Op – Ophthalmology
EXAMINATION OF THE BLIND ANIMAL

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1. History

a)Is the blindness of sudden onset, or gradual onset?
b)Was deterioration of vision associated with preferential loss of night vision or day vision?
c)Is the animal healthy? Are there other signs of illness, besides loss of vision?

2. Localizing the Lesion in the Blind Animal

The blind patient, like all patients, should undergo a comprehensive physical examination. This is because in many cases, the cause of blindness is a systemic disease. Likewise, a neurological evaluation should be performed to rule neuro-ophthalmological causes of blindness.
Next, a full ophthalmological examination is conducted. It is described in detail in a separate part of these proceedings ("the Ocular Examination"). Particular attention is paid to methods of assessing vision (e.g., the menace response and obstacle course), and to the subcortical tests (e.g., the pupillary light reflex, dazzle reflex, etc.). Additional tests, such as imaging (ultrasound, CT) or electroretinography (see elsewhere in the Proceedings) may also be conducted.
Based on the results of the ocular examination and the pupillary light reflex (PLR), the blind patient may be categorized into one of 4 general categories:

a)Abnormal ophthalmic findings combined with a normal/diminished PLR:

- Opacity of the ocular media: severe blepharospasm, corneal edema, hyphema/hypopyon, cataract, vitreal hemorrhage
- Retinal disease – outer retinal degeneration (PRA), chorioretinitis

b)Abnormal ophthalmic examination and an absence of PLR:

- Glaucoma
- Retinal detachment
- Optic neuritis involving the proximal portion of the optic nerve, and causing papilledema
- Optic nerve hypoplasia/atrophy

c)Normal ophthalmic examination and an absence of PLR:

- Sudden Acquired Retinal Degeneration (SARD)
- Optic neuritis involving the distal portion of the optic nerve
- Neoplasia of the optic nerve or neoplasia compressing the optic nerve or chiasm
- Lesions affecting the contralateral optic tract, up to the level of the lateral geniculate nucleus.

d) Normal ophthalmic examination, and normal PLR: These are usually neurological cases, caused by central lesions affecting the visual pathways from the lateral geniculate nucleus to the contralateral visual cortex.

II. DISCUSSION OF SELECT CAUSES OF BLINDNESS

Many of the diseases listed above, including corneal opacities, cataracts, hereditary retinal diseases and glaucoma, are discussed in other parts of these Proceedings. Below is a discussion of the leading causes of acute blindness (other than glaucoma).

1. Retinal Detachment

Retinal detachment is a separation between the retina and choroid (more specifically, between the retina and retinal pigment epithelium). A result of the separation is ischemia of the photoreceptors. If the separation is not quickly resolved, and blood supply restored, cones & rods will begin dying, leading to irreversible blindness.
There are 3 types of detachments, depending on the mechanism of their formation. *Serous detachment* is caused by accumulation of fluid in the subretinal space, between the retina and choroid. This fluid, which originates in the choroid, may be blood or exudates. *Traction detachment* is caused by a force which pulls the retina off the choroid. This force may be generated by forward movement of the vitreous

body (for example, following anterior lens luxation) or due to traction by fibrin clots. *Rhematogenous detachment* is due to penetration of liquefied vitreous into the subretinal space, through retinal holes.

Causes of Retinal Detachment

The list of possible causes for retinal detachment depends on the type of detachment.

- Rhematogenous detachment may be caused by senile changes, trauma or inflammation (see below)
- Traction detachment may be caused by lens luxation, or by inflammation (see below)
- Serous detachments are caused by bleeding or inflammation.

Causes of exudative (serous) detachment

An inflammation that leads to retinal detachment is usually one that involves the choroid and retina (chorioretinitis or retinochorioditis). As is the case for anterior uveitis, it is conceivable that any systemic or ocular inflammation will lead to chorioretinitis. However, chorioretinitis is usually an inflammation caused by an infectious agent. These can be viral (distemper in the dog; FIP, FeLV and FIV in the cat), rickettsia (Ehrlichia canis), protozoal (Leishmania, Toxoplasma) or fungal infections.

