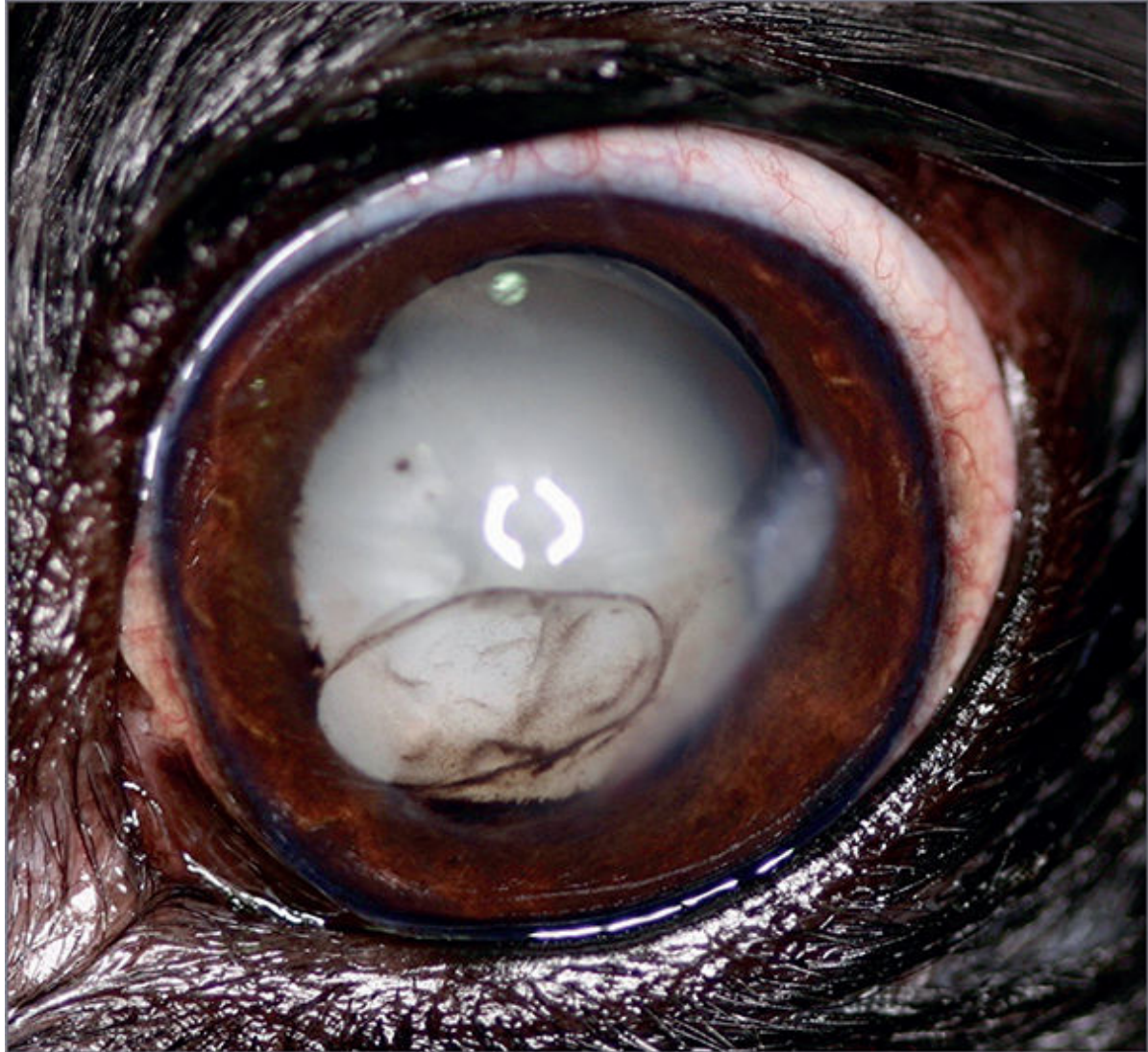


Figure 19. Traumatic cataract due to a cat's scratch. The crystalline lens rupture has caused extrusion of part of the contents leading to severe phacoclastic uveitis.

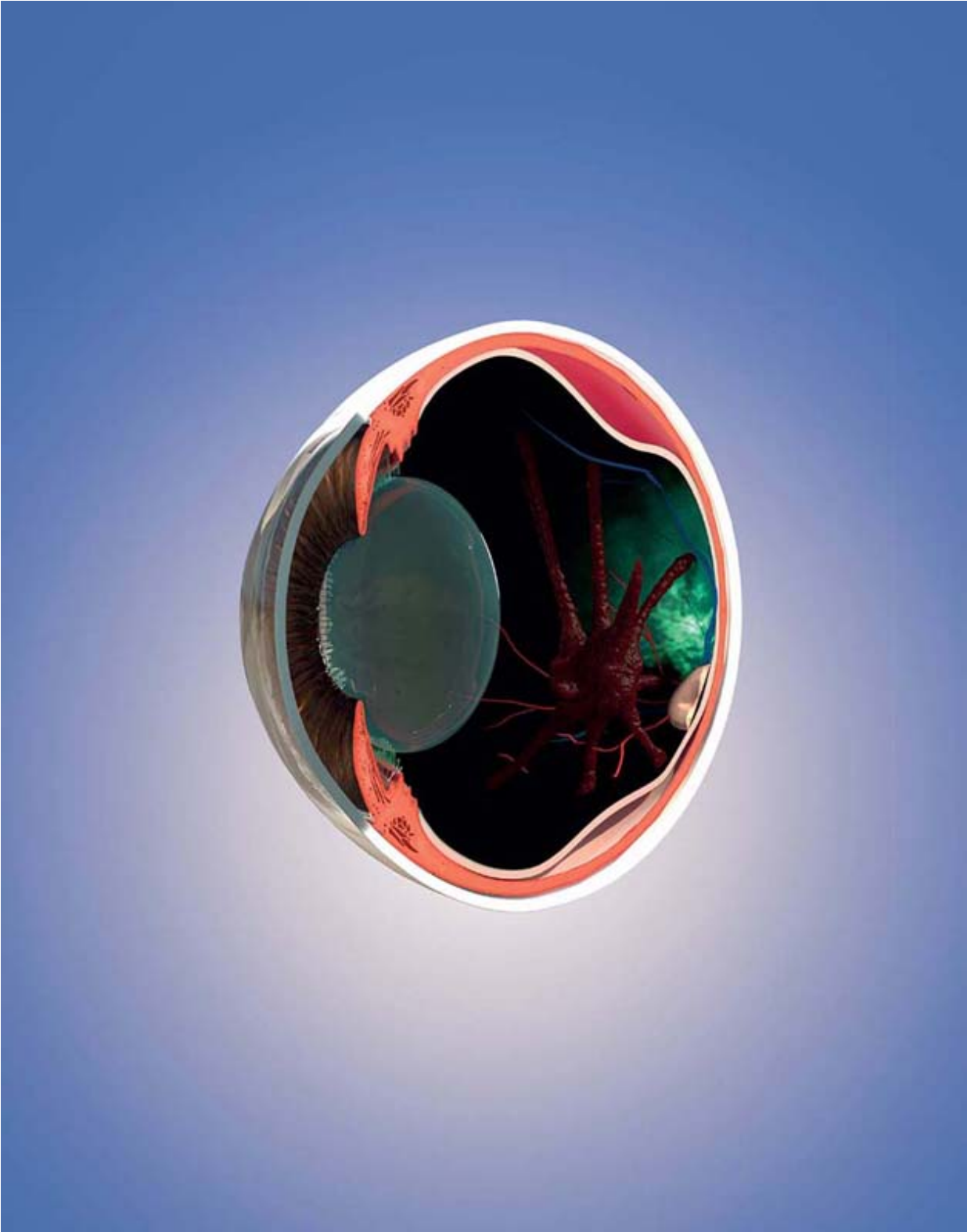
Treatment of crystalline lens capsule ruptures

Bear in mind that this is an extremely serious situation, and it will cause severe uveal inflammation due to the release of substances with a very potent antigenic activity in the aqueous and vitreous humours.

Treatment, which should be performed as soon as possible and even when there are still no signs of uveitis, is of a surgical nature and consists of phacoemulsification of the crystalline lens and, if the lens capsule rupture allows, the insertion of an intraocular lens.

09

The retina



Anatomy and physiology of the retina

The retina is the most internal layer of the eyeball and is where phototransduction takes place, that is, the process of transforming photons of light into electrical signals. As such, it is a highly specialised tissue with a low capacity for repair and regeneration.

The retina is traditionally divided into 10 layers (Fig. 1), but from a practical perspective it can be reduced to the communication between three types of specialised neurons: photoreceptors (cones and rods), bipolar cells, and ganglion cells.

The innermost layer of the retina is called the retinal pigment epithelium (RPE) and it is not a neural tissue. The RPE fulfils the essential role of nourishing and protecting the retina as this is where the blood–retinal barrier is located. When the retina becomes detached, the separation occurs between the RPE and the neuroretina.

The layer of photoreceptors contains two types of photosensitive cells:

- **Rod cells** are more sensitive to low light levels, so they are essential for vision in dim environments (scotopic vision). They are also responsible for detecting movement.
- **Cone cells** require more intense light levels, so they function in brighter environments (photopic vision). Cone cells contain different photopigments, which allows them to detect colours. Furthermore, due to their position in the area centralis and connection to ganglion cells, they provide visual acuity, or in other words, the fine details of sight.

When a photon reaches the receptors, it stimulates chemical changes in the photopigments, thereby altering the sodium channels in the membrane and hyperpolarising the cells. The resulting action potential travels along the bipolar cells and finally stimulates the ganglion cells. During these interactions, some other cells that are very important for vision, called interneurons, are also involved in convergence, attenuation, and integration processes.

Finally, the retinal ganglion cells have a very long axon that extends to the brain. First, they form a layer of nerve fibres and then, after passing through the cribriform plate, they emerge from the eye to form the optic nerve.

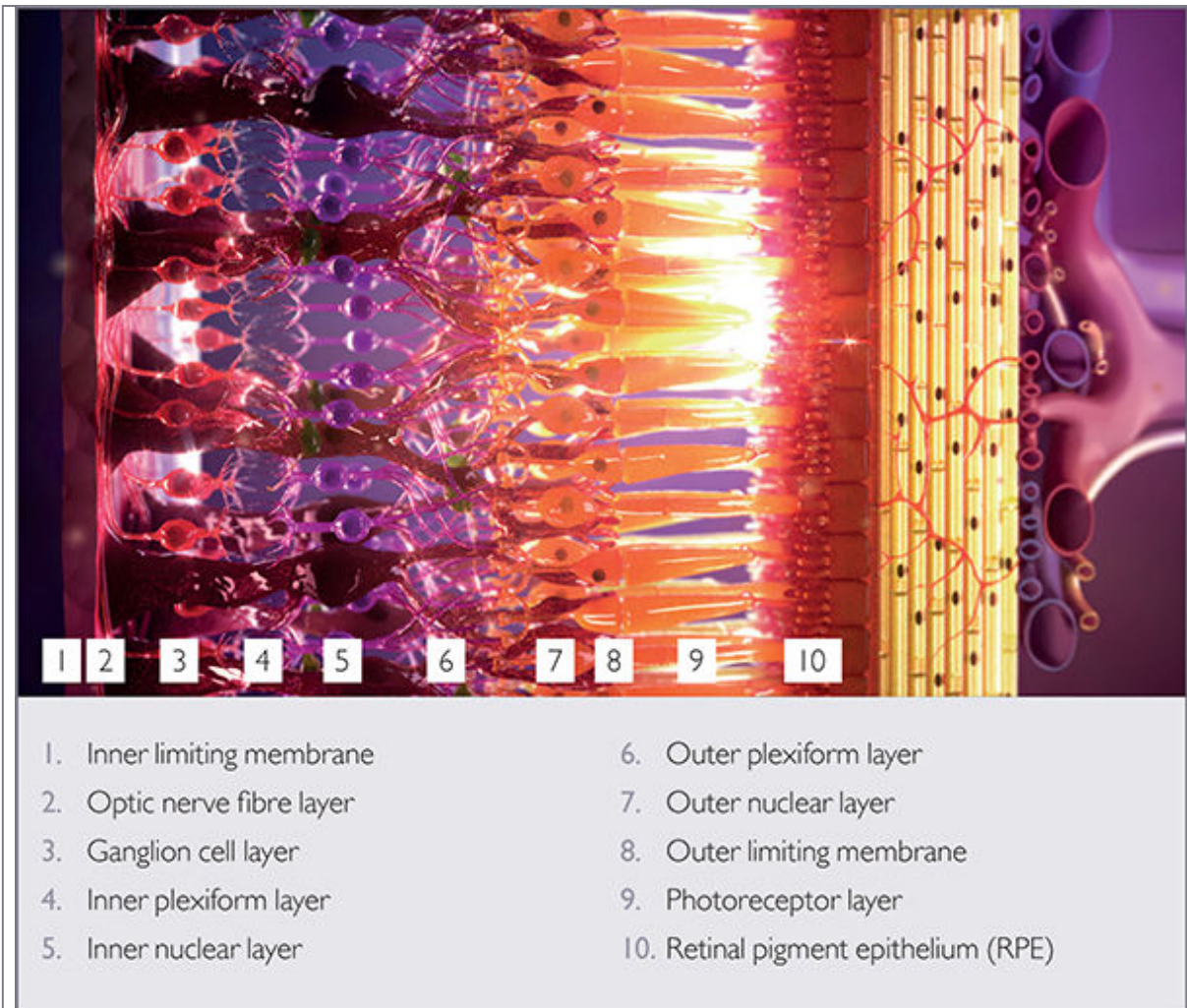


Figure 1. Layers of the retina.

Examination of the retina

To identify different retinal diseases, the vet must be familiar with the range of normal physiological variations, as the retina's structure can differ enormously between healthy animals.

The fundus of the eye consists of (Fig. 2):

- **Tapetal fundus** : also known as the tapetum, it is located in the upper portion of the retina and occupies around one-third of the fundus' surface. The tapetum's colour varies and in some breeds, it is smaller (toy dogs) or completely absent (some Beagles, albino dogs) (Fig. 3).

- **Nontapetal fundus** : located ventrally to the tapetal fundus, this region is pigmented. Humans do not have a tapetum and so the RPE is pigmented across the entire ocular fundus, but in animals the RPE lacks pigment in the tapetal region.
- **Optic disc** : also called the optic nerve head. This is the point where ganglion cell axons join to form the optic nerve and pass through the sclera via the cribriform plate.
- **Retinal vasculature** : dogs have a holangiotic retinal vascular pattern, which means they have visible vessels and multiple arteries emerging from the optic disc in a centrifugal arrangement and returning in a centripetal pattern.
- **Area centralis** : like humans, dogs have a central region with a greater density of cone cells; however, unlike humans, it is not called the macula. It is an avascular region situated dorsally and temporally to the optic nerve. Injuries in this region can have major consequences for an animal's vision.

The retina can be examined using either a direct or indirect ophthalmoscope. The examination should also be performed on dilated pupils after the application of tropicamide eye drops and a 15-minute wait.

Direct ophthalmoscopes are more typically used in general clinical practice and can incorporate greater magnifications. Although the examiner may lose some overall perspective because they can only display a small field, the increased detail is important when examining the ocular fundus in veterinary patients.

Indirect ophthalmoscopes require more training as the examiner must incorporate an aspheric lens. However, they offer a broad field of observation and, since the examiner can use both eyes at once, the instrument produces a stereoscopic image that helps identify lesions more accurately. Always remember that the image produced via indirect ophthalmoscopy is inverted.

