Lacrimal Apparatus

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1 Introduction

The tear film actively and passively protects the cornea and conjunctiva (it contains e.g. lysozyme, lactoferrin, IgA, IgM and IgE),[1,2,3] keeps them clean, moist and lubricated, supplied with nutrients, and transports white blood cells. The eyelids spread the tear film over the surface of the eyeball and maintain its thickness within very narrow limits.[4] The upper eyelid, in particular, has a very important function in this regard. In birds, the thickness of the tear film is mainly maintained by the nictitating membrane. Some cats have a relatively low frequency of blinking and often a markedly lower tear production than dogs. This may be one of the causes of the frequent occurrence of central corneal sequester formation in the cat, especially the Persian, Himalayan and Burmese which have very large, protruding eyes.
The tear film is approximately 0.04-0,07 mm thick and consists of three layers.[5,6] The outer layer is the lipid layer, which is produced by the sebaceous glands in the margins of the lids, and reduces evaporation, lubricates and prevents adhesion of debris. The middle layer is formed by the aqueous fraction of the tears. It washes away foreign bodies and contains, for example, antimicrobial agents, soluble mucins, immunoglobulins, enzymes and cellular debris. This layer blends gradually into the inner, mucous layer of the tear film, which is mainly produced by the intra-epithelial goblet cells of the conjunctiva. This hydrophilic layer attaches to the glycocalyx,[7] which serves as an adhesive between the hydrophobic microvilli of the corneal epithelium and the mucous layer of the tear film, thus lubricating and protecting the cornea. The mucous layer also entraps debris and microbes in a mucous thread, which moves continously, via the lower cul-de-sac, to the medial canthus. There it dries and is rubbed away.
About 60% of the aqueous portion of the precorneal tear film is produced by the lacrimal gland, which is located dorsolateral to the globe, under the zygomatic process of the frontal bone. The remaining 40% of the tears is produced by the gland (or glands in some species) of the nicitating membrane (main producer of tears in birds) and by the accessory tear glands.[8]
Stimuli that are painful or irritating to the cornea and conjunctiva or to the adnexa can increase tear production, while anesthesia, atropine, and drugs such as some sulfonamide derivatives and etodolac can reduce it.
Part of the tear film evaporates. The remainder drains away via the lacrimal punctae (oval, 2–3 mm diameter;, which are located 6–10 mm from the medial canthus in the conjunctiva at the mucocutaneous junctions of the lower and upper lids. From there, the tear fluid drains via the lacrimal canaliculi, the saccus, and nasolacrimal duct to the external nares. In some animals, the nasolacrimal duct does not extend all the way to the nose but empties into the nasopharynx at the level of the canine tooth. Obstruction or compression along the path of drainage and/or overproduction of tear fluid quickly leads to epiphora and tear stripe formation (tear staining syndrome).
In the cat, this is often accompanied by pigmented detritus, possibly resulting from oxidized catacholimines in the tears that can accumulate as small pigmented stripes on the margins of the lids and in the tear stripe, and which can infiltrate the corneal surface resulting in sequestra.[9]
In rabbits and swine only a lower punctum exists.

2.1 Keratoconjunctivitis sicca (KCS)

KCS can be defined as a tear film deficiency of the cornea and conjunctiva, and hence it is not a primary keratitis. KCS is usually caused by an inadequate production of tears by the lacrimal glands, thus of the watery fraction, and therefore the STT values are low and the tear film break-up time is less than 20 sec. This deficiency can also occur because the tear film breaks up due to an abnormal composition (e.g. of the mucous layer). In these cases, there are often low-normal STT values in addition to minor sicca signs. .

