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Cats are increasingly popular as pets. By the end of the previous century, the number of cats exceeded the number of dogs in the USA.

Principally, in cats, similar ocular diseases as in other mammalian companion animals are seen. Yet, some ophthalmic diseases occur in felines that are not seen in other (small) companion animals. Examples of these are: the corneal sequestrum, and eosinophic keratitis and conjunctivitis, blindness due to taurine deficiency. Some other eye diseases show a different expression, course, or different etiological or pathobiological signs in the cat as compared to, for example, the dog. Examples of this type comprise ocular herpes problems, iris melanoma and some types of retinal atrophy.

In this paper, the most important ophthalmological disorders in felines will be discussed, as well as some specific diagnostic and therapeutic aspects for cats. Cats are not small dogs!

Palpebral aplasia

In palpebral aplasia the margins of the eyelids are completely or partly undeveloped. This anomaly is congenital, most likely hereditary (recessively), and usually bilateral, affecting the lateral part of the upper eyelid. It occurs relatively frequent in Persian cats and Persian crossbreeds. Palpebrae aplasia may be associated with other congenital anomalies.

Affected kittens are often born with the palpebral fissure partly or fully open. There are frequently hairs in that area, that are directed toward the globe which predispose to chronic irritation of the corresponding area of the cornea. The eyelids cannot partly or completely close. As a result, parts of the cornea are exposed and cannot be protected by an adequate lid function, leading to lesions of chronic irritation (edema, neovascularization, pigmentation, ulceration and sequestration).

If there are few ectopic hairs in the area, these have to be permanently removed. If the cornea is only slightly irritated, administration of topical lubricants, to effect, on a daily basis will be sufficient. If the lesions are larger, a substitute eyelid should be created by blepharoplasty.

The breeder/owner should be informed that these anomalies may be hereditary, and that the parents and littersmates should be examined carefully for these and other anomalies. Affected animals, and ideally also members of their immediate family should not be bred.

Dermoid

Dermoids are ectopic and abnormally developed islands of skin in or on the the cornea and conjunctiva, sometimes extending to the eyelids. In the cat, it is a rare anomaly. In the Burmese this condition most often bilateral, and is probably hereditary in this breed. An isle or fold of skin often disrupts the lid margin and is continuous with the conjunctiva. Blinking is abnormal and hairs generally grow toward the cornea causing chronic irritation, and resulting in edema, vascularization, and pigmentation.

Treatment consists of a very precise keratectomy with a rounded scalpel (e.g., Beaver 6400), beginning at the central margin and removing the dermoid and the superficial stromal layers into the direction of the limbus. Magnification of 5-10x is necessary to be certain that no hair follicles remain and that the cornea is not incised too deeply.

If the lids are affected, the affected parts are removed as well. In these cases, blepharoplasties are seldom necessary, as the fissure length is sufficient in most cases.

The prognosis is favorable. Parents and littersmates should also be examined. Affected animals and ideally also members of their immediate family should not be used for breeding.

Lid neoplasia

Neoplasms of the eyelids are quite common in dogs, horses and cattle but rare in cats.

In the cat, lid neoplasms usually are malignant (squamous cell carcinomas, mastocytomas). Squamous cell carcinomas are more often seen in white cats (possibly in connection with UV hypersensitivity). In cats, eosinophilic granulomas must be considered in the differential diagnosis.
Carcinomas are usually flat, slowly growing, ulcerating defects at or near the margin of the eyelid. They may initially show as an hyperemic area, with some dark exudate. They are often accompanied by neoplasms on other parts of the body (e.g., ears, lips). Therefore, these parts have to be examined thoroughly. The diagnosis is established by means of biopsy or, in the case of small neoplasms, by direct radical excision.

Therapy: Neoplasias of the eyelids should be carefully surgically removed (blepharoplasty). The tumor itself should never be squeezed during surgery, otherwise causing artefacts for the pathologist. The tumor, in 5% formaldehyde, is at least stored, but better directly sent for histopathologic evaluation.