Causes of hemorrhagic (serous) detachment

Any cause of systemic bleeding could result in a hemorrhagic retinal detachment. Common causes include systemic hypertension, thrombocytopenia (Ehrlichia canis), coagulopathies, hyperviscosity, anemia and trauma

Clinical Signs Of Retinal Detachment

- Blind eye (no menace response)
- Fixed dilated pupil. A consensual PLR will be present when stimulating the contralateral eye.
- When performing an ophthalmoscopic examination, the clinician will find it difficult to focus on the retina (since it moved from its natural place). It is possible to see a "sheet" floating in the posterior part of the eye. This sheet, which is the retina, may be transparent, white (i.e., edematous), or hemorrhagic, depending on the cause of detachment. Retinal blood vessels may be seen on it even without the use of an ophthalmoscope.
- Ultrasound. A 10 MHz probe can image the detached retina. This image is called "the seagull sign", because the detached retina usually remains anchored to the eye in the optic disc and to the ora serrata. An ultrasound examination is particularly useful when an ophthalmoscopic examination can not be conducted due to severe corneal edema, hyphema, etc.

Treatment of Retinal Detachment

- It is imperative to diagnose the primary cause of the detachment, and to treat it. Therefore, systemic workup has to be performed. Depending on the type of detachment, this workup should be aimed at diagnosing cardiovascular or infectious diseases.
- Lens extraction surgery is indicated in when the detachment is secondary to anterior lens luxation
- Fibrin clots and strands can be dissolved by injecting tissue plasminogen activator (TPA) into the eye, thus preventing traction detachments.
- Treatment of exudative serous detachments involves draining the subretinal fluid. This may be done using hyperosmotic agents. Systemic carbonic anhydrase inhibitors should also be considered. If the cause of the exudate is inflammatory, systemic steroids should be considered.
- Specialized referral centers may perform surgery to re-attach the retina, or to "seal" retinal holes.

2. Sudden Acquired Retinal Degeneration (SARD)

This is an acquired disease of an unknown cause, typically appearing in middle-aged (female) dogs. The history provided is one of sudden onset blindness. The typical patient is "cushinoid". In many dogs, owners will report a history of lethargy, weight gain and PU/PD during the last few months. Bloodwork is also suggestive of Cushing's disease

Examination will reveal a blind eye with a fixed, dilated pupil. The fundus appears normal during the first few months. Degenerative changes may appear at a later stage (few months). The ERG is flat, indicating lack of retinal activity.

Currently there is no treatment for SARD. Hopefully, once the cause is identified, treatment can be offered.

3. Optic Neuritis

A. Cause

An inflammation of the optic nerve caused by:

- Any cause of meningitis
- Infectious causes – distemper, fungal diseases (e.g., Cryptococcus), toxoplasma, bacteremia, etc. In many of the systemic disease, the ocular signs may be the presenting complaint.
- Neoplasia, trauma or an abscess in regions where the optic nerve passes (especially at the optic chiasm!)
- CNS diseases – GME, reticulosis, etc.
- Idiopathic – probably the most common cause

B. Diagnosis

• Blind eye with a fixed, dilated pupil
• ERG is normal, since the retina is not affected (thus distinguishing optic neuritis from SARD)
• The optic disc appears normal or inflamed, depending on which part of the nerve is involved. If the proximal part of the optic nerve is involved, papilledema and vascular congestion of the optic disc are seen on examination of the fundus. Atrophy of the optic disc is noticed as the disease resolves. Inflammation of more distal parts of the nerve may present with a normal-looking disc.

C. Treatment

Treatment is based on identifying and treating the primary cause. Systemic steroids should be administered if no systemic cause is found. Prognosis is guarded.

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OCULAR EXAMINATION

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An ophthalmic examination should not be a scary experience! Though admittedly interpretation of the findings may sometimes be challenging, the examination itself follows a logical, anatomical order. Furthermore, it does not require expensive equipment. In fact, the most important items required are non-ophthalmic in nature: a room that can be darkened, a good source of focal light and a magnifying loupe. A hand held lens, a direct ophthalmoscope, a Schiotz tonometer and some disposable items (stains, solutions, etc.) complete the list of basic equipment.