There are many known causes of KCS:
1. KCS can be congenital and possibly inherited.
2. Traumatic injuries to the lacrimal glands or in the area of the innervation near the base of the ear can result in inflammation and/or atrophy of the glands themselves or in a disturbance to their innervation.
3. Surgical trauma to the nerve supply or the tear glands or their ducts can cause KCS. Also, the removal of the nicitatans gland may result in KCS.[10] KCS, after hypophysectomy has been ascribed to traumatic or ischemic neuropraxia of the major petrosal nerves.[11] In rats, hypophysectomy-induced regression of the lacrimal gland was partially restored by the administration of androgens and prolactin, suggesting trophic action of these hormones on the gland.[12]
4. Nutritional deficiencies such as hypovitaminosis A.
5. Intoxications such as locoweed,[13] belladonna, and botulism can cause KCS.
6. Medications such as phenazopyridine,[14] etodolac and sulpha derivatives (note: sometimes used in geriatrics or for chronic kidney/bladder disease) can cause permanent KCS in less than 14 days. Atropine can cause temporary KCS.[15,16] Tear production ceases during general anesthesia.[17,18] An unprotected cornea will dry out very quickly, especially under anesthesia during which the eyelids remain open (e.g. anesthesia with ketamine in the cat), and certainly if the eye also lies directly under the heat of the surgical lights.
During anesthesia the eyes should be protected by application of a neutral eye ointment.
7. Inflammation, especially due to infection of the tear glands, is an important cause of KCS. Infections of the conjunctival sac presumably provide an important port of entry for infection of the tear glands, but can also result in blockage of the drainage system. In addition, inflammation in the middle ear can cause a disturbance to the parasympathetic innervation of the tear glands.
8. Autoimmune diseases resulting in a plasmacytic, lymphocytic adenitis, can cause KCS.[19] This is a T-cell reaction and justifies using T-cell inhibitors for treatment. This is the most frequent cause of KCS in the dog. Autoimmune and degenerative diseases such as Sjögren's syndrome and the dysautonomia syndrome have been reported to cause KCS. Sjögren's syndrome, in which all mucous membranes are excessively dry, is the most important cause of KCS in humans. Sjögren's syndrome has also been described as a cause of KCS in dogs[20], but not as yet in cats. In dysautonomia syndrome in cats and dogs, the sympathetic and parasympathetic neurons of the autonomic ganglia are degenerated. Mydriasis is found in approximately 90% of these cats, and in 80% there is a distinct KCS.
9. Neoplasia can directly or indirectly destroy the lacrimal glands.
10. Idiopathic causes. In many patients with KCS, the primary cause cannot be determined. The glands atrophy or no longer function because of denervation, for example.
11. Secondary to lid defects, exophthalmos, proptosis, luxation of the globe, lagophthalmos etc., a local KCS or KCS over the whole conjunctiva and cornea may develop.

In the dog, KCS occurs regularly and since only a small proportion of these patients recover completely, they will continue to require regular follow-up examinations and attention. In the cat, KCS is seen much less frequently. However, tear film that breaks up too quickly in the central area of the cornea could easily be the initiating factor in the development of mummification or sequestration of the cornea in large-eyed and often short-nosed cats . KCS is usually bilateral (about 60%) and occurs more often in female animals (about 65%). Breed predisposition includes small dogs in general, with a high prevalence in the Long-haired Dachshund, Cavalier King Charles Spaniel, and West Highland White Terrier.
Symptoms: The cornea and conjunctiva lose their normal luster and have a dull matted appearance. The reflected image can no longer be seen sharply on the "cornea" . There are variable signs of mucopurulent conjunctivitis, such as redness, swelling, slight blepharospasm, and mucopurulent discharge, although without the expected overproduction of tears and/or wet hair around the eye. If KCS has existed for some time, signs of a chronic superficial keratitis are to be expected, often beginning in the centrolateral part of the cornea, which is the part most exposed to drying. These signs include edema, injection of vessels, epithelial defects, fibrosis and pigmentation. Usually there is no edema, but epithelial