Treatment of larger tumors, and tumors with distinct aspects of malignancy, or recurrences consists of radical removal of the neoplasm. These methods usually require more extensive blepharoplasties. In malignant tumors, adjunctive therapy (cryo, hyperthermia, radiation, brachytherapie, immunotherapy, et cetera) is advisable in most cases. In all cases, a preoperative metastases detection is to be performed.

Conjunctivitis
In conjunctivitis exudate is mixed with the tear film. Viscosity increases and lacrimal drainage decreases, resulting in epiphora and tear stripe formation below the medial canthus. This reaction may be caused by wind, dust, or allergens. Other causes are acute infections such as in the upper respiratory disease in felines caused by agents as viruses, Mycoplasma and Chlamydoephila. This is followed by the production of more mucus and more desquamated epithelial cells. Production of purulent material is usually more prominent in bacterial and fungal infections. Purulent conjunctivitis often follows catarhral conjunctivitis, caused by mechanical or infectious agents.

Catarhral conjunctivitis is characterized by epiphora, redness, slight swelling, and mucous discharge. In the acute phase, epiphora is especially prominent. In the chronic phase (after 1-2 weeks), redness, swelling, and discomfort increases.

 Conjunctivitis usually causes more itching than pain. The predominance of pain is a sign of corneal involvement. The pruritus may result in rubbing and a minor blepharospasm. Often some follicular activity will follow, increasing when the conjunctivitis persists. Having much more serious consequences for the eye are epithelial defects in the conjunctiva and cornea which may occur in upper respiratory disease in cats. When two opposite layers are affected, the surfaces may adhere and grow together (symblepharon). This process is often found in the lacrimal canaliculi, between the palpebral conjunctiva of the nictitating membrane and the lids, and between the palpebral conjunctiva and the cornea.

Therapy: Therapy in acute conjunctivitis consists of removal of discharge by irrigation, 1-4 times daily. The conjunctiva usually begins to regenerate in a matter of days. If there is no spontaneous improvement, broad spectrum ocular antibiotic drops or ointment can be prescribed. In cats, oxytetracycline or chlortetracycline ointment is often preferred.

In acute purulent conjunctivitis, therapy consists of dissolution of the purulent exudate by acetylcysteine, followed by irrigation, at least 4 times daily. Antibiotic therapy (topical and/or systemic) may be prescribed as well. In acute conjunctivitis due to upper respiratory disease in the cat topical therapy includes acetylcysteine (decreasing the risk of symblepharon; see below).

Symblepharon
Symblepharon is an adhesion between parts of the conjunctiva or between the conjunctiva and the cornea. It is especially found in young cats as a complication of feline ocular herpesinfection (FHV-1). The virus and the secondary bacterial infection destroy the superficial layers of the epithelium. Together with the mucous discharge this easily results in conjunctival layers adhering and subsequently growing together. In particular, there are frequently adhesions between the walls of the lacrimal canaliculi and/or the palpebral conjunctiva of the nictitating membrane and the rest of the conjunctiva. Strangely enough, symblepharon is usually unilateral. Signs of symblepharon include epiphora, tear-stripe formation, permanent protrusion of the nictitating membrane, and blepharospasm. The adhesions are found during examination of the conjunctival sac. If the cornea is involved there may be membranes of scar tissue spreading over and loosely attached to the cornea.

Treatment consists of loosening the adhesions. Installation of a soft bandage contact lens covers the globe and may facilitate regrowth of the epithelium and thus prevent recurrence of adhesion. Still, recurrence is possible. Surgical treatment should only be performed after the underlying infectious disease has run its course.
Adhesions may be prevented when, during the acute phase of upper respiratory disease in cats, the adhesive mucous discharge is removed very thoroughly by topical acetylcysteine and irrigation.