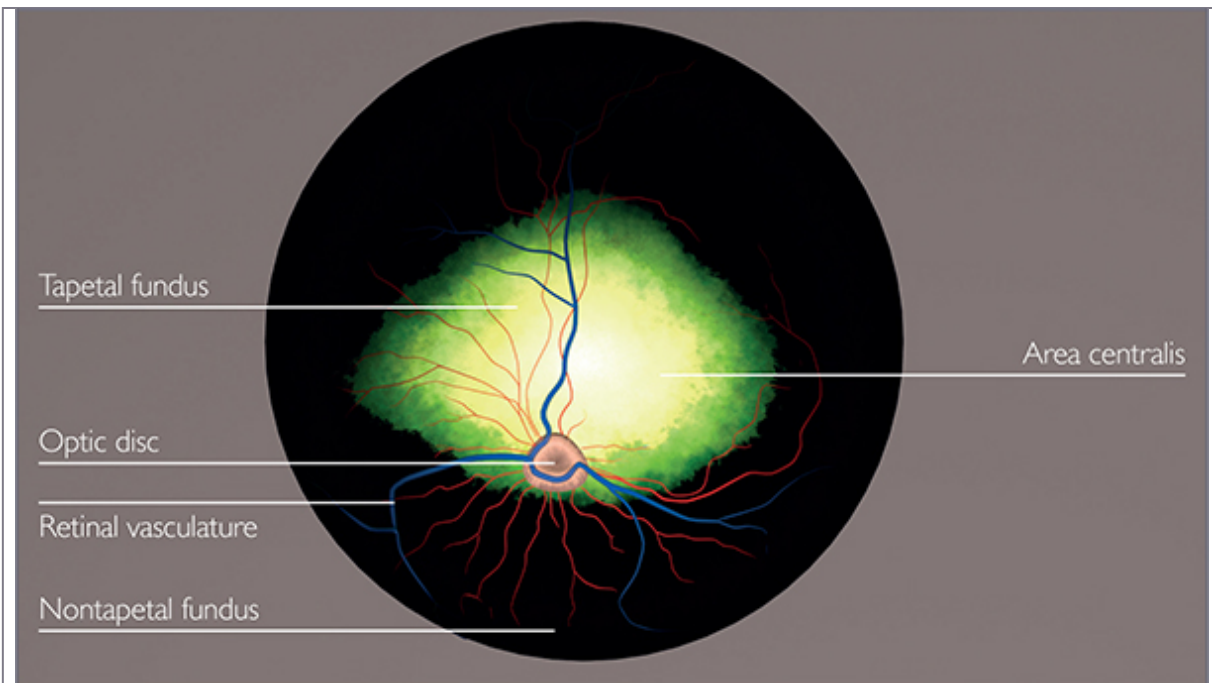


Figure 2. Elements visible in an ocular fundus examination.

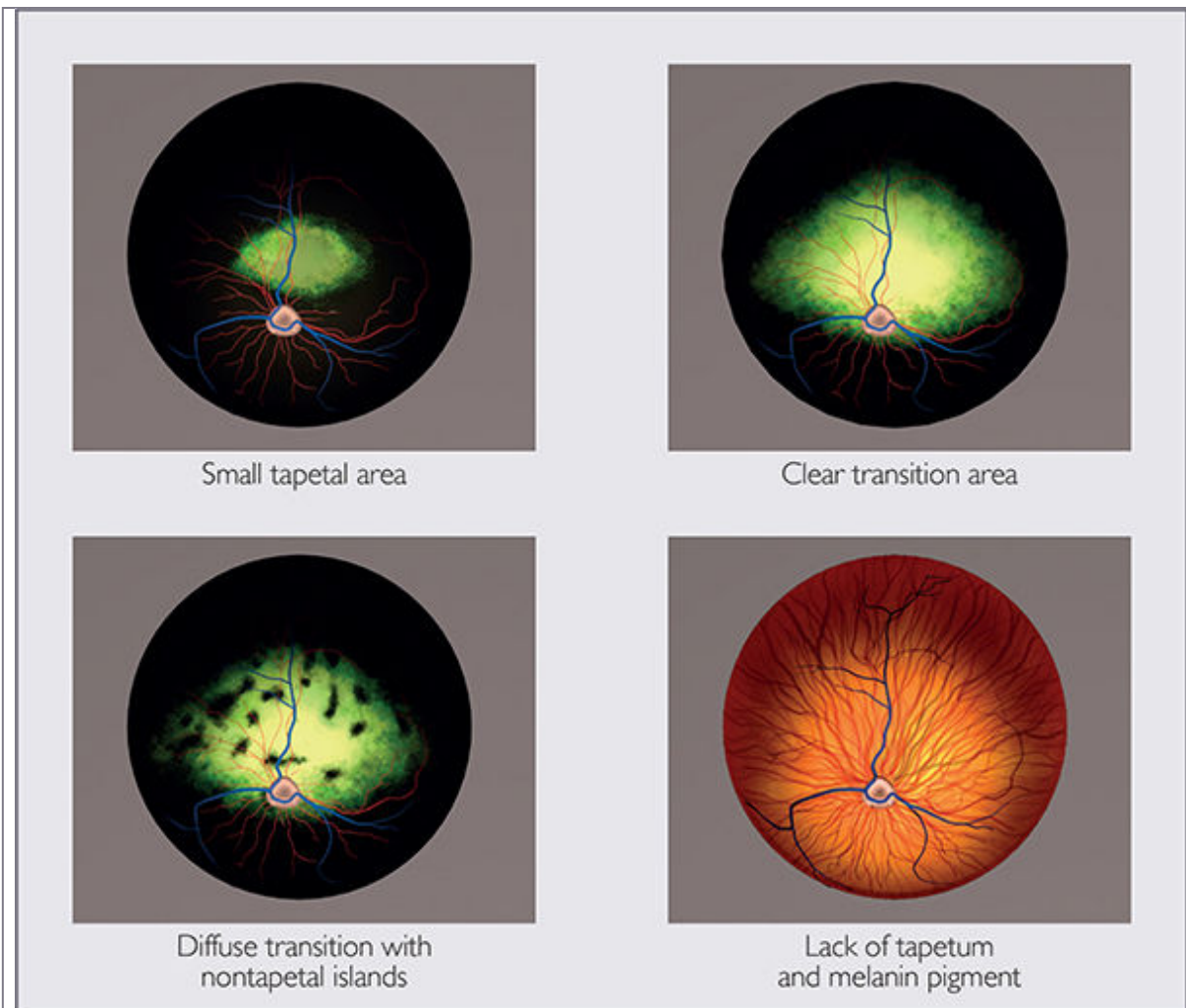


Figure 3. Different types of physiological tapetal fundi.

Signs of retinopathy

- Hyporeflectivity: this is the result of a thickened retina which consequently attenuates passing light more than usual. It may be a symptom of retinal oedema, an early detachment, or retinal folds due to dysplasia, amongst others.
- Hyperreflectivity: the tapetal fundus is more reflective due to retinal thinning. It occurs in cases of progressive retinal atrophy.
- Vascular attenuation: any reduction in the calibre of vessels also tends to be associated with progressive retinal atrophy.
- Optic nerve pallor.
- Retinal haemorrhages.

- Folds.
- Perivascular cuffing.
- Chorioretinal scars (Fig. 4).

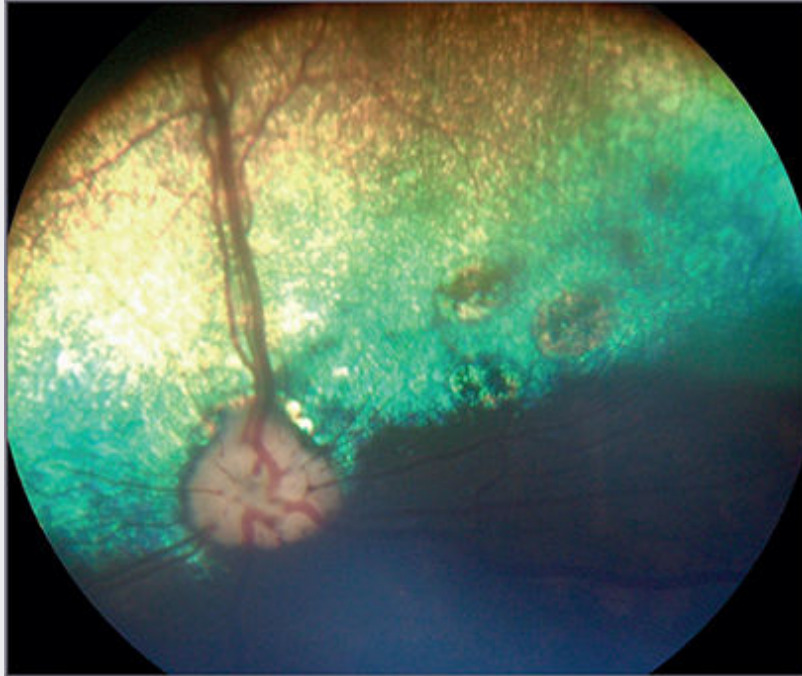


Figure 4. Chorioretinal scars.

Congenital retinal diseases

These conditions are discussed in detail in the specific chapter on congenital diseases.

Acquired retinal diseases

Inherited retinopathies

Although traditionally they have all been included under the umbrella of progressive retinal atrophy (PRA), it is actually a large group of highly variable diseases in which different genetic mutations provoke abnormalities in RPE metabolism and the phototransduction of cone and rod cells. These disorders can appear in several different breeds and at any age. The field is in constant evolution as researchers are continually discovering new diseases

and their underlying mutations. Different types of PRA can even be observed in a single breed. They can only be diagnosed and characterised through an electroretinogram.

Patients suffer from a progressive loss of vision, with most animals losing their nocturnal vision because rod cells are affected first. Later on, cone cell degeneration also becomes apparent. An obstacle avoidance test can be performed in the consultation room under normal and low lighting conditions to determine the patient's visual capacity in different settings. In the long term, besides blindness, dogs with retinal degeneration can develop cataracts due to the release of substances from the retina.

Retinal degeneration is untreatable, although gene therapy experiments are currently being conducted.

Retinopathies secondary to infectious diseases

The choroid is closely related to the retina, in fact cases of posterior uveitis are called chorioretinitis. There are several infectious diseases that can induce clinical signs in the retina, while the specific possibilities depend on the diseases endemic to each region. Many produce retinal inflammation and haemorrhage, whereas others may even lead to retinal detachment.

Electroretinography

Electroretinography (ERG) is a noninvasive test of retinal function. Electrodes are placed around the eye in order to record the retina's response to different light stimuli. By varying the light's intensity, wavelength, and using light- and dark-adapted ERGs, it is possible to discern between cone and rod cells. Electroretinography can be used to diagnose these diseases long before any ophthalmoscopic changes become apparent. The technique can also determine whether blindness is of a retinal or central origin.

Genetic tests

Nowadays, genetic testing is often used in pedigree dogs. The main advantage is that it can identify mutation carriers before animals reach sexual maturity and, therefore, before the disease is expressed. However,

genetic tests only identify a specific mutation, so they do not rule out the possibility of patients developing retinopathies due to other mutations. The aim of screening should be to eliminate a disease from the breed without detriment to the genetic pool. What is more, there is not a genetic test available for every disease, as even though many conditions have been well characterised, the specific mutation remains to be identified.

Ocular defects certificate

These certificates are issued following a painstaking ophthalmological exam in search of inherited eye diseases. The certificate is valid for 1 year and attests to the ocular health of a breeding animal. As many retinal disorders develop in mature dogs (3–5 years in the case of PRA in Cockers, for example), the examinations need to be completed throughout the animal's lifetime.

The clinical signs of chorioretinitis are those typical of inflammation, plus retinal oedema, haemorrhages, vitritis, subretinal exudate, perivascular cuffing, and so on. Here are some of the diseases that can generate chorioretinitis in dogs:

- Bacterias: *Borrelia burgdorferi* , *Bartonella* , any source of infection that produces sepsis.
- Viruses: distemper.
- Rickettsias: *Ehrlichia canis* (Fig. 5), *Babesia canis* , *Anaplasma phagocytophilum* .
- Protozoa: *Leishmania* , *Toxoplasma* , *Neospora* .

The treatment of infectious chorioretinitis revolves around the resolution of the primary cause and initiation of a topical and systemic anti-inflammatory therapy in an attempt to reduce tissue damage in the retina.

Old lesions due to chorioretinitis can leave chorioretinal scarring.

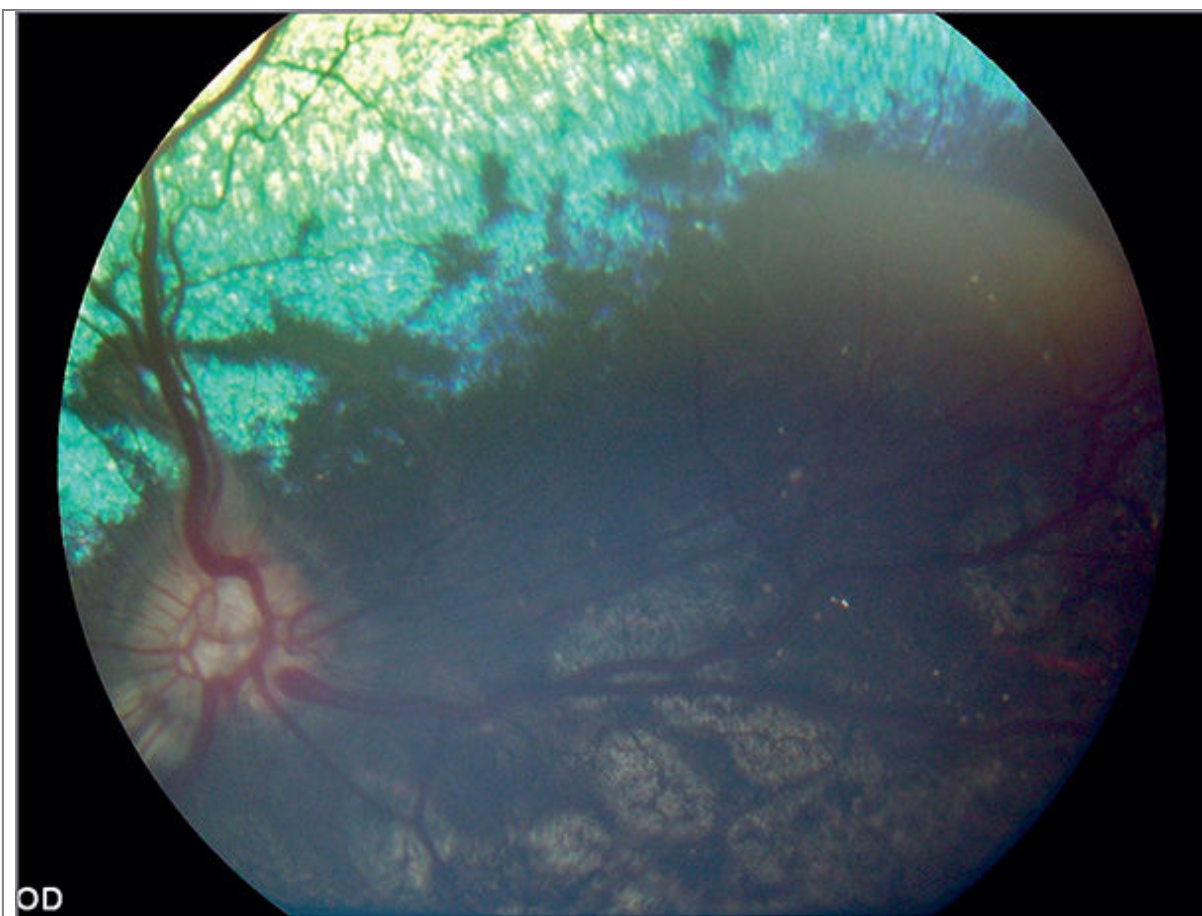


Figure 5. Multifocal chorioretinitis in a *Ehrlichia canis* -positive dog.