keratinization and fibrosis cause the grayish-white corneal cloudiness. In the cat, there may be sequestration of the cornea. In addition, the ipsilateral half of the nasal plane and the oral cavity can have a dry appearance. Sometimes, the dry nasal plane is the only sign of KCS that has come to the attention of the owner.
Diagnosis: The diagnosis is based on the lowered STT value *and* the clinical signs. If the value is normal or low-normal, but there are still clinical signs consistent with KCS, referral for rose bengal staining of the cornea and tear film break-up time measurement, both with the aid of a slit-lamp biomicroscope, can be of additional diagnostic value.
Many forms of conjunctivitis and keratitis come into consideration in the differential diagnosis, but in these disorders, normal or even elevated tear production is to be expected.
Therapy: Treatment of mild KCS is medical. Severe chronic forms are treated medically or in specific cases surgically. If there are only minor signs of KCS, together with a low-normal STT and a positive rose bengal staining, then initially it will be sufficient to wash the eyes and apply e.g. cromoglycate and vitamin A oil, or dexamethasone, both 4 times daily, or 0.2% cyclosporin 2 times daily.
In severe, distinct acute KCS, antibiotics and corticosteroids are given orally for 7–10 days.[21] The client is educated to understand the nursing care necessary to provide comfort and function for the patient. Further treatment consists of dissolving the mucopurulent discharge with 10% acetylcysteine, washing the eye, and then applying 0.2% cyclosporine 2 times daily.[22] Cyclosporine can also be applied to both eyes even though the condition may only be unilateral. It takes at least 3 weeks before the drug reaches an effective level, and therefore ancillary therapy must be provided until the drug becomes effective. In a number of patients, an increased tear production does not occur, but the subjective signs of the disease are markedly reduced and the patient is much more comfortable. Cyclosporine 2% (or the stronger 0.02-0.03% tacrolimus[23] or Pimecrolimus, all T-cell inhibitors; *Note*: the latter two are more cytostatic and not yet registered for the use on the eye) are more potent alternatives, but they are not yet registered in most countries.
Fifteen minutes after the cyclosporin ointment, a topical, “initial choice” antibiotic and if there is no overt ulceration, a corticosteroid or an “initial choice” antibiotic-corticosteroid combination are used. This procedure is repeated 4 times daily for 4–6 weeks. Artificial tears should be used as necessary at times other than those described. Additional pilocarpine can be administered orally[24].
On the day of the follow-up examination (after 3-4 weeks), the topical medications are not given but, if prescribed, the pilocarpine should be given orally as usual. If the signs of KCS have disappeared and the STT value has become normal, the antibiotic-corticosteroid combination is adjusted to fit the patient's needs. Often one can discontinue them and maintain the patient on the T-cell inhibitor and the artificial tears or other mucocinomimetic (e.g. *i-drops*®. If the STT value is still low or has declined, a maintenance level is determined and the patient is kept on that level and monitored as needed. At the last resort, if the STT remains 0 and the owner is not able to continue the treatment, referral for a parotid duct transposition (PDT) can be considered. There must, however, be a normal production of saliva.
In the PDT operation[25,26] (difficult in the cat), the terminal portion of the parotid duct is freed in the mouth and is brought to the conjunctival sac either via a tunnel running totally under the skin of the cheek[27] or from an incision in the skin of the cheek. The opening of the duct is implanted in the ventrolateral fornix, between the nictitating membrane and the palpebral conjunctiva. The disadvantages of PDT are that calcium deposition can occur on the cornea and an excess of saliva/mucus can lead to inflammation of the skin of the cheek.
Prognosis: Prognosis for healing of distinct, acute KCS is relatively good. Prognosis for chronic KCS is less favorable. The prognosis for KCS is often somewhat better in cats than in dogs. Because only the smaller proportion of this group will heal completely, the majoritiy of patients will need continuous veterinary care and thus, the owner must be motivated to do so.

2.2 (Sialo)dacryoadenitis

Dacryoadenitis of the deep gland of the nictitating membrane in rats and mice may cause porphyrine-containing tears and result in a redness of the skin around the eyes (chromodacryorrhea).
The sialodacryoadenitis virus in the rat is a highly infectious coronavirus of the respiration apparatus of the rat. It causes rhinotracheitis, bronchitis and alveolitis. It may also cause an inflammation of the salivary glands and a necrotizing inflammation of the deep (Hardens) gland of the nictitating membrane and/or the other lacrimal glands
Symptoms: There may be exophthalmos, epiphora and keratoconjunctivitis. The symptoms will generally disappear spontaneously within a week, but may be complicated by uveitis and multifocal retinal degeneration.