**Eosinophilic keratitis**
Eosinophilic keratitis is a superficial keratitis in the cat in which the granulation component over the cornea is prominent. The etiology is not known but is believed to be related to (previous) herpes infection. It occurs fairly infrequently and can be unilateral or bilateral. A zone of opacity followed by pink granulation tissue invades the superficial layer of the cornea, usually from the lateral side. Sometimes the granulation is locally covered by white-yellow (“cottage cheese”) necrotic plaques (staining fluorescein positive). The diagnosis is made on the basis of the finding of large numbers of eosinophils among other inflammatory cells by cytologic or histologic examination.

The therapy consists of topical treatment with dexamethasone drops (0.1%) 3-4 times daily until the signs disappear, after which maintenance administration to effect is adequate. Treatment with β-irradiation (Sr90), megestrol acetate (orally), or cyclosporin (topically) is also possible. Megestrol acetate can be used, but the progestagenic and diabetogenic side effects should be considered.

The prognosis is favorable, even if recurrences have to be suppressed repeatedly.

**Intraocular haemorrhage**
Intraocular haemorrhage may be caused by a number of underlying causes such as trauma, uveitis, coagulopathy, vasculopathy, intraocular neoplasia and systemic hypertension.

Especially in cats with intraocular haemorrhage aged older than 11-12 years, systemic hypertension should be considered as the most probable cause. A general physical examination is of great importance and measurement of the (systolic) blood pressure should be performed. Systolic pressure values over 170 mm Hg should be considered abnormal. If confirmed, examinations for underlying diseases (hyperthyroidism, chronic kidney failure, hyperaldosteronism, etcetera) should be performed.

Initial symptomatic anti-hypertension treatment consists of oral amlodipine. Hypertension patients should be closely monitored, preferably by an internist.

**Lens luxation**
The lens can dislocate or luxate by rupture of the zonular fibers. This disorder occurs much more frequently in the dog than in the cat (about 9:1). In the cat, lens (sub)luxation is seen predominantly in elderly cats suffering from a chronic anterior uveitis (often subclinical). Secondary glaucoma occurs usually less acutely, less rapidly, and less often in the cat than in the dog.

The earliest recognizable sign of lens luxation is the presence of vitreous in the anterior chamber, observable as very thin white treads or clouds. If the lens is displaced, an “aphakic crescent” will be visible between the pupil margin and the lens equator. If the lens is fully luxated, the anterior chamber is deeper (in posterior luxation). The loss of support for the iris can also result in an iridodonesis. In lens luxation towards anterior the lens is completely visible in the anterior chamber (slit lamp!), directly behind the cornea.

Medical (miotic) and surgical therapies are available. The surgical therapy consists of complete (hence intracapsular) removal of the lens. Because secondary glaucoma occurs less rapidly in the cat, removal of the lens is less urgent in most cases.

**Hereditary photoreceptor degenerations**
The term “PRA” is used, especially in breeders’ circles, for a large group of hereditary primary retinal atrophies with the collective clinical denomination “PRA”. PRA is always hereditary, bilateral and symmetrical. The most important types of PRA are < gekenmerkt > by progressive, irreversible photoreceptor disease with secondary degeneration of the other retinal components.

PRA can be divided into many types, most of which start with degeneration of the rods and, hence, with night blindness. This is mainly of importance in connection with the age at which the signs of night-blindness first appear. For the cat, two main types are of importance. If the rods and/or cones are abnormally developed (dysplastic), they will also degenerate early in life. One form of PRA of this type is rod cone dysplasia which occurs in Abyssinian and Somali (Rdy). The mutation is a single base pair deletion in a different gene (CRX), which results in a defective protein that is critical for ophthalmic development. Cats carrying one copy of this mutation have a retarded development and subsequent degeneration of photoreceptor cells, which leads to early-onset blindness by 7 weeks of age. The Rdy mutation is inherited as a dominant trait. This mutation is rare.
In the late onset forms, the rods are normally developed and the degeneration starts later in life. In Abyssinian, Somali and some Ocicat breeds, an inherited late-onset PRA has been identified. This disease has been designated rdAc. Cats affected with this form of blindness have a normal vision at birth, with electoretinographic (ERG) demonstrable signs of degeneration at about seven months of age. Vision loss is progressive, with most cats becoming blind by 3-6 years of age.