Hypertensive retinopathy

High blood pressure can cause microvascular dysfunction and damage leading to target organ damage (kidneys, brain, eyes). In the retina, arterial hypertension may produce highly varied clinical signs including retinal haemorrhage (Fig. 6), small bullous retinal detachment, complete exudative retinal detachment, and so on. The most common causes of secondary hypertension in dogs are acute or chronic kidney disease, endocrine diseases (hyperadrenocorticism, diabetes, primary aldosteronism), or neoplasms such as phaeochromocytomas. Primary hypertension is uncommon in dogs.

Examination of the ocular fundus is often enough to confirm arterial hypertension after measuring the patient's blood pressure during consultation.

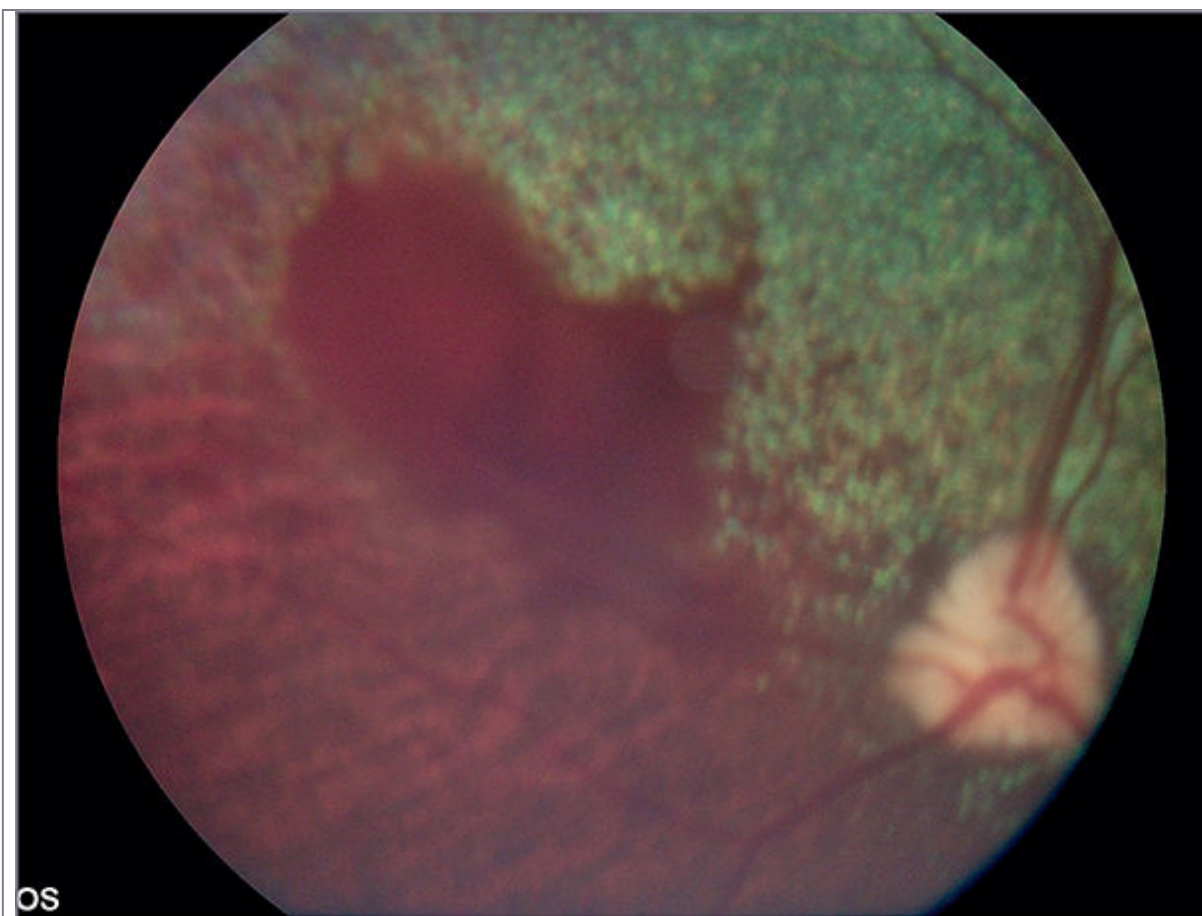


Figure 6. Retinal haemorrhages in a dog with arterial hypertension.

Sudden acquired retinal degeneration syndrome (SARDS)

SARDS has the potential to cause a form of blindness that develops rapidly and aggressively. Patients have fairly unreactive and dilated pupils. While examining chromatic pupillary light reflexes, dogs may demonstrate areflexia under the red light and a pupil response to the blue light (see the chapter on neuro-ophthalmology).

The fundoscopic appearance is normal for the first few weeks of the disease, so an electroretinogram is necessary to distinguish between SARDS and central blindness.

The syndrome is most prevalent among middle-aged, castrated females and a lot of patients may have polyuria, polydipsia, polyphagia, and anosmia. Although it is believed to be an autoimmune disease, the primary aetiology of SARDS has not yet been determined. At present, there is no effective treatment for this syndrome.

Retinal detachment

Retinal detachment occurs when the retina loses its connection to the RPE, thus precluding its function (Fig. 7).

It can transpire due to different mechanisms:

- An embryonic lack of attachment associated with a congenital disease, for example Collie eye anomaly.
- Exudative detachment is when the presence of subretinal fluid separates the retina from the RPE, such as in the case of hypertensive choroidopathy, tumours, or infectious chorioretinitis. This is the most frequent cause.
- Tractional detachment is due to the postinflammatory formation of fibrovascular membranes, which retract, pull on the retina, and eventually provoke its detachment.
- Rhegmatogenous detachment occurs when vitreous liquefaction results in a retinal tear, thus allowing vitreous humour to penetrate under and separate the retina. This is the most common type of retinal detachment in predisposed breeds, such as the Shih Tzu or Whippet.

It is essential to find the cause of detachment since the process is often reversible, particularly if the retina remains connected to the optic nerve head and the ora ciliaris retinae. For instance, when dealing with hypertensive choroidopathies, if the systemic blood pressure can be normalised, the subretinal exudate will often reabsorb. In some cases, this is suffice to induce retinal reattachment and the patient will regain their vision. The same thing can happen in patients with retinal detachment secondary to an infectious disease once the infection has been eliminated. However, if the retina is torn (retinal dialysis), medical treatment is basically ineffective. Furthermore, whenever the retina is detached for too long, blindness may be irreversible even if it is reattached.

In the case of partial retinal detachment, the problem can be corrected by performing laser retinopexy through the pupil. The laser is used to create small burns around the defect which subsequently form a barrier that prevents the detachment from progressing further.

In recent years, vets have started performing vitreoretinal surgery in dogs. Correct patient selection is vital in these interventions as the procedure has a very high economic and technical cost.

When a blind dog is brought to the clinic due to bilateral retinal detachment, one of the eyes may have suffered detachment some time before

the other and the owners may have only noticed changes once the dog lost vision in its second eye.

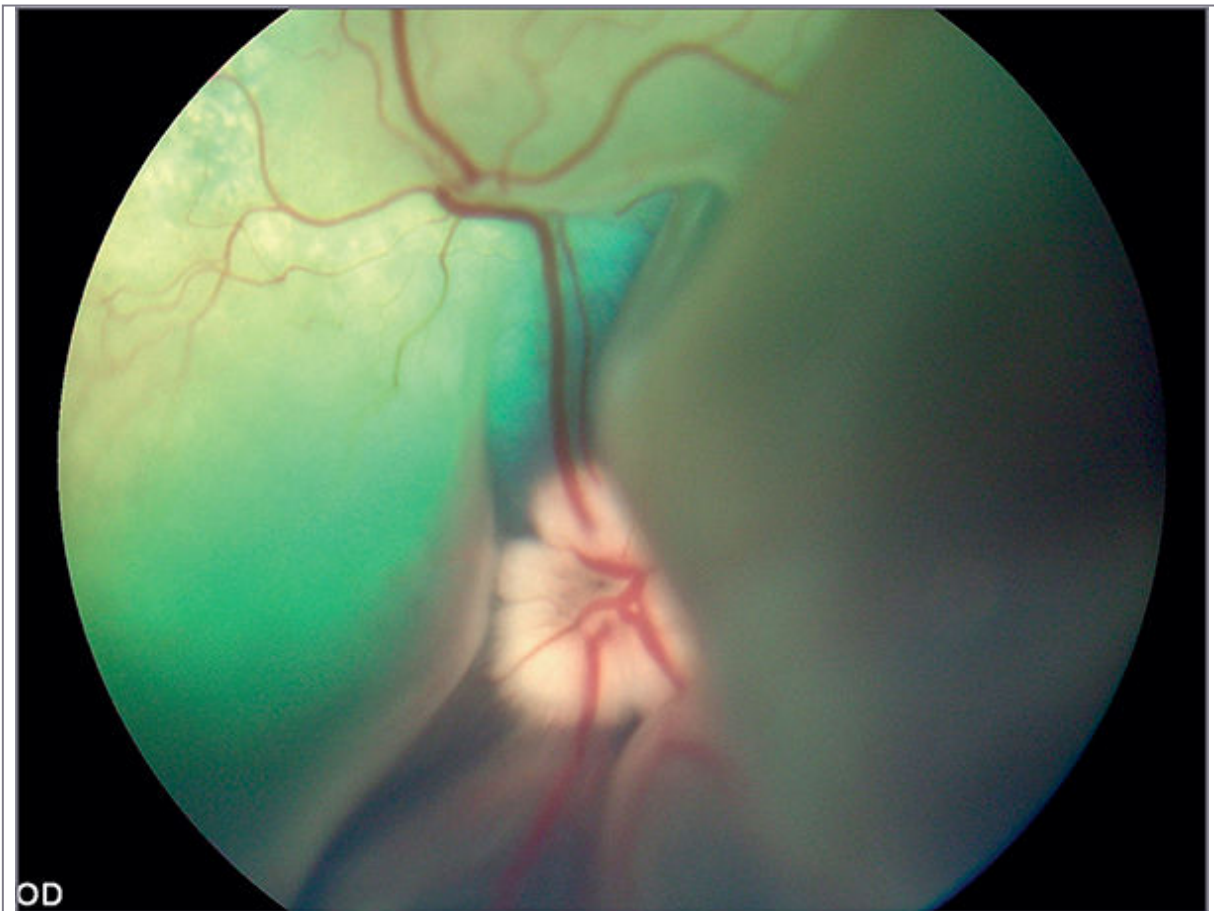


Figure 7. Bullous retinal detachment in a dog.



Retinal detachment

Diseases of the optic nerve

Because the optic nerve is enveloped by the meninges, many of the diseases that affect the nerve originate in the central nervous system.

Congenital diseases of the optic nerve

These are described in the chapter on congenital diseases.

Optic neuritis

When the optic nerve is inflamed, it interrupts axonal transport and therefore produces blindness. It can manifest as intraocular (inflammation focused on the optic nerve head) or retrobulbar optic neuritis. Additionally, while the presentation is normally bilateral, it can also be unilateral. There are many causes of optic neuritis:

- Meningoencephalitis: the inflammation may be disseminated or limited to the optic nerve (also called isolated optic neuritis).
- Infectious diseases: ehrlichiosis, distemper, neosporosis.
- Orbital traumas.
- Retrobulbar tumours, above all those which originate in nerve cells (meningiomas).

The main reason for consultation in animals with optic neuritis is blindness. Under examination, these patients will not have a direct pupillary reflex or menace response in the affected eye or eyes. An ocular fundus examination reveals a prominent optic disc with poorly defined borders and there is often peripapillary retinal detachment. Optic disc haemorrhages are also common (Fig. 8).

The patient should undergo a complete physical and ophthalmological examination in the neurological service, as well as complementary tests to characterise the disease (blood chemistry, haematology, serology, MRI, cerebrospinal fluid analysis). The prognosis for the dog's vision depends on the duration of the optic nerve inflammation and its cause. Some diseases that cause optic neuritis have a poor vital prognosis.

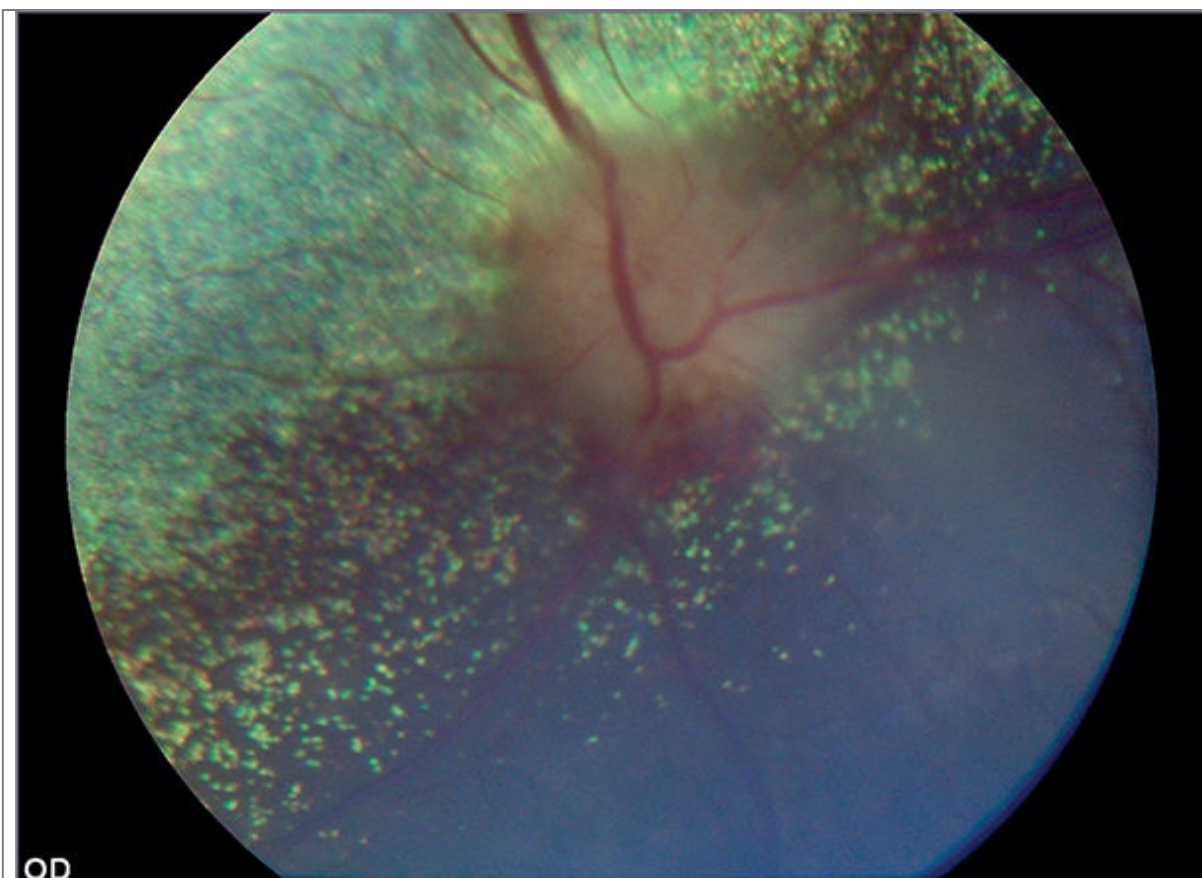


Figure 8. Optic neuritis. Note the prominent optic disc with a poorly defined border and haemorrhages.

Optic neuropathy

This is atrophy of the optic nerves secondary to another eye disorder. The affected optic nerve will look paler and smaller than normal. It can be caused by retinal degeneration, glaucoma, chronic optic neuritis, or chronic orbital diseases. There is no effective treatment, although the use of neuroprotective drugs is recommended.