3 Tear stripe formation

By means of the “zip-closure action” of the blinking of the lids, tear fluid is moved towards the medial canthus, where, if there is any functional obstruction to its drainage or simply excessive production, it flows over the edge of the eyelid. If this situation persists, a tear stripe will form under the medial canthus; this being a brown-colored, moist stripe (tear staining syndrome. In cats, small aggregates of pigmented material can be found between and on the hairs. Tear stripe formation can, therefore, be caused by a problem in the drainage of tear fluid or by overproduction of tears, or a combination of the two. During the diagnosis, possible causes of overproduction should be excluded first, such as medial entropion (short-nosed Persian cat), distichiasis, trichiasis, conjunctivitis, or keratitis. Causes of a disturbance to the drainage should then be considered. These include:

3.1 Micropunctum or stenosis of the lacrimal punctum

Narrowing of a punctum only causes a problem in drainage if it involves the inferior punctum. It can be a congenital defect. In the cat, it is usually a residual complication of upper respiratory disease e.g. feline herpes virus type 1 (FHV-1), calici virus, chlamydia infections.[28,29]
The most important sign is chronic tear stripe formation. The passage of fluorescein is retarded or absent. A lacrimal tear duct cannula can only be introduced with difficulty or not at all, but the passage is otherwise good.
Therapy: Treatment consists of stretching the punctae with a lacrimal dilator and irrigating the drainage apparatus with a collyrium. In easily handled patients, this can be done under local anesthesia, but in most cats it is necessary to carry out the stretching and flushing under general anesthesia.

3.2 Atresia and secondary closure of the punctum

One or both puncta and/or canaliculi may have failed to open or have become closed. In the dog, this abnormality is often congenital (inheritance unknown), while in the cat the cause is usually adhesions after damage to the epithelial lining of the drainage system in the course of upper respiratory disease e.g. FHV-1 and calici virus and/or Chlamydia infection. Examination will reveal either no recognizable punctum, or that the lacrimal cannula almost immediately encounters an obstruction preventing the outflow of tears.
Therapy: The treatment, carried out under anesthesia, consists of opening the relevant punctum. This requires somewhat specialized instruments and skill, without which the patient should be referred. If the opposite punctum is open, this can be used for insertion of a round-tipped, hooked probe with a hole in the tip (e.g. a modified Worst “pigtail” probe). The epithelium at the level of the punctum is raised and snipped off. In order to prevent the punctum from closing again, a silicone tube with a diameter of 0.7–1.5 mm can be introduced. After-care consists of a “specific choice” antibiotic and corticosteroid in artificial tear drops (4 times daily) and a protective collar. The tube is left in place for about 3-6 weeks. It is then cut and both remaining parts removed.
If both puncta are closed, the dorsal one is opened with a pointed blade and the above procedure is carried out. If one or both canaliculi are found to be closed over a greater distance, opening in the above manner will not usually be possible. If the abnormality is not

too severe a hindrance for the patient, further attempts at opening should be avoided. In exceptional cases, conjunctivo-rhinostomy can be considered (see 4 Dacryocystitis). In horses, atresia of the lacrimal drainage apparatus[30] (and dacryocystitis) is characterized by copious mucous or mucopurulent exudate from the involved eye. This is usually seen in young horses. The tear ducts can be irrigated (cat catheter) via the tear ducts; nevertheless , to start with, it is more simple to try to irrigate from the ostium in the nose (urinary catheter, diameter 3–6 mm). However, the obstruction is most frequently at the distal opening and can easily be corrected by antegrade irrigation with a collyrium and observing the “bulging” at the distal end in the nose. Under local anesthesia, this bulge can be incised, either with a pointed scalpel or by electrocautery. After opening the distal end, the duct is irrigated thoroughly with a collyrium and an antibiotic (some clinicians include corticosteroids). Cannulation is possible, but is usually not necessary for maintaining a patent duct.

4 Dacryocystitis

Dacryocystitis is an infrequent disorder and consists of inflammation of the lacrimal sac, canaliculi, and nasolacrimal duct. It often follows a viral, bacterial, or mycotic infection, or a foreign body. Additionally, trauma to the nose or neoplasms can predispose to obstruction of the drainage apparatus. In horses, it can also be caused by a *Thelazia* spp. infection (see Chap. Conjunctiva).[8] The secondary inflammation causes swelling, by which the drainage system is compromised and the passage further reduced. The problems occur especially in the upper part of the duct, where the duct is surrounded by a bony canal. Swelling in this part can only press inwards, so obstruction develops rapidly. The purulent exudate that is formed seeks an exit and it usually takes the path of least resistance, which is upwards towards the conjunctival sac, or it eventually forms a fistula to the lower lid skin, a few millimeters under the medial canthus. The purulent exudate often causes and sustains a chronic conjunctivitis.
Diagnosis: Dacryocystitis has the appearance of a chronic purulent conjunctivitis, with a normal or elevated STT value. After the conjunctival sac has been irrigated, pressure on the medial canthus produces a purulent exudate from the puncta. Dacryocystorhinography and/or CT scanning can be used for diagnosis and also to determine the most effective method of correcting the lesion.
Therapy: For purposes of both diagnosis and therapy, the tear drainage system is irrigated (5% acetylcysteine can be added to the irrigation fluid) to remove the exudate. If the passage is thereby opened, a viscous antibiotic-corticosteroid combination solution (preferably after sensitivity testing) is introduced into the lacrimal sac. This treatment is repeated 2–4 times daily for at least 1 week. If the animal resists or is too difficult to handle, the procedure must be carried out under anesthesia, but since repeated anesthesia is impossible, referral for catheterization should be arranged instead.[9]
The first step in catheterization is the introduction of a 3-0 monofilament nylon suture (its tip rounded by heating), preferably via the dorsal punctum and thence through the nasolacrimal duct to the nose. Retrograde irrigation of the nasolacrimal drainage apparatus can be performed, but is less easy. A silicone tube (0.7–1.3 mm) is passed over this thread and pulled through to the nose. After removal of the nylon thread, the end of the tube is fixed to the skin with sutures near the medical canthus and the angle of the nose. After-care consists of administering the same antibiotic-corticosteroid preparation 4 times daily. The tube is left in place for at least 3 weeks and the administration of the medication is continued for an additional 10 days. A protective collar is placed on the animal to prevent removal of the tube.
If the passage cannot be opened up by irrigation and/or if catheterization is not possible, the only solution is to perform a dacryorhinostomy, for which the patient has to be referred.
The simplest but also the most traumatic and least controlled method is a conjunctivorhinostomy. By using a Kirschner wire, a canal is drilled into the nose. As the wire breaks through into the nasal cavity, uncontrollable hemorrhage can occur. A silicone or polyethylene tube can be passed over the wire to keep the canal open.
A better method is a conjunctivomaxillorhinostomy[30] in which a hole about 7 mm in diameter is made rostral to the medial canthus, passing into the maxillary sinus in dogs and into the nasal passage in cats. The hole is then enlarged in the direction of the medial canthus until it reaches the edge of the orbit, so that the nasolacrimal duct is laid open to its orbital entrance.

The two ends of a silicone tube are introduced through the existing puncta (or newly created if necessary) to the maxillary sinus (dog) or nasal passage (cat). The ends of a silk or nylon suture, which have previously been passed through the tube, are tied and the knot is turned into the tube. The tube is inserted through the hole into the maxillary sinus and the skin over the hole is closed. After-care consists of the previously described topical treatment and a protective collar. The tube should be left in place for at least 3 weeks. Removal of the tube is as described for atresia.