The diagnosis of retinal atrophy is based on the signs and the findings in ophthalmic examination (fundoscopy), and can be confirmed by an ERG. In differential diagnosis the disease could be confused with non-inherited, bilateral, diffuse, progressive retinal degenerations, such as taurine deficiency or quinolone-associated retinal atrophy in the cat.

There is no available therapy. The prognosis for vision is hopeless. Blind cats can, however, manage quite well in their familiar environment.

Prevention: Animals with signs of PRA should be considered as having the disease until proven otherwise. It is also of great importance that the diagnosis is confirmed by DNA testing (if available).

**Drug-associated photoreceptor degeneration**
Quinolone-associated retinal atrophy was first diagnosed in cats in 1997. This photoreceptor degeneration progresses very fast, and may occur after one dosage.

Predispositions: elderly cats, outgoing, summer/Sunny weather, and a (too) high dose vrijloop, zomer/zonnig weer, (te) hoge dosering. The dosage should not exceed 2.5 mg/kg (once every 12 hours max), and should be administered as shortly as possible.

**Taurine deficiency**
Taurine is an acid amine that - although not a building stone for protein - is still sometimes described as an amino acid. In most animal species it is not an essential nutritional element but cats cannot produce sufficient taurine themselves. High concentrations of taurine are found in the retina, brain, liver, and heart. Considerable taurine is also present in other animal "products" such as milk, meat, fish, and shellfish.

Taurine is especially important in neurotransmission and for the cell membranes and photoreceptors, but it also has a function as a bile acid conjugator and in energy transport.

Taurine deficiency can be expected in cats that are fed an exclusively vegetarian diet or only dog food (often containing much less meat than the label suggests!).

**Symptoms:** Disturbances in the ERG occur only after 5 weeks on a taurine-free diet. After about 20 weeks, granulation develops in the central area, which is the area with the highest concentration of cones. Hyper-reflection in this area then occurs and spreads out dorsally along the papilla, in a more or less discus or stripe form (Plate 14.20). Finally there is generalized hyper-reflection and vessel atrophy, usually after more than a year of deficient taurine intake, and the cat is completely blind.

**Diagnosis:** The diagnosis can be made on the basis of the history and can eventually be confirmed by measuring the concentration of taurine in plasma. The reference values are of the order of 15-150 μmol/l. The costly measurement is seldom performed. Blood from healthy reference animals should thus also be included. In differential diagnosis consideration must be given to such abnormalities as PRA, FCRD (see 14.16) and, in the end stage, all abnormalities associated with bilateral generalized, diffuse retinal atrophy.

**Therapy:** Treatment consists of changing the food and if necessary adding meat, fish, shellfish, or taurine in powder form. The degeneration already present is irreversible but the process will, however, be stopped.

To prevent the disease, owners should be made to realize that cats are emphatically carnivores and should be fed a diet that is appropriate to this.
infusion set tubing to prevent the sutures from cutting into the tissue. Then the canthotomy incision is closed. Before the sutures are knotted, prophylactic antibiotic ointment is applied behind the nictitating membrane. Aftercare consists of applying the same medication in the lateral canthus, between the eyelids. After 3-5 days the medial suture is removed. If there is still an apparent tendency to luxation, then the remaining suture is left in place for a few more days.

Blunt trauma
Blunt trauma can be recognized by damage to the tissues of the orbit, the adnexa, the globe and its contents and all the consequences of this. Hemorrhages and post-traumatic inflammation can cause severe swelling. Tearing of the zonular fibers of the lens can induce lens luxation and subsequent glaucoma. There may also be fractures. Appropriate therapy requires an accurate diagnosis, in which radiographic and ultrasound examination are of great importance.