Papilloedema

Given that the optic nerve is surrounded by the meninges, papilloedema occurs when there is an increase in intracranial pressure. This process occurs as a comorbidity of up to half of all intracranial tumours.

Unlike optic neuritis, papilloedema does not produce signs of inflammation (haemorrhages, vitritis, retinal detachment, etc.).

10

The orbit



Anatomy and physiology of the orbit

The eyeball is a fragile organ, which is why it is located within a solid structure called the orbit that protects the globe and its adnexa, besides supplying its vasculature and innervation.

The orbit has a conical shape with an anterior base, where the eyeball is situated, and a posterior vertex, where the extraocular muscles originate. The posterior region of the orbit is also home to most of the foramina, that is, the holes through which emerge a significant portion of the eye's innervation and irrigation.

Dogs, like all carnivores, have an open orbit where four-fifths of the orbital rim are osseous, while the rest is formed by the orbital ligament which extends from the zygomatic process of the frontal bone to the frontal process of the zygomatic bone. This 'open' design allows carnivores to open their mouths wider.

The orbit's location evidently determines the eyeball's position in the animal's head and this, in turn, has a definitive impact on their field of vision.

Since dogs are predators, the position of their orbits near the front of the head gives them a large binocular field of vision and a reduced monocular field.

The breed has a strong influence on the orbit's anatomy; dolichocephalic dogs have a deep orbit, whereas brachycephalic breeds have a shallower arrangement. This means the eyeballs of flat-faced breeds are more exposed and therefore more susceptible to conditions such as proptosis.

The orbit houses important accessory structures of the eye, such as the extraocular musculature which is formed by four recti muscles, two oblique muscles, and the retractor bulbi muscle. The orbit also contains three fasciae, namely the periorbita, orbital septum, and Tenon's capsule. These fasciae delimit what is known as the muscle cone and they help fix the position of orbital masses with respect to the orbit as either intraconal or extraconal.

There is a fat pad at the back of the orbit that affords the eye a certain degree of backward movement within its socket; during examination, this is called ocular retropulsion.

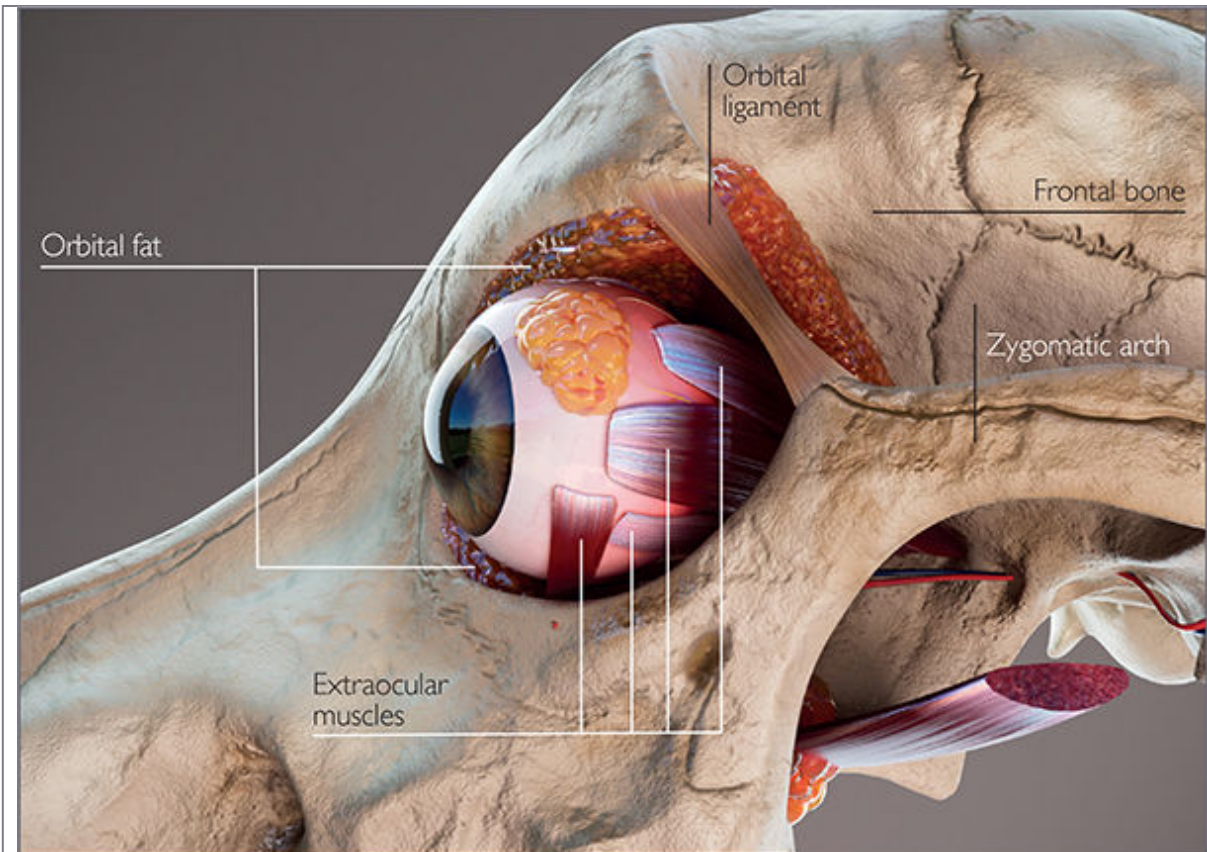


Figure 1. Structures of the orbit.

Examination of the orbit

As the orbit is a complex region with blood vessels, nerves, muscles, and fat all contained within a confined space, any change in the volume of these structures will result in the displacement of the eyeball or its adnexa. Consequently, the clinical signs of orbital diseases may include:

- Exophthalmos.
- Strabismus.
- Prolapsed nictitating membrane.
- Periocular inflammation.
- Pain upon opening the mouth.

It is important to differentiate clearly between exophthalmos, buphthalmos, and eyeball proptosis:

- **Exophthalmos** is the anterior displacement of the eyeball with respect to the orbital rim (Fig. 2). This situation is not an acute process, nor is it

accompanied by rupture of the ocular musculature. Furthermore, the intraocular pressure usually remains constant, or any increases are only very small. Exophthalmos usually courses with a prolapsed nictitating membrane and, since the eyeball is forcefully pushed outwards, retropulsion is impossible.

- **Buphthalmos** is a completely different scenario because it is due to an increase in eyeball volume and is not accompanied by any orbital disease. It is usually the consequence of a sustained increase in intraocular pressure caused by primary glaucoma or secondary glaucoma due to an intraocular disease, such as the presence of a tumour. Buphthalmos is not accompanied by a prolapsed nictitating membrane and retropulsion is usually possible, although the extent of retropulsion may be less than in the contralateral eye because of the increase in eyeball volume.
- **Proptosis** is the acute displacement of the eyeball secondary to trauma. It may or may not be associated with a bone fracture. As such, it corresponds to a medical emergency with a bleak prognosis in terms of preserving the patient's vision (Fig. 3).

If we revisit the concept of exophthalmos, we can appreciate that it does not always occur along the anteroposterior axis. When the causal mass is located extraconally, the eyeball will displace dorsally, ventrally, medially, or laterally depending on the mass' position on the orbit. If the mass is located intraconally, the eye will be displaced rostrally.

A proper examination must include a comparative study of both eyes viewed from above, a comparison of retropulsion, observation of the eyes at a distance, and assistance from complementary imaging techniques, such as an ocular ultrasound, CT, and MRI, is particularly helpful in reaching the correct diagnosis.



Figure 2. Overhead view used to compare both eyes. The right eye in the photo has exophthalmos and divergent strabismus.



Figure 3. An ocular proptosis just a few minutes after it occurred.

Diseases of the orbit

The diseases of the orbit can be categorised as either congenital or acquired.

Congenital diseases of the orbit

Briefly, the main congenital diseases of the orbit are:

- **Anophthalmos** : complete absence of the eyeball. This is very rare, and a detailed examination generally reveals the presence of a cystic eye.
- **Cystic eye** : an eyeball is present, although it is very small. The eyeball is nonfunctional and is usually devoid of any intraocular structures which are replaced by a fluid-filled tissue (Fig. 4).
- **Microphthalmos** : an eyeball of reduced size that may retain its functionality and which is associated with other congenital anomalies, such as a persistent pupillary membrane.

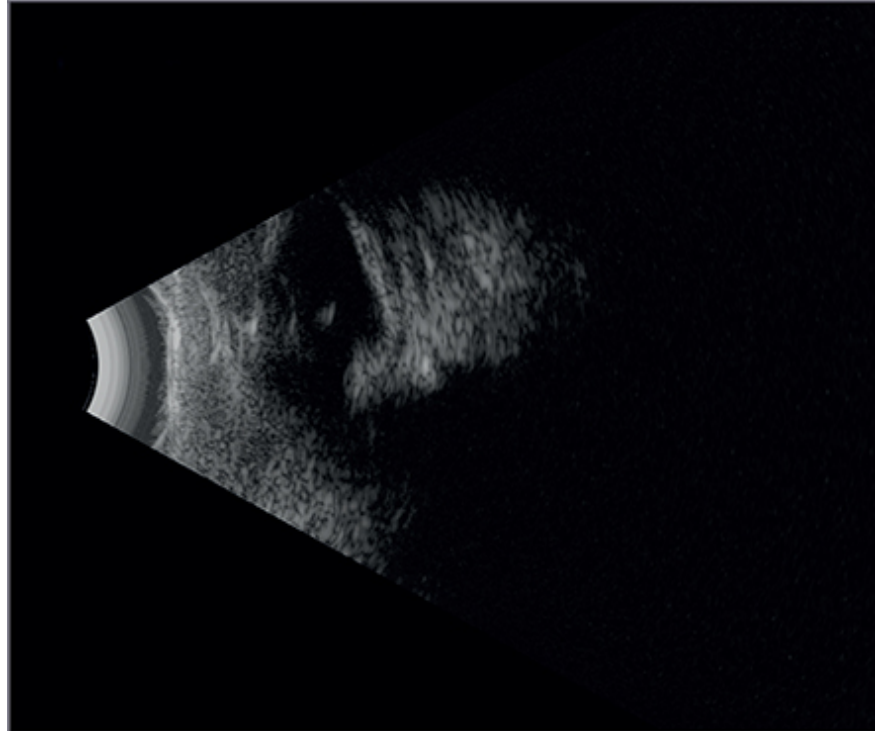


Figure 4. Cystic eye in a puppy detected by ultrasound.

Acquired diseases of the orbit

These may or may not be of an inflammatory nature.

Orbital cellulitis and abscesses

These diseases have different aetiologies, normally affect just one eye, and follow an acute evolution. Inflammation can occur in such a fashion that it is spread diffusely throughout the orbital structures or, alternatively, it may form an encapsulated collection of fluid, as in the case of an abscess.

Unlike neoplasms, which tend to develop at an average age of 9.5 years, cellulitis is diagnosed in younger animals, although vets must take into account that some tumours are surrounded by an inflammatory process and there may even be concomitant infection.

A complete eye exam is essential as it can sometimes reveal the entry point of foreign bodies or fistulous tracts.

Ultrasound provides a first impression of the location, appearance, and organisation of the area containing the accumulation of fluid (Fig. 5). The ultrasound study often needs to be complemented, if the focus can be

pinpointed, with a fine-needle aspiration to collect a sample for cytology and culture. Nevertheless, further imaging tests are usually necessary.



Figure 5. Left eye affected by an orbital abscess (a). The ultrasound image shows the fluid collection behind the eyeball (b).

Mucocele and salivary cysts

Mucocele and salivary cysts are two different conditions that develop when saliva accumulates in the orbit or other parts of the head.

Mucocele are the result of salivary gland trauma and do not have a genuine capsule. Salivary cysts, on the other hand, do have a capsule and their aetiology tends to be closely related to alterations of the oral mucosa which hamper correct drainage of saliva.

Myositis of the masticatory and extraocular muscles

Myositis of the muscles of mastication is an autoimmune disease common to breeds such as the German Shepherd Dog, Weimaraner, Golden Retriever, and Labrador Retriever.

This disease is characterised by very painful inflammation of the musculature which equates to acute discomfort upon opening the mouth. The presentation is bilateral, although not always symmetrical, and the clinical signs normally include fever, reduced appetite, periocular inflammation, exophthalmos, and a prolapsed nictitating membrane.

An MRI scan will highlight any inflammation of the masticatory muscles, which could evolve into fibrosis if the process becomes chronic.

Myositis of the extraocular muscles, also known as extraocular polymyositis, can affect any breed, but it is most often encountered in Golden Retrievers. It usually manifests in young dogs with exophthalmos and exotropia (divergent strabismus), but typically without a prolapsed nictitating membrane, and in many cases an ultrasound reveals an increase in the size of the affected musculature (Fig. 6).



Figure 6. Patient with extraocular myositis and severe periocular inflammation (a). Close-up of the right eye showing signs of severe conjunctival hyperaemia (b). Ultrasound image with increased echogenicity of the extraocular musculature (c).

Other orbital diseases

Orbital fat prolapse

Prolapse occurs due to weakness of the orbital fasciae, resulting in a subconjunctival mass consisting of normal fat tissue.

Resection of the mass and elimination of all the prolapsed tissue is generally curative.

Cranio-mandibular osteopathy

Above all, this problem tends to be found in West Highland White Terriers and Scottish Terriers, and it is particularly severe when it affects the temporomandibular joint. It is a self-limiting process and usually requires treatment with anti-inflammatories.

Tumours of the orbit

Tumours located in the orbit cause progressive and slightly painful exophthalmos, although if this courses with an infectious process, patients often develop acute clinical signs and fever.

They usually manifest in elderly animals and affect just one eye which does not allow positive retropulsion. Frequently they are primary malignant tumours and, depending on their character, are associated with nasal discharge, particularly if there is osteolysis (Fig. 7), or blindness when they originate in the optic nerve.

Osteolysis, the size of the mass, and its intracranial extension are considered negative prognostic factors, while treatment depends on several factors, although euthanasia is sometimes the most appropriate indication.