5 Lacerations

Lacerations of the medial canthus are rare. They are usually in the lower lid and accompanied by laceration of the lacrimal ducts. The canaliculi are located in the swollen wound by intubation. An S-shaped 000 probe or Worst pigtail probe (preferably with an eye in the tip) is introduced via the upper punctum into the wound. A 0.7–1.3 mm diameter silicone tube is either placed over the tip of the probe or cut on a slant and inserted into the eye in the tip of the probe. The tube is pulled through the upper punctum via the entrance in the wound and the sacculus. The probe is then introduced through the lower punctum to the wound and the other end of the silicone tube is pulled through the rest of the lower canaliculus to the lower punctum. The ends are tied together by a 5-0 silk suture. This method is quick, but both ends may irritate the cornea. Irritation from the ends can be avoided by making a circular tubing or if the ends are fixed to the medial canthal skin using a simple interrupted suture. A strand of 6-0 monofilament nylon is passed through the tube, the length of the tube is adjusted and the strand is knotted, leaving the tube in a ring through the canaliculi and sacculus. The wound in the lid margin is closed with a figure-of-eight suture and the rest of the wound by single interrupted sutures. The tubing is left in place for 2–3 weeks.

6 Cysts and neoplasia

The occurrence of congenital cysts or the development of neoplasms of the tear glands or of the lacrimal drainage system are rare. When such abnormalities are suspected, the patient should be referred for diagnosis and eventual treatment.

Literature

1. Ginel, P. J., Novales, M., Garcia, M., et al.: Immunoglobulins in stimulated tears of dogs. Am. J. Vet. Res. **54**:1060, 1993.

2. German, A. J., Hall, E. J. & Day, M. J.: Measurement of IgG, IgM, and IgA concentrations in canine serum, saliva, tears and bile. Vet. Imm. Immunopath. **64**: 107, 1998.

3. Davidson, H. J. & Kuonen, V. J.: The tear film and ocular mucins. Vet. Ophthalmol. **7:** 71, 2004.

4. Carrington, S. D., et al.: Polarized light biomicroscopic observations on the pre-corneal tear film. I. The normal tear film of the dog. J. Small Anim. Pract. **28**: 605, 1987.

5. Prydal, J. I. & Campbell, F. W.: Study of precorneal tear film thickness and structure by interferometry and confocal microscopy. Invest. Ophthalmol. Vis. Sci, **33**: 1996, 1992.

6. King-Smith, P. E., Fink, B. A., et al.: The thickness of the human precorneal tearfilm: evidence from reflection spectra. Invest. Ophthalmol. Vis. Sci. **41**: 3348, 2000.

7. Gipson, I. K., Yankauckas, M., Spurr-Michaud, S. J., et al.: Characteristics of a glycoprotein in the ocular surface glycocalyx. Invest. Ophthal. Vis. sci. **33**: 218, 1992.

8. McLaughlin, S.A., et al.: Effect of removal of lacrimal and third eyelid glands on Schirmer tear test results in cats. JAVMA **193**: 820, 1988.

9. Sams, R.: Ohio State University, personal communication. 2003.

10. Peiffer R. L. & Harling, D. E.: Third eyelid. *In*: Textbook of small animal surgery. Edited by D. H. Slatter, Philadelphia, Lea & Febiger, 1501–1509, 1985.

11. Meij, B. P., Voorhout, G., et al.: Transsphenoidal hypophysectomy in beagle dogs: evaluation of a microsurgical technique. Vet. Surg. **26**: 295, 1997.

12. Azzarolo, A. M., Bjerrum, K., et al.: Hypophysectomy induced regression of female rat lacrimal glands: partial restoration and maintenance by dihydrotestostrone and prolactin. Invest. Ophthalmol. Vis. Sci. **36**: 216, 1995.

13. Slatter, D.: Fundamentals of veterinary ophthalmology. Philadelphia, W.B.Saunders, 249, 1990.
14. Bryan, G. M. & Slatter, D. H.: Keratoconjunctivitis sicca induced by phenazopyridine in dogs. Arch. Ophthalmol. **90**: 310, 1973.
15. Ludders, J. W. & Heavner, J. E.: Effect of atropine on tear formation in anesthetized dogs. JAVMA **175**: 585, 1979.
16. Hollingsworth, S. R., Canton, D. D., Buyukmihici, N. C. & Farver, T. B.: Effect of topically administered atropine on tear production in dogs. JAVMA **200**: 1481–1484, 1992.
17. Vestre, W. A., et al.: Decreased tear production associated with general anesthesia in the dog. JAVMA **174**: 1006, 1978.
18. Arnett, B. D., Brightman, A. H. & Musselman, E. E.: Effect of atropine sulfate on tear production in the cat when used with ketamine hydrochloride and acetylpromazine maleate. J. Am. Vet. Med. Assoc. **185**: 214, 1984.
19. Kaswan, R. L., Martin, C. L. & Dawe, D. L.: Keratoconjunctivitis sicca: immunological evaluation of 62 canine cases. Am. J. Vet. Res. **46**: 376, 1985.
20. Staman, J., Goudswaard, J., Stades, F. C. & Wouda, W.: Sjögrenssyndrome (Keratoconjunctivitis in combination with xerostomy) in the dog, Proceedings Voorjaarsdagen, Neth. Sm. Anim. Vet. Assoc. 10–11 April, 1978.
21. Lettow, E. & Hildebrand, B.: Keratoconjunctivitis sicca beim Hund. Tierärtzl. Prax. **5**: 351, 1977.
22. Kaswan, R. L., Salisbury, M. A. & Ward, D. A.: Spontaneous canine keratoconjunctivitis sicca: A useful model for human keratoconjunctivitis sicca. Treatment with cyclosporine eye drops. Arch. Ophthalmol. **107**: 1210, 1989.
23. Bardoulay, A, English, R. V., et al.: The effect of topical Tacrolimus aqueous suspension on tear film production in dogs with keratoconjunctivitis sicca. Trans. Am. Coll. vet. Ophthalmologists. **34**: 33, 2003.
24. Rubin, L. F. & Aguirre, G. D.: Clinical use of pilocarpine for keratoconjunctivitis sicca in dogs and cats. JAVMA **151**: 313, 1969.
25. Lavignette, A. N.: Keratoconjunctivitis sicca in a dog treated by transposition of the parotid salivary duct. JAVMA **148**: 778, 1966.
26. Wolf, E. D. & Merideth, R.: Parotid duct transposition in the horse. J. Equine Vet. Sci. **1**: 143, 1981.
27. Stades, F. C.: Apparato Lacrimalia. *In* Atlante di Oftalmologia veterinaria. Ed.: C. Perrucio, Torino, Edzione medico scientifico, 152–156, 1985.
28. Hoover, E. A., Rohovsky, M. W. & Griesemer, R. A.: Experimental feline viral rhinotracheitis in the germ free cat. Am. J. Pathol. **58**: 269, 1270.
29. Nasisse, M. P., Guy, J. S., Davidson, M. G., et al.: Experimental ocular herpesvirus infection in the cat. Sites of virus replication, clinical features and effects of corticosteroid adminstration. Invest. Ophthalmol. Vis. Sci. **30**: 1758, 1989.
30. Lundvall, R. L. & Carter, J. D.: Atresia of the nasolacrimal meatus in the horse. JAVMA **159**: 289, 1971.
31. Ladouceur, C. A. & Kazocos, K. R.: *Thelaxia lacrimalis* in horses in Indiana. JAVMA **178**: 301, 1981.
32. Severin, G. A.: Nasolacrimal duct catheterization in the dog. JAAHA **8**: 13, 1972.
33. Stades, F. C.: Dacryozystomaxillorhinostomie. Eine neue chirurgische Therapie zur Dacryozystitis: Tierärztl. Prax. **6**: 243, 1978.