Hyphaema (hemorrhage in the anterior chamber) is a frequent complication after trauma. There is little evidence that any therapy is of much benefit, however, keeping the animal quiet (e.g., cage rest) is beneficial. Within 1-2 days the erythrocytes in the anterior chamber will settle to the bottom, resulting in a dense red horizontal level with clearing above. A hyphaema that does not resorb, or bilateral hyphaema, is usually not the result of trauma but more likely a sign of a systemic underlying cause and further examination should be carried out in that direction.

Penetrating or perforating trauma
Perforating trauma to the lids or the globe is usually caused by thorns, splinters, claws, or teeth. In horses, training whips and nails in the stalls are often incriminated. In this abstract, only perforating corneal trauma is dealt with.

Corneal wounds
Corneal wounds can occur with or without a foreign body and can be superficial, deep, or perforating. The patient has severe blepharospasm, profuse tear production, corneal edema, and possibly a prolapse of coagulated material, even iris prolapse, in the wound.

Perforating corneal defects
Perforating corneal defects are usually caused by objects that strike the cornea with great speed, such as cat claws, thorns, air rifle or shotgun pellets. The associated trauma
to the iris can cause hemorrhage into the anterior chamber as well as outside the eye, via prolapse of the iris through the corneal wound. This will be accompanied by a severe inflammatory reaction in the uvea. When there is deeper penetrating trauma, cataract or hemorrhage in the vitreous or retina can occur. The eye should not be irrigated, and any substance prolapsing through the wound should be left undisturbed until just before suturing. If the animal is tractable and further examination to determine course of action is necessary, a topical anesthetic should be administered. In addition topical antibiotics and atropine if indicated may be administered.

Thorns which are in part still clearly visible above the corneal surface can be extracted with a foreign body forceps. If the thorn is deeper, attempts to remove it by forceps only cause edema and there is a great risk that the thorn may be propelled from the forceps into the anterior chamber, or worse yet, into the iris and/or lens. An attempt can be made to remove a thorn lying deep in the cornea by lifting it out with two 0.45 mm (26 gauge) hypodermic needles.

Perforating wounds in the cornea should be sutured under general anesthesia with muscle relaxation, using 8-0 or 9-0 monofilament nylon with a spatula-shaped needle. Muscle relaxation is preferred, in order to get the eye to rotate in a standard gaze, and resulting in less traction during surgery to the compromised eye. Corneal tissue is scarce and thus the laceration is not debrided. Small, clean iris prolapses can be replaced through the cornea with a spatula. A large or contaminated iris prolapse should be excised by electrocautery.

Aftercare consists of appropriate broad spectrum antibiotics, atropine and steroid anti-inflammatory drops to treat the anterior uveitis that is present in every patient that suffers this type of injury. These agents should be used frequently 6 times daily for the first few days and frequency modified as the situation dictates. In many patients, systemic steroid or non-steroidal anti-inflammatory drugs are added. After 1-2 days the eye should be examined for and evaluated for severity of post-traumatic uveitis and secondary glaucoma. The sutures are removed after 17 days.

**THE DISCOLORED EYE**

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A fully problem oriented clinical work-up of the general ophthalmologic patient is feasible nor useful in every single case. Ophthalmic diagnoses are diagnosed by a a good case history, careful description of the signs, the results of a thorough clinical examination, a proper differential diagnosis (including breed predispositions), and further examinations, if applicable.

As ocular reactions are often rather repeatable, it can be useful to have the assistance of lists of groups of abnormalities or signs that differentially may be the cause of the problem. In this case the sign “discoloration” is taken as the main lead. Recognition and differentiation of these signs may lead to a diagnosis in an easier and more structured way. This will be discussed by use of cases. The presented lists are indicative only.