As with any other system, the clinician should pay particular attention to the signalment. Numerous ocular diseases may be breed- or age-related. Since many ophthalmic disorders may be manifestations of systemic diseases, a general history should be taken and a comprehensive physical examination should be conducted. Similarly, if neuro-ophthalmological abnormalities are present (blindness, strabismus, anisocoria, etc.), the neurological system should be evaluated, as these may be signs of a nervous system disease.

1. Gross Inspection

The patient should be observed as it walks into the room, since this is an unfamiliar environment which may highlight visual deficits; these will be further evaluated later on. Following the anamnesis and physical examination, the ocular assessment begins by careful observation of the patient from a distance, without touching the patient (as this may cause distortion of palpebral fissure). While observing, ask yourself:

• Are both eyes open normally? Is there evidence of pain or photophobia? Is the animal blinking normally?
• Are the eyes of normal size and position? Is there evidence of exophthalmous or buphthalmous? Are the pupils of equal size?
• Is the eyelid conformation normal? Is there evidence of entropion or ectropion (usually of the lower lid)? Is the upper lid prolapsed? Is the 3rd eyelid elevated?
• Is there ocular discharge? What is its nature?

Next, the orbital area is palpated to detect any

fractures, abnormal swellings, etc. Use the opportunity to press on the globe through the upper lid. This serves both as a retropulsion test (which indicates the presence of a retrobulbar mass), and to proptose the 3rd eyelid, allowing inspection of its outer surface. It is *NOT* an effective way of evaluating intraocular pressure (IOP).
Inspect (grossly) the eyelids. Examine their skin surface, the mucocutaneous junction, and evert them slightly to visualize the palpebral conjunctiva and the two punctas. Use the opportunity to test the blink reflex in response to touching of the canthal skin. Continue by examining the bulbar conjunctiva and the cornea surface.

2. Assessing Vision

a) Menace Response: This involves making a sudden threatening gesture which is supposed to elicit a blink response. The afferent arm of the response consists of the retina, optic nerve axons, and the optic tract and radiations. The efferent component of the response includes the primary motor cortex, cerebellum, and the nucleus and nerve of cranial nerve VII (facial nerve).
It is important to note that the menace response involves cerebral cortical integration and interpretation and therefore is not a reflex. Rather, it is a cortical response that requires the entire peripheral and central visual pathways, as well as the visual cortex and the facial nucleus and cranial nerve, to be intact. Also, remember that the menace response is a very crude test of vision, and in fact requires visual acuity of only 6/600!
The menace response should be evaluated in one eye, while the other eye is being covered. Be careful not to touch the eyelashes/hair of the patient, or to cause wind movement, as this may lead to a "false positive" response; consider making the menace gesture behind a glass partition. Likewise, "false negative" results (lack of a menace response in a visual animal) are also possible. One possible reason is facial nerve paralysis, which is ruled out using the blinking reflex. The menace response is absent in very young (<10-12 weeks) animals, and may also be affected by the patient's mental state.
b) Additional Visual Tests: Vision can also be evaluated using an obstacle course. You should be consistent in the obstacle course that you construct, and make sure it can be navigated by normal animals! Test the patient in light and dim conditions, and consider patching one eye.
Another test is the visual placing response, which is useful when results of the obstacle course and menace response are equivocal. It is conducted by lifting the animal towards the table, allowing it to see the approaching surface. A normal animal will extend its leg towards the surface before its paw touches the table.

3. Examination in the dark

After the light has been dimmed, the dilatation of the pupils should be evaluated. Use a dim light (to prevent constriction), and stand at a distance so you can visualize both pupils simultaneously, using the tapetal reflection. The tapetal reflection also serves to highlight (by means of retro-illumination) any ocular opacities, particularly in the lens or vitreous.
Next, use a bright light to evaluate the Pupillary Light Reflex (PLR). Unlike the menace response, the PLR is a subcortical reflex. Therefore, it does NOT test vision, and a normal PLR may be found in a cortically blind animal. Furthermore, the PLR is usually present (though it may be diminished or slow) in animals suffering from outer retinal degeneration (PRA), cataracts, and other causes of subcortical blindness. Nevertheless, the PLR is a very important test, which helps localize the lesion which causes loss of vision.
If one of the pupils does not react to light, or if it can not be visualized (e.g., in cases of severe corneal edema or hyphema), the consensual PLR should be checked. Alternatively, you can check the dazzle reflex. This is also a subcortical reflex, which is manifested as a bilateral, partial blink in response to a bright light.
Magnification is required for the next stages of the examination. Once again, the lid margins, conjunctiva and corneal surface are examined. Use the magnification to check for aberrant eyelashes (trichaisis, distichia); these can be best visualized against the white background of the conjunctiva, by slightly pulling the eyelid. Following the anatomical order, next inspect the anterior chamber (looking for opacities in the aqueous), the iris surface and the anterior segment of the lens.

4. Ophthalmoscopy

This part of the examination is the one which clinicians usually dread the most. Part of this undoubtedly stems from the large range of normal variations in the appearance of the canine (and, to a lesser extent, the feline) fundus. Admittedly, if you are not in the habit of examining fundii, you will find it difficult to diagnose abnormalities. You should therefore make a habit of examining, however briefly, the fundus of every patient that you see. Your clients will appreciate the extra touch, and you will gain the required proficiency.
Due to the high cost of an indirect ophthalmoscope, only a direct ophthalmoscope is available in most general practices. This instrument provides a high magnification (x16 in an average dog). The unfortunate consequence of the high magnification is a small viewing field (4°), extending the time required to examine the entire fundus. A quick

overview of the fundus may be obtained using a bright light source and a handheld lens (20-30D), providing a means of monocular "indirect ophthalmoscopy". The direct ophthalmoscope comes with several features:

• A grid (graticule) - use it to compare the size of the lesion to the size of the optic disc
• Red-free filter (emits green light) - helps evaluation of hemorrhage and blood vessels, which appear black.
• Apertures of varying diameter-use the largest one that is appropriate for the patient's pupil
• Changing lenses permits the examiner to evaluate the depth/height of a lesion, or to examine more anterior structures, such as the lens. A raised lesion will come into focus by adding convex/converging lenses (+). A depression/coloboma will come into focus by adding concave/diverging lenses (-). In dogs, each diopter you add is equivalent to 0.28 mm.
• Use of a narrow beam allows to evaluate depressions and elevations of fundus lesions

Ophthalmoscopy should be conducted in a dark room, following dilatation of the pupil. First evaluate the tapetal reflection from a distance, to detect any lenticular or vitreal opacities. As you approach the patient, focus on successively more posterior structures- cornea, iris, lens and vitreous- till you are focused on the fundus. Carefully inspect the *entire* fundus, evaluating changes in the tapetum, non-tapetum, blood vessels and optic disc. It is best to stay in stationary position and let the patient's eye movements bring the structures to you, instead of trying to "chase" them.

5. Additional tests

• Schirmer tear test is used to evaluate tear production and diagnose keratoconjunctivitis sicca. It should be conducted at an early stage of the examination, as any ocular manipulation may induce reflex tearing.
• Fluorescein staining is used to diagnose corneal ulcers. Superficial ulcers may be stained with Rose Bengal
• Samples for bacteriology, mycology and cytology may be taken as indicated. The first two should be taken before any drops are put in the eye, as ophthalmic solutions frequently contain preservatives.
• Nasolacrimal patency is evaluated by passage of fluorescein from the eye to the nose, by cannulating the nasolacrimal system and by dacryocystorhinography.
• Ultrasound is frequently used in ophthalmology. The main indications are imaging of the retrobulbar area, and imaging of the posterior segment when it can not be visualized (e.g., due to hyphema or cataract). CT and MRI techniques may be used in certain cases.
• Tonometry-measuring IOP to diagnose glaucoma.
• Additional tests, including gonioscopy (evaluation of the iridocorneal angle as part of the diagnosis of glaucoma) and electroretinography (recording electrical responses of the retina to flashes of light, to determine retinal function) may be available in referral centers, and are discussed elsewhere in these proceedings.