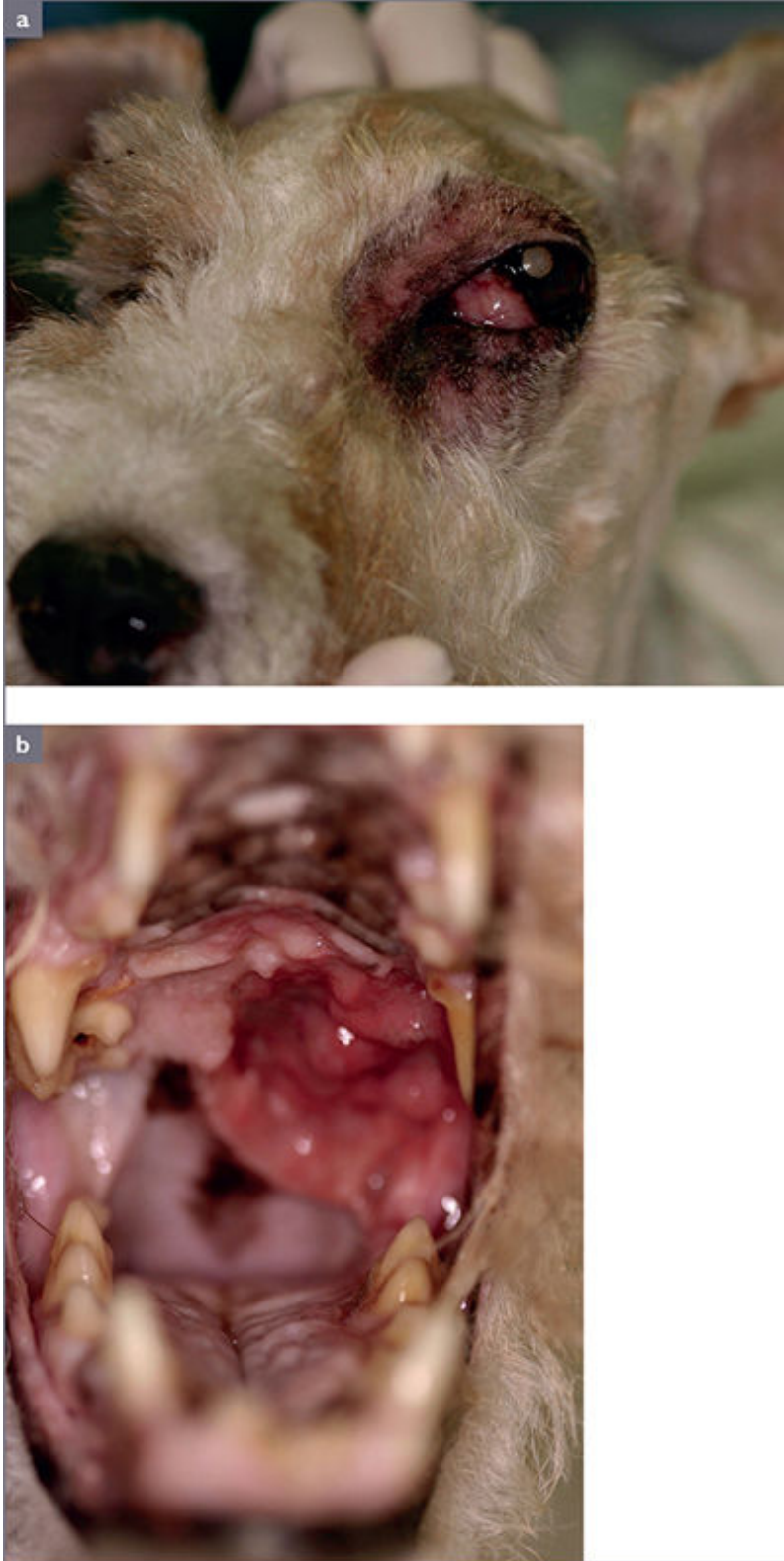


Figure 7. Dog with a bone tumour on the left orbit (a). Close-up of the palate region (b) with evident ulceration and tissue proliferation on the left-hand side.

Treatment of diseases of the orbit

Good management of the inflammation is a fundamental factor in diseases such as myositis because if the process turns chronic, the affected musculature may develop fibrosis and the associated sequelae. The autoimmune origin of these diseases means that corticosteroids should be prescribed at immunosuppressive doses before assessing changing to other immunosuppressants or immunomodulators once the first phase is under control.

For accumulations of fluid in the orbit, as with abscesses, after determining their location a microbiological study should be conducted, so a suitable antibiotic therapy can be administered and the collection drained if possible.

The intracavitary injection of sclerosing agents is another option for the management of cystic masses, such as mucoceles.

The drainage of abscesses through the oral cavity must be carried out with the utmost care to avoid damaging the eyeball when accessing the orbit via the palate.

The removal of orbital tumours represents a difficult surgical challenge which should be planned after assessing its size, location, and the viability of the affected eye (Figs. 8 and 9).

An orbitotomy is the approach of choice to access the orbit when trying to preserve the eye.

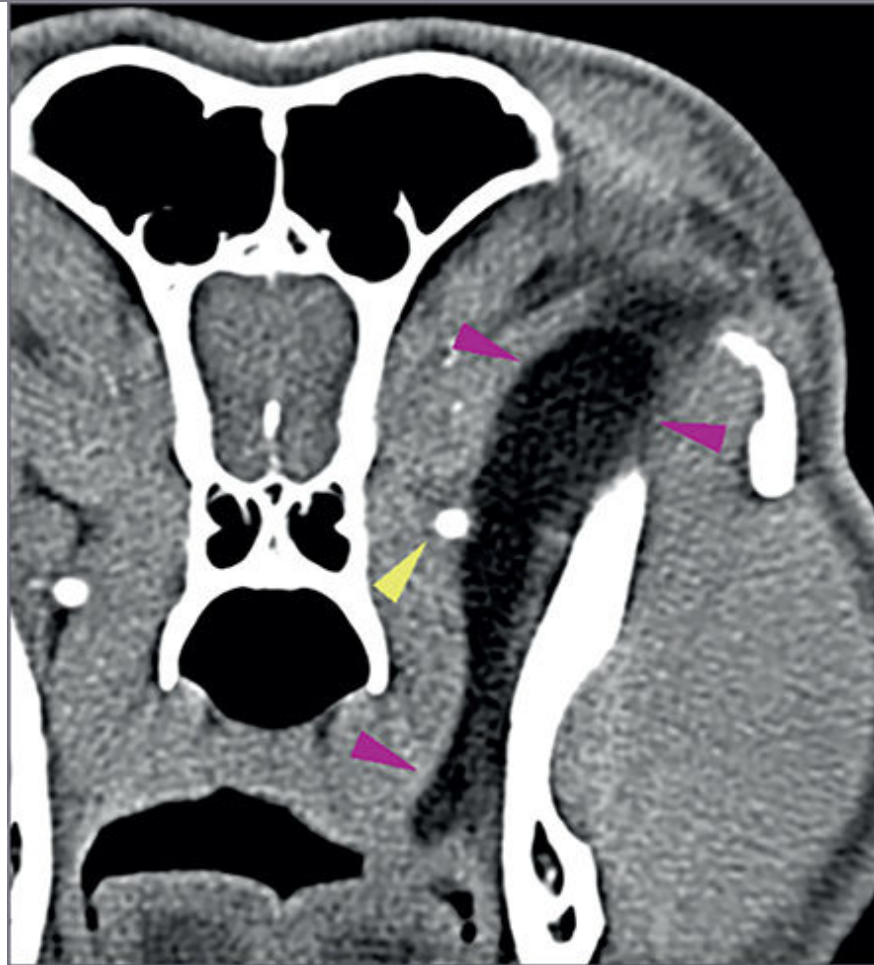


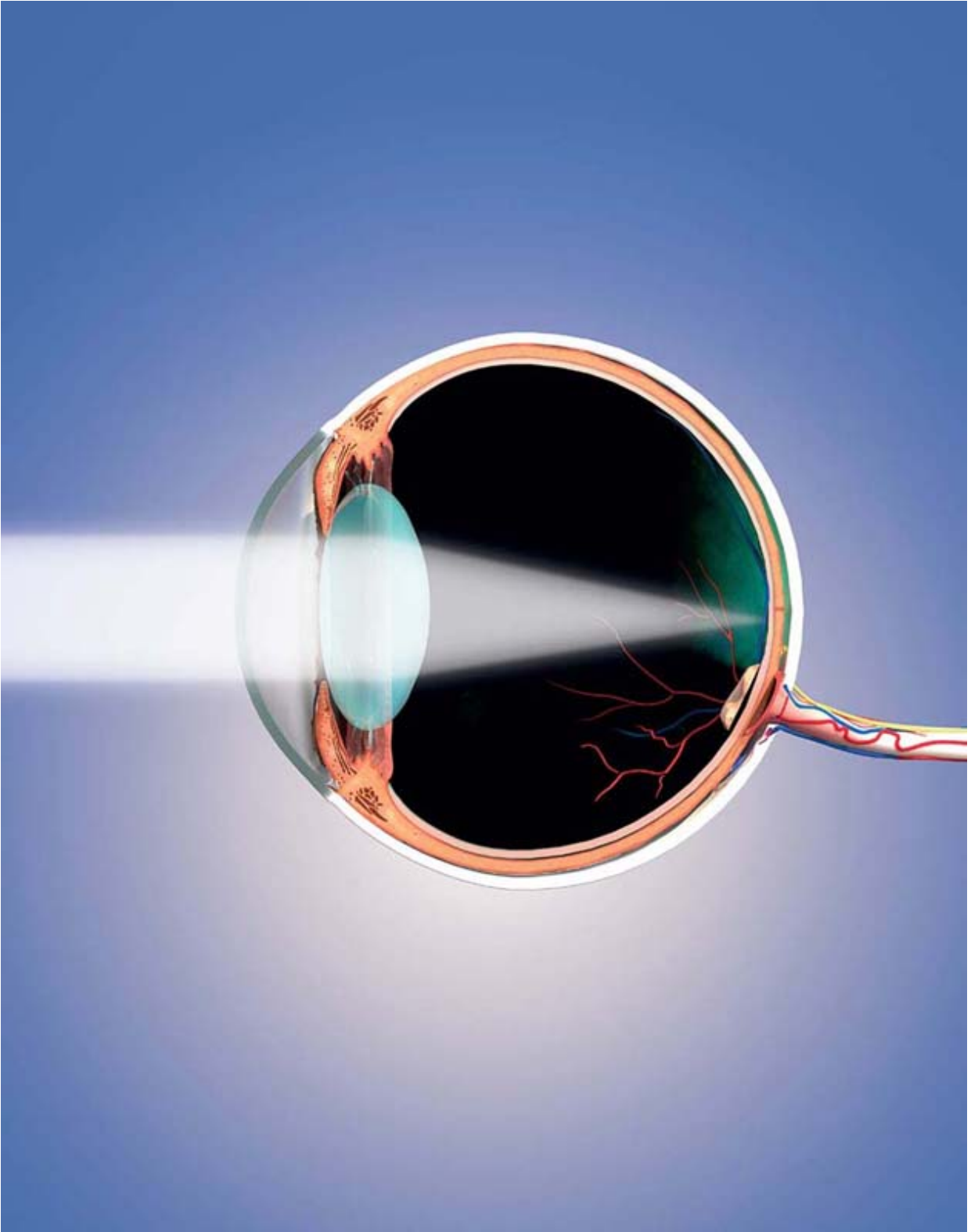
Figure 8. Computed tomography of orbital lipoma.



Figure 9. Full removal of the lipoma through the oral cavity.

11

Neuro-ophthalmology



The process of sight, in which light is converted into an electrical signal and sent to the visual cortex, the eye's innervation, and its accessory structures are all complex entities. Palpebral aperture, nociception, eyeball position, pupil aperture, and other processes are all controlled by afferent and efferent pathways.

All ophthalmological examinations should routinely include a neuro-ophthalmological assessment.

Neuroanatomy

Visual pathways

Ganglion cell axons merge to form the optic nerve (cranial nerve II), which passes through the sclera by means of the cribriform plate and then through the orbit until it reaches the optic foramen in the presphenoid bone. The optic nerves later join to form the optic chiasm. In dogs, 25 % of the fibres continue on the same side of the brain, while the other 75 % crossover. After the optic chiasm, the continuations of the optic nerve fibres are called optic tracts until they reach the lateral geniculate nucleus in the thalamus. At this point, 20 % of the fibres connect with different brainstem nuclei in order to transmit the signal for different reflexes, such as the pupillary reflex (Fig. 1). The remaining fibres project to the visual cortex where the image is recreated.

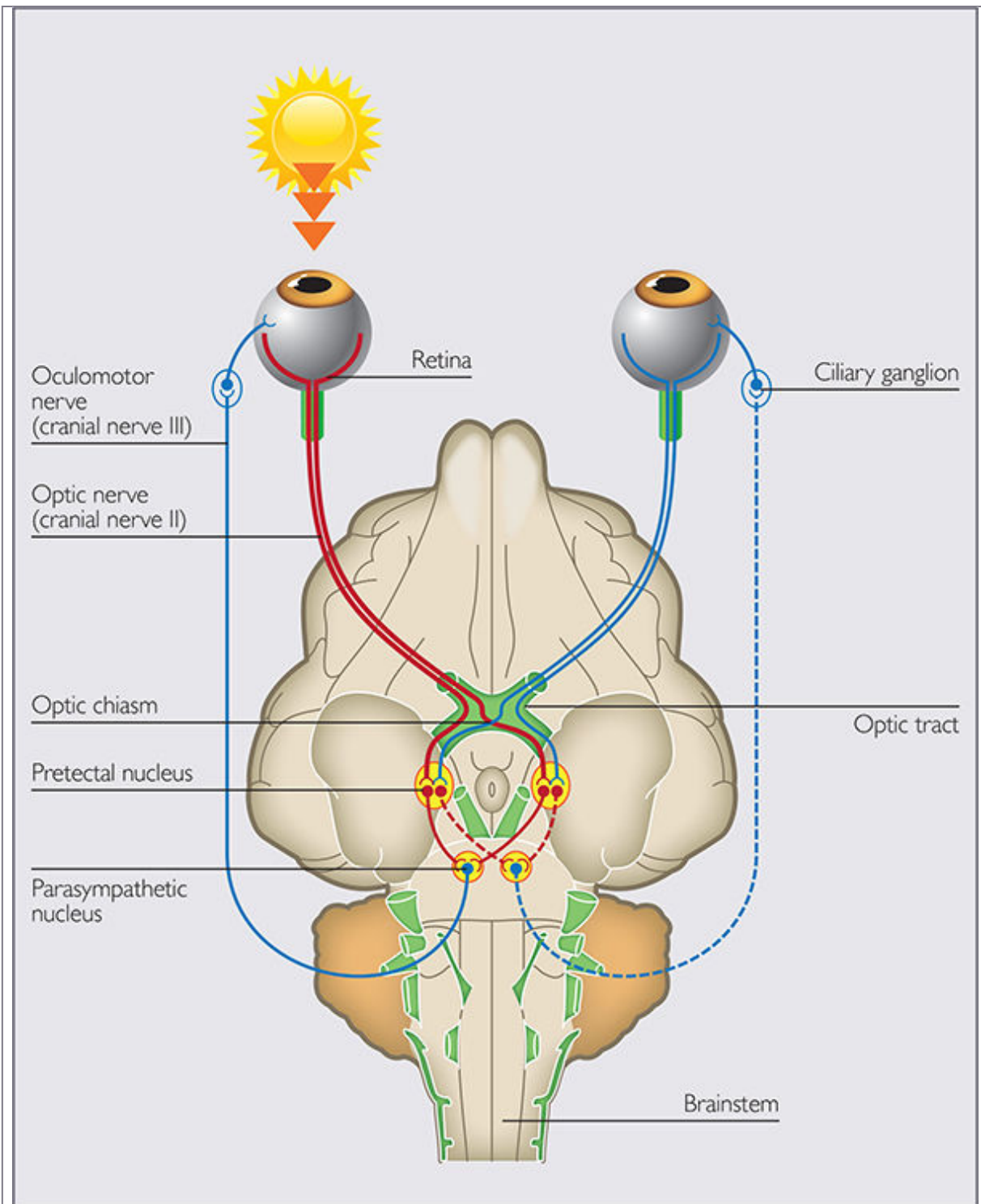


Figure 1. Neuroanatomy of the pupillary reflex.

Pupillary reflexes

The fibres that enter the brainstem to control the pupillary reflexes reach the pretectal nuclei where there is another decussation. They then emerge from the brainstem to innervate the ventral oblique and dorsal, ventral, and medial recti muscles. These fibres also supply parasympathetic nerves to the smooth muscles of the iris and ciliary body.

Neuro-ophthalmological examination

A neuro-ophthalmological exam is used to assess a patient's visual capacity and various reflexes and responses.

Menace response

This test confirms that the patient blinks when the examiner's hand approaches one eye while the other is covered (Fig. 2). The examiner must take care not to touch the patient or create a draft of air that could be detected by the animal and cause them to blink, thus producing a false positive.

In a physiological menace response, the afferent stimulus arises in the retina, the signal then travels along the optic nerve, chiasm, optic tracts, and lateral geniculate nucleus, before it finally emits optic radiation in the visual cortex. The stimulus is sent via association fibres to the motor cortex where it undergoes decussation at the bridge and continues on to the cerebellum. The efferent fibres run to the facial nerve nuclei (cranial nerve VII) which innervates the orbicularis oculi muscle (Fig. 3).

The menace response is learnt, so it is not completely developed until dogs are 14 weeks old.



Figure 2. Assessing the menace response in a cat.

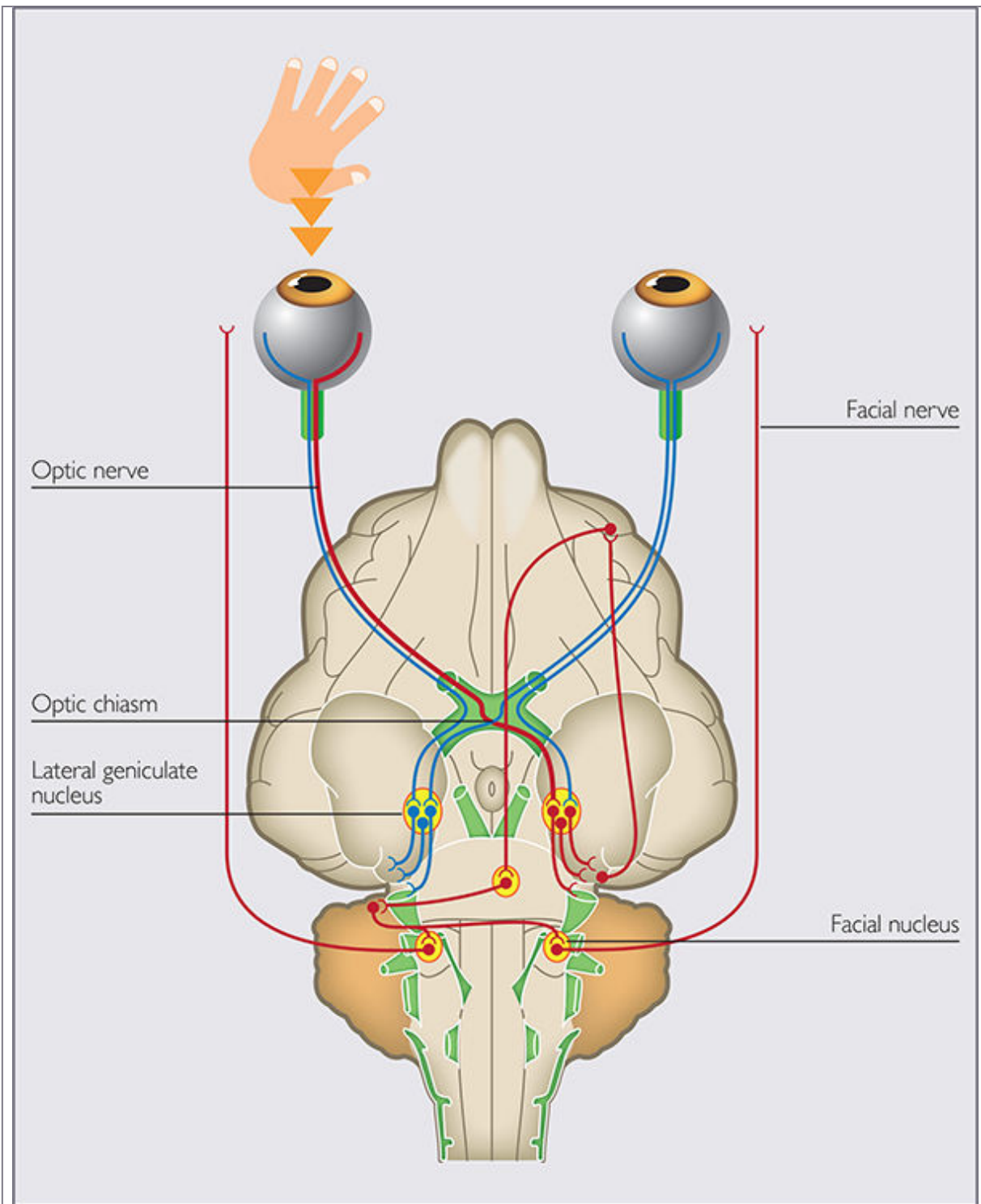


Figure 3. Nuclei and neural pathways involved in the menace reflex.

Palpebral reflex

Observe how the animal blinks when touching the skin close to its eye. Induce blinking by touching the skin close to the medial and lateral canthi, which are innervated by the ophthalmic and maxillary branches, respectively, of the trigeminal nerve (cranial nerve V). Then check the facial nerve (cranial nerve VII) produces a blink reflex by stimulating the orbicularis oculi muscle.

Dazzle reflex

This is a subcortical reflex (it is independent of what the animal sees) induced by stimulating the eye with a strong light. The dazzle reflex is positive in animals with media opacities (marked keratitis, cataracts) and a functional retina (Fig. 4).

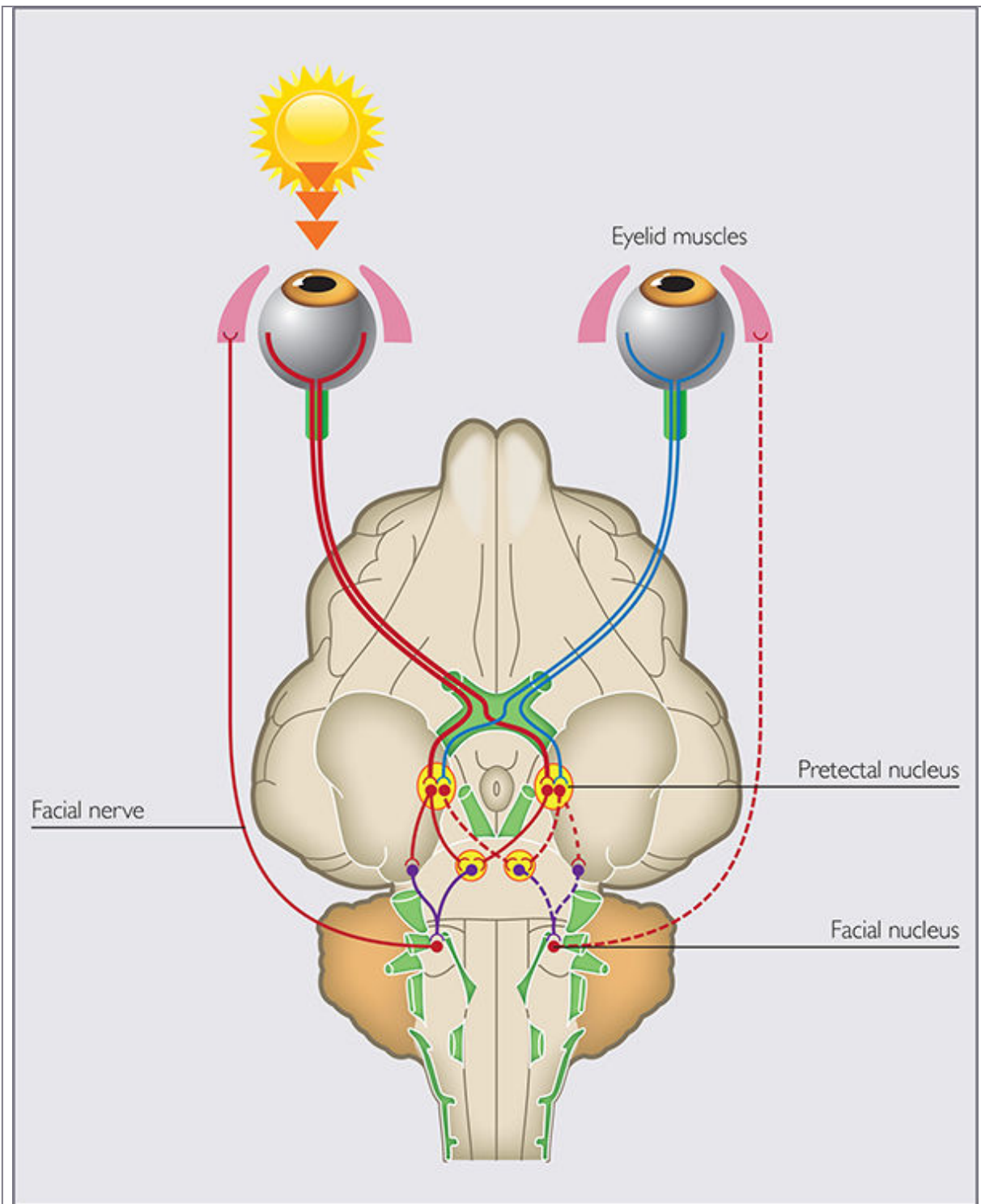


Figure 4. Nuclei and neural pathways involved in the dazzle reflex.

Pupillary reflexes

When assessing the pupillary reflexes, start by examining the pupils under normal room lighting conditions. Then turn off the light and confirm there is normal symmetrical dilation of the pupils. The pupils are controlled by the combined, antagonistic action of the iris sphincter and dilator muscles.

Other vision tests

Besides those described already, other tests may be conducted during the neuro-ophthalmological exam. The maze test consists of placing some simple obstacles around the examination room and observing how the patient navigates through them. It should be repeated under normal lighting and darkened conditions. The examiner can also use cotton balls or a laser pointer to study the patient's ability to track objects or lights.

Differential diagnosis of acute blindness

- Ocular media opacity:
 - Very marked keratitis
 - Corneal perforation
 - Anterior luxation of the crystalline lens
 - Cataracts
 - Glaucoma
 - Intraocular haemorrhage
 - Severe uveitis
- Sudden acquired retinal degeneration syndrome (SARDS)
- Retinal detachment
- Optic neuritis
- Cortical tumours

Alterations responsible for defects in either the visual or pupillomotor pathways

From a neuroanatomical perspective, an animal with normal pupillary reflexes but a lack of vision in both eyes must have suffered damage to the cerebral cortex. The injury may have an inflammatory origin, such as in the case of granulomatous meningoencephalitis, contusions, or cerebral oedema.

Brain tumours can also cause blindness due to the mass effect or perilesional inflammation. Lastly, such defects may be secondary to infectious diseases, postanaesthetic hypoxia, or hepatic encephalopathy.

Curiously, sometimes the lesions are located at the point before the pupillomotor fibres separate from the visual fibres, given that a pupillary reflex can still be induced with just a few fibres. So, it is possible to come across blind animals with retinal degeneration or cataracts but a normal pupillary reflex.

Alterations responsible for defects in both the visual and pupillomotor pathways

All lesions located before the lateral geniculate nucleus will result in a total loss of vision and pupillary reflexes because the fibres share the same path. The most common causes affect the retina or optic nerve, namely glaucoma, retinal detachment, retinal degeneration, and optic neuritis. If the problem is unilateral, the patient will show signs of anisocoria at rest with mydriasis in the affected eye. If the chiasm is compromised, there will be bilateral areflexic mydriasis and blindness. Lesions affecting the optic tracts are rare.

Alterations of the pupillary reflexes without visual deficit

The causes of pupillary deficits with unaffected vision are located in the efferent nerve pathway. The lesion may affect the innervation or muscles of the iris. The most common neurological causes are Horner's syndrome (as detailed below), damage to the oculomotor nerve (cranial nerve III) affecting only the autonomic fibres (internal ophthalmoplegia) or also the extraocular muscles (external ophthalmoplegia), and cavernous sinus syndrome. Other conditions that can result in abnormal pupillary reflexes include:

- Uveitis
- Glaucoma
- Synechiae
- Iris atrophy
- Persistent pupillary membrane

Horner's syndrome

Horner's syndrome involves sympathetic denervation of the eye and its adnexa. Consequently, it has the following clinical signs:

- Palpebral ptosis due to a loss of tone of Müller's muscle.
- Enophthalmos caused by a loss of tone in the periorbita.
- Prolapsed nictitating membrane secondary to enophthalmos.
- Miosis due to denervation of the iris dilator muscle.

Given the complicated neuroanatomy of the eye's sympathetic innervation, the lesion may be located in the:

- Spinal cord (C1 to T3): focal myelopathy, fibrocartilaginous embolisms, tumours.
- Brachial plexus: avulsion, tumours.
- Cranial mediastinum: lymphoma, thymoma.
- Middle ear: otitis, polyps, tumours.
- Retrobulbar region: tumours, abscesses.

In dogs, Horner's syndrome is idiopathic in up to 60 % of cases. The disease is particularly prevalent among Golden Retrievers, above all females. As many as 90 % of cases in this breed are classified as idiopathic.

An eye drop test with 1 % phenylephrine is very useful in the diagnosis of Horner's syndrome since postganglionic lesions may cause hypersensitivity due to denervation. This means that the clinical signs will return within 20 minutes of instilling the eye drops. Additionally, the vet should perform an ear exam, a neurological exam, and take chest/neck X-rays. Phenylephrine or apraclonidine eye drops can be prescribed as symptomatic treatment.

Facial paralysis

This condition courses with dysfunction of the facial motor nerve (cranial nerve VII). The primary clinical signs are facial asymmetry, a drooping lip on the affected side of the face, absence of blinking, and dry eye. Dogs with facial paralysis can still withdraw the eyeball because the corresponding muscle is not innervated by the facial nerve.

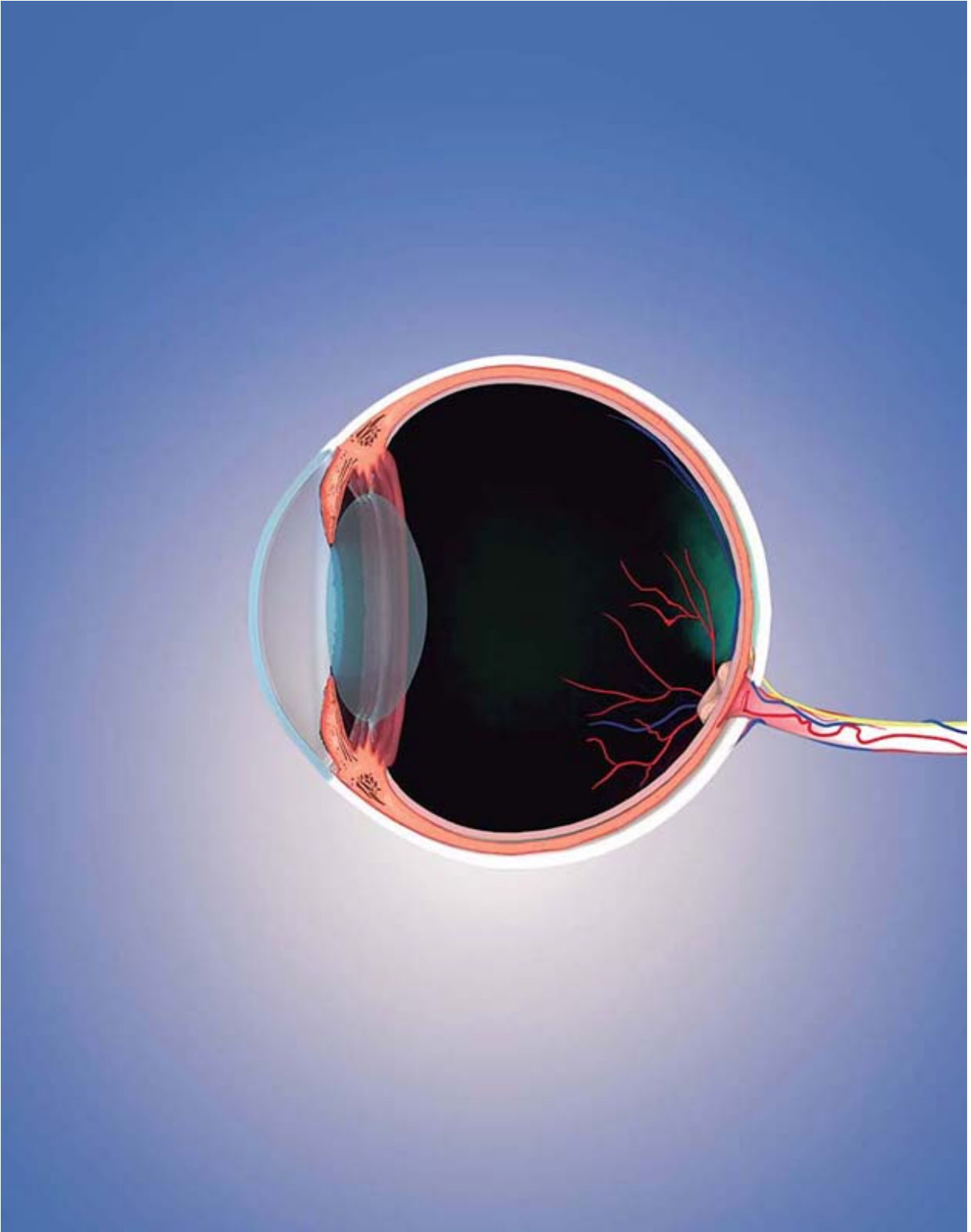
The facial nerve is essential for maintaining a healthy ocular surface as it innervates the main lacrimal gland, and because blinking spreads the tears that clean the surface.

The main aetiologies of facial paralysis are idiopathic, ear canal surgery, trauma, tumours, and otitis. There may also be concomitant diseases, such as hypothyroidism, Horner's syndrome, keratoconjunctivitis sicca, and so on.

Treatment of facial paralysis is based on the use of artificial tears and ocular lubricants to compensate for the lack of lacrimal secretion. This can be supplemented with lacrimal secretion stimulants, such as oral pilocarpine or cyclosporine. If the paralysis cannot be reversed, or drops cannot be instilled frequently, we recommend a temporary tarsorrhaphy to reduce evaporation and protect the ocular surface.

12

Congenital eye diseases



The organogenesis and tissue differentiation phenomena that occur during the embryonic development of the eye and its accessory structures can suffer deviations, of either a genetic or nongenetic origin, and therefore have a certain degree of heritability. Accordingly, it is important to highlight that these developmental alterations may be triggered by circumstantial causes, such as embryonic diseases or substance intoxications, or as a result of hereditary diseases.

These anomalies will cause what are known in ophthalmology as ocular defects; defects in one or more structures that entail a minor or major deficit in the eyes' functionality and have a variable impact on vision depending on when they occur during embryogenesis, their location, and their severity.

Broadly speaking, the eyes can be said to start their embryonic development from the optic pits or sulci, which originate on both sides of the end of the neural tube. The neural tubes later close to form two vesicles that grow until they come into contact with the surface ectoderm, at which point the crystalline lens placode forms. Subsequently invagination gives rise to a bilayer structure called the optic cup.

The optic cup gradually invaginates and its initially incomplete closure produces the optic fissure, the point of origin of the developmental anomalies described below. Finally, the two layers of the optic cup fuse to form the first "intraocular space".

In parallel to the above, the ocular vasculature develops from the mesenchyme that enters the optic cup via the optic stalk (which, in turn, connects it to the neural tube) and forms the origin of the hyaloid artery.

The lens placode thickens and transforms into a vesicle that is profusely irrigated from the hyaloid artery via the vascular tunic. These blood vessels should atrophy and disappear completely in the adult eye by the time the dog is approximately 4 months old.

It is important to bear in mind that developmental alterations can appear immediately after birth, so they can be classified as congenital, or at any other moment in the animal's lifetime; either way, this is independent of whether or not it is a hereditary disease.

Another significant factor to consider is that developmental anomalies often occur in clusters, so the affected eye will present more than one ocular defect.

The identification of each and every ocular deficit will allow vets to determine their origin, select a possible treatment, and make a prognosis about the future functionality of the organ.

Colobomata

Colobomata are worthy of special mention in this chapter in order to clarify their definition. The term refers to the underdevelopment of a segment of a specific structure in the eye.

Returning to the optic fissure's role in the embryonic development of the eye and its inferonasal location, it is comprehensible that its incomplete closure may result in a lack of tissue in a given ocular structure and its position will always coincide with this quadrant of the eye. This situation is an example of a so-called typical coloboma.

There is also a possibility that errors during embryonic development may cause a coloboma in an ocular structure that is not directly related to the incomplete closure of the optical fissure and, as such, may appear in other locations. These are known as atypical coloboma.

Anophthalmos and microphthalmos

The complete absence of the eyeball (anophthalmos) is quite rare in dogs, unlike the occurrence of abnormally small eyes (microphthalmos) which occupy a smaller volume than the space available in the orbital cavity.

Very small eyeballs may be concealed behind the nictitating membrane and an ultrasound could be required to determine the presence of a residual optic vesicle. In such cases the eyelashes often brush against the conjunctivae and patients may have entropion.

Vets should be aware that a microphthalmic eye does not necessarily imply blindness as all the intraocular structures may be present, but they will be affected by the corresponding reduction in their dimensions (Fig. 1).

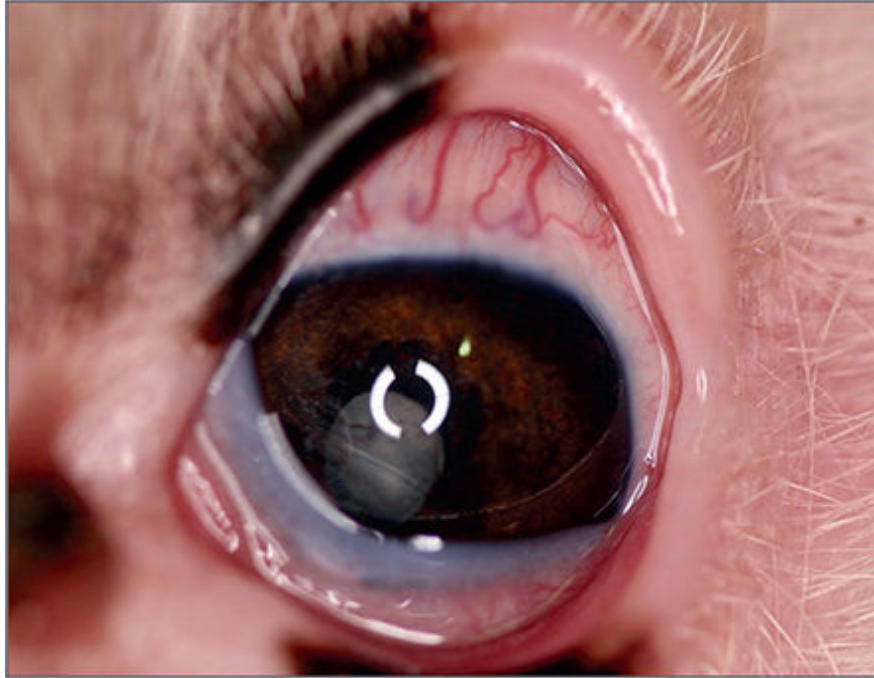


Figure 1. Eye with congenital cataracts and microphthalmos.

Congenital diseases of the eyelids and conjunctivae

Eyelid colobomata

This corresponds to a partial developmental defect of one or more eyelids. Complications of varying severity will arise depending on which area is affected, for example the appearance of keratitis due to exposure and excessive tear evaporation, abrasions from eyelashes and periocular hairs, and so on.

Treatment revolves around techniques to reconstruct the defective segment of eyelid and therapies to prevent hairs and eyelashes from rubbing against the ocular surface (cryotherapy, electrolysis, etc.).

Dermoids or choristomas

These have already been covered in Chapter 4 . They are caused by the abnormal presence of dermal tissue, including hairs and glands, on the eyelid margins, conjunctivae, or cornea, or even on several structures at the same time (Fig. 2).

The extension and position of these dermoids can sometimes imply a decline in vision and even blindness.

Their removal is essential to avoid constant irritation of the ocular surface and the subsequent consequences, which may include ulcers due to abrasive hairs or the formation of pigment deposits on the cornea (Fig. 3).



Figure 2. Dermoid affecting the eyelids, conjunctiva, and cornea.

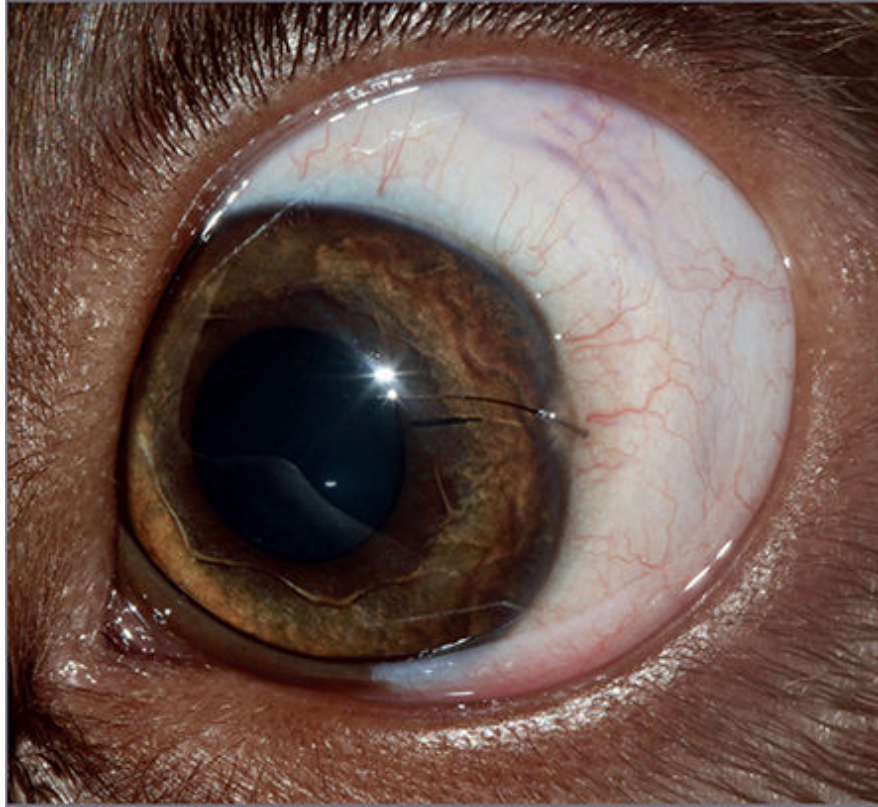


Figure 3. Small conjunctival dermoid with just a single hair. Remnants of the pupillary membrane are also visible.

Congenital diseases of the lacrimal system

Aggenesis or hypoplasia of the lacrimal glands

A total or partial lack of development of the lacrimal glands produces the corresponding deficit in the aqueous component of the tear film (Fig. 4). The severity is directly proportional to the manifestation of clinical signs such as dry secretions at the eyelid margins, strands of mucus in the conjunctival sacs, a lack of corneal lustre, and signs of severe discomfort, such as photophobia, blepharospasm, and a prolapsed nictitating membrane.

The affected eye is prone to the appearance of complications including corneal ulcers and, in the medium-to-long term, melanin deposits which will affect the cornea's transparency.

Treatment of this defect is based on the frequent instillation of artificial tears and ocular lubricants, and once the animal reaches adulthood, surgical techniques such as parotid duct transposition can be considered.



Figure 4. Congenital alacrima in the left eye of a Pinscher.

Imperforate and minimal aperture of the lacrimal puncta

Congenital defects of the lacrimal puncta may affect one or both ducts; in patients this manifests as epiphora and signs of conjunctival irritation such as hyperaemia and hyperplastic follicles. Similarly, dogs often have dermatitis near the medial canthus because the region is always moist due to the epiphora.

Surgical enlargement or opening of the affected puncta is the treatment of choice.

Agensis of the nasolacrimal duct

This refers to the absence of all or part of the lacrimal drainage system, with clinical presentations similar to those described in the previous section.

The use of complementary techniques, such as contrast radiography (dacryocystography) and other imaging techniques (computed tomography), provides detailed information on the extent of the defect.

Nevertheless, sometimes it proves impossible to pinpoint the affected segment, especially if it is relatively large.

Alternative tear drainage techniques, such as conjunctivobuccostomy and conjunctivorhinostomy, are not exempt from complications and recurrences.

Congenital diseases of the cornea and sclera

Corneal dermoids

These have been discussed previously. Nevertheless, it is important to underline that in puppies a dermoid may appear to be just a small spot on the cornea without any signs of discomfort, but as the dog grows, the dermoid will also develop, and the gradual appearance of hairs will trigger severe lesions.

Subepithelial dystrophy

Subepithelial dystrophy is expressed as the loss of transparency in regions of the cornea, which are observed as whitish opacities, usually located near the centre, and with no signs of discomfort (Fig. 5). It tends to disappear progressively in the first 2 months of life.

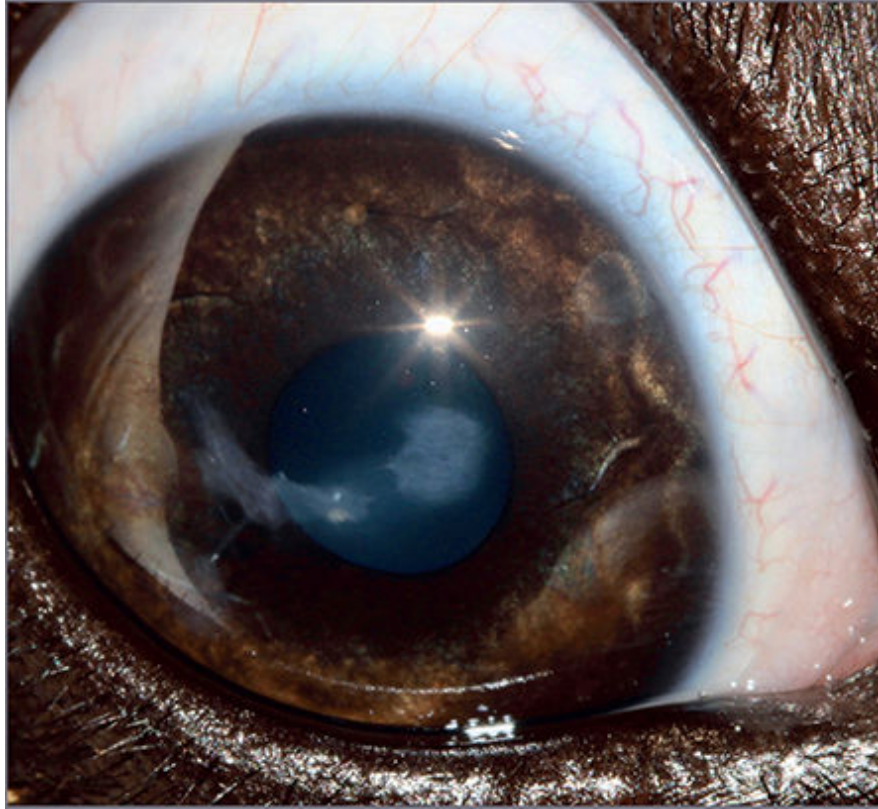


Figure 5. A mild case of congenital corneal dystrophy.

Iridocorneal persistent pupillary membrane

The failure of the uveal tract to fully regress leads to strands of tissue that can extend from the iris collarette to the corneal endothelium. This contact with the cornea provokes the appearance of opacities and, over time and depending on the extension, may even produce oedema and melanisation (Fig. 6).

If they are only small and do not affect the visual axis, the patient will not display any signs of vision deficit.



Figure 6. Persistent pupillary membrane and congenital cataract.

Scleral coloboma

This is the congenital absence of part or the entire thickness of the sclera, manifesting as a dark patch under the conjunctiva. This coloured area is the underlying uvea, and if the defect is extensive, it can herniate and require a graft of scleral tissue or another biomaterial.

Scleral colobomata are not uncommon at the back of the eye, close to the optic nerve head. If they are large, they can cause retinal detachment and even originate a retroocular cyst.

Congenital diseases of the iris

Persistent pupillary membrane

This occurs when the pupillary membrane, which nourishes the anterior region of the crystalline lens, fails to regress as it normally does in the final stages of the eye's development, which includes the first few weeks of life.

It presents with strands of tissue that span from the iris collarette to the cornea (iridocorneal persistent pupillary membrane), to the crystalline lens

(iris-to-lens), and even bridge across the pupil to another section of the iris (iris-to-iris). Since it is not an inflammatory process, there are no associated signs of uveitis.

More often than not, the visual axis remains unaffected and so there is no visual impairment.

Abnormal pupils

This section does not relate to diseases that course with iris synechiae which distort the shape and location of the pupil, as these are acquired alterations secondary to other processes that are generally of an inflammatory origin. Here we deal with congenital disorders of the pupil, such as:

- Aniridia: absence of the iris. This is rarely encountered in dogs.
- Coloboma: defect in a segment of the iris. If it is a typical coloboma, the defect will be located in the inferonasal quadrant.
- Dyscoria: pupil with an irregular, noncircular shape. May be due to the presence of a coloboma.
- Corectopia: pupil displaced from its central position.
- Polycoria: presence of more than one pupil.

Congenital hypoplasia

This involves the presence of underdeveloped regions of iris, including authentic holes that can tend to grow larger with age. They are more common in light-coloured irises.

Iris discolorations

These may include:

- Albino iris: very uncommon in dogs and manifests as a pinkish iris.
- Subalbinotic iris: a reduced amount of pigment, observed as a bluish colour in part or all of the iris.
- Iris heterochromia: is the presence of colour variations in a patient's irises. When the two irises present different colours, it is known as heterochromia iridium or complete heterochromia. If the different colours are present in the same iris, the anomaly is known as heterochromia iridis or sectoral heterochromia (Fig. 7).

It is interesting to note that these deviations in the coloration of the iris will also manifest as differences in the distribution of the retinal pigment, so the appearance of the iris can give us an indication about what to expect in the retina.

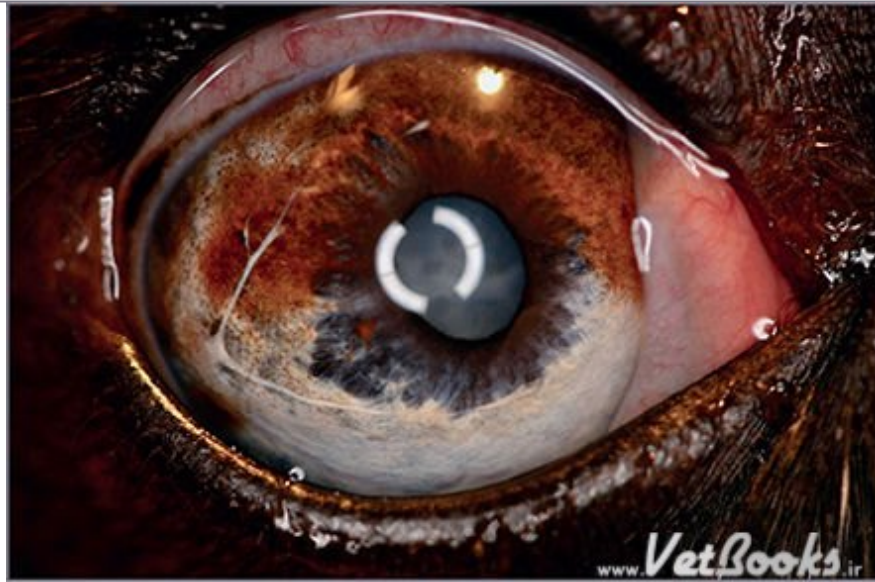


Figure 7. Partial heterochromia iridis and remnants of the pupillary membrane in a Collie.

Iris nevus

Nevi are hyperpigmented, generally circular regions in the iris.

As they are nonneoplastic, vets should take advantage of each visit to the clinic to check that a patient's nevus or nevi do not grow or change shape over time.

Waardenburg syndrome

This can present in dogs with a white coat and in combination with heterochromia iridis and deafness.

Congenital diseases of the crystalline lens

Alterations in size and shape

For example:

- Aphakia: total absence of the crystalline lens. This is a rare defect in dogs.

- Microphakia: abnormally small crystalline lens. The characteristic image is that of a clearly visible crystalline lens equator and insertion of the zonular fibres which, in this case, are stretched in order to reach the lens from the ciliary processes.
- Spherophakia: the crystalline lens becomes more spherical than its usual lenticular shape.
- Lenticonus: can affect anterior or posterior regions. From a lateral perspective, the affected pole loses its normal curvature and bulges, giving it a very characteristic ultrasound appearance. With posterior lenticonus, the clinician must take into account its association with other developmental abnormalities, such as persistent primary vitreous and even persistent hyaloid artery.
- Coloboma: appears as a defect on the equator which loses some of its spherical nature.

Congenital cataracts

Contrary to popular belief, cataracts can have a congenital origin and may be accompanied by other ocular defects, such as the persistence of the pupillary membrane, the tunica vasculosa lentis, and remnants of primary vitreous or the hyaloid artery (Fig. 8).

The location and appearance of a cataract detected in a young patient may provide insight as to whether it is a congenital process. This is important in certain breeds given their confirmed heritability.

These congenital cataracts, which can manifest as small, posterior subcapsular opacities, may be stable throughout the animal's lifetime or they may progress towards mature cataracts and therefore require treatment.

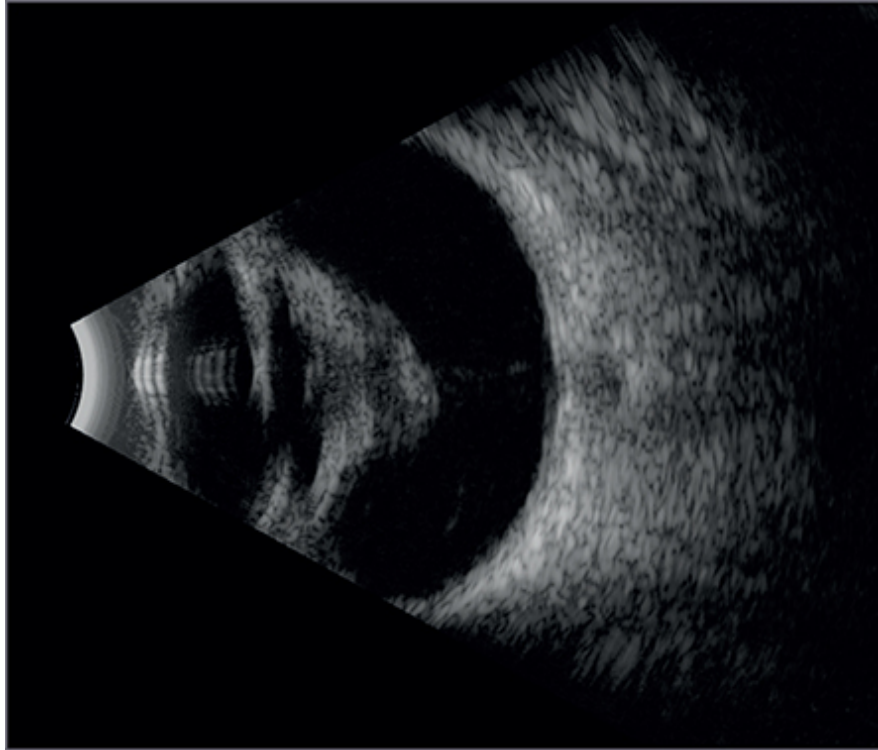


Figure 8. An eye presenting a congenital cataract, persistent hyperplastic primary vitreous, and remains of hyaloid artery.

Congenital diseases of the vitreous

These are not uncommon and, as indicated previously, may be accompanied by other abnormalities in other areas of the eye.

Persistent hyaloid artery

The hyaloid artery corresponds to the primary embryonic irrigation of the crystalline lens and normally disappears in the first few weeks of life. However, remnants or the entire artery can persist and even remain functional (confirm with Doppler ultrasound).

This vasculature, which arises from the optic nerve head and runs to the posterior pole of the lens, may present with other developmental abnormalities, such as posterior subcapsular or capsular cataracts.

Persistent tunica vasculosa lentis

The tunica vasculosa lentis is the branch of the hyaloid artery running along the posterior pole of the crystalline lens. If it does not regress as normal before eyeball maturation, a latticework structure will appear adhered to the posterior capsule.

Persistent hyperplastic primary vitreous

Besides persistent hyaloid artery and tunica vasculosa, another defect is persistent hyperplastic primary vitreous.

The anomaly manifests as a plaque in the posterior pole of the lens and ultrasound reveals a triangular structure emerging from the pole. The remnants of blood vessels persist in this structure and may be accompanied by vitreous haemorrhage, posterior lenticonus, and even lenticular haemorrhage.

The surgical approach to these cases should consider removing all of these embryonic structures to provide transparency along the visual axis.

Congenital diseases of the retina

Retinal dysplasia

This is a significant anomaly in dogs that produces folds within the layers of the sensory retina. There are three well-differentiated patterns:

1. Geographic retinal dysplasia: appears as irregular areas delimited by greyish-white regions. These irregularities correspond to retinal thinning, observed as hyper-reflective foci in the tapetal fundus, while other areas are hyperpigmented due to hypertrophy of the retinal pigment epithelium which is found under the neuroretina.
2. Focal or multifocal retinal dysplasia: in this case the examiner can observe retinal folds. These folds appear as greyish streaks in the tapetal fundus and whitish streaks in the nontapetal area (Fig. 9).
3. Retinal detachment secondary to complete dysplasia: in this defect, the retina is only attached around the optic nerve head. There is significant visual impairment, if not blindness, which could also be complicated by vitreous haemorrhage.

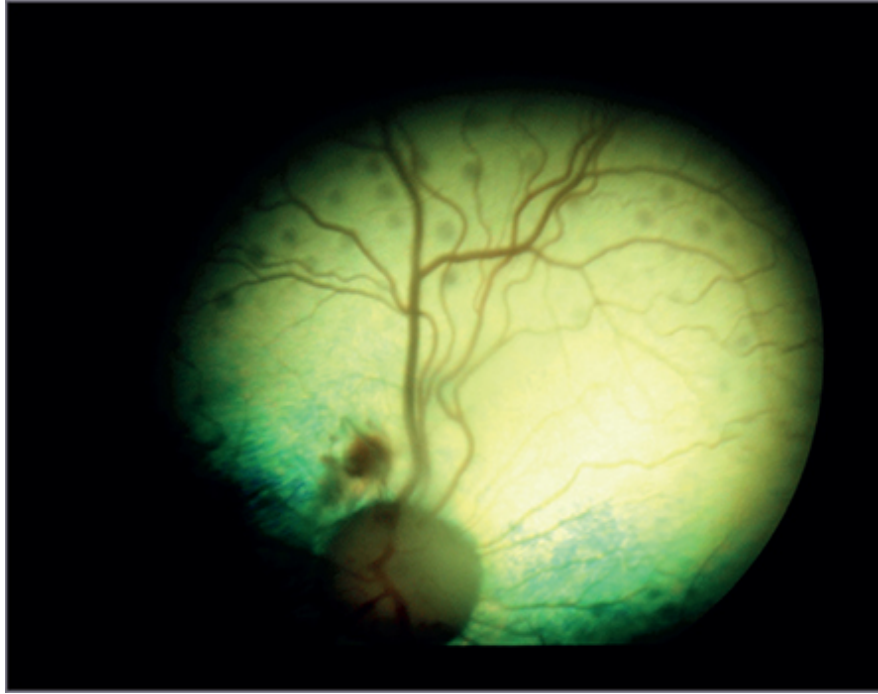


Figure 9. Remnants of hyaloid artery and retinal dysplasia.

Collie eye anomaly (CEA)

CEA is a congenital defect seen in a high proportion of Collies, but it has also been reported in Rough Collies, Australian Shepherds, and Whippets.

The disorder manifests with a combination of various anomalies, such as choroidal hypoplasia in the temporal region, retinal dysplasia, and abnormal retinal blood vessels.

The hypoplasia allows the examiner to observe a choroid with thick, abnormal vessels separated by large spaces and set against the clear background of the sclera.

Affected eyes often suffer retinal detachment and haemorrhages, which can lead to blindness. Colobomata of the optic nerve head are also common in these patients (Fig. 10).

Retinal colobomata

As we have already seen, these are related to the incomplete closure of the optic fissure, and they may affect the retina and other ocular structures.

In the case of colobomata affecting the retina and choroid, they appear as hyperpigmented regions where only the sclera is visible. If the sclera is also

compromised by the defect, the given area will appear as an indentation under fundoscopy and ultrasound.

Congenital diseases of the optic nerve

Among the developmental defects with an impact on the optic nerve are:

- **Aplasia:** total absence of the optic nerve and accompanying retinal vessels. It is a rare condition in dogs and results in blindness.
- **Hypoplasia:** this anomaly is much more frequent. The optic nerve forms from the convergence of fibres that emerge from the retinal ganglion cell layer. Fewer ganglion cells give way to fewer nerve fibres and therefore a smaller optic nerve. This means there is a decline in vision and pupillary light reflexes, whose intensity is directional proportional to the number of fibres in the optic nerve.
- **Colobomata of the optic nerve head:** these can affect the optic disc or peripapillary areas. As we saw for the iris, typical colobomata are located in the inferomedial quadrant of the optic nerve head and atypical ones in any other region. Colobomata do not necessarily imply blindness, provided that they do not compromise the entire optic disc and therefore leave some functional nerve fibres. Fundoscopy typically reveals blood vessels “sinking” at the edges of the coloboma (Fig. 10).

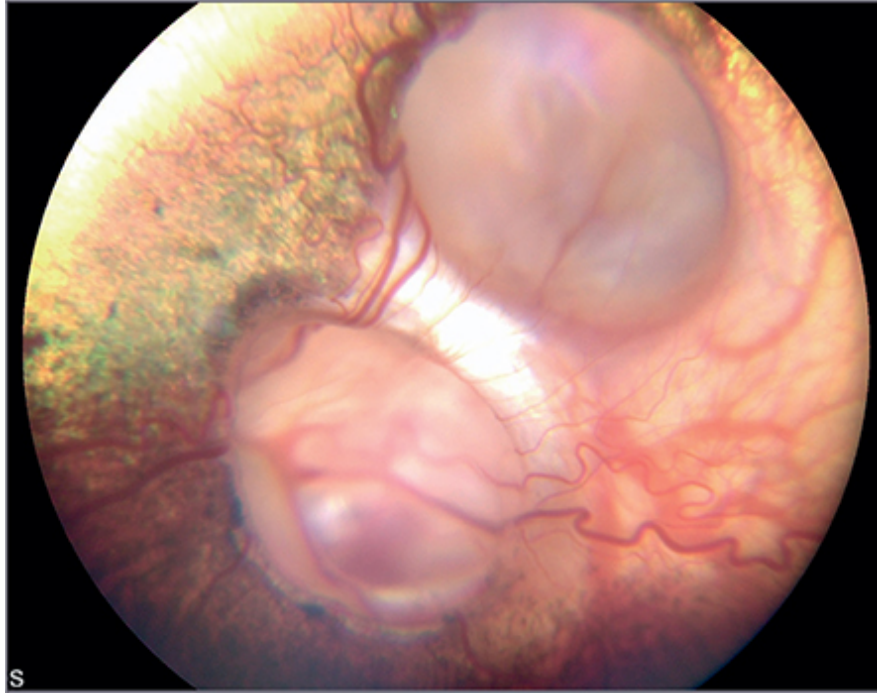


Figure 10. Double retinal coloboma affecting the optic nerve head.