The “red” eye:

**Mainly localised, with “extra” tissue:**

- hyperplasia of the tear gland of the nictitating membrane
- (epi)scleritis (nodular)
- corneal granulation tissue, e.g., in a secondary healing corneal ulcer
- neoplasia

**Predominantly diffuse:**

- severe excitement (conjunctiva)
- conjunctivitis/dacryocystitis (e.g., keratoconjunctivitis sicca, “trauma”, infection)
- keratitis
- (epi)scleritis (nodular)
- intraocular haemorrhage (e.g., hyphaema due to numerous causes)
- rubeosis iridis (uveitis)

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Systemic diseases may induce ocular signs and diseases. Neurological and vascular diseases are particularly prone to ocular manifestation. In this abstract, only a limited number of examples of systemic diseases and diseases of other organ systems corresponding with ocular signs will be described.

Disorders of the eyelids, caused by extraocular etiologies comprise blepharitis, juxtapalpebral defects, and inflammation/infection of the tarsal glands ("meibomitis; chalazion-like lesion such as seen in the course of Leishmaniosis). Blepharitis, although not a commonly found disorder, may have a bacterial, parasitic (demodicosis), mycotic, atopic/allergic or auto-immune mediated (uveodermatologic syndrome; lupus) etiology. Another ophthalmological "link" with dermatological disease may be the eosinophilic granuloma complex.

Conjunctival/sclera signs related to extraocular causes comprise plasmoma/plasmacellular conjunctivitis (immunogenic), episcleritis, symblepharon (due to FHV1-infection), icterus, hemorrhages (hematologic and hepatic causes), (venous) hyperemia (circulatory causes), and nictitans protrusion as a non-specific sign.

The position of the globe may indicate ophthalmic signs due to a loss or increase of orbital fat, or due to orbital wall changes (enophthalmos; exophthalmos, the latter with or without nictitans protrusion. The amount (increase or decrease) of orbital fat, changes of the orbital wall (bony and muscular components), the size of the (peri)orbital glands, inflammation/infection/abscessation, foreign bodies, vascular abnormalities (including hemorrhage), and last but not least (para)neoplastic diseases may affect the position of the globe and of the nictitans. In patients with a "retrobullbar process" as orbital disease opening of the mouth may be painful and/or mechanically limited. This is predominantly the case in dog. Sudden opening of the mouth (barking, yawning) is especially
painful and is avoided first (history!). At last, eating and drinking may be impossible.

Corneal signs caused by systemic factors comprise superficial keratitis (pannus), punctate keratitis, and corneal lipidosis (“dystrophy”). Corneal lipidosis may be due to local causes, may be hereditary determined, or may be due to systemic disorders of the fat metabolism (e.g., hypothyroidism). Rather uncommon systemic diseases such as mucopolysaccharidosis and GM1 and GM2 gangliosidosismay cause corneal signs.

The uvea is composed of the iris, ciliary body, and choroid. Although diseases may be limited to the anterior or posterior portion of the uvea, the uvea is generally regarded a “functional entity”. The degree of iridal pigmentation may be an (additional) indication for “generalized problems”. In cases of oculocutaneous albinism, the iris is unpigmented, i.e., more or less red. In Van Waardenburg syndrome, such absence of pigmentation is associated with deafness. In rare cases, in mink and sometimes in the cat, partial oculocutaneous albinism is associated with an increased susceptibility for (infectious) diseases, and blood clotting disorders (Chediak-Higashi syndrome). This syndrome is based on an autosomal recessive mutation. Besides hypopigmentation, cataract, photophobia and nystagmus may be present. Another pigmentation disorder of the iris is formed by hyperpigmentation, which may be difficult to assess. If the hyperpigmentation is brownish, and if bilaterally present, these pigmentation are regarded benign (“freckles”). If iridal hyperpigmentation is dark-brown to black, melanoma may be suspected. In dogs, iris melanoma is generally elevated and may even grow out through the sclera. Iris melanoma in dogs has been published to have a low metastasis rate (4%). In cats, iris melanoma tends not to be elevated (diffuse iris melanoma), is multifocal of nature, and may cause (severe) dyscoria. Different form dog, metastasis rate in cat is believed to be about 63%.

By far the most important uveal disease with extracocular associations is uveitis. The main pathogenetic momentum is generally believed to be immunogenic. Infections are an important underlying factor for uveitis, by causing an (aberrant) immune response. On the other hand, the infectious agent is believed not to be a direct factor in most cases of uveitis. However, one should try to detect (or exclude) specific etiological agents as well as possible. The occurrence of such diseases strongly depends on the geographical location where the patient is, or has been, held (ask about traveling!). Infectious diseases in dog and cat for which diagnostic tests may be performed comprise (examples):  
- **Viruses**: CAV-1, FeLV, FIP, FIV  
- **Bacteria**: Borrelia, Leptospira  
- **Fungi**: Cryptococcus, Aspergillus, Histoplasma, Candida, Coccidioidomycosis  
- **Algae**: Prototheca  
- **Protozoa**: Leishmania, Toxoplasma, Ecephalitozoan, cuniculi  
- **Metazoa**: Toxocara, Dirofilaria, Onchocerca, Habronema

In addition, the process of uveitis may be triggered by non-infectious causes. Examples of these are: blunt or penetration trauma, metabolic (e.g., hyperlipoproteinemia) and (para)neoplastic disorders. Notwithstanding the many possible causes for uveitis, the underlying cause remains unknown in many case (‘idiopathic’ uveitis). Although uveitis is the most common cause for “aqueous flare” or “clots” of coagulated fibrin in the anterior chamber, there are other causes for a non-transparent anterior chamber. Free blood in the anterior chamber (hyphema) may be caused by trauma, coagulopathy, vasculopathy (collie eye anomaly), uveitis, systemic hypertension, or neoplasia. Hence, a general physical examination must be performed in such (ophthalmic) patient! The presence of lipids in the anterior chamber may be associated with hyperlipoproteinemia or hyperlipidemia. Pus (hypopyon) indicates a (mostly bacterial) infection (endophthalmitis).

The most common lental disorders of interest are cataract (lental opacification) and lens luxation. A lens luxation is regarded a hereditary trait in small and medium sized terriers, but may also be traumatic, or predominantly in cat and horse – may be caused by anterior uveitis (iritis, iridocyclitis). Cataract has many etiologies, the most important being heredity. Other possibilities are: congenital (mostly in combination with lental malformation, persistent intraocular vascular components, and/or microphthalmia), radiation, electricity (electrical chord biting, lightning strike), intoxication (e.g., naphthalene) alimentary deficiencies (vitamin A, essential amino acids), metabolic (diabetes mellitus, hyper- and hypocalcaemia), trauma and inflammatory causes.

Vitreous disorders are rare. In relation to systemic diseases, only hemorrhage and developmental anomalies such as...
Companion Animal Programme

1. Collection of findings (ophthalmic examination)
2. Interpretation of the signs (differential diagnosis)
3. Plan

1. Concerning the findings, three main issues are of importance:
   - Exact location
   - Color
   - Increase (any swelling or proliferation) or decrease (e.g., degenerative or ulcerative disease)

Findings are of course collected during the ophthalmic examination.

2. For the interpretation of the findings, it is important to follow a structured process. It may be helpful to use a personalized checklist, which contains all or most groups of diseases. This will prevent a whole group from being “forgotten”. An example of such a checklist may be as follows:
   - Congenital/hereditary
   - Trauma (blunt, perforative, foreign body)
   - Alimentary (including deficiency, intoxication)
   - Physical (electricity, radiation, etcetera)
   - Metabolic
   - Inflammatory
   - Degenerative
   - Neoplastic

3. Plan (diagnostic, therapeutic)
   At last, the diagnosis is established, or several differential diagnostic possibilities remain. In case of a definitive diagnosis, a therapy can be advised (therapeutic plan). In more possibilities remain, a diagnostic plan is needed. For ophthalmic cases, further diagnostic procedures comprise: diagnostic imaging (ultrasound, CT, MRI), electrophysiology (ERG, VEP), biopsies (cytology, pathology), microbiology (culturing, PCR), laboratory (clinical chemistry, hematology), etcetera.

Possible therapeutic modalities may be: expectative (no therapy), medical, surgical, laser treatment, cryotherapy, et cetera.

Following the above-described procedure, ophthalmic cases will be discussed.

Specific wishes from the audience can be discussed, preferably after a timely request before the start of this session.

During the “meet the specialist” session, cases will be discussed interactively, on the basis of projected slides showing ophthalmic signs and eye diseases.

The basis of the diagnostic process is divided into three parts:

THE DISCOLORED EYE
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The eyes and vision are closely related to the central nervous system. It must be realized that the only observable part of the nervous system is located in the ocular fundus. Neuro-ophthalmological signs include: dyscoria, abnormal pupil size (mydriasis, miosis, anisocoria), abnormal papillary light reactions, impaired vision, abnormal global movements and adnexal tone. Specific disorders affecting pupil size are the Horner syndrome (sympathetic denervation) and feline dysautonomia (Key-Gaskell syndrome). In addition, facial palsy may be caused by hypothyroidism and in borreliosis.

In all parts of the eye(s), (para)neoplastic changes may occur. The uvea is predominantly involved. Ocular neoplasia is most often primary, ocular metastases of neoplasia in other organs have been described (e.g., due to mammary carcinoma in a cat). Additionally, uveitis may be caused by systemic (para)neoplastic disease (e.g., lymphoma, malignant histiocytosis).

Persistent hyperplastic primary vitreous (PHPV) come to mind.

Fundus abnormalities, relevant within the frame of systemic diseases, comprise posterior uveitis, retinal detachment, retinal dysplasia, retinal atrophy (hereditary, vitamin A deficiency, taurin deficiency in cat, fluoroquinolone-induced retinal atrophy in cat), papilledema and (para)neoplastic syndromes. Fundus manifestations caused by vascular diseases comprise hyperlipidaemia (“lipemia retinalis”), arterial hypertension, and hyperviscosity.

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   - Increase (any swelling or proliferation) or decrease (e.g., degenerative or ulcerative disease)

Findings are of course collected during the ophthalmic examination.

2. For the interpretation of the findings, it is important to follow a structured process. It may be helpful to use a personalized checklist, which contains all or most groups of diseases. This will prevent a whole group from being “forgotten”. An example of such a checklist may be as follows:
   - Congenital/hereditary
   - Trauma (blunt, perforative, foreign body)
   - Alimentary (including deficiency, intoxication)
   - Physical (electricity, radiation, etcetera)
   - Metabolic
   - Inflammatory
   - Degenerative
   - Neoplastic

3. Plan (diagnostic, therapeutic)
   At last, the diagnosis is established, or several differential diagnostic possibilities remain. In case of a definitive diagnosis, a therapy can be advised (therapeutic plan). In more possibilities remain, a diagnostic plan is needed. For ophthalmic cases, further diagnostic procedures comprise: diagnostic imaging (ultrasound, CT, MRI), electrophysiology (ERG, VEP), biopsies (cytology, pathology), microbiology (culturing, PCR), laboratory (clinical chemistry, hematology), etcetera.

Possible therapeutic modalities may be: expectative (no therapy), medical, surgical, laser treatment, cryotherapy, etcetera.

Following the above-described procedure, ophthalmic cases will be discussed.

Specific wishes from the audience can be discussed, preferably after a timely request before the start of this session.

During the “meet the specialist” session, cases will be discussed interactively, on the basis of projected slides showing ophthalmic signs and eye diseases.

The basis of the diagnostic process is divided into three parts: