

## CHAPTER 1

# Equine ocular examination basic techniques

---

Riccardo Stoppini and Brian C. Gilger\*

### Introduction

The basic and essential aspect of equine ophthalmology is a complete, thorough ocular examination. In this chapter, ophthalmic examination of the horse is discussed, with emphasis on techniques, tools, and instruments, and basic diagnostic modalities. Understanding normal equine ocular anatomy is integral to interpreting the ocular examination. Anatomy of every equine ocular compartment (Figure 1.1) is described in detail in subsequent chapters and is not discussed here, although cross-references to relevant sections in the book will augment our discussion. Excellent reviews of equine eye and head anatomy can also be found in other sources [1–6].

Routine ophthalmic diagnostic techniques are described here, while advanced diagnostic techniques are discussed in Chapter 2. Examination of the equine eye includes obtaining a detailed history and signalment, inspecting the patient in a well-lit environment, examining the ocular structures in a darkened environment, facilitating the examination with restraint, sedation, and local nerve blocks, and collecting relevant diagnostic samples or data [4, 7–10].

### Medical history

A thorough medical history relevant to the ocular examination should include how the animal is used (e.g., pet or performance) and its living environment. Additional information that should be collected includes any history of travel, vaccination history, deworming schedule, and presence of concurrent or previous medical problems such as nasal discharge, presence of stridor,

previous trauma to the head, and status of other horses on the premises with similar signs (Box 1.1). Characterization of the primary complaint should include the onset and initial clinical signs, treatment given and response to that treatment, progression and duration of the ocular problem, and current therapy. Signalment can provide an important clue as to the cause of many ophthalmic conditions (e.g., congenital stationary night blindness in the Appaloosa, hereditary cataracts in the Morgan horse). Existing medical therapy can also greatly influence findings on ophthalmic examination. For example, a finding of mydriasis on the ophthalmic examination could be caused by use of topical atropine, which may result in mydriasis for up to 14 days in horses [11]. Depending on the specific complaint, further information may be required, such as a thorough description of vision loss (e.g., light versus dark, moving objections, one eye or both) or changes in behavior during sport activity.

### Routine and advanced equipment required for the ophthalmic examination

Prior to any examination, the proper equipment to perform the examination is needed. Although there are some differences in opinion and personal preferences among equine ophthalmologists, Box 1.2 lists the routine and advanced equipment a clinician should have available for the ophthalmic examination. Routine materials for the examination are shown in Figures 1.2 and 1.3.

### Overview and methods of the equine ocular examination

Ocular examination in the horse, like any physical examination, should be performed in a systematic manner, and to help remember the steps, the compilation of a clinical report or

---

\* The authors would like to thank Dr Tammy Miller Michau for her contributions. She was the author of this chapter in the first edition of *Equine Ophthalmology*.



(A)



(B)

**Figure 1.1** (A) Normal external appearance of the equine eye. The horse's palpebral fissure, cornea, and pupil are oval horizontally. The lateral canthus (L) is more rounded than the medial canthus (M). There are prominent folds in the upper and lower eyelids. Numerous eyelashes are present along the lateral two-thirds of the upper eyelid, and vibrissae are located dorsonasal to the upper lid and ventral to the lower lid (arrow). The leading edge of the third eyelid is usually partially pigmented (N). The lacrimal caruncle (Lc) is prominent. (B) Normal external appearance of the equine eye when eyelid pigment is absent. Note the lack of pigment on the third eyelid, conjunctiva, and sclera as well.

**Box 1.1** Anamnesis and horse history.

- Horse attitude and use (jumping, trotter, dressage, pleasure, etc.)
- Travel and environment history
- Medical history (previous trauma, incident, etc.)
- Time of purchase and PPE (pre-purchase examination)
- Horses in contact and eventual infectious diseases
- Vaccination status
- Deworming history
- Reaction/drug anaphylaxis events
- Breeding info (in case possible presence of heritable pathologies)
- Previous medical history (PMH) (nasal discharge, coughing, colic, etc.)
- Previous ocular history
- Previous ocular treatments (medical/surgical)
- Previous abnormal/changed behavior

examination sheet may be helpful (Box 1.3). The general order of steps to be taken is shown in Box 1.4. The initial examination of the equine eye should occur prior to sedation and should take place in a well-lit area. The examination area should be quiet, away from distractions, and if possible away from other horses. The menace response and other subjective vision testing (e.g., maze testing) and evaluation of the pupillary light reflex (PLR) should be performed before sedation. For accurate evaluation of PLRs, a bright focal light source and a darkened examination area are often required. To adequately examine the cornea and internal structures of the eye, the horse must be examined in a darkened stall or in stocks in a room where the lights can be dimmed. Ideally, stocks are preferred because they will better protect the examiner from accidental (or purposeful!) movements of the horse, which can be exaggerated and unpredictable when the horse is tranquilized.

A thorough ocular examination usually requires appropriate restraint, tranquilization, regional nerve blocks, and topical anesthesia. Methods of restraint required to examine the ocular

**Box 1.2** Equipment and supplies for general equine ophthalmic examination.

**Routine equipment and supplies**

- Bright focal light source: Finnoff halogen transilluminator
- Direct ophthalmoscope, PanOptic ophthalmoscope, or indirect ophthalmoscope with 20-D lens
- Sterile dye strips (fluorescein, rose bengal, lissamine green)
- STT
- Sterile culture swabs for cytology and culture
- Kimura platinum spatula, #10 to #15 sterile surgical blade (cytology)
- Glass slides (cytology)
- Sterile eyewash
- Ophthalmic lubricant, artificial tears ointment
- Topical anesthetic: proparacaine HCl 0.5%
- Short-acting dilating agent: tropicamide HCl 1%
- Sedatives: detomidine HCl, xylazine, acepromazine, romifidine, etc.
- Local nerve block agents: mepivacaine HCl, lidocaine HCl, bupivacaine HCl
- Graefe fixation forceps (to manipulate conjunctiva and third eyelid)
- Digital tonometer (Tono-Pen or TonoVet tonometer)
- Open-ended tomcat urinary catheter for nasolacrimal irrigation
- 1- and 3-mL syringes, 18- and 25-G needles
- 12-mL syringes for nasolacrimal irrigation

**Advanced equipment and supplies**

- All supplies and instruments for routine examination
- Handheld slit-lamp biomicroscope
- Binocular indirect ophthalmoscope and lenses (15-, 20-, 30-D or 2.2 panretinal)
- Ultrasound (7.5-, 10-, and 20-MHz probe)
- Sterile methylcellulose gel for ultrasound
- Electroretinogram
- 19-G, 3½-inch spinal needle for retrobulbar block
- 27-G needle for aqueocentesis
- Digital camera (color and infrared)
- Advanced imaging: radiography, computed tomography (CT), magnetic resonance imaging (MRI)



**Figure 1.2** Materials and supplies for a routine ophthalmic examination in the horse. (A) 18-G, 1½-inch needle, 25-G, 1-inch needle, 1 mL and 3 mL syringes. (B) Tranquilizer (detomidine). (C) 2% Lidocaine HCl. (D) STT strips. (E) Fluorescein dye strips. (F) 1% Tropicamide HCl. (G) Sterile eyewash. (H) Direct ophthalmoscope. (I) Finnoff transilluminator. (J) Indirect ophthalmoscopy lens (20 D). (K) Panoptic ophthalmoscope.



**Figure 1.3** Materials for collection of corneal culture and cytology. (A) 0.5% Proparacaine HCl. (B) 25-G, 1-inch needle and 3-mL syringe. (C) 2% Lidocaine HCl. (D) Kimura platinum spatula. (E) #15 surgical blade. (F) Microscope slides. (G) Blood agar culture plate. (H) Sabouraud agar culture plate. (I) Thyoglycolate medium.

**Box 1.3** Equine ophthalmology examination form.

**Equine Ophthalmology Examination Form**

Owner: \_\_\_\_\_

Referring Veterinarian: \_\_\_\_\_

Horse: \_\_\_\_\_

First visit: \_\_\_\_\_

Remote history: \_\_\_\_\_

Recent history: \_\_\_\_\_

**RIGHT EYE**

PLR dir: \_\_\_\_\_

PLR cons: \_\_\_\_\_

Dazzle Refl: \_\_\_\_\_

Corneal Rfl: \_\_\_\_\_

Palpebral Rfl: \_\_\_\_\_

Menace response: \_\_\_\_\_

IOP: \_\_\_\_\_

SIT I-II: \_\_\_\_\_

TBUT: \_\_\_\_\_

Globe and orbit: \_\_\_\_\_

Eyelids \_\_\_\_\_

Conjunctiva: \_\_\_\_\_

NLD: \_\_\_\_\_

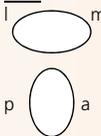
Cornea: \_\_\_\_\_



Iris and AC: \_\_\_\_\_



Lens: \_\_\_\_\_



Vitreous: \_\_\_\_\_

Fundus: \_\_\_\_\_



**LEFT EYE**

PLR dir: \_\_\_\_\_

PLR cons: \_\_\_\_\_

Dazzle Refl: \_\_\_\_\_

Corneal Rfl: \_\_\_\_\_

Palpebral Rfl: \_\_\_\_\_

Menace response: \_\_\_\_\_

IOP: \_\_\_\_\_

SIT I-II: \_\_\_\_\_

TBUT: \_\_\_\_\_

Globe and orbit: \_\_\_\_\_

Eyelids \_\_\_\_\_

Conjunctiva: \_\_\_\_\_

NLD: \_\_\_\_\_

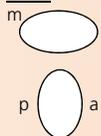
Cornea: \_\_\_\_\_



Iris and AC: \_\_\_\_\_



Lens: \_\_\_\_\_



Vitreous: \_\_\_\_\_

Fundus: \_\_\_\_\_



structures of the horse range from a halter and lead rope to mechanical restraint in stocks with use of a lip twitch. Use of restraint is dependent on temperament of the horse and experience of the handlers and examiner. Use of a tranquilizer is generally recommended, and frequently needed, to facilitate the complete routine ophthalmic examination.

**Initial examination**

With the examiner positioned in front of the horse, the head, bony orbits, eyelids, globes, and pupils should be examined for symmetry (Figure 1.4) before touching the eyelids, giving sedation, or use of eyelid nerve blocks. Ocular comfort may be assessed by evaluation of palpebral fissure size and symmetry,

**Box 1.4** General order of steps in the routine equine ocular examination.

- 1 Obtain medical and ocular history.
- 2 Examine horse in its environment.
  - Observe walking on a lead or loose in a stall or round pen.
  - Observe horse performing activity such as jumping, cutting, etc.
- 3 Evaluate for symmetry from the front of the head.
  - Observe globe, orbit, pupils, eyelash direction, ear and lip position.
- 4 Perform vision testing.
  - Menace response, dazzle reflex, maze testing.
- 5 Perform palpebral and pupillary light reflexes.
- 6 Perform STT if indicated.
- 7 Administer sedation if required.
- 8 Perform palpebral nerve block.
- 9 Use direct transillumination for gross disease of eyelids, cornea, anterior chamber, and iris.
- 10 Collect samples for culture and cytology if indicated.
- 11 Perform detailed examination of the eyelids, cornea, anterior chamber, and iris with transillumination and biomicroscopy.
- 12 Test corneal reflex.
- 13 Apply topical fluorescein and examine the cornea.
- 14 Apply topical anesthesia if indicated.
- 15 Perform tonometry.
- 16 Induce mydriasis (tropicamide HCl) if not contraindicated.
- 17 Perform detailed examination of the lens and vitreous, via transillumination, retroillumination, and biomicroscopy.
- 18 Perform direct and/or indirect ophthalmoscopy.
- 19 Irrigate nasolacrimal duct(s) if indicated.

position of the eyelashes, ocular discharge, and blink rate [4, 8, 9]. The upper eyelashes of the healthy horse are nearly perpendicular to the cornea (Figure 1.5A) [8]. A ventral or downward direction of the eyelashes in relation to the cornea may indicate blepharospasm, enophthalmos, or ptosis (see Figure 1.4B–D and Figure 1.5B), while an upward deviation may indicate exophthalmos or an enlarged eye (see Figure 1.4C) [8].

The examiner should then be positioned at the side of the horse's head to examine each eye individually. An assistant may be required to elevate the head of a sedated horse to the same level as the examiner's eyes. The examiner may need to use a stool for an extremely tall horse or kneel on the ground for an extremely short horse (e.g., Miniature horse), although this should be done with great caution and never directly in front of the horse.

A cranial nerve (CN) evaluation (specifically, CN II, III, IV, V, VI, VII) is then performed before any sedation is induced. These CNs are assessed via the menace response, pupillary light and dazzle reflexes, globe and eyelid position and mobility, and sensation of ocular and adnexal structures [8, 9]. Examination of the CNs is discussed in more detail in the following section.

The cornea should be examined for abnormalities (e.g., opacities, ulceration, blood vessels, edema) by using transillumination and/or slit-lamp biomicroscopy. Evaluation of resting pupil size, shape, and mobility, and appearance of the anterior chamber structures should follow, including the assessment for aqueous flare. The attachment of the iridocorneal angle

pectinate ligaments to Descemet's membrane (i.e., gray line) can be observed medially and laterally in the adult horse (Figure 1.6A, B) and allows for direct visualization of the horse's iridocorneal angle. Collection of cultures and Schirmer tear values are performed, when indicated, prior to placing any medications into the eyes. When indicated, cytology is collected next, usually after application of topical anesthetic (e.g., proparacaine HCl 0.5%, oxibuprocaine chloridrate 0.4%, tetracaine). Fluorescein staining of the cornea is then performed. Examination of the nasolacrimal system, third eyelid, and conjunctiva is performed concurrently. Fluorescein staining is followed by induction of topical anesthesia, if not already given to collect cytology and perform tonometry. The ocular media (cornea, aqueous humor, lens, and vitreous) are evaluated for clarity and transparency by transillumination and ophthalmoscopy [4, 9]. The anterior surface of the third eyelid can be examined by gently retropulsing the globe to produce passive prolapse of the nictitans. For evaluation of the posterior surface, the third eyelid can be gently grasped with Graefe fixation forceps or manipulated with a strabismus hook.

For complete examination of the lens and posterior segment, mydriasis is required. The most common mydriatic used is 1% tropicamide HCl eyedrops, which takes effect in approximately 10–20 minutes and lasts 4–6 hours [12, 13]. In the case of severe intraocular inflammation or reflex uveitis because of corneal disease or trauma, a single application of tropicamide may not be sufficient to dilate the pupil. Topical phenylephrine (2.5% or 10%) does not cause mydriasis in normal horses, nor does it enhance the mydriatic effect of tropicamide [14, 15]. The use of atropine for routine examination is not recommended because of its longer duration of action and potential adverse effects in the horse [11, 16]. After mydriasis has been achieved, the clarity, position, and size of the lens, vitreous body, optic nerve, retinal blood vessels, and the tapetal and nontapetal fundus are evaluated. With full mydriasis, the edge of the lens and attachment of the zonular fibers may be visible [17].

### Cranial nerve examination

For a breakdown of the CN examination see Table 1.1.

### Vision testing

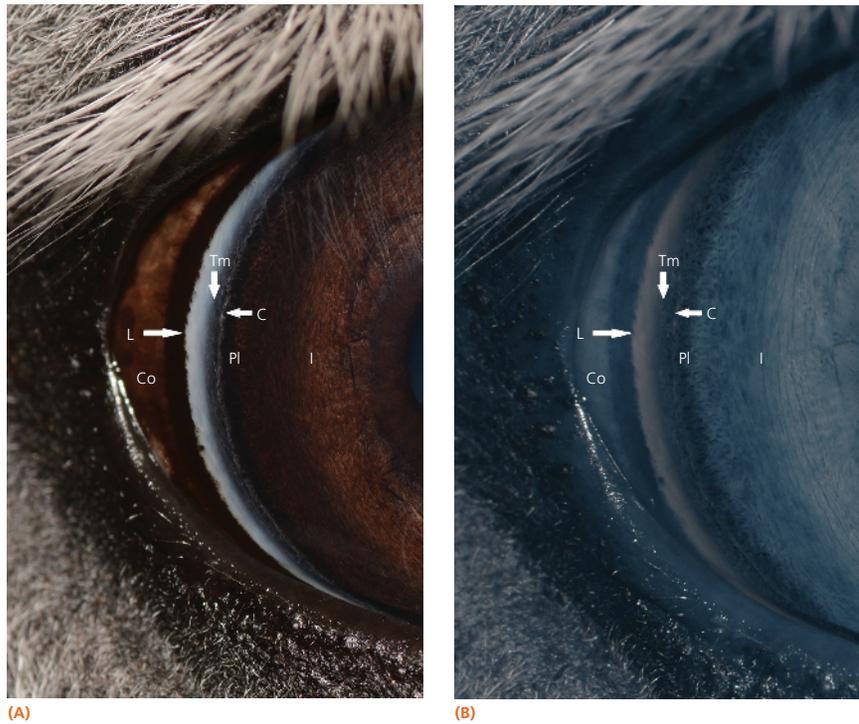
Vision testing in horses is subjective. Environmental observation, menace response, dazzle reflex, and maze testing provide only rough data. Determining total blindness is possible with these tests, but determining whether a horse has decreased vision is not easily done. Advanced diagnostic testing, such as electroretinography (see section **Electroretinography**), may help to determine if there are abnormalities in retinal electrical function but do not test vision per se. If visual function is in doubt in one or both eyes, the horse can have a unilateral blindfold and be subjected to a maze test. However, horses that are depressed, are ataxic, or have vestibular disease may stumble over objects despite having vision [9]. Equine vision and vision testing are discussed more extensively in Chapter 12.



**Figure 1.4** (A) The head (including ears and nostrils), bony orbits, eyelids, globes, and pupils should be examined for symmetry, with the examiner positioned in front of the horse. (B) Horse with orbital asymmetry due to an orbital fracture on the right side. (C) Horse with asymmetry due to a retrobulbar mass resulting in exophthalmos on the left side. (D) Horse with orbital asymmetry due to an orbital fracture resulting in enophthalmos on the left side.



**Figure 1.5** (A) The cornea and eyelids as viewed from the front in a healthy horse. The upper eyelashes are nearly perpendicular to the cornea. (B) The cornea and eyelids as viewed from the front in a horse with ocular pain. The eyelashes are no longer at 90 degrees from the corneal surface but are pointed downward.



**Figure 1.6** (A) The attachment of the iridocorneal angle pectinate ligament to Descemet's membrane (i.e., gray line) can be observed medially and laterally in the adult horse. Iris (I), pectinate ligaments (PI), attachment of pectinate ligaments to corneal endothelium (C), trabecular meshwork (Tm), limbus (L), and conjunctiva (Co). (B) More details are visible at infrared picture.

**Table 1.1** Cranial nerve examination.

CN	Name	Motor (M) or sensory (S)	Function	Dysfunction	Assessment
II	Optic	S	Vision Pupillary light pathway	Blindness	Menace response Pupillary light reflex Dazzle reflex
III	Oculomotor	Medial, dorsal, ventral rectus (M), ventral oblique Pupillary sphincter muscle (M) Levator palpebrae superioris (M)	Globe movement Pupillary function Upper eyelid elevation	Lateral strabismus Mydriasis Ptosis	Eye position Pupillary light reflexes
IV	Trochlear	Superior oblique (M)	Globe movement	Dorsomedial strabismus	Move head and observe eye position
V	Trigeminal	Maxillary branches: skin of face/eyelid (S) Ophthalmic branches: eye (S)	Sensation of skin, eyelids, and cornea	Neurotrophic keratitis	Corneal reflex
VI	Abducens	Lateral rectus (M), retractor bulbi	Globe movement	Medial strabismus	Move head and observe eye position
VII	Facial	Orbicularis oculi (M) Lacrimal gland (M)	Close palpebral fissure Stimulates tear production	Exposure keratitis KCS	Observation Palpebral reflex STT
VIII	Vestibulocochlear		Equilibrium	Spontaneous nystagmus	Observation

### Menace response

The menace response is a learned protective response in which a menacing movement toward the eye results in closure of the eyelids and possibly retraction of the globe or an avoidance movement of the head [12]. The threatening movement can be performed with the examiner's hand, but care should be taken to avoid contacting the vibrissae and to avoid causing an air current that could be detected even in a blind eye. For detection

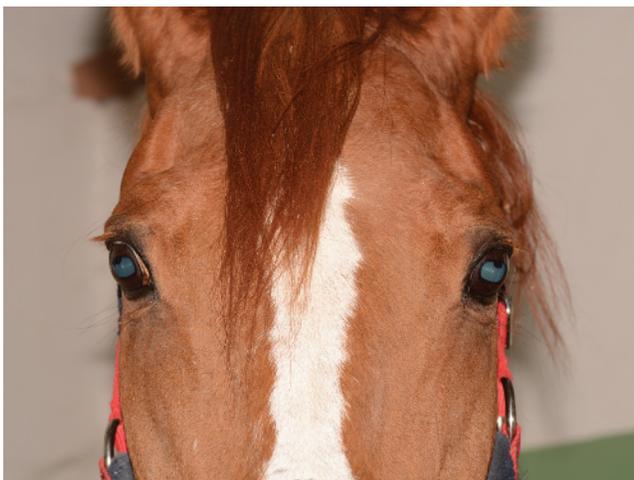
of a visual deficit in one field, the menacing gesture is directed first toward the nasal visual fields and then toward the temporal visual fields [18]. However, partial visual deficits can be extremely difficult to detect using a menace response. The afferent arm of the menace response is the retina and CN II, and the efferent arm is the palpebral branch of CN VII, which innervates the orbicularis oculi muscle (see Chapter 14) [19]. A horse that has intact vision but is extremely stoic, depressed,

or frightened may have a diminished menace response. Lightly tapping the medial or lateral canthus before attempting to induce the menace response again may heighten the response from an uninterested, stoic horse. A pathologic lack of menace response may result from a lesion in the retina, CN II, visual cortex, or CN VII [19]. Cerebellar disease can also cause bilateral deficiency in the menace response in the absence of blindness or CN VII paralysis, possibly because of a loss of cerebellar modulation of cerebral visual function [18, 19].

### Pupillary light reflexes

Both eyes should be examined for pupil size and symmetry and for abnormalities such as synechia that may affect the PLR. One method to observe pupil symmetry in the horse is to use an indirect or direct ophthalmoscope directed at the center of the horse's head from a distance of 1.5–2.0 m. This technique will illuminate the pupils via both tapetal reflexes and make it possible to evaluate pupil symmetry (Figures 1.7 and 1.8) [20]. Anisocoria may be a normal finding in horses with bilateral heterochromia iridis or unilateral heterochromia iridis in which the larger pupil is ipsilateral to the heterochromic eye (Figure 1.9) [6].

When stimulated by light during the PLR, vertical movement of the pupil is much faster and excursion is greater than horizontal movement. The pupil's shape is a horizontal ellipse that becomes rounder when dilated (Figures 1.10 and 1.11) [21]. Pupillary light reflexes can be used to simultaneously evaluate function of the retina, CN II, midbrain, and CN III [18–20]. Light directed into one eye should result in constriction of both that pupil (direct response) and the pupil of the contralateral eye (indirect or consensual response). This results from bilateral excitation of the parasympathetic component of CN III in the pretectal region [18–20]. The normal equine pupil responds



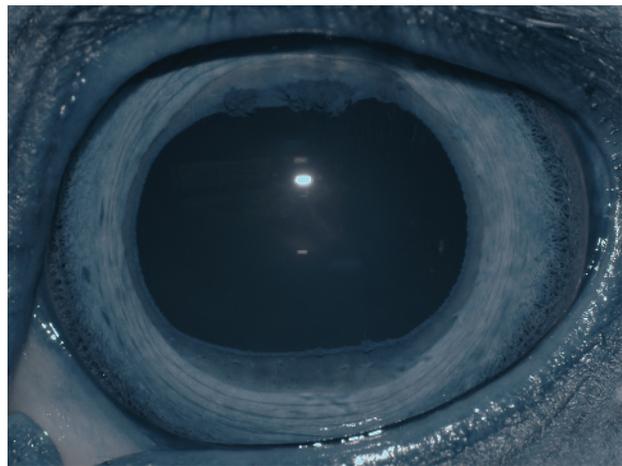
**Figure 1.7** With the examiner 1.5–2.0 m in front of the horse, an indirect ophthalmoscope directed at the center of the horse's head should illuminate both tapetal reflexes and make it possible to determine pupil symmetry.



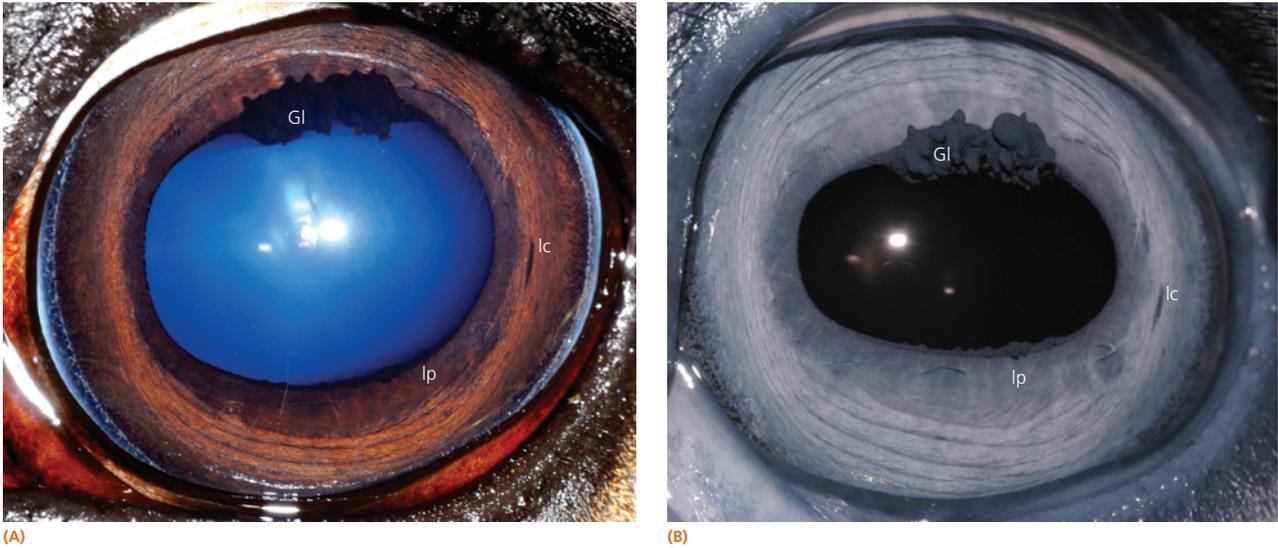
**Figure 1.8** In this case, a different pupil diameter is present (anisocoria), with slight miosis in the left eye.



**Figure 1.9** Anisocoria may be a normal finding in horses with bilateral heterochromia iridis or unilateral heterochromia iridis in which the larger pupil is ipsilateral to the heterochromic eye.



**Figure 1.10** Pupil of the adult horse appears rounder when dilated. At infrared photograph, details of lateral and medial parts of the iridocorneal angle are more visible.



**Figure 1.11** Normal anatomy of the pupil and corpora nigra in a horse. Granula iridica (GI) are present on the dorsal and ventral pupillary margins but are normally more prominent on the dorsal margin. The iris can be separated into a pupillary zone (Ip) and a more peripheral ciliary zone (Ic). (A) Color photograph. (B) Infrared photograph.

somewhat sluggishly and incompletely to light, in a biphasic manner [8, 19]. The first part is a brisk but small reaction, followed by the second slower complete movement. The magnitude and time of response depends on the brightness of the light source and the mental state of the horse. A very focal and bright light source is required to stimulate a rapid and complete response. Pupillary light responses are most vigorous if the beam is directed towards the visual streak in a direction that is temporal (lateral) and slightly dorsal to the optic disc. Consensual responses can be difficult to evaluate in the horse because they tend to be weaker than the direct response and can be awkward for an examiner to determine alone. The indirect PLR is less prominent because of decussation at the chiasm (85%) in the horse, which results in more efferent pupillomotor fibers that return to the ipsilateral side of the brain [6, 12, 20]. This is referred to as **dynamic contraction anisocoria** [17]. Evaluation for the consensual light reflex is unnecessary if the horse has vision and a direct response in both eyes. The consensual light reflex can be extremely valuable in evaluating problems when the posterior segment cannot be visualized (e.g., corneal edema, hyphema) for assessment of retinal function in the affected eye. Pupillary escape, a slight dilation that follows constriction under direct light stimulation, is a normal response in the horse [20].

### Dazzle reflex

The dazzle reflex, in contrast to the cortically mediated menace response, is a subcortical reflex that requires function of the retina, CN II, rostral colliculus, possibly the supraoptic nuclei of the hypothalamus, CN VII, and orbicularis oculi [18–20]. A very bright focal light source is directed into the eye, and blinking or blepharospasm is a normal response. Care should be

taken if the light source generates heat, because this can be detected by the horse if the light source is close to the cornea and can also result in blepharospasm.

### Palpebral reflex

Horses normally blink approximately 5–25 times per minute at rest [22]. The blink is synchronous between both eyes approximately 30–100% of the time [22, 23]. Two types of normal blinking occur in the horse at rest: complete and incomplete [23]. Incomplete blinking is most common and consists predominantly of upper eyelid motion downward [23]. Complete blinking is associated with an upward movement of the lower lid to meet the upper lid and is highly variable in occurrence [23]. The blink rate slows when the horse is sedated, anxious, or focused on an object of interest.

The palpebral reflex is elicited by touching the medial and lateral canthi and results in closure of the eyelids [18–20]. If CN V or CN VII is abnormal (e.g., facial nerve paralysis) or if the eyelids are unable to close (e.g., with severe trauma and swelling), the blink may be absent or incomplete.

### Corneal reflex

The corneal reflex is elicited by lightly touching the unanesthetized cornea with a sterile cotton-tipped swab and results in closure of the eyelid and retraction of the globe [18–20]. This subcortical reflex occurs in response to a tactile or painful stimulus to the cornea. The afferent pathway of the corneal reflex is via the ophthalmic branch of CN V [20]. The result should be closure of the eyelid and retraction of the globe, mediated by CN VII and CN VI, respectively [20]. If CN V or CN VII is abnormal or if the eyelids are unable to close, the blink may be absent or incomplete. Corneal sensitivity can be quantitated by

a technique called **corneal esthesiometry**, which is described later in this chapter. Lack of corneal sensation may be the cause of corneal ulceration and other corneal abnormalities (see Chapter 7).

### **Autonomic (sympathetic and parasympathetic) nervous system**

The autonomic nervous system is composed of two main divisions: the sympathetic and parasympathetic nervous systems (see Chapter 14). The sympathetic nervous system begins in the hypothalamus and extends down the intermediolateral cell columns of the spinal cord to synapse at C8 to T3. The axons from these cells exit the anterior nerve root of the spinal cord through the white ramus. The fibers (preganglionic fibers) course through the brachial plexus, through the thoracic inlet, and course cranially in the neck in the cervical sympathetic trunk. This trunk is associated with the vagus nerve forming the vagosympathetic trunk in the carotid sheath. Ventromedial to the tympanic bulla, the cervical sympathetic trunk separates from the vagus nerve and terminates in the cranial cervical ganglion. Postganglionic fibers proceed rostrally between the tympanic bulla and petrous temporal bone through the orbital fissure and form the sympathetic root of the ciliary ganglion [18–20]. Short ciliary nerves (along with contributory fibers to the long ciliary nerves) extend from the ciliary ganglion to the dilator muscle of the iris.

The sympathetic nervous system controls dilation of the pupils and other motor functions of the eyes and face. Damage to sympathetic innervation to the head may result in Horner's syndrome (miosis, ptosis, enophthalmos, increased sweating on the face and ear of the affected side, ipsilateral distention of facial blood vessels, and ipsilateral hyperemia of the conjunctiva and nasal mucosa) [24–30]. Chapter 15 offers a detailed description of the clinical signs and diagnosis of Horner's syndrome in horses. Causes reported in the horse include jugular vein and carotid artery injections, cervicothoracic spinal cord injury, cervical abscesses, guttural pouch disease or surgery, neoplasia or trauma of the neck and thorax, trauma to the vagosympathetic trunk, middle ear disease, traumatic lesions of the basisphenoid area, polyneuritis equi syndrome, equine protozoal myelitis of the cervical spinal cord, esophageal rupture, and trauma to the neck and thorax [8, 20, 24, 26, 27, 29–31].

The parasympathetic nervous system to the eye originates in the parasympathetic nucleus of the oculomotor nerve, located in the ventral part of the central gray matter next to the median plane and anteriorly to the general somatic efferent motor nucleus of the oculomotor nerve [32]. The fibers from this nucleus travel with the oculomotor nerve (CN III) and exit in the motor root of the ciliary ganglia where they synapse. The postsynaptic fibers travel to the eye in the short ciliary nerves to the constrictor muscles of the iris. Parasympathetic fibers also run with the facial nerve (CN VII) to the lacrimal gland to result in lacrimation when stimulated.

### **Restraint and sedation**

Some horses can undergo an ocular examination without sedation, but most horses require sedation for a complete, detailed ophthalmic examination. Use of restraint and sedation depends on the temperament of the horse, availability of equipment, and comfort level of the handlers and examiner. Tranquilization with detomidine HCl 0.02–0.04 mg/kg, administered intravenously (IV) is preferred for ophthalmic examinations because it provides rapid tranquilization without an excitation phase (either on induction or during recovery) and a steady and low head position without movement (i.e., fine tremors). Xylazine 0.5–1 mg/kg IV with or without butorphanol tartrate (0.01–0.02 mg/kg IV) can also be used [8, 9]. Butorphanol is commonly added for painful procedures or additional restraint [8, 9]. However, xylazine can have a profound excitation phase on induction or recovery. Both butorphanol and xylazine are associated with fine head tremors, or head “jerks,” which can be very disruptive during slit-lamp biomicroscopy and minor procedures around the eye such as cytology collection. Addition of acepromazine (0.02–0.04 mg/kg IV; 20 mg IV in a 500-kg horse) [33] 10–15 minutes prior to or together with detomidine is recommended to avoid the fine tremors when additional tranquilization is needed for extended examination or minor standing surgical procedures. For prolonged examination, need of additional tranquilization, or standing surgical procedures, addition of romifidine (50–75 µg/kg IV) may be helpful [34, 35]. Additional restraint includes lip (rope or chain) and neck twitch (manual). Although these techniques are generally very effective for short-term restraint (i.e., less than 10 minutes), some horses react adversely to these techniques, so caution is advised when using them.

### **Regional nerve blocks**

Two ophthalmic nerves are frequently denervated (“blocked”) during the equine ocular examination: the auriculopalpebral, or more precisely the palpebral branch of the facial nerve (CN VII), and the frontal (supraorbital) branch of the trigeminal nerve (CN V) [36, 37]. When these nerves are blocked, akinesia and anesthesia, respectively, of the upper eyelid occurs.

### **Regional akinesia**

The most common nerve blocked is the palpebral branch of the auriculopalpebral nerve, which innervates the orbicularis oculi muscle, responsible in part for eyelid closure. The orbicularis oculi muscle in horses is very strong, and therefore akinesia of this muscle is required to open the eyelid for examination in many horses, especially horses that are in pain. It is extremely important in conditions in which the structural integrity of the globe can be compromised, because the pressure applied by the muscle during manipulation for examination or during blepharospasm could result in rupture of the globe. Akinesia of the eyelids may be induced for routine eye examination, diagnostic procedures (e.g., corneal cytology and culture), therapy (e.g., subconjunctival injections, placement of a subpalpebral lavage), and standing surgeries [36, 37].

A volume of 1–2 mL of an anesthetic is injected subcutaneously with a 25-gauge (G), 5/8-inch needle adjacent to the nerve, and the injection site is massaged to facilitate anesthetic diffusion [8, 9, 12, 36, 37]. Anesthetics most frequently used for eyelid blocks include 2% lidocaine HCl, which has an onset of action of 4–6 minutes and a duration of 60–90 minutes, and 2% mepivacaine, with an onset of action of 3–5 minutes and a duration of 90–120 minutes [8, 9, 12]. Procaine or bupivacaine can also be used. Repeated injections of anesthetic may result in a refractory phenomenon, requiring higher volumes of drug and longer times to achieve akinesia [12]. The auriculopalpebral nerve block results in paralysis of the orbicularis oculi muscle of the upper eyelid and variable paralysis of the lower eyelid for approximately 1–2 hours [8, 9, 12]. Duration of anesthesia can be prolonged with the addition of 1:10,000 epinephrine [12], but this is not usually required for most examinations or minor surgical procedures. Ptosis, narrowing of the palpebral fissure, and easy manual elevation of the upper eyelid should result [8, 9, 12]. Sensation to the eyelids and some palpebral function remains intact, so the horse can usually blink and continue to protect the cornea. Once the examination is concluded, if the horse is not blinking well, topical ophthalmic ointments should be used every 30 minutes to protect the cornea until palpebral function returns.

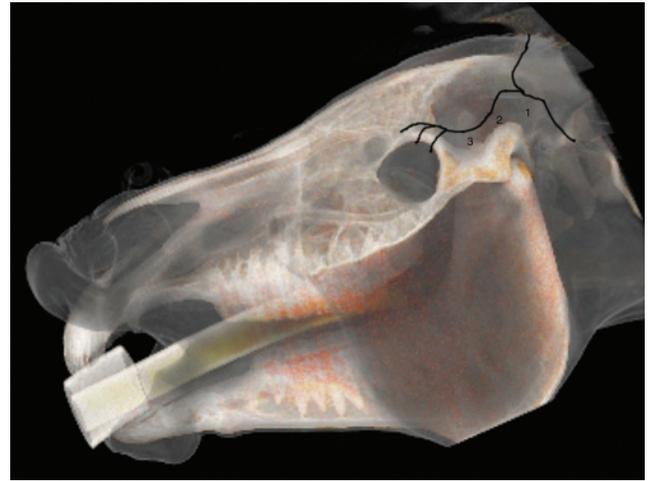
The auriculopalpebral nerve branches from the main trunk of the facial nerve, where it is protected by the parotid gland for the full length of the caudal border of the ramus of the mandible [12, 20]. It then emerges from beneath the gland just caudal to the caudal border of the condyle of the mandible, where it is covered by thin facial muscles and lies close to the rostral auricular artery and vein [12]. The branches then pass rostrally and dorsally to reach their destination (Figure 1.12).

The auriculopalpebral nerve can be blocked subfascially in the depression just anterior to the base of the ear where the caudal border of the coronoid process of the mandible meets the zygomatic process of the temporal bone. At this point, the nerve emerges from the parotid salivary gland and becomes subcutaneous on the lateral aspect of the dorsal tip of the coronoid process (Figures 1.12 and 1.13) [12, 36, 37].

The palpebral branch of the auriculopalpebral nerve can be blocked just lateral to the highest point of the caudal zygomatic arch, where the nerve can be palpated through the skin by running a finger forcefully over the dorsal border of the bone (see Figures 1.14 and 1.15) [36, 37]. The palpebral branch of the auriculopalpebral nerve can also be blocked where it lies on the zygomatic arch caudal to the bony process of the frontal bone (see Figures 1.14 and 1.15) [36, 37].

### Regional anesthesia and analgesia

Sensation to the eyelids is provided by the ophthalmic and maxillary divisions of the trigeminal nerve (CN V) (Figure 1.14) [12]. The frontal, lacrimal, and infratrochlear nerves arise from the ophthalmic branch of CN V, and the zygomatic nerve arises from the maxillary branch of CN V [6, 12]. The frontal

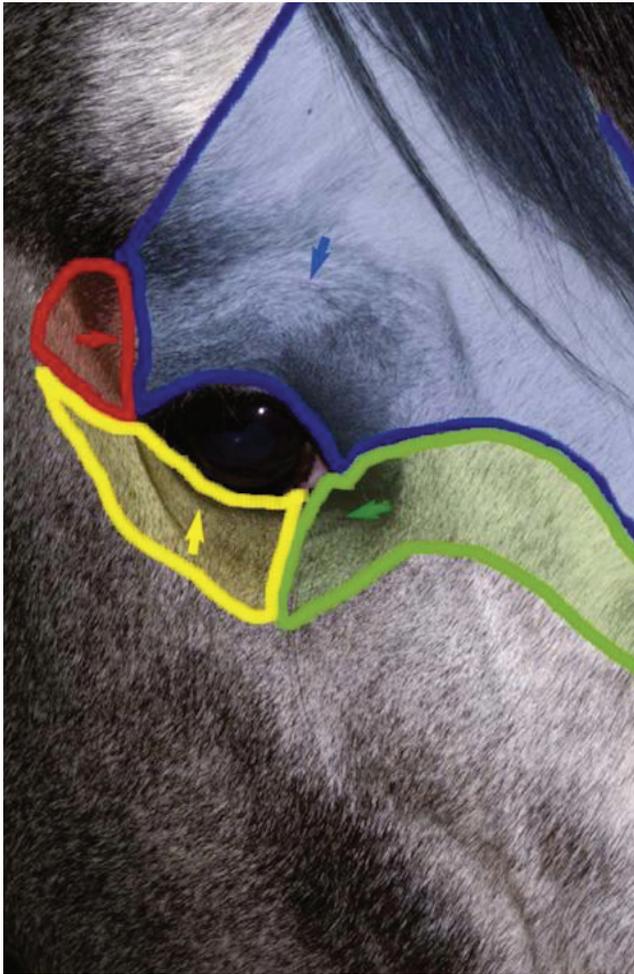


**Figure 1.12** Three sites at which the auriculopalpebral nerve can be blocked: caudal to the posterior ramus of the mandible (1), dorsal to the highest point of the zygomatic arch (2), and where it lies on the zygomatic arch caudal to the bony process of the frontal bone (3).



**Figure 1.13** Location of the palpebral nerve block. A 25-G needle is inserted, and 1–2 mL of lidocaine or mepivacaine is injected subcutaneously.

(supraorbital) nerve innervates most of the central upper eyelid and is the only sensory block normally required for examination [12]. The lacrimal nerve provides sensory innervation for the lateral upper eyelid [12]. The infratrochlear nerve provides sensory innervation for the medial canthus [12]. The zygomatic nerve innervates the majority of the lateral lower eyelid [12]. The nasociliary nerve, a branch of the maxillary branch of CN V, provides sensory innervation to the cornea [12]. Anesthesia of these nerves is sometimes necessary for eyelid and conjunctival biopsies or simple surgeries, as well as subpalpebral lavage placement in the horse. The anesthetics most frequently used



**Figure 1.14** Sensation to the eyelids is provided by the ophthalmic and maxillary divisions of the trigeminal nerve (CN V). The frontal, lacrimal, and infratrochlear nerves arise from the ophthalmic branch of CN V, whereas the zygomatic nerve arises from the maxillary branch of CN V. The approximate areas of sensation that would be blocked with each nerve are indicated as follows: frontal (blue), lacrimal (red), zygomatic (yellow), infratrochlear (green).

are the same as those used for akinesia and include lidocaine HCl and mepivacaine.

The four main sensory nerve branches can be blocked directly as follows: the frontal (supraorbital) nerve is blocked as it emerges from the supraorbital foramen within the frontal bone (Figures 1.15 and 1.16) [12, 17]. This foramen can be easily palpated by placing the thumb below the dorsal orbital rim and the middle finger in the supraorbital fossa. The index finger is placed straight down, midway between the thumb and middle finger, to locate the supraorbital foramen (see Figure 1.16). A depression is usually palpable. A 25-G,  $\frac{3}{8}$ -inch needle is then inserted subcutaneously over the foramen, and 1–2 mL of anesthetic is injected (Figure 1.17). Passing the needle into the foramen is not recommended because this may damage the supraorbital artery and vein, which exit the skull through the



**Figure 1.15** The frontal (supraorbital) nerve is blocked as it emerges from the supraorbital foramen within the frontal bone, as indicated by the white dot.

supraorbital foramen. Furthermore, if the needle inadvertently enters the periosteum surrounding the supraorbital foramen, this can be painful and the horse may react negatively. The frontal nerve is mainly sensory, but this block can result in partial upper eyelid akinesia as well, likely by further denervating the branches of the palpebral nerve [9].

The lacrimal nerve can be blocked by injecting 1 mL of lidocaine adjacent to the lacrimal notch, a depression that can be palpated on the dorsolateral bony orbital rim, or by using a line block along the lateral third of the dorsal orbital rim (Figures 1.14 and 1.18). The zygomatic nerve can be blocked with a line block along the ventrolateral orbital rim (Figures 1.14 and 1.19).

The infratrochlear nerve can be blocked as it runs through the trochlear notch located medially on the dorsal orbital rim (Figures 1.14 and 1.20). The notch can be palpated along the orbital rim.

### Globe and orbit examination

Anatomy of the globe and orbit is reviewed in detail in Chapter 5. Initial examination of the globe and orbit should be made with the examiner positioned in front of the horse (see Figure 1.4), where symmetry between the eyes is carefully assessed. Palpebral fissure size and symmetry, relative globe position, and direction of the eyelashes are evaluated. The upper eyelashes of the healthy horse are nearly perpendicular to the cornea (see Figure 1.5A) [8]. A change in the angle between the eyelashes and the cornea may indicate blepharospasm, enophthalmos, exophthalmos, or ptosis (see Figure 1.5B) [8].



**Figure 1.16** The supraorbital foramen can be palpated with the thumb below the dorsal orbital rim and the middle finger in the supraorbital fossa. The examiner then places the index finger straight down midway between the thumb and middle finger to locate the supraorbital foramen.

Apparent changes in globe size (e.g., buphthalmos (Figure 1.21)) should be differentiated from changes in globe position (e.g., exophthalmos (Figure 1.22)). Cornea globosa (Figure 1.23) has been reported in the Rocky Mountain horse and may be difficult to distinguish from buphthalmos [38, 39]. The orbit should be examined by observation, palpation of the bony orbital rim, and retropulsion of the globe through a closed eyelid [4, 8, 9]. Forceful manipulation of the eyelid and retropulsion should not be performed if the structural integrity of the cornea or globe may be compromised.

### Eyelids and conjunctiva examination

Anatomy of the eyelids and conjunctiva is reviewed in detail in Chapter 6. Examination of the eyelid should include assessment of function and detailed examination using diffuse illumination with magnification (e.g., using a slit-lamp biomicroscope). The periocular tissues including the eyelids, conjunctiva, sclera, and nictitans should be inspected with transillumination [17], the technique of direct focal illumination for inspection of the



**Figure 1.17** A 25-G, 5/8-inch needle is inserted into or just over the foramen.



**Figure 1.18** The lacrimal nerve can be blocked by using a line block along the lateral third of the dorsal orbital rim.

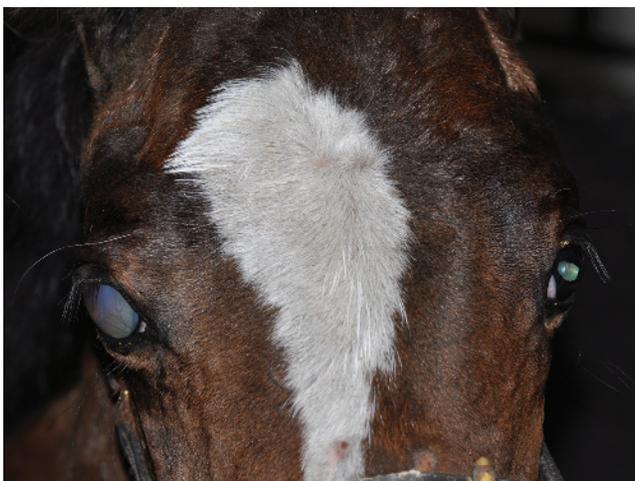
anterior structures of the eye. It can be performed with a Finnoff transilluminator (Welch-Allyn, Skaneateles Falls, NY), direct ophthalmoscope, slit-lamp biomicroscope, or even a penlight. Eyelids should be examined for position, movement,



**Figure 1.19** The zygomatic nerve can be blocked with a line block along the ventrolateral orbital rim.



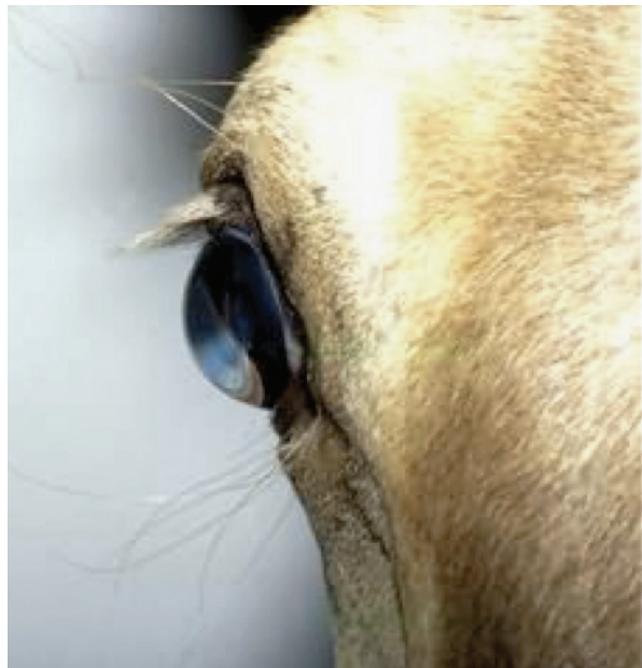
**Figure 1.20** The infratrochlear nerve can be blocked as it runs through the trochlear notch, located medially on the dorsal orbital rim. The notch can be palpated.



**Figure 1.21** Right eye congenital glaucoma and hydrophthalmus in a foal.



**Figure 1.22** Exophthalmos of the right eye of a horse with a retrobulbar mass.



**Figure 1.23** Corneal globosa of a Rocky Mountain horse with multiple congenital ocular anomalies. (Source: Photograph courtesy Dr David Ramsey.)

and conformation prior to the use of eyelid blocks [4, 8, 9] Attempts to forcefully elevate the upper eyelid should be avoided if a palpebral nerve block has not yet been performed, and each eye should be examined with minimal handling of the adnexal tissues. Culture and cytology should be collected prior to instillation of any medications (see section **Culture and cytology of corneal lesions** and Chapter 3). Biopsy of the eyelid or conjunctiva should be considered if indicated (see Chapter 6).

#### **Lacrimal and nasolacrimal system examination**

See anatomy of the lacrimal and nasolacrimal system in Chapter 6. Assessment of the lacrimal system is done by microscopic examination of the tear film via slit-lamp biomicroscopy



**Figure 1.24** (A) Normally a single distal punctum of the nasolacrimal system is present and can be located in the skin of the floor of the nostril near the mucocutaneous junction (arrow). (B) Methyl methacrylate cast of the left nasolacrimal duct in the horse with the medial bony orbit and medial wall of the lacrimal canal removed. (A) Lacrimal sac. (B) Duct within the lacrimal canal. (C) Narrowing of nasolacrimal duct at the exit from the lacrimal canal. (D) Exit of the duct from the lacrimal canal. (E) Duct pressed laterally by cartilaginous plate in alar fold. (F) Duct within basal fold. (Source: (B) Latimer et al., 1984. Reproduced with permission from American Veterinary Medical Association.)

(or other means of magnification) and adjunctive diagnostic tests, such as the Schirmer tear test (STT), the phenol red thread (PRT) tear test and Tear film Break-Up Time (TBUT) test. The examiner should also inspect the openings of the proximal (eyelid) and distal (nasal) nasolacrimal puncta (Figure 1.24).

The STT is used commonly to measure aqueous tear production [40–43]. In the test, a filter paper strip is placed in the lower conjunctival sac, and wetting is then measured in millimeters per 60 seconds (Figure 1.25). Commercial filter paper strips available include standardized Sno-Strips (Akorn) and Color Bar (Eagle Vision/Schering-Plough). Strips can also be made from Whatman filter paper (#40, 5 × 40 mm with a notch 5 mm from the end) [12]. The STT I, in which no topical anesthesia is used, measures the approximate amount of basal and reflex tearing. The STT II, performed after the application of topical anesthesia, theoretically only measures basal secretion of aqueous tears. Some residual tear volume may make both of these measurements slightly inaccurate. The STT should be performed before manipulation of the eye and orbit during examination to minimize reflex tearing. There are no reports of the effect of an auriculopalpebral nerve block on the STT in horses.

The PRT test consists of a thread of 75 mm in length with a fold 3 mm from the end, impregnated with phenol red, which is a pH indicator. The thread is placed in the lower conjunctival fornix for 15 seconds. The pale yellow thread turns orange due to the alkaline tears composition. Even if the main advantage of this test is that it can be used in small eyes (e.g., nondomestic species), it has been used in horses too, with a mean tear production of  $30.22 \pm 0.99$  and  $31.00 \pm 1.4$  in healthy Arabian and Thoroughbred horses [44]. The TBUT test is a noninvasive ocular diagnostic test that allows an assessment of the quality of the precorneal tear film (mucin and/or lipid layers) by observing



**Figure 1.25** The STT is used to measure aqueous tear production. In the test, a filter-paper strip is placed in the conjunctival sac, and wetting is then measured in millimeters per 60 seconds.

its stability over the corneal surface. It corresponds to the time required for the ocular surface to lose cohesive surface wetting after each blink. To perform the TBUT test, fluorescein solution is applied to the corneal surface, the horse is allowed to blink, and then the eyelids are held open until a dry spot forms, which

appears dark compared to the fluorescein green color in the tear film. The observation of these dark areas is facilitated by using a slit-lamp biomicroscope with the cobalt blue filter. Mean basal TBUT values reported in horses are  $8.3 \pm 1.3$  seconds [45]. Topical use of tetracaine HCl eyedrops in a normal horse results in a quicker TBUT [45].

Deficiencies in aqueous tear production have rarely been reported in the horse [23, 42, 43, 46–51]. This may be because the STT is not a part of the routine ophthalmic examination in the horse. Specific indications to perform STT include evidence of CN VII dysfunction (e.g., after trauma, facial paralysis), desiccated cornea or conjunctiva, presence of tenacious mucoid discharge, and presence of unexplained corneal vascularization or ulceration. Keratoconjunctivitis sicca (KCS) is most commonly the result of CN V or VII trauma but has also been reported in cases of fractures of the mandible and stylohyoid bone, post anesthesia, locoweed poisoning, eosinophilic dacryoadenitis, hypothyroidism, and in association with corneal stromal sequestration [23, 41, 43, 46–51].

The effects of age, season, gender, environment, sex, time of day, and placement of strips on STT results in healthy horses and ponies have also been reported [40, 42]. In general, the STT value in the horse is much greater than that in cats and dogs [41, 52, 53]. STT values are highly variable between eyes and between the same eye during different times of the day, and this appears to be unrelated to signalment, housing, or season [40]. One study found a diurnal variation in horses housed in a 12-hour light and 12-hour dark setting [42]. These horses had STT values that gradually increased during the light phase, peaked at 4–6 hours, then decreased during the dark phase [42]. Healthy horses have been reported to have an STT I range of 11–>30 mm wetting/min and 15–20 mm/30 s. Both sick and healthy neonatal foals have been reported to have lower STT values than adults [54, 55]. STT I values for sick neonatal foals ( $14.2 \pm 1$  mm wetting/min) and healthy neonatal foals ( $12.8 \pm 2.4$  mm/min) were not significantly different but were lower than STT I values from healthy adult horses ( $18.3 \pm 2.1$  mm/min) [54, 55].

Comparisons of STT I and STT II values revealed minimal differences in one study (i.e., STT I and STT II values of  $12.7 \pm 9.1$  mm wetting/min and  $9.9 \text{ mm} \pm 4.25$  mm wetting/min) [53], while the second study did not reveal a difference between STT I and STT II values [40]. This is in contrast to the dog, in which the STT I value is significantly higher than the STT II value [56]. Sedation with xylazine does not affect the STT value; however, general inhalant anesthesia with halothane does lower the STT value for up to 3 hours [41].

Borderline STT measurements (e.g., measurements of 10–15 mm wetting/min) should always be repeated. Comparison of tear test results between the two eyes should be cautiously interpreted in clinical assessment of decreased tearing [40]. In general, repeatable measurements of less than 10 mm wetting/min should be considered abnormal in conjunction with clinical signs [4]. See Chapters 6 and 7 for more information on KCS.



**Figure 1.26** Passage of fluorescein dye to the distal punctum in the nares (Jones test I) is timed and should occur within 5 minutes but may take up to 20 minutes in the horse.

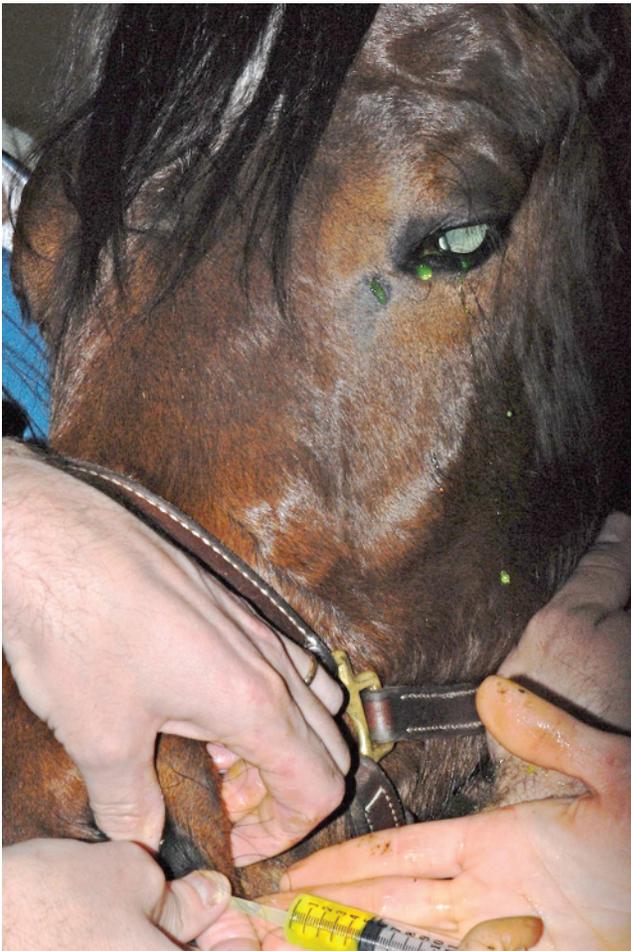
### Nasolacrimal duct patency

The physiologic patency of the nasolacrimal system can be evaluated with topical sodium fluorescein, which is not rinsed from the eye [12]. Passage of the fluorescein to the distal puncta in the nares (Jones test I) is timed and should occur within 5 minutes but may take up to 20 minutes (Figure 1.26) [17]. The required time for passage is influenced by the amount of fluorescein placed, tear production, and length of the individual horse's nasolacrimal system [17]. A positive test result is definitive for a patent nasolacrimal duct but does not prove that both proximal puncta are patent [17]. A negative test result is only suggestive of a problem and may even be normal in the horse because of the large volume capacity of the nasolacrimal duct [17, 57, 58]. However, the nasolacrimal duct should be irrigated if the dye fails to appear and clinical signs suggest a problem such as epiphora (watery ocular discharge) without an obvious cause, mucopurulent ocular or nasal punctal discharge, or dacryohemorrhhea [17, 57, 58].

Irrigation of the nasolacrimal duct can be performed retrograde (i.e., from the distal nares opening) or normograde (i.e., from the proximal eyelid puncta) [12]. Sedation is usually required to perform either procedure in the horse. Retrograde irrigation through the nasal meatus to the nasolacrimal duct is easiest to perform (Figure 1.27) because of the larger size of the opening [17, 58]. The distal nasolacrimal puncta can usually be cannulated by a 5 or 6 polyethylene urinary catheter [17]. Suitable catheters are 4- to 6-French canine urinary catheters, 5-French feeding tubes, or polyethylene tubing [17]. The largest catheter that will pass through the bony canal in an adult horse is a 6-French urinary catheter [17]. The tip of the catheter, after it has been coated with lidocaine gel, is inserted into the distal punctal opening for a distance of at least 5 cm. Digital pressure should be applied to the opening to close it and prevent normograde loss of fluid. A 12- to 20-mL syringe previously filled with



(A)



(B)

**Figure 1.27** Retrograde irrigation through the distal opening to the nasolacrimal duct is easiest to perform. (A) The tip of the catheter, after it has been coated with lidocaine gel, is inserted into the distal punctal opening. Digital pressure should be applied to the opening to prevent normograde loss of fluid. (B) The 12-mL syringe, previously filled with eyewash, is attached; gentle irrigation of the nasolacrimal duct is performed until fluid exits the proximal punctum near the medial canthus of the eye.

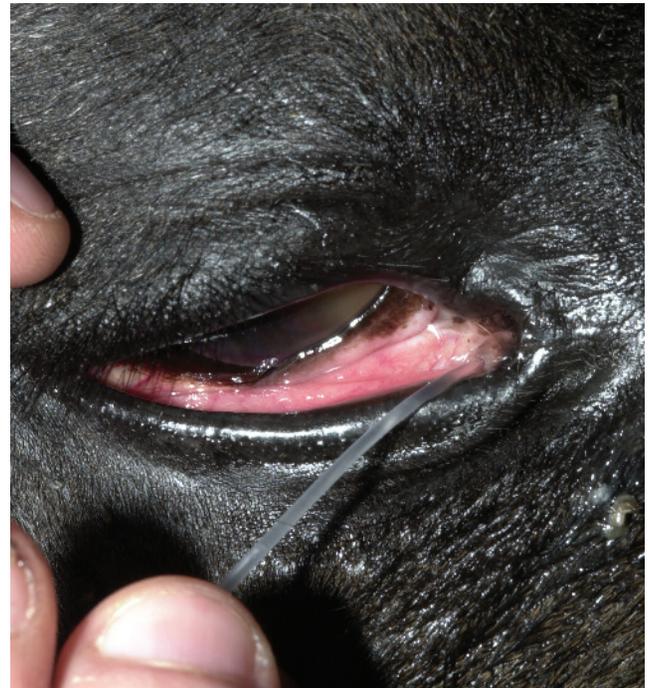
**Box 1.5** Supplies needed for irrigation of the nasolacrimal duct.

#### Retrograde irrigation

- Lidocaine gel for retrograde
- Open-ended tomcat catheter, 4- to 6-French polyethylene urinary catheter, 5-French feeding tubes, or polyethylene tubing
- A 12- to 20-mL syringe
- Sterile eyewash or balanced salt solution

#### Normograde irrigation

- Topical anesthetic (proparacaine HCl)
- Open-ended tomcat catheter, lacrimal cannula, 18- to 20-G IV cannula
- A 12- to 20-mL syringe
- Sterile eyewash or balanced salt solution



**Figure 1.28** Normograde irrigation from the proximal puncta should be attempted if the result of the Jones test I is negative and the duct cannot be irrigated from the distal nasal puncta. Catheterization of the proximal punctum using an open-ended tomcat catheter in the lower eyelid is demonstrated.

eyewash is attached, and gentle irrigation of the nasolacrimal duct is performed until fluid exits the proximal puncta near the medial canthus of the eye. Sneezing by the horse is common during this procedure and may be violent. A list of supplies needed to perform nasolacrimal duct irrigation can be found in Box 1.5.

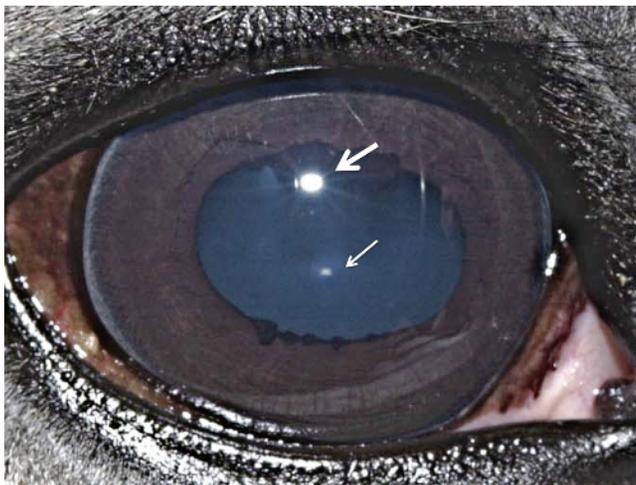
If retrograde irrigation is unsuccessful, then normograde irrigation from the proximal puncta should be attempted with a lacrimal cannula, open-ended tomcat catheter, 18- to 20-G IV cannula, or teat tube syringe (Figure 1.28) [17]. The punctum in

the lower eyelid is usually slightly larger and easier to cannulate than the punctum in the upper eyelid [17]. Gentle pulse pressure may be required to unblock an obstructed duct. Excessive force in the placement of the catheter or during irrigation should be avoided because significant damage to the nasolacrimal duct could result [17]. Ducts that are compromised by a foreign body or other anatomic obstruction (e.g., after trauma, mass effect) may not be effectively irrigated [59]. Skull radiographs or computed tomography (CT) scans and a contrast dye study (e.g., dacryocystorhinography) should be performed next if the duct cannot be irrigated. Endoscopic examination of the nasolacrimal duct also has been described [60]. See descriptions of these diagnostic techniques in the Chapter 2.

### Cornea and sclera examination

The anatomy and diseases of the equine cornea are described in Chapter 7. To examine the cornea of the horse, diffuse and focal direct illumination (or transillumination) with magnification is used first, followed in most cases by biomicroscopy using a slit-lamp. The corneal examination should be performed with the observer located rostral to the eye. Light directed diagonally across the cornea will reveal opacities of the cornea against the dark background of the pupil [17].

The Purkinje–Sanson reflexes are three reflections from the eye produced by the light source during transillumination (Figure 1.29) [17]. Disease may alter the sharpness and location of these reflexes. The first, largest, and most anterior originates from the cornea. The second originates from the anterior lens capsule, and the third and most posterior originates from the posterior lens capsule. If a slit-lamp biomicroscope is used, two corneal reflexes are seen, one from the anterior surface and the other from the endothelium [17]. The corneal and anterior lens



**Figure 1.29** The Purkinje–Sanson reflexes are three reflections produced by the light source during transillumination. The largest is produced by the cornea (**larger arrow**). The second is produced by the anterior lens capsule (**smaller arrow**). The third is not easily visible and is produced by the posterior lens capsule.

capsule reflexes are virtual and noninverted and will move in the same direction as a change in the light position. The image on the posterior surface is real and inverted and will move in the opposite direction to the light [17]. The images are valuable in determining corneal clarity, depth of the anterior chamber, thickness and position of the lens (after mydriasis), and in locating lesions within the lens [17].

### Biomicroscopy for corneal examination

The technique of biomicroscopy, in which a slit-lamp binocular microscope with an external pivoting light source is used, is the same for horses as for humans and small animals and has been well described elsewhere [12, 61–64]. Slit-lamp biomicroscopy improves visualization and localization of lesions of the cornea, anterior chamber, lens, and anterior vitreous by means of transillumination and retroillumination [12]. It can also be used to assess corneal thickness (i.e., pachymetry), anterior chamber depth, and aqueous flare [12].

The availability of portable handheld models of the slit-lamp biomicroscope has made biomicroscopy for equine ophthalmology easy and efficient. Portable models are available from Clement-Clark, Kowa, Nippon, Dioptrix, and Zeiss. The Kowa SL-14, SL-15, and SL-17 ( $\times 10$  or  $\times 16$  magnification) is light and powered by battery, and therefore of excellent use in examination of a horse. However, lack of magnification above  $\times 16$  and inherent movement of the examiner and horse limit the ability to see fine structure and lesions. An alternative to a biomicroscope is using magnification (i.e.,  $\times 2.5$ – $3.0$  magnifying head loupes) and the slit beam on the direct ophthalmoscope. Very small “slit-lamps” are also made by Heine (HSL 150 (Heine USA, Dover, NH); Eidolon Hand Held Slit Lamp Model 510L (Eidolon Optical LLC, Natick, MA)) that resemble a penlight with a magnifier on the end (Figure 1.30). Although these instruments are inexpensive and portable, their lack of magnification and illumination limits their usefulness [12].



**Figure 1.30** Use of a small, portable slit-lamp (Heine Handheld Slit Lamp (Heine USA, Dover, NH)). (Source: Photograph courtesy Dr David Wilkie.)

The light beam of the biomicroscope should be angled at 20–45 degrees from the axis of the microscope and thus the visual plane of the observer (Figure 1.31). The light beam width, length, orientation, and color can then be modified by a series of diaphragms and filters [12]. The focal distance of the instrument is 7–10 cm, and fine focus is achieved by moving either toward or away from the eye within this range [12].

Particular attention needs to be used during biomicroscopy examination in horses, as during this examination the examiner's eyes are in contact with the slit-lamp without direct visualization of the entire animal. In this situation, sudden head movement could be very dangerous for the examiner and for the instrument as well. To avoid possible trauma from the horse, it is important to stay in contact with the horse's head with your hand (Figure 1.31). It is also very important to remove glasses, if used, during this examination.

The initial examination of the horse should proceed with diffuse illumination: a wide, low-intensity slit beam should be used, and the microscope should be defocused from the light [12]. The surfaces of the eyelids, cornea, conjunctiva, and iris should be inspected. With the use of low magnification, a broad slit beam is focused on the cornea, creating a parallelepiped (i.e., a three-dimensional section) of illuminated tissue [12, 61]. This allows visualization of transparent structures such as the cornea and lens in three dimensions. In the cornea, the anterior surface, stroma, and posterior surface of the cornea can be visualized [61]. Nontransparent structures such as the sclera only yield a magnified two-dimensional surface or external view. The slit beam is then narrowed and intensified to reveal a two-dimensional cross-section of the cornea and lens, allowing the examiner to accurately determine lesion depth and axial positioning [61]. This is extremely important in evaluating the depth of corneal lesions (e.g., stromal ulcerative keratitis, stromal abscesses) in the horse.



**Figure 1.31** The light beam of the biomicroscope should be angled at 20–45 degrees from the axis of the microscope (Kowa SL-17) and thus the visual plane of the observer. The examiner should always be in contact with the horse's head, avoiding possible trauma from head movements.

Direct and indirect retroillumination are performed by reflecting the slit beam from deeper structures while focusing on more superficial structures [61]. Other techniques that can be performed with slit-lamp biomicroscopy, such as specular reflection, are difficult to impossible in a horse because of continuous slight ocular movements.

### Culture and cytology of corneal lesions

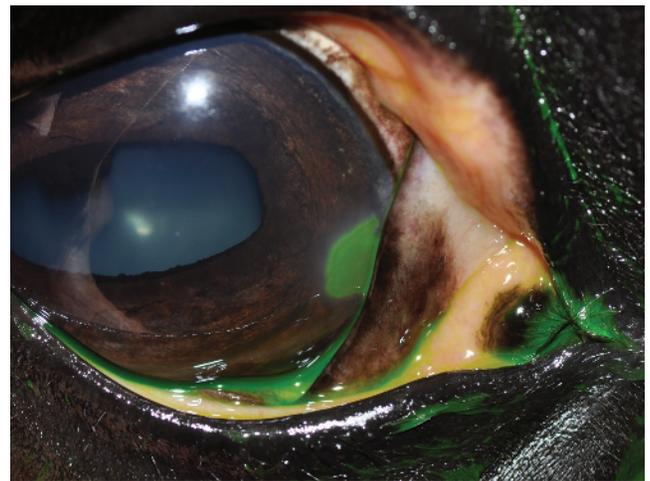
See Chapter 3 and online material for detailed information on collecting and interpreting ocular cultures and cytology.

### Ophthalmic dyes

Topical ophthalmic dyes are routinely used in veterinary medicine to aid in the diagnosis of corneal, conjunctival, and nasolacrimal diseases. Commonly used topical ophthalmic dyes include sodium fluorescein, rose bengal, lissamine green, alcian blue, trypan blue, and methylene blue [12, 65–67]. Sodium fluorescein and rose bengal are the two most commonly used dyes in clinical veterinary ophthalmology [12, 66, 68, 69]. Indications for the use of topical ophthalmic dyes in the horse include determining the health and integrity of the precorneal tear film, corneal epithelium, and conjunctival epithelium, and the physiologic flow of the nasolacrimal system. Intravenous sodium fluorescein is used to perform fluorescein angiography and is discussed in Chapter 2.

### Sodium fluorescein

The most common use for topical sodium fluorescein is detecting ulcerative keratitis, but it will also stain conjunctival ulcerations and abrasions. In a corneal ulceration, the hydrophilic fluorescein binds to the corneal stroma but not to the lipophilic epithelium or to Descemet's membrane (Figure 1.32). Small quantities can also pool or diffuse through intact epithelial cell intercellular spaces to reveal weakly staining epithelial



**Figure 1.32** In a corneal ulceration, the hydrophilic fluorescein binds to the corneal stroma but not to the epithelium or to Descemet's membrane, resulting in a green stain.

microcysts and partial-thickness microerosions [12, 67]. From the stroma, the dye can then readily pass through Descemet's membrane and the corneal endothelium to enter the aqueous humor and can be quantified (e.g., by fluorophotometry).

Fluorescein staining of the cornea is indicated for almost every condition in the horse involving its eye and orbit—a red or painful eye, discharge from the eye, an obvious corneal irregularity, history of ocular trauma—and for assessment of the tear film stability (TBUT) and physiologic nasolacrimal function. Fluorescein is available as a sterile 0.5–2% alkaline solution or as a sterile impregnated paper strip [70]. The impregnated paper strips should be used for topical application, because the solutions have been associated with bacterial contamination [71]. Other equipment needed to perform the stain includes sterile eyewash solution and a 3-mL syringe with a 25-G needle. The easiest method of applying topical fluorescein to horses is to place a sterile fluorescein strip in a 3-mL syringe, fill the syringe with sterile eyewash, replace the plunger, and then squirt the solution through the **hub** of a 25-G needle from which the needle has been manually broken off (Figure 1.33) [8, 17]. After application, the eyelids should be closed or the animal allowed to blink to distribute the stain evenly across the ocular surfaces. Excess fluorescein can be removed with gentle irrigation with eyewash if required. Alternatively, undiluted fluorescein applied topically may allow for better visualization of corneal epithelial erosions or early corneal trauma. The use of an ultraviolet or blue light, usually available on a direct ophthalmoscope or the slit-lamp biomicroscope, may improve visualization of the stain. False-positive results may occur after the use of proparacaine topical anesthesia or if direct contact between the paper strip and cornea occurs, which may leave a mark that resembles a corneal defect [12, 17].

Fluorescein may also be used to detect leakage of aqueous humor through the cornea (i.e., the Seidel test) [12, 17]. The Seidel test can be used to detect full-thickness corneal injuries or determine whether a corneal suture is leaking. The application of sodium fluorescein without subsequent irrigation results in a high dye concentration in which the dye fluoresces at wavelengths closer to the yellow and orange spectra. With or without gentle pressure on the cornea, aqueous leakage locally dilutes the fluorescein, and the dye fluoresces green [12].

### Rose bengal

Rose bengal (i.e., tetrachloro-tetra-iodo-fluorescein) has been used to aid in the diagnosis of precorneal tear film disorders, mucin precorneal film deficiencies, and superficial corneal epithelial abnormalities in horses (Figure 1.34). It can be used primarily or after sodium fluorescein application. Rose bengal stains dead and degenerating cells and mucus [67]. However, rose bengal has a dose-dependent ability to stain normal cells, and this ability is normally blocked by tear film components [12, 67, 72]. Therefore stain uptake may indicate tear film abnormalities, such as a mucin deficiency, more accurately than cell viability [12, 67, 72]. Rose bengal, like sodium



**Figure 1.33** The easiest method of applying topical fluorescein to horses is to place a sterile fluorescein strip in a 3-mL syringe, fill the syringe with sterile eyewash, replace the plunger, then squirt the solution through the hub of a 25-G needle from which the needle has been manually broken off.



**Figure 1.34** Rose bengal positive stain of epithelial multipunctate lesions suspected of superficial primary fungal infection.

fluorescein, is available as both an impregnated paper strip and a solution [12, 66, 69]. Use of the 0.5% or lower concentrations can minimize the irritation that can be associated with the 1% solution [12]. The dye has also been shown to be toxic to corneal epithelium at routine concentrations [73]. Slit-lamp biomicroscopy may be necessary for adequate visualization of rose bengal stain.

The use of rose bengal would be indicated in the horse in any of the conditions in which corneal or conjunctival ulceration is suspected, but especially when a viral or fungal cause is suspected. One report suggested that rose bengal staining is present as a result of ocular surface damage in the presence of keratomycosis (Figure 1.34) [74]. Specimens obtained from horses with painful eyes should be stained with both fluorescein and rose bengal; superficial keratitis may be negative for fluorescein but positive for rose bengal [23, 74].

### Other ophthalmic dyes

Lissamine green is an organic acid dye widely used in human ophthalmology [67]. It is a true vital dye as it does not stain healthy epithelial cells even in the absence of the tear film, differently from rose bengal. Despite this difference, lissamine green has a similar staining pattern to rose bengal and it has been used in the case of KCS, with the advantage that it is less toxic and irritant than rose bengal [69, 75, 76]. Different dyes can be applied alone or at the same time, as mixing of the dyes does not affect staining properties [69, 77].

Trypan blue, an azo dye derived from tuluidine, is widely used in human and veterinary cataract surgery, as it stains the anterior lens capsule and thus helps its visualization during capsulorrhexis. Also it has been used in two recent studies on equine cornea: an *in vitro* model for corneal scarring treatment with mitomycin C and a novel model method of gene delivery [78, 79].

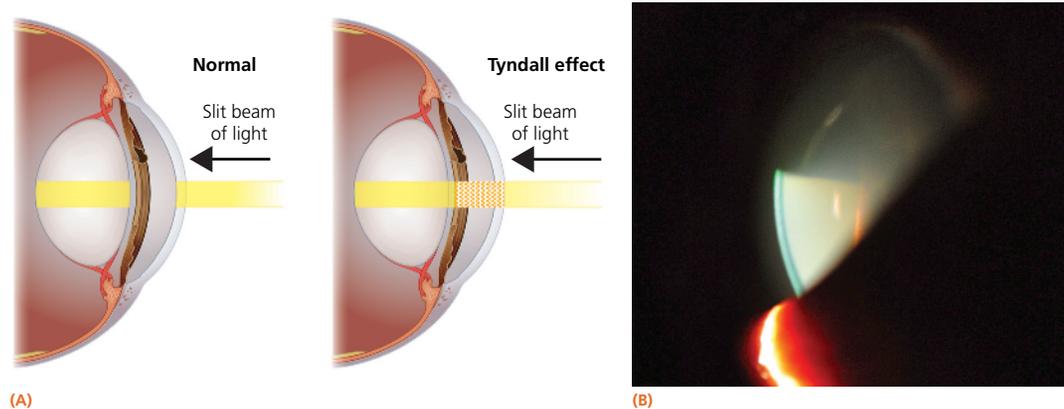
### Anterior chamber and iris examination

The intraocular portion of the ocular examination is conducted next. See Chapter 8 regarding the anatomy and diseases of the anterior chamber and uvea of the horse's eye. With a thin beam (slit) or small circular beam of light from the light source directed at a 45-degree angle to the eye, the anterior chamber depth and clarity are inspected. In a healthy horse, this will not result in any internal reflection of light from the aqueous, and the anterior chamber should appear clear (Figure 1.35). If solids (e.g., protein, cells) are present in the aqueous, there will be a reflection of light from these particles (i.e., the Tyndall effect) [12]. This turbidity results in visualization of the beam of light traversing the anterior chamber and is referred to as **aqueous flare**. Aqueous flare (and other ocular lesions such as corneal edema, corneal pigment, and conjunctival redness) can be graded on a subjective scale from 1+ to 4+, with 4+ being the greatest degree of severity.

### Tonometry

Measurement of intraocular pressure (IOP) in the horse has been revolutionized by the development of handheld portable digital tonometers. Direct tonometry via a manometer is the most accurate but invasive method for recording the IOP and is not practical for clinical use. Indirect tonometry, the measurement of corneal tension, is the technique used to determine IOP in clinical veterinary ophthalmology [17]. Digital, indentation, and applanation tonometry have all been described in the horse [12]. Digital tonometry, the estimation of IOP by digital palpation, can be considered just approximate but not accurate. Indentation tonometry, commonly performed with a Schiottz tonometer, requires general anesthesia (a lateral position) in the horse and is impractical. However, normal values have been reported for the Schiottz tonometer in the horse and are 14–22 mm Hg [17]. Applanation tonometry measures the amount of flattening (area of contact) of the cornea when a weight touches it [12, 17]. The force it takes to flatten this portion of the cornea is an estimate of the IOP (pressure = force/area). Rebound tonometry (TonoVet tonometer (ICare Finland Oy, Helsinki)) is another method to measure IOP in horses. This uses a disposable probe that is electromagnetically propelled to contact the cornea, then rebound to the instrument, which then uses these rebound characteristics to estimate IOP [80]. Accurate measurement of IOP in the standing horse requires use of applanation or rebound tonometry (Figure 1.36) [8, 12]. In one study involving normal horses, the TonoVet rebound tonometer correlated well to manometry IOP measurements, and IOPs obtained with the TonoVet rebound tonometer (Figure 1.37) were higher than with the Tono-Pen applanation tonometer (Figure 1.38) [80].

Tonometry is part of routine ocular examination and it is particularly indicated in horses with focal or diffuse corneal edema, a red or painful eye, orbital trauma, ocular blunt trauma, exophthalmos or hydrophthalmos, a history of glaucoma in the



**Figure 1.35** (A) With a thin beam (slit) or small circular beam of light from the light source directed at a 45-degree angle to the eye, the anterior chamber depth and clarity are inspected. In a healthy horse, this will not result in any internal reflection of light from the aqueous, and the anterior chamber should appear clear. If solids (e.g., protein, cells) are present in the aqueous, there will be reflection of light from these particles (i.e., the Tyndall effect). (Source: Illustrations by Samantha J Elmhurst BA Hons/LivingArt.) (B) This turbidity results in visualization of the beam of light traversing the anterior chamber and is referred to as aqueous flare.



**Figure 1.36** Materials needed to perform tonometry in the horse. Accurate measurement of IOP in the standing horse requires tonometry, either using a Tono-Pen digital tonometer (TP) or a TonoVet rebound tonometer (TV), or similar instrument. The Tono-Pen tonometer requires use of a topical anesthetic, such as 0.5% proparacaine HCl (P), while the TonoVet does not.



**Figure 1.37** Tonometry in the horse using a TonoVet tonometer.

opposite eye, or a lens subluxation/luxation; it is also indicated for follow-up examinations of animals with medically controlled glaucoma [8, 12]. Three separate readings with less than 5% standard error are averaged to obtain the IOP in millimeters of mercury [12, 17]. If significant corneal disease is present, the



**Figure 1.38** Tonometry in the horse using a Tono-Pen tonometer.

most normal part of the cornea should be used to take readings [17]. A fibrotic or edematous cornea may result in a falsely elevated IOP [17]. See Chapter 10 for more information on the diagnosis and treatment of glaucoma.

Horses that require sedation for ocular examination may show dramatic decreases in IOP, as illustrated by one study in which xylazine decreased IOP by 23% [81]. In another report, the IOP range was 20.5–39.8 mm Hg and showed that both acepromazine and xylazine decreased IOP [82]. A combination of xylazine and ketamine had no effect [83, 84]. The effect of IV administration of romifidine on IOP in normal horses has been recently described by two publications. In the first report, a consistent decrease of IOP after romifidine IV administration (75 µg/kg) was noted, with the largest change from baseline values (16.7%) at 15 minutes after injection [85]. The second report describes a highly significant IOP decrease 45 minutes after IV injection of romifidine (40 µg/kg) in right eyes (58.5% decrease) and left eyes (55.2% decrease) from the baseline values. In this second report, IOP values returned to normal 120 minutes after the IV injection [86]. It has been suggested that horses without an auriculopalpebral block will have an elevated IOP due to eyelid tension [17, 84] but this has not been supported in two other studies in normal horses [81, 87]. However, horses with ocular disease such as glaucoma may be blepharospastic; therefore auriculopalpebral blocks are recommended prior to tonometry. IOP in normal horses without an auriculopalpebral nerve block has been reported as ranging from 24.5 ± 4.0 mm Hg to 28.6 ± 4.8 mm Hg, while baseline IOP values in horses with auriculopalpebral nerve block has been reported as ranging from 24.25 ± 3.5 mm Hg to 26.35 ± 5.57 mm Hg [80, 85, 86, 88]. In one study of normal horses in which horses were tranquilized with detomidine and given auriculopalpebral nerve blocks, 87% of horses with their head positioned lower than their hearts had elevated IOP [89]. In another study, circadian rhythm was shown to have an influence on horses' IOP [90]. In this study, IOP was found to be low during the dark phase and high during the light phase, with a peak at the end of the light phase [90]. Evaluating the results of these studies together, it is

recommended that the examiner be consistent when measuring IOPs in horses in terms of time of day, nerve blocks, head position, and tranquilization, especially when performing repeated IOPs on the same horse. Tranquilization (always the same drug), auriculopalpebral nerve blocks, and an elevated normal head position are recommended for tonometry in horses.

Countertop electronic applanation tonometers may also still be available (e.g., MacKay-Marg (Biotronics, Redding, CA); the Alcon pneumotonograph) and can be used to measure IOP in horses. The MacKay-Marg tonometer was shown to produce reliable results in comparison with direct tonometry in the dog, rabbit, and horse [88, 91, 92]. It has been used to study IOP in the horse [81, 83, 87, 88]. IOP measured in the normal horse using the MacKay-Marg tonometer in one report was  $20.6 \pm 4.7$  mm Hg [81].

The Alcon pneumotonograph is an applanation tonometer-tonographer that measures IOP via a gas-suspended plunger. Measurements can be permanently recorded on heat-sensitive paper. However, pneumotonography may provide falsely elevated pressure readings in the horse [93].

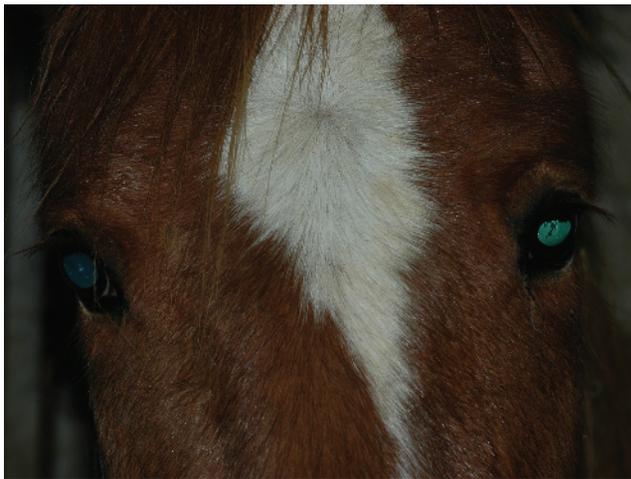
### Tonography

Tonography is the use of continuous tonometry to noninvasively estimate the pressure-sensitive facility of conventional aqueous humor outflow [12, 94]. In theory, the weight of the tonographic probe on the cornea increases both IOP and the rate of aqueous humor outflow without changing the rate of aqueous humor production [12]. The subsequent decline in IOP (decay curve) is measured over 2–4 minutes, thereby allowing an estimation of conventional outflow (corneoscleral trabecular outflow) [12]. The unconventional outflow (uveoscleral) is pressure independent and thus not estimated by tonography. Uveoscleral outflow in the horse is substantial and may be the major outflow pathway, but it cannot be measured by

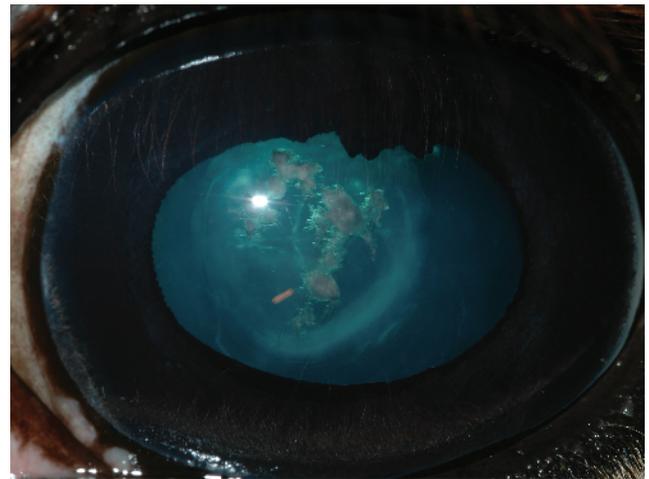
tonography [83]. Tonography has been used in the normal horse to estimate the facility of aqueous humor outflow (C-value). A measured equine C-value of  $0.88 \pm 0.65$  mL/min per mm Hg is substantially higher than that reported for the healthy dog and cat (0.24–0.27 mL/min per mm Hg) [94–96]

### Lens examination

For details on the anatomy and diseases of the lens see Chapter 9. Complete evaluation of the lens requires a dilated pupil; therefore use of 1% tropicamide HCl is recommended 15–20 minutes prior to initiating the lens examination. The lens is initially evaluated using direct focal illumination (i.e., transillumination) and retroillumination. Transillumination of the lens is performed by directing a beam of light at a 45-degree angle into the lens and directly observing the lens using this light [17]. Retroillumination is examining the lens using light reflected by the tapetum and posterior segment structures of the eye. This is done by using a direct ophthalmoscope or other focal light source (e.g., a Finnoff transilluminator) and starting about an arm's length from the horse, directing the light to obtain a bright tapetal reflex, then moving toward the animal to bring the lens structures into focus. This technique improves detection of opacities in the cornea, anterior chamber, lens, and vitreous because these opacities reflect, refract, or obstruct returning light (Figure 1.39A) [17]. Normal Y sutures can be visualized with transillumination but not with retroillumination [17]. With transillumination, nuclear sclerosis (i.e., the normal aging change of the lens due to increased lens density) will appear as a greater central translucence, with the cortex remaining clear [17]. On retroillumination, the lens will appear clear with the pupil filled with the tapetal reflex, and a junction “ring” will be seen at the nuclear–cortical junction. With transillumination, the attachment of the lens zonules may be seen in a well-dilated eye immediately behind the edge of the



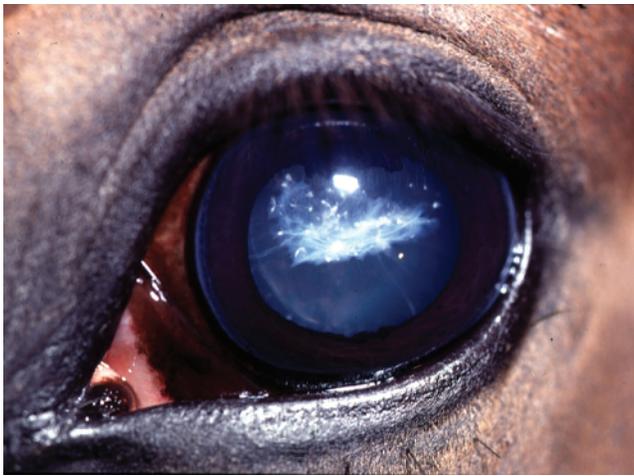
(A)



(B)

**Figure 1.39** (A) Retroillumination from distance with direct ophthalmoscopy in this foal shows dark multipunctate central spots in the left eye. (B) Transillumination of the same lesions shows multipunctate nuclear cataracts that appear white.

pupil [17]. Cataracts will appear white on transillumination (Figures 1.39B and 1.40) and black if they are not complete (i.e., light can pass around them) when observed on retroillumination (Figure 1.41) [17]. Direct retroillumination is performed by placing the objectives in the path of the refracted light, which causes opaque lesions to appear dark against a light background and transparent lesions to appear clear within a dark halo [17]. Indirect retroillumination allows improved detection of transparent lesions by taking advantage of differences between their refractive indices and those of surrounding tissues [17]. Lesions are observed against a darker background because the reflected light is directed away from the objectives [17]. Any noted opacities should be further investigated with the slit-lamp biomicroscope.



**Figure 1.40** Direct transillumination of a cataract. The lesion appears white.



**Figure 1.41** Retroillumination of an incomplete cataract. The lesion appears black.

### Biomicroscopy for lens examination

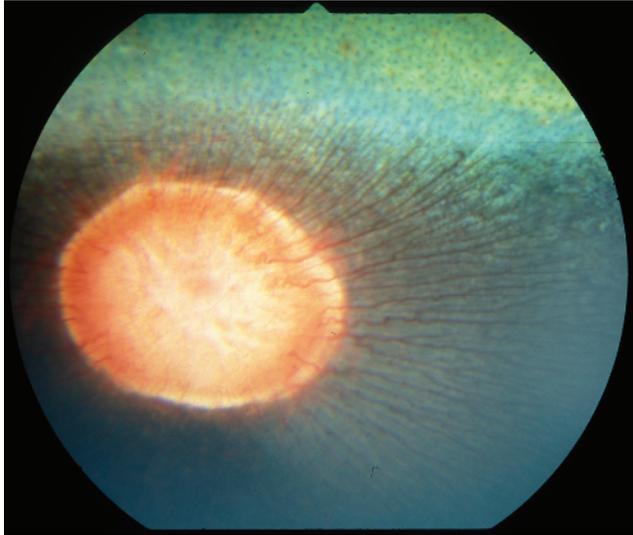
The light beam of the biomicroscope should be angled at 20–45 degrees from the axis of the microscope and thus the visual plane of the observer (see Figure 1.31). The initial examination of the lens should proceed with diffuse illumination; a wide, low-intensity slit-beam should be used, and the microscope should be defocused from the light [12]. The anterior surfaces of the lens and vitreous should be inspected. The slit-beam is then narrowed and intensified to reveal a two-dimensional cross-section of the lens, allowing the examiner to accurately determine lesion depth and axial positioning [61]. This is extremely important in evaluating the depth and location of lens opacities. Direct and indirect retroillumination are performed by reflecting the slit beam from deeper structures while focusing on more superficial structures [61].

### Posterior segment ocular examination: vitreous and retina

Normal anatomy and diseases of the posterior segment of the equine eye are discussed in Chapter 11. Examination of the equine ocular posterior segment involves direct focal illumination (i.e., transillumination) using a focal light source, as previously described in the lens section. Examination of the vitreous is performed first. Transillumination of the vitreous can reveal small posterior polar remnants of the hyaloid artery and areas of light reflection between vitreous planes [17]. Neither can be seen with retroillumination. Transillumination can also be used to look directly at the ocular fundus of the horse [17]. This is done by holding a light source against the examiner's face and directing the light into the horse's pupil (Figure 1.42). The examiner moves forward toward the eye until the ocular fundus becomes visible. A large fundus area, approximately six times greater than that seen with an ophthalmoscope, can be visualized [17]. This is used as an initial screen for



**Figure 1.42** Transillumination can also be used to look directly at the ocular fundus of the horse. This is done by holding a light source against the examiner's face and directing the light into the horse's pupil.



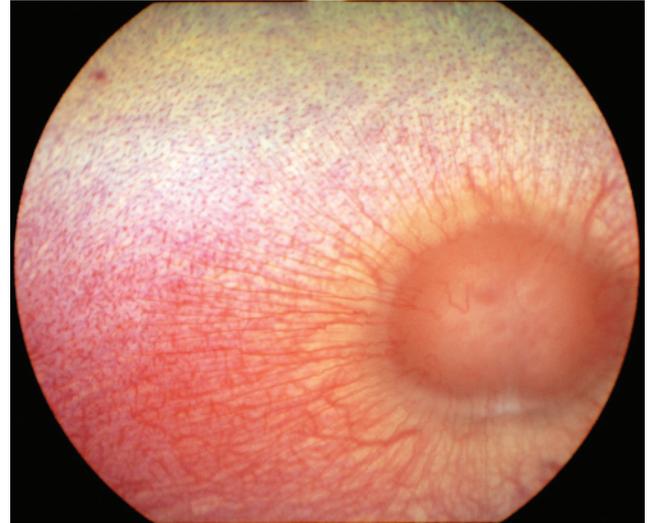
**Figure 1.43** In most horses, a triangular fibrous tapetum in the dorsal choroid can be seen on ophthalmoscopic examination. End-on choroidal capillaries can be visualized as small dark dots throughout the tapetal fundus (i.e., stars of Winslow). The nontapetal area is usually dark brown.

disease. Details of any observed abnormality can then be evaluated with direct or indirect ophthalmoscopy.

**Ophthalmoscopy** is the examination of the ocular fundus (i.e., choroid, retina, and optic nerve) and is an integral part of any ophthalmic or physical examination. There are two common methods of performing ophthalmoscopy in the horse: direct and indirect. Direct ophthalmoscopy can be performed by means of transillumination, as previously described, or with the use of a direct ophthalmoscope. Indirect ophthalmoscopy requires the use of a light source and a handheld lens.

Advantages of the direct ophthalmoscope are its upright image, availability of options such as slit and graticule, ability to alter the dioptric power of the ophthalmoscope, and the high magnification provided [12, 17]. Disadvantages include the short working distance to the horse's head, a small field of view, lack of stereopsis, difficulty in examining the peripheral fundus, and greater distortion of the image when the visual axis is not clear [12, 17]. Advantages of indirect ophthalmoscopy include a wider field of view, a safer working distance from the horse's head, potential for stereopsis (i.e., depth perception) if binocular equipment is used, a greater view of the peripheral fundus, and the ability to alter the magnification by changing the diopter strength of the lens being used [12, 17]. Disadvantages of indirect ophthalmoscopy include the inverted and reversed image, the expense of binocular equipment, and the initial difficulty of mastering the technique [12, 17].

The ocular fundus can be visualized without mydriasis in the horse, but a complete examination requires mydriasis. The use of a short-acting mydriatic (1% tropicamide) is recommended, as previously described, resulting in complete mydriasis within 10–20 minutes with a duration of 4–6 hours [13]. The menace response, dazzle reflex, resting pupil size, and direct and consensual



**Figure 1.44** The tapetum may be undeveloped in animals with albinotic or subalbinotic coat colors. Melanin in the retinal pigment epithelium may be absent, depending on coat and iris coloration.

PLRs in each eye in bright and dim lighting should be evaluated before mydriasis is induced. Tonometry before pharmacologic mydriasis, indicated in small animals, is not absolutely necessary in the horse unless glaucoma is high on the list of differential diagnoses.

The normal ophthalmoscopic appearance of the ocular fundus of the horse (Figure 1.43) is described in Chapter 11 [7, 97–102]. The equine ocular fundus is dominated by the tapetum fibrosum, which occupies the dorsal two-thirds of the posterior segment, and the stars of Winslow, which are end-on views of choroidal blood vessels traversing the tapetum. The tapetal color varies from green-yellow (most common) to aquamarine or turquoise [8, 99]. Changes in tapetal color intensity from the central to peripheral tapetum have also been described [103]. The tapetum may be undeveloped in animals with albinotic or subalbinotic coat colors (Figure 1.44) [8, 104]. The nontapetal area is usually dark brown, but this melanin in the retinal pigmented epithelium (RPE) may be absent, depending on coat and iris coloration. If the pigment is absent, the choroidal vessels can be visualized. The optic disc is horizontally oval, usually located slightly temporal and ventral in the nontapetal area, and salmon pink [8, 104]. Retinal vessels radiate out only a short distance from the optic disc and are usually absent at the ventral disc border [8, 104].

### Direct ophthalmoscopy

Direct ophthalmoscopy is extremely useful for rapid ocular examination in horses, and it can be used to identify most lesions of the equine ocular fundus (Figure 1.45) [8]. In the horse, direct ophthalmoscopy can be performed with a transilluminator. The light source should be held against the examiner's face, near the eye, and directed through the horse's pupil. This provides a larger view of the fundus than can be seen with the

direct ophthalmoscope, but it is not entirely in focus. This is a quick and useful method to screen for obvious signs of disease.

The direct ophthalmoscope consists of a halogen coaxial optical system and a power source [12]. A series of concave and convex lenses can be rotated through the viewing aperture by means of a dial (Figure 1.46) [7, 97, 105]. Green or black numbers represent convex or converging lenses, and red numbers represent concave or diverging lenses [7, 12, 97, 105]. The size, shape, and color of the light beam can be adjusted by a second dial which produces large and small circles of light, a slit beam of light, a graticule, and two filters [12, 17]. The size of the circular spot of white light should be adjusted to the patient's pupil size to minimize light reflections from the corneal surface [12]. The slit beam also aids in the detection of elevations or

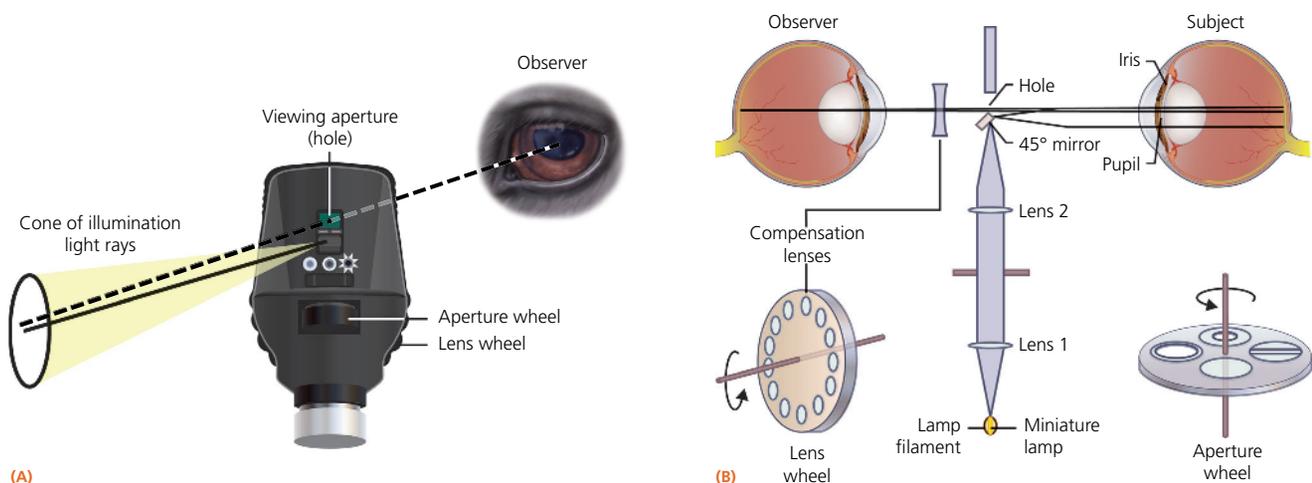


**Figure 1.45** Direct ophthalmoscopy is extremely useful for rapid ocular examination in horses and can be used to identify most lesions of the equine ocular fundus. There is a short working distance to the horse's head.

depressions in the ocular fundus, and distances can be estimated by changing the dioptric power of the ophthalmoscope [12]. When the retina is in focus at 0 diopter (D), the lesion is elevated if the lesion surface is in focus at a positive diopter setting (i.e., black numbers) and depressed if the lesion surface is in focus at a negative diopter setting (i.e., red numbers) [12]. The graticule is a grid that can be used to size the optic disc and estimate the size of fundic lesions [12]. A red-free filter (appears green) is used to evaluate retinal vessels and differentiate hemorrhage (which appears black) from pigmented lesions (which appear brown) [12].

Most ocular fundi are in focus at 0 to  $-2$  D if the examiner's vision is emmetropic, and therefore the ophthalmoscope should initially be set at 0 D [12, 17]. The direct ophthalmoscope should be placed against the examiner's brow, and using the dominant eye, the examiner should identify the horse's fundic reflex from a distance of approximately 0.5–0.75 m [12, 17]. Once the fundic reflex is identified, the examiner moves toward the horse to a point approximately 2–3 cm from the eye to visualize the fundus [12, 17]. Ophthalmoscopy should then proceed to identification and examination of the optic nerve, retinal vasculature, nontapetal fundus, and tapetal fundus in quadrants. The resulting image with a direct ophthalmoscope is upright and magnified several times above normal with a millimeter equivalent per dioptic change of 1.33 in the axial plane [12, 106]. Magnification of the image varies with working distance; lesions should be compared with optic disc diameter rather than by units of measurement [12, 17].

Welch-Allyn has developed a new monocular indirect ophthalmoscope (PanOptic (Welch-Allyn, Skaneateles Falls, NY)) that can be used in an undilated pupil and has five times greater magnification than a routine ophthalmoscope (Figure 1.47). The PanOptic ophthalmoscope also has a wider visual field than the direct ophthalmoscope, with an upright nonreversed image.



**Figure 1.46** (A, B) Direct ophthalmoscope schematics. (Source: Illustrations by Samantha J Elmhurst BA Hons/LivingArt.)



**Figure 1.47** PanOptic ophthalmoscope, which has a wider visual field than the direct ophthalmoscope and an upright, nonreversed image.

### Indirect ophthalmoscopy

Monocular or binocular indirect ophthalmoscopy involves the use of a handheld converging lens held near the patient and a light source near the examiner's eye. A larger area of ocular fundus can be visualized with indirect versus direct ophthalmoscopy and may allow the examiner to more easily detect disease.

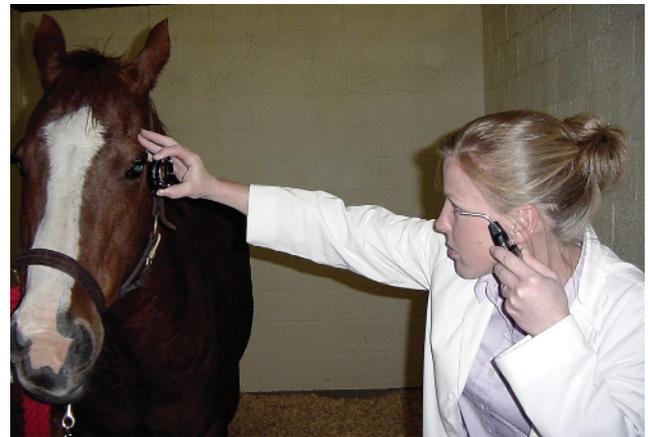
In binocular indirect ophthalmoscopy, a mirror and a light source fitted onto a headband are used to direct light into the patient's eye. The handheld lens is used to magnify the reflected image, and two prisms are used to split the reflected beam so it can be directed into both the examiner's eyes, permitting stereopsis (Figure 1.48) [12]. The light intensity should be adjusted to permit adequate illumination without causing patient discomfort [12].

In monocular indirect ophthalmoscopy, a handheld light source is used in addition to the handheld lens (Figure 1.49). The light source should be placed near the examiner's eye and temple so that both the head and light source function as one unit. A direct ophthalmoscope can be used for indirect ophthalmoscopy, and the dioptric power of the ophthalmoscope should be adjusted to +4 or +6 D [12].

The handheld lens provides a virtual image (i.e., the image is inverted and reversed) of the patient's fundus. A variety of lenses are available, ranging from +14 to +90 D in strength. The +20-D and +28-D lenses are the most useful in the horse, providing a fundus view of approximately 40 degrees. A +15-D is useful for a more detailed image of the fundus, while the 2.2 Pan-Retinal lens is a good compromise, giving a wide field of view (similar to +30-D lens) with magnification similar to a +20-D lens. The quality of the handheld converging lens affects the ease and clarity of the evaluation. The smaller the lens diopter rating, the greater the fundic magnification [12]. In the horse, lateral magnification has been reported for 14-D (1.18), 20-D (0.79), 30-D (0.51), and 40-D (0.38) lenses [106]. Axial magnification in the horse has also been reported for 14-D (1.86), 20-D (0.84), 30-D (0.35), and 40-D (0.19) lenses [106].



**Figure 1.48** In binocular indirect ophthalmoscopy, a mirror and a light source fitted onto a headband are used to direct light into the patient's eye. The handheld lens magnifies the reflected image, and two prisms split the reflected beam so it can be directed into both the examiner's eyes, permitting stereopsis.



**Figure 1.49** In monocular indirect ophthalmoscopy, a handheld light source is used in addition to the handheld lens. The light source should be placed near the examiner's eye near the temple so the head and light source function as one unit.

The refractive error of an animal can be semiquantified during indirect ophthalmoscopy by slowly withdrawing the lens toward the examiner and further from the eye and observing any change in magnification. The fundic image will get larger (myopic) or smaller (hyperopic), or it will remain static (emmetropia) [104]. For a more accurate measurement of refractive error, streak retinoscopy can be performed. This technique is described in the section **Retinoscopy (skiascopy)**.

Binocular indirect ophthalmoscopes suitable for use in horses, some with battery-powered illumination, are available from Heine, Keeler, Propper, Topcon, Welch-Allyn, Zeiss, and others.

## Advanced ophthalmic procedures

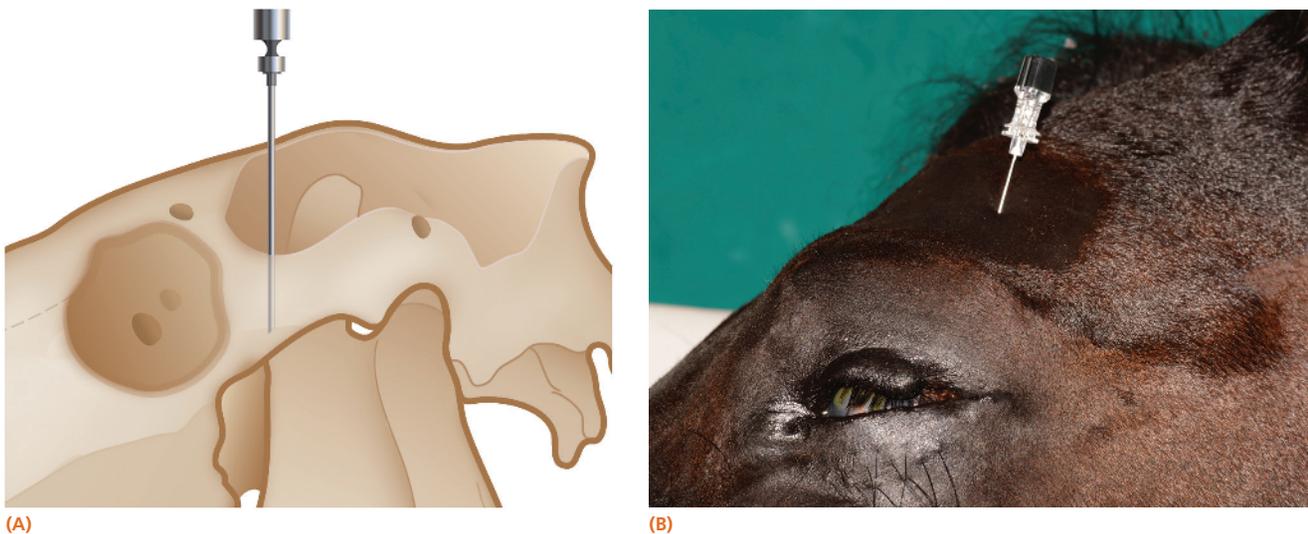
### Retrolbulbar nerve block

The retrolbulbar nerve block temporarily blocks the optic (CN II) and oculomotor (CN III) nerves, the abducens nerve (CN VI), trochlear nerve (CN IV), and the maxillary and ophthalmic branches of the trigeminal nerve (CN V). Retrolbulbar anesthesia can be used as an adjunct to general anesthesia in horses to reduce nystagmus and enophthalmos during corneal and intraocular surgery and prevent the need to give the horse neuromuscular blocking agents [107]. It can also be used to perform standing eyelid and corneal surgeries, as well as to perform anterior or posterior chamber paracentesis for diagnostic purposes. It can be used for the primary purpose of analgesia during the immediate post-operative period after an enucleation, and its use has been widely described for enucleation in standing sedated horses [108, 109]. A recent study reports that retrolbulbar nerve block can prevent heart rate decrease and bradyarrhythmias caused by vagal stimulation occurring during enucleation [110]. Retrolbulbar nerve block has been used also during cataract surgery, with the patient under general anesthesia, to provide extraocular muscle akinesia, thereby reducing the need for muscle relaxants and assisted ventilation [111]. Three methods have been described for retrolbulbar anesthesia in the horse: the four-point block, modified Peterson block, and direct injection into the orbital cone above or below the zygomatic arch.

The site for the retrolbulbar injection above the zygomatic arch and caudal to the temporal process of the malar bone is preferred because it requires a single needle penetration, is not located near the globe, and, if performed properly, avoids the direct location of the optic nerve. The orbital fossa above the dorsal orbital rim and zygomatic arch is clipped and aseptically

prepped with povidone-iodine (Betadine) solution. Care must be taken to avoid getting surgical scrub (Betadine or Nolvasan) or alcohol on the ocular surface, because severe irritation and corneal ulceration may develop. Therefore these substances are avoided around the eye. A 22-G, 2½-inch spinal needle (BD, Franklin Lakes, NJ) is placed through the skin perpendicular to the skull, in the orbital fossa just posterior to the posterior aspect of the dorsal orbital rim (Figure 1.50). The needle is advanced posterior to the globe until it reaches the retrolbulbar orbital cone. When the needle advances to this location, the eye will have a slight dorsal movement as the needle passes through the fascia of the dorsal retrolbulbar cone into the retrolbulbar space. The needle is advanced until it just passes into the cone, evidenced by the sudden release of the eye back to normal position or a slight “popping” sensation. Once the needle is positioned, 10–12 mL of 2% lidocaine HCl is injected into the retrolbulbar space. Mepivacaine 2% (Carbocaine) and bupivacaine are also used with a longer action and reduced tissue hypersensitivity reaction. Before injection, aspiration should be performed to make sure the needle is not positioned within a blood vessel. During the injection, the globe is pushed externally (i.e., slight exophthalmos), indicating an accurate placement of anesthetic. Onset of anesthesia usually occurs within 5–10 minutes. The duration of effect is approximately 1–2 hours. Ocular sensation, blink reflex, and vision will be compromised during this time, so stall rest and protection of the eye with lubricants or a temporary tarsorrhaphy are recommended for 2–4 hours after anesthesia.

Very few complications have been documented from this procedure. The most common complication is a hypersensitivity to lidocaine, which results in generalized formation of hives and severe retrolbulbar swelling after injection. These lesions will resolve with the use of systemic nonsteroidal



**Figure 1.50** Retrolbulbar block. (A) Placement of the needle for the retrolbulbar block. (Source: Illustrations by Samantha J Elmhurst BA Hons/LivingArt.) (B) The orbital fossa above the dorsal orbital rim and zygomatic arch is clipped and aseptically prepped. A 22-G, 2½-inch spinal needle is placed through the skin perpendicular to the skull, in the orbital fossa, just posterior to the posterior aspect of the dorsal orbital rim.

anti-inflammatory and antihistamine medications within 3–5 days after the injection. Another complication is development of exposure corneal ulcers, likely caused by exposure of the cornea due to poor eyelid function and corneal desensitization. Careful monitoring of eyelid function and frequent topical lubrication until full function returns will prevent this complication. Retrobulbar block denervates the optic nerve; therefore the horse will have decreased vision or be blind in that eye for 1–3 hours after injection. Thus it is recommended that the injection be performed unilaterally so that complete blindness does not occur after injections.

The four-point block, a local muscle block, has also been described for retrobulbar anesthesia in the horse [17]. Lidocaine or mepivacaine, 5–10 mL, can be deposited laterally by passing a 20-G, 3-inch needle through the lateral canthus skin and following the globe posteriorly. Ventrally, the needle passes through the skin or bulbar conjunctiva posteriorly. The needle should be directed slightly nasally to avoid the optic nerve. Nasally, the needle passes through the base of the elevated third eyelid and posteriorly into the orbit. Dorsally, the needle is passed through the center of the upper eyelid following the globe posteriorly. This technique should not be used when intraocular surgery is performed, because it may put pressure on the globe [17]. Failure to inject anesthetic into the muscle cone or injecting it in front of the orbital septum may cause the drug to migrate forward under the conjunctiva and cause severe chemosis [17]. There is no advantage of this technique over the single-injection retrobulbar block; therefore the four-point block is not recommended unless adequate anesthesia has not been achieved with the first block.

Use of a modified Peterson block has been described in the horse, although it would rarely have application in most types of ocular surgery and is not recommended [17].

Although few complications have been seen after retrobulbar and eyelid nerve blocks in horses, rare problems associated with the injections can occur during or after the surgical procedure [107]. Bacteria can be deposited in the orbit by the spinal needle if the skin surgical site was not aseptically prepared. This may result in orbital abscess or cellulitis formation. Laceration of the extraocular muscles, optic nerve, sclera, or ophthalmic arteries by the needle is also possible during the injection. Traumatic injury during needle introduction could result in retrobulbar hemorrhage or optic neuritis. An isolated case of oculocardiac reflex elicitation during the block has also been reported [112]. These complications can be mostly avoided by use of appropriate tranquilization, eyelid nerve blocks, antiseptic technique, and added restraint methods to restrict movement by a standing horse. The relatively low complication rate associated with the injection techniques far outweighs the risks associated with general anesthesia in horses.

### Orbital aspiration

Aspiration of a lesion (e.g., mass, fluid) in the equine orbit can be performed for cytology, culture, and histopathologic examination [12]. An 18-G, 4-inch, slightly curved needle is inserted

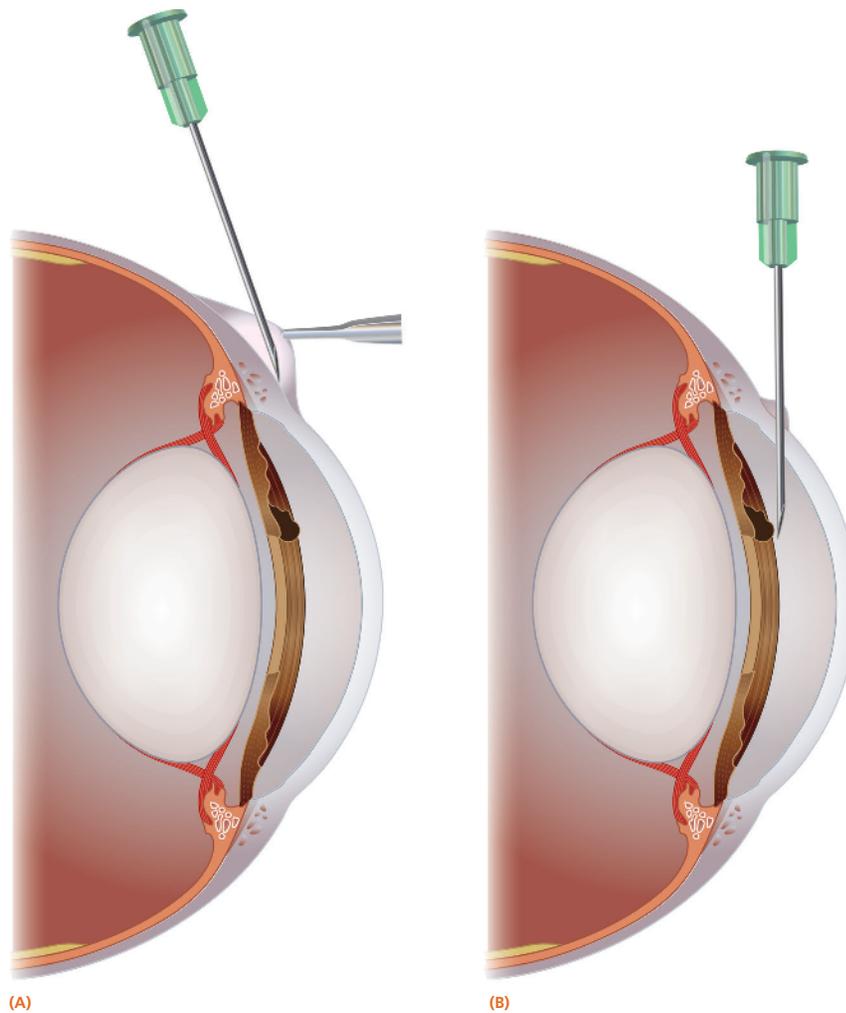
1 cm lateral to the lateral canthus and then directed posteriorly in a line parallel to the medial canthus [8, 113]. Approaching the retrobulbar space via the supraorbital fossa (as described in the section **Retrobulbar nerve block**) is also possible, especially if the lesion is in the dorsal retrobulbar area. For accurate and less risky sample collection, ultrasound- or CT-guided fine-needle aspiration, versus blind aspiration, may decrease the risk of injuring orbital structures [114].

### Aqueous paracentesis

Aqueous humor fills the anterior segment of the eye, supplies nutrients to the avascular cornea and lens, and removes waste products from the interior of the eye. Small amounts of aqueous humor (0.2–0.5 mL) can be aspirated from the anterior chamber in the horse [12, 115, 116]. Aqueous paracentesis can be performed with the horse under general anesthesia or standing with sedation, topical anesthesia, and a retrobulbar block [12]. The site for aspiration should be the dorsal or dorsotemporal limbus to take advantage of the scleral extension beyond the iris base (Figure 1.51) [17]. The bulbar conjunctiva should be cleaned with dilute Betadine (5%) solution and sterile saline solution or eyewash [12]. Use of a topical broad-spectrum antibiotic, such as fluoroquinolones, before and after paracentesis is recommended to further decrease possible bacterial contamination. The bulbar conjunctiva is grasped with thumb forceps near the site of entry (0.5–0.7 mL subconjunctival injection of 2% Mepivacaine helps in this procedure), and a 27- to 30-G needle is directed through the limbal cornea or subconjunctival limbus (bevel up) anterior and parallel to the iris (see Figure 1.51) [12]. The needle should be tunneled for several millimeters through the limbus into the anterior chamber, which may facilitate rapid formation of a seal after the needle is withdrawn [12]. A small volume (0.2–0.5 mL) of aqueous humor is slowly aspirated, and the needle is withdrawn [12]. A syringe can be attached to the needle when this is first performed or after the needle is placed into the anterior chamber; or no syringe may be used, and the aqueous that fills the hub can be drawn into a capillary tube. The tip of the needle should be visualized at all times to avoid lacerating the iris or lens capsule.

Injections of therapeutics into the anterior chamber (e.g., tissue plasminogen activator, antibiotics) can also be performed using the same technique, except that injection of solution is made instead of aspiration. If an aqueous humor diagnostic sample is also required, this sample should be collected first, followed by injection of the medication.

Possible complications of aqueous paracentesis include hyphema, anterior lens capsule rupture with subsequent phacoclastic uveitis, endophthalmitis, anterior uveitis, corneal edema associated with endothelial damage, and choroidal edema and hemorrhage [12]. In an attempt to minimize development of inflammation and other complications after the procedure, the aqueous humor should be very slowly removed, and the volume removed should be 0.5 mL or less. Diagnostic procedures that can be performed with aqueous humor samples in the horse



**Figure 1.51** Aqueous paracentesis. (A) The bulbar conjunctiva is grasped with thumb forceps near the site of entry. (B) A 27- to 30-G needle is directed through the limbal cornea or subconjunctival limbus (bevel up) anterior and parallel to the iris, avoiding the lens. A drilling and tunneling motion will facilitate entry through the sclera and may facilitate rapid formation of a seal after the needle is withdrawn. (Source: Illustrations by Samantha J Elmhurst BA Hons/LivingArt.)

include cytology, culture and sensitivity, protein measurement, antibody titers (e.g., *Leptospira* species), and polymerase chain reaction (PCR) [117–121]. See Chapter 8 for more information on the diagnostic value of aqueocentesis.

### Vitreous paracentesis

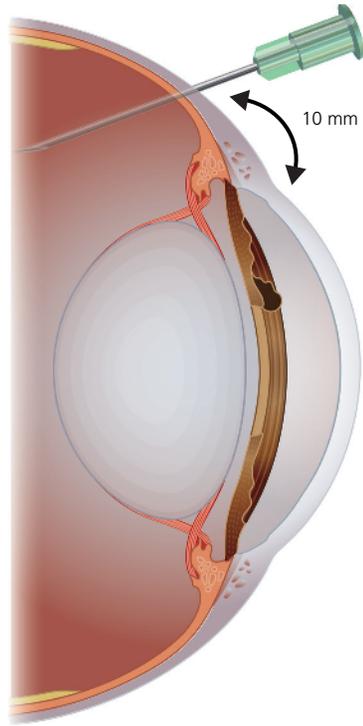
The location for vitreous paracentesis is approximately 10–12 mm from the dorsolateral limbus. This will place the needle through the pars plana and avoid introducing the needle through the sensory retina [122, 123]. A 23- to 25-G needle is inserted through the conjunctiva and sclera, with the needle directed posteriorly to avoid the lens (Figure 1.52). As with aqueous humor, diagnostic procedures performed on vitreous humor samples include culture and sensitivity, cytology, protein measurement, antibody titers, and PCR [12, 124, 125]. Vitreous paracentesis can also be performed to instill therapeutic medications, such as gentamicin for ciliary body ablation in cases of

chronic glaucoma and antibiotics in cases of endophthalmitis. Complications of vitreal paracentesis include hemorrhage, retinal detachment, and endophthalmitis—risks versus benefits of this procedure have to be considered.

## Advanced ophthalmic diagnostics

### Corneal esthesiometry

The corneal reflex is one of the most sensitive reflexes of the body, and its purpose is to protect the eye. Corneal sensitivity can be tested empirically by touching the cornea with a sterile Dacron-tipped applicator and observing a corneal reflex (response of blinking, globe retraction, or avoidance), or using an esthesiometer. The afferent arm of corneal reflex is mediated by the ophthalmic branch of the trigeminal nerve (CN V), and the efferent arm by the abducens (CN VI, globe retraction) and



**Figure 1.52** Vitreous paracentesis. The dorsal scleral overhang requires a greater distance from the limbus with a dorsal site than with a temporal site. However, 10 mm, rather than 15 mm, is probably more appropriate dorsally to avoid introducing the needle through the sensory retina. A 23-G needle is inserted through the conjunctiva and sclera, with the objective being to pass through the pars plana of the ciliary body. The needle should be directed toward the optic nerve to avoid the lens. (Source: Illustration by Samantha J Elmhurst BA Hons/LivingArt.)

facial nerve (CN VII, eyelid closure). Corneal esthesiometers quantitatively evaluate corneal sensitivity by measuring the corneal touch threshold (CTT), which is the threshold of the stimulus that results in a corneal reflex [126]. Estimation of corneal sensitivity may be used to diagnose and monitor corneal diseases, evaluate progression of corneal diseases after surgery, and monitor the effects of surgery and topical medications. The Cochet–Bonnet esthesiometer (Luneau Ophthalmologie, Chartres Cedex, France) has been described to evaluate CTT in horses [54, 126]. The instrument contains a nylon or platinum filament with an adjustable length (0.5–6 cm) and 0.12-mm diameter. The filament is applied in different lengths to the cornea until a corneal reflex is elicited with the same pressure or length of filament [54]. The length of the nylon filament estimates the applied pressure on the corneal surface and is readable on a millimeter scale. The shorter the filament, the more pressure is applied to the cornea, and vice versa. The pressure ranges from 5 to 180 mg/0.0113 mm (0.4 to 15.9 g/mm).

The CTT in the central area of the equine cornea, similar to that in other species, has been shown to be the most sensitive, and the dorsal region the least sensitive [54, 126, 127]. A decrease in corneal sensitivity was shown in sick neonatal foals

compared with normal adults [54]. Corneas in healthy foals were slightly more sensitive than those in healthy adults [54].

In humans, it has been shown that the CTT is influenced by age, mental status, iris color, hormone cycle, gravidity, time of day, humidity, room temperature, esthesiometric method, and investigator [128–132]. A decrease in sensitivity has been described in cats with herpes keratitis, in dogs with spontaneous chronic corneal epithelial defects, and in dogs after neodymium:yttrium aluminium garnet (Nd:YAG) laser photocoagulation [133, 134]. Further investigation into the effects of corneal disease, cyclophotocoagulation, and surgeries such as penetrating or lamellar keratoplasty on CTT needs to be performed in the horse.

### Ultrasonic pachymetry

Pachymetry is an accurate and reliable ultrasound technique to measure the corneal thickness *in vivo* in animals and humans [81, 135, 136]. Use of spectral-domain optical coherence tomography (SD-OCT) or *in vivo* confocal microscopy (IVCM) may be more accurate methods to measure corneal thickness, but the cost of these modalities and the lack of portable equipment make them not as practical for use in the horse [137, 138]. Prior to use of ultrasonic pachymetry, corneal thickness was measured from gross post-mortem specimens and histologic specimens, which overestimated corneal thickness because the cornea swells after death [139]. Ultrasonic pachymetry measures the time required for ultrasonic energy to traverse the cornea, with a preset constant for velocity of sound, and converts this to a measure of thickness [135, 136, 140]. A transparent cornea is required for ultrasonic pachymetry full-thickness corneal measurements [12].

A 20-MHz ultrasonic pachymeter (DGH500 (DGH Technology Inc., Exton, PA)) has been used to study the horse. It has been used to determine corneal thickness in healthy juvenile and adult horses, Rocky Mountain horses with cornea globosa, horses that have been given an auriculopalpebral nerve block and xylazine, and healthy Miniature horses [38, 81, 140, 141]. Corneal thickness of enucleated globes measured by a caliper was approximately 858 mm centrally, 914–939 mm dorsally and ventrally, and 861–898 mm laterally and medially [140]. With ultrasonic pachymetry, central corneal thickness (CCT) was reported to be 793 mm, and thicker peripherally at 831–924 mm *in vivo* [81]. Three reports indicate that the dorsal and ventral portions of the cornea are thicker than the central cornea [38, 81, 140]. CCT of healthy equine eyes has been recently measured also with high-frequency (HF) ultrasound and ultrasound microscopy (UBM), with the mean value of  $818 \pm 41 \mu\text{m}$ , and with SD-OCT, with the mean values of  $800 \pm 50$  and  $812.8 \pm 42.9 \mu\text{m}$  [142, 143]. Eyelid block, sedation, age, and sex did not affect corneal thickness [81]. However, thickness of the central portion of the cornea may increase up to the age of 6 months in a healthy horse [38]. Corneal thickness in the normal Miniature horse is 785 mm centrally [141]. Corneal thickness was measured using a portable ultrasonic pachymeter in Quarter horses

affected by hereditary equine regional dermal asthenia (HERDA) [144, 145]. In several different corneal regions (dorsal, ventral, nasal, temporal, and central) the corneal thickness was significantly lower than in the control group, with a CCT mean value of  $741.2 \pm 43.6 \mu\text{m}$  compared with  $819.9 \pm 45.9 \mu\text{m}$  mean value in the control group [145]. See Chapter 7 for more information on corneal thickness and diseases of the cornea in the horse.

### Electroretinography

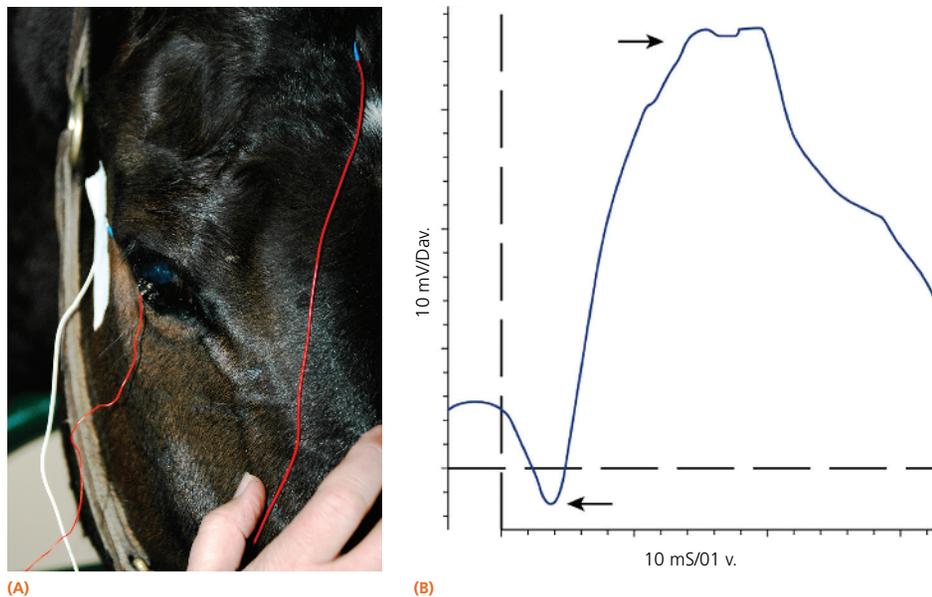
Electroretinography is used to determine function in the outer layers of the retina by recording the summation of electrical response when the retina is stimulated by light [146, 147]. The electroretinogram (ERG) is the recorded total electrical response of the retina to that light. The ERG is not a measure of vision but only a measure of functional integrity of the outer portion of the retina and RPE. A blind animal (e.g., with disease of the inner retina, optic nerve, or central nervous system) can have normal ERG findings. The ganglion cells, their axons, and the optic nerve do not contribute to the ERG recording.

An ERG is indicated whenever visual problems in the outer retina are suspected. In the horse, an ERG is indicated to assess for retinal function in cases of equine recurrent uveitis (ERU), corneal or lens opacities that preclude visualization of the ocular fundus (e.g., cataracts, diffuse corneal edema), suspected congenital stationary night blindness (CSNB), neuroaxonal dystrophy (NAD), equine degenerative myeloencephalopathy (EMD), non-ERU-related retinitis and chorioretinitis, drug toxicity, retinal detachment, ocular trauma, and chronic glaucoma [12, 148–154]. A recent ERG evaluation of eyes with extensive bullet-hole lesions shows that these lesions do not appear to

compromise outer retinal function [155]. See Chapter 11 for more information on these diseases and their effect on the ERG.

The primary components of an electroretinograph are a light source (photostimulator), a high-gain amplifier, and a recorder [147]. Three electrodes are needed: a positive (i.e., active) corneal electrode, a negative (i.e., reference) electrode, and a ground (i.e., indifferent) electrode (Figure 1.53A) [147]. The clinical ERG consists of three basic waveforms: the a-wave, b-wave, and c-wave (Figure 1.53B) [147]. The a-wave is an initial negative deflection. This is followed by a positive deflection, the b-wave, which has a higher potential (amplitude) and is followed by an after-negativity. In addition to the a-wave and b-wave, there is usually a second positive deflection, the c-wave, which is more prolonged. The origin of each wave component is complex and poorly understood. Latency and implicit times, the time from onset of a stimulus to the peak of a particular response, are also important in evaluating the ERG. In a diseased eye, the amplitudes typically decrease while the implicit times increase.

One of the intrinsic factors that can affect the ERG is the eye's state of light adaptation, classified as scotopic (dark-adapted) and photopic (light-adapted) [147]. The scotopic state of the eye is primarily a rod response. The photopic state of the eye is primarily a cone response. Rods are more numerous than cones in the horse, so the ERG is primarily a rod response in this species [147]. The rod and cone responses can be separated by repetitive stimuli or flickers of varying frequency, light adaptation state of the retina, and variation in the intensity of the stimulus [147]. Other intrinsic factors that may affect the ERG include age, transparency of the ocular media, retinal integrity, and retinal circulatory disturbances [147]. Amplitudes



**Figure 1.53** (A) Three electrodes are in place for performance of an electroretinogram (ERG) in the standing horse. (B) The clinical ERG consists of three basic waveforms (a-wave, b-wave, and c-wave). The a-wave, an initial negative deflection (**bottom arrow**), and the b-wave, positive deflection, which follows the a-wave (**top arrow**), are most prominent in the horse.

can vary according to species, state of adaptation to light, ocular movements, pupillary dilation, and pathologic state of the eye [147, 156].

Extrinsic factors that affect the ERG include the light stimulus used, electrodes used, and recording equipment [147, 156]. The light stimulus may have variations in duration of flash, light intensity, frequency, and color. The positioning and type of electrode affect the level of background noise and amplitude of the ERG. Both extrinsic and intrinsic factors can be minimized and must be monitored by establishing a protocol for evaluating the ERG in the horse [147, 156].

Three main techniques used in clinical veterinary ophthalmology are the flash ERG, pattern ERG, and visual evoked potentials. In contrast to the flash ERG, the pattern ERG originates in the inner retina and therefore is helpful in diagnosing diseases such as glaucoma. Limits to visual resolution can also be established by pattern ERG. Visual evoked potentials, although not ERGs, can be recorded with the same equipment and can record activity from the visual cortex. This activity has been mapped in dogs and cats but not in the horse [157].

Flash electroretinography is the most common method for assessment of retinal function in veterinary ophthalmology [147, 153–156]. Proper ERG recording requires general anesthesia in animals to prevent recording artifacts from muscle activity and to allow ideal positioning of the eyes. General anesthesia is inherently risky, expensive, and more labor intensive in the horse. Also, the inhalant anesthetics, halothane and isoflurane, have been shown to have a negative effect on the ERG amplitude and inner retinal function in other species [147, 158]. Electroretinography in the standing horse can be difficult because sedation will not eliminate the almost constant head movement. Sedated or unsedated, the horse may still shy from the closeness of the stimulator, and even small head movements can easily dislodge the other electrodes and require their replacement [147]. However, with proper restraint, auriculopalpebral nerve blocks, sedation, and patience, an adequate ERG can be recorded in the standing horse [147, 156].

The normal dark adaptation curve has been investigated, suggesting that full-field ERG examination should be performed after a minimum 20 min of dark adaptation [159]. Findings from flash ERGs in horses have been reported, and the implicit times and amplitudes were similar in the various reports [147, 149, 151, 156, 160, 161, 163]. Mean reported implicit times and amplitudes of the photopic a- and b-waves are  $5.19 \pm 1.56$  and  $26.63 \pm 2.26$  ms and  $40.89 \pm 20.50$  and  $184.75 \pm 63.26$  mV, respectively [147]. Mean implicit times and amplitudes of low-intensity flash (0.33 cd/m<sup>2</sup> with 5 minutes of dark adaptation) and high-intensity flash (4.62 cd/m<sup>2</sup> with 5 minutes of dark adaptation) scotopic a- and b-waves, with pseudo-Ganzfeld stimulation, DTL (Dawson, Trick, and Litzkow) microfiber electrodes (Retinographic Technologies, Scranton, PA), and detomidine sedation in the standing horse have been reported [147]. Low-intensity flash scotopic a- and b-wave implicit times and amplitudes are  $5.73 \pm 1.88$  and  $36.95 \pm 3.89$  ms and

$103.18 \pm 120.72$  and  $409.30 \pm 319.36$  mV, respectively [147]. High-intensity flash scotopic a- and b-wave implicit times and amplitudes are  $5.13 \pm 1.34$  and  $34.75 \pm 1.87$  ms and  $153.68 \pm 94.19$  and  $374.09 \pm 161.93$  mV, respectively [147].

More recently, a study on ERG responses has been performed in 30 normal Thoroughbreds, sedated with detomidine HCl (0.015 mg/kg) using the Handheld Multispecies ERG (HMsERG) Model 1000 (RetVetCorp Inc., Columbia, MO), with its mini-Ganzfeld half-sphere bowl system and a gold foil contact lens electrode (ERG-jet™ (Fabrinal SA Tuilerie 42, 2300 La Chaux-de-Fonds, Switzerland)). After 20 min of dark adaptation, the International Society for Clinical Electrophysiology of Vision (ISCEV) standard protocol was used measuring implicit time (IT) and amplitudes (AMP). The protocol included a scotopic setting for rod function testing (10 mcd s/m<sup>2</sup> with 0.2-s intervals, b-wave: mean IT =  $57.35 \pm 9.36$  ms, variation 41.8–72.9 ms, AMP =  $96.53 \pm 46.19$  μV, variation 19.8–173.3), followed by a mixed scotopic–photopic (rod and cone) stimulation (3000 mcd s/m<sup>2</sup> with 2.5-s intervals, a-wave: mean IT =  $13.94 \pm 0.44$  ms, range 13.2–14.7 ms, AMP =  $106.20 \pm 22.75$  μV, range 68.4–144 μV, b-wave: mean IT =  $35.13 \pm 3.84$  ms, range 28.7–41.5, mean AMP =  $188.60 \pm 49.88$  μV, range 105.7–271.5 μV) and then by photopic (cone) stimulation (10,000 mcd s/m<sup>2</sup> with 5-s intervals, a-wave: mean IT =  $13.95 \pm 0.56$  ms, range 13–14.9, mean AMP =  $136.25 \pm 30.39$  μV, range 85.7–186.8 μV, b-wave: mean IT =  $36.01 \pm 5.66$  ms, range 26.6–45.4 ms, mean AMP =  $177.64 \pm 43.83$  μV, range 104.7–250.6 μV). Lastly, after 10 minutes of light adaptation, a cone stimulation was recorded using a 30-Hz flicker response test at 3000 mcd s/m<sup>2</sup> (b-wave: mean IT =  $25.84 \pm 1.86$  ms, range 22.8–28.9 ms, mean AMP =  $80.59 \pm 21.94$  μV, range 44.1–117.1 μV) [156]. ERG flicker-photometry has been used to assess the spectral sensitivities of cones in the horse, [147, 156] but oscillatory potentials have not yet been recorded [147, 149, 151, 156, 160, 161, 163].

### Retinoscopy (skiascopy)

Retinoscopy, or skiascopy, is the technique used to determine the refractive state of the eye. Retinoscopy is the only clinical and practical method of refraction in horses. Commonly used in pediatric human ophthalmology, this technique has been used in veterinary medicine to define the normal, pathologic, and surgically induced refractive state of the eyes of the horse [39, 163–180]. The instrumentation and technique used can be challenging, and the reader is referred to an article by Davidson for further information [170].

Light rays projected onto an eye from infinity emerge from an emmetropic eye as parallel rays, from a myopic (near-sighted) eye as converging rays, and from a hyperopic (far-sighted) eye as diverging rays [170]. The location at which these emergent light rays form a focal point is called the **far point** [170]. The far point is at infinity, in front of infinity, and beyond infinity for emmetropic, myopic, and hyperopic eyes, respectively [170].

The retinoscope is either spot or streak, but streak retinoscopes are the most commonly used in veterinary medicine [170].



**Figure 1.54** Retinoscopy with a skiascopy bar in the horse.

Both streak and spot retinoscopes are available from Copeland, Heine, Keeler, Propper, Reichert, and Welch Allyn. Plus or minus spherical lenses, available in increments of 0.25 D, are placed between the retinoscope and the horse to quantitate the refractive error of the eye [170]. A simple and inexpensive skiascopy bar or rack contains a series of plus and minus lenses in increments of 0.5–1.0 D [170].

Retinoscopy is performed in a darkened room with a handler restraining the horse's head. Mydriasis is often unnecessary and can even make the technique more difficult; the limited accommodative ability of the horse makes cycloplegia less important [170–172]. The retinoscope is placed against the examiner's brow, and the examiner is positioned 0.67 m (working distance, approximately an arm's length) from the patient's eye (Figure 1.54) [170]. The streak is swept horizontally across the horse's pupil, rotated horizontally, and then swept vertically across the pupil [170]. Finally, a trial lens or skiascopy bar is placed 1–2 cm from the patient's cornea, and the process is repeated [170].

As the streak is slowly swept across the pupil, the fundic reflex will move in either the same or the opposite direction, depending on the refractive state of the patient. With no refractive lens, the fundic reflex will move in the same direction as the sweep with emmetropic and hyperopic eyes (a **with** motion) and in the opposite direction of the sweep with more than 1.5-D myopic eyes (an **against** motion) [170]. If a with motion is observed, plus lenses of increasing dioptric strength are placed in front of the patient's eye until an against motion is observed or neutralization is reached [170]. Neutralization is characterized by a fundic reflex that completely fills the pupil without any noticeable direction of movement [170]. If an against motion is observed, minus lenses of increasing dioptric strength are used to achieve neutrality [170]. At a working distance of 0.67 m, a +1.5-D lens is needed to achieve neutralization with an emmetropic eye; therefore the refractive error of an eye is determined by subtracting 1.5 D from the gross refraction needed to achieve neutrality [170].

Retinoscopy may allow selection of intraocular lens implants in horses and assist with evaluation of performance problems in working animals. However, refractive error has an unknown effect on horses, and is discussed further in Chapter 12. In healthy horses, refractive error has been reported to range from  $-3$  to  $+3$  D [173], but most appear to be within 1 D of emmetropia [168–176]. In one study, the mean refractive state was  $-0.06 \pm 0.68$  D, with emmetropia in 77 of 158 horses (48.7%), hyperopia, with a maximum value of  $+1.5$  D, in 38 (24.1%), and myopia, with a maximum value of  $-1.5$  D, in 40 (25.3%) horses. In this study, just 1.3% of horses (2/158) were myopic to the degree of  $-2.0$  D, and 0.6% (1/158) were myopic to the degree of  $-3.0$  D [175]. In another study on 333 horses and ponies (666 eyes), emmetropia was found in 83.63% of eyes (557/666), with 68.5% (228/333) of horses/ponies with emmetropia in both eyes. Refractive errors greater than 1.50 D were found in 2.7% of eyes tested. Ametropic eyes included hyperopia (54%) and myopia (46%). Anisometropia (when the refractive power of the two eyes differs by more than 0.5 D) was found in 30.3% of horses/ponies [176]. No significant differences in the refractive states in both axes, horizontal and vertical, were found after mydriasis induced with tropicamide 1%, but it is suggested to perform retinoscopy examination before or 40–45 min after application of topical tropicamide 1%, as mydriasis may subjectively influence a novice retinoscopist's ability to obtain accurate results [172]. The aphakic equine eye after cataract surgery has been reported to be hyperopic ( $+9.94$  D) [177, 178]. An intraocular lens implant (IOL) with a refractive power of 25 D resulted in  $-3.48$  D myopia at post-operative retinoscopy [177, 178]. In a second study, a 14-D IOL was placed, after cataract phacoemulsification, in five eyes with a mean post-operative refractive error of  $+0.4 \pm 1.1$  D [179]. In another study, the same +14-D IOL was placed in a foal after phacoemulsification, resulting in a post-operative refractive error of  $+0.31$  D [180]. Astigmatism, a state of unequal refraction along the different meridians of the eye (i.e., vertical versus horizontal), has not been investigated by retinoscopy, but in horses has been measured by keratometry [145, 175]. Variation in the reported refractive error in horses can be explained by the technique used, skill of the examiner, sample size, and accommodative state of the animal.

### Additional content

The book companion website at

[www.wiley.com/go/gilger/ophthalmology](http://www.wiley.com/go/gilger/ophthalmology)

includes additional text and figures that are not in the printed book or E-book.

Please see the “**About the companion website**” page at the start of the book for details on how to access the website.

## References

1. Budras K. *Anatomy of the Horse: An Illustrated Text*, 3rd edn. Hannover: Schlutersche; 2001.
2. Dyce K, Sack W, Wensing C. *Textbook of Veterinary Anatomy*, 4th edn. Philadelphia: Saunders; 2010.
3. Kainer RA. Clinical anatomy of the equine head. *Vet Clin North Am Equine Pract* 1993;**9**:1–23.
4. Lavach J. *Large Animal Ophthalmology*. St Louis: Mosby; 1990.
5. Prince J. *Anatomy and Histology of the Eye and Orbit in Domestic Animals*. Springfield: Charles C. Thomas; 1960:128–153.
6. Samuelson D. Ophthalmic anatomy. In: Gelatt KN, Gilger BC, Kern TJ (eds). *Veterinary Ophthalmology*, 5th edn. Ames: Wiley Blackwell; 2013:39–170.
7. Bistner S. Fundus examination of the horse. *Vet Clin North Am Large Anim Pract* 1984;**6**:541–551.
8. Brooks D, Matthews A. Equine ophthalmology. In: Gelatt KN (ed). *Veterinary Ophthalmology*, 4th edn. Ames: Wiley Blackwell; 2007: 1165–1274.
9. Cooley PL. Normal equine ocular anatomy and eye examination. *Vet Clin North Am Equine Pract* 1992;**8**:427–449.
10. Meredith R, Wolf E. Ophthalmic examination and therapeutic techniques in the horse. *Comp Cont Ed Pract Vet* 1981;**3**:s426–s433.
11. Davis JL, Stewart T, Brazik E, Gilger BC. The effect of topical administration of atropine sulfate on the normal equine pupil: influence of age, breed and gender. *Vet Ophthalmol* 2003;**6**: 329–332.
12. Featherston HJ, Heinrich C. Ophthalmic examination and diagnostic. Part I: the eye examination and diagnostic procedures. In: Gelatt KN, Gilger BC, Kern TJ (eds). *Veterinary Ophthalmology*, 5th edn. Ames: Wiley Blackwell; 2013:533–613.
13. Gelatt K, Gum G, Mackay E. Evaluation of mydriatics in horses. *Vet Comp Ophthalmol* 1995;**5**:104–107.
14. Hacker DV, Buyukmihci NC, Franti CE, Bellhorn RW. Effect of topical phenylephrine on the equine pupil. *Am J Vet Res* 1987;**48**:320–322.
15. Hahn CN, Mayhew IG. Studies on the experimental induction of ptosis in horses. *Vet J* 2000;**160**:220–224.
16. Williams MM, Spiess BM, Pascoe PJ, O'Grady M. Systemic effects of topical and subconjunctival ophthalmic atropine in the horse. *Vet Ophthalmol* 2000;**3**:193–199.
17. Severin G. *Veterinary Ophthalmology Notes*, 3rd edn. Fort Collins: Colorado State University; 1995.
18. George L. Localization and differentiation of neurologic diseases. In: Smith B (ed). *Large Animal Internal Medicine*. St Louis: Mosby; 1990:145–170.
19. Mayhew I. *Large Animal Neurology: A Handbook for Veterinary Clinicians*. Philadelphia: Lea & Febiger; 1989.
20. Scagliotti R. Comparative neuro-ophthalmology. In: Gelatt KN (ed). *Veterinary Ophthalmology*, 3rd edn. Philadelphia: Lippincott Williams & Wilkins; 1999:1307–1400.
21. Crispin SM. Developmental anomalies and abnormalities of the equine iris. *Vet Ophthalmol* 2000;**3**:93–98.
22. Gum G, Gelatt K, Ofri R. Physiology of the eye. In: Gelatt KN (ed). *Veterinary Ophthalmology*, 3rd edn. Philadelphia: Lippincott Williams & Wilkins; 1999:151–181.
23. Crispin SM. Tear-deficient and evaporative dry eye syndromes of the horse. *Vet Ophthalmol* 2000;**3**:87–92.
24. Bacon CL, Davidson HJ, Yvorchuk K, Basaraba RJ. Bilateral Horner's syndrome secondary to metastatic squamous cell carcinoma in a horse. *Equine Vet J* 1996;**28**:500–503.
25. Green SL, Cochran SM, Smith-Maxie L. Horner's syndrome in ten horses. *Can Vet J* 1992;**33**:330–333.
26. Neer T. Horner's syndrome: anatomy, diagnosis, and causes. *Comp Contin Educ Pract Vet* 1984;**6**:740–746.
27. Purohit RC, McCoy MD, Bergfeld WA 3rd. Thermographic diagnosis of Horner's syndrome in the horse. *Am J Vet Res* 1980;**41**:1180–1182.
28. Simoens P, Lauwers H, De Muelenare C, Muylle E, Steenhaut M. Horner's syndrome in the horse: a clinical, experimental and morphological study. *Equine Vet J Suppl* 1990;**62**–65.
29. Smith J, Mayhew I. Horner's syndrome in large animals. *Cornell Vet* 1977;**67**:529–542.
30. Sweeney RW, Sweeney CR. Transient Horner's syndrome following routine intravenous injections in two horses. *J Am Vet Med Assoc* 1984;**185**:802–803.
31. Geiser D, Henton J, Held J. Tympanic bulla, petrous temporal bone, and hyoid apparatus disease in horses. *Comp Contin Educ Pract Vet* 1988;**10**:740–754.
32. De Lahunta A, Glass E, Kent M. Lower motor neuron: general visceral efferent system. In: De Lahunta-Glass-Kent (ed). *Veterinary Neuroanatomy and Clinical Neurology*, 4th edn. St Louis: Elsevier Saunders; 2015:197–221.
33. Muir W, Hubbell J. Anesthetic techniques. In: Muir W, Hubbell J (eds). *Equine Anesthesia: Monitoring and Emergency Therapy*. St Louis: Mosby; 1991:444–460.
34. Stine MJ, Michau TM, Williams MK, et al. The effects of intravenous romifidine on intraocular pressure in clinically normal horses and horses with incidental ophthalmic findings. *Vet Ophthalmol* 2014;**17** Suppl 1:134–139.
35. Ramsey DT, Hauptman JG, Petersen-Jones SM. Corneal thickness, intraocular pressure, and optical corneal diameter in Rocky Mountain horses with cornea globosa or clinically normal corneas. *Am J Vet Res* 1999;**60**:1317–321.
36. Ramsey DT, Ewart SL, Render JA, Cook CS, Latimer CA. Congenital ocular abnormalities of Rocky Mountain horses. *Vet Ophthalmol* 1999;**2**:47–59.
37. Rubin L. Auriculopalpebral nerve block as an adjunct to the diagnosis and treatment of ocular inflammation in the horse. *J Am Vet Med Assoc* 1964;**144**:1387–1388.
38. Ramsey DT, Hauptman JG, Petersen-Jones SM. Corneal thickness, intraocular pressure, and optical corneal diameter in Rocky Mountain horses with cornea globosa or clinically normal corneas. *Am J Vet Res* 1999;**60**:1317–321.
39. Ramsey DT, Ewart SL, Render JA, Cook CS, Latimer CA. Congenital ocular abnormalities of Rocky Mountain horses. *Vet Ophthalmol* 1999;**2**:47–59.
40. Beech J, Zappala RA, Smith G, Lindborg S. Schirmer tear test results in normal horses and ponies: effect of age, season, environment, sex, time of day and placement of strips. *Vet Ophthalmol* 2003;**6**:251–254.
41. Brightman AH, 2nd, Manning JP, Benson GJ, Musselman EE. Decreased tear production associated with general anesthesia in the horse. *J Am Vet Med Assoc* 1983;**182**:243–244.
42. Piccione G, Giannetto C, Fazio F, Giudice E. Daily rhythm of tear production in normal horse. *Vet Ophthalmol* 2008;**11** Suppl 1:57–60.

43. Schwarz BC, Sallmutter T, Nell B. Keratoconjunctivitis sicca attributable to parasympathetic facial nerve dysfunction associated with hypothyroidism in a horse. *J Am Vet Med Assoc* 2008;**233**:1761–1766.
44. Sindak N, Kandemir L, Mugdat Y, et al. Measurement of phenol red thread tear test in Arabian and Thoroughbred horses. *Vet Ophthalmol* 2010;**13**:219–221.
45. Monclin SJ, Farnir F, Grauwels M. Determination of tear break-up time reference values and ocular tolerance of tetracaine hydrochloride eyedrops in healthy horses. *Equine Vet J* 2011;**43**:74–77.
46. Reilly L, Beech J. Bilateral keratoconjunctivitis sicca in a horse. *Equine Vet J* 1994;**26**:171–172.
47. Collins B. Immune mediated keratoconjunctivitis sicca in a horse. *Vet Comp Ophthalmol* 1994;**4**:61–65.
48. Spurlock SL, Spurlock GH, Wise M. Keratoconjunctivitis sicca associated with fracture of the stylohyoid bone in a horse. *J Am Vet Med Assoc* 1989;**194**:258–259.
49. Spiess BM, Wilcock BP, Physick-Sheard PW. Eosinophilic granulomatous dacryoadenitis causing bilateral keratoconjunctivitis sicca in a horse. *Equine Vet J* 1989;**21**:226–228.
50. Wolf E, Meredith R. Parotid duct transposition in the horse. *J Equine Vet Sci* 1981;**12**:143–145.
51. Joyce JR, Bratton GR. Keratoconjunctivitis sicca secondary to fracture of the mandible. *Vet Med Small Anim Clin* 1973;**68**:619–620.
52. Marts B, Bryan G, Prieur D. Schirmer tear test measurement and lysozyme concentration of equine tears. *J Equine Med Surg* 1977;**1**:427–430.
53. Williams R, Manning J, Peiffer R. The Schirmer tear test in the equine: normal values and the contribution of the gland of the nictitating membrane. *J Equine Med Surg* 1979;**3**:117–119.
54. Brooks DE, Clark CK, Lester GD. Cochet-Bonnet aesthesiometer-determined corneal sensitivity in neonatal foals and adult horses. *Vet Ophthalmol* 2000;**3**:133–137.
55. Clark C, Brooks D, Lester G. Corneal sensitivity and tear production in hospitalized neonatal foals. *Am Coll Vet Ophthalmol* 1996;**134**–**136**.
56. Hamor RE, Roberts SM, Severin GA, Chavkin MJ. Evaluation of results for Schirmer tear tests conducted with and without application of a topical anesthetic in clinically normal dogs of 5 breeds. *Am J Vet Res* 2000;**61**:1422–1425.
57. Dziezyc J. Nasolacrimal system. In: Auer J (ed). *Equine Surgery*. Philadelphia: Saunders; 1992:630–634.
58. Latimer CA, Wyman M, Diesem CD, Burt JK. Radiographic and gross anatomy of the nasolacrimal duct of the horse. *Am J Vet Res* 1984;**45**:451–458.
59. Cassotis NJ, Schiffman P. Calcification associated with the nasolacrimal system of a horse: case report and mineralogic composition. *Vet Ophthalmol* 2006;**9**:187–190.
60. Spadari A, Spinella G, Grandis A, et al. Endoscopic examination of the nasolacrimal duct in ten horses. *Equine Vet J* 2011;**43**:159–162.
61. Berliner M. *Biomicroscopy of the Eye*. New York: Paul B. Hoeber; 1949.
62. Martin CL. Slit lamp examination of the normal canine anterior ocular segment. III. Discussion and summary. *J Small Anim Pract* 1969;**10**:163–169.
63. Martin CL. Slit lamp examination of the normal canine anterior ocular segment. II. Description. *J Small Anim Pract* 1969;**10**:151–162.
64. Martin CL. Slit lamp examination of the normal canine anterior ocular segment. I. Introduction and technique. *J Small Anim Pract* 1969;**10**:143–149.
65. Feenstra RP, Tseng SC. What is actually stained by rose bengal? *Arch Ophthalmol* 1992;**110**:984–993.
66. Gelatt K. Vital staining of the canine cornea and conjunctiva with rose bengal. *J Am Anim Hosp Assoc* 1972;**8**:17–22.
67. Kim J. The use of vital dyes in corneal disease. *Curr Opin Ophthalmol* 2000;**11**:241–247.
68. Laroche RR, Campbell RC. Quantitative rose bengal staining technique for external ocular diseases. *Ann Ophthalmol* 1988;**20**:274–276.
69. Slatter DH. Differential vital staining of the canine cornea and conjunctiva with fluorescein-rose bengal and alcian blue. *J Small Anim Pract* 1973;**14**:291–296.
70. Kimura SJ. Fluorescein paper. A simple means of insuring the use of sterile fluorescein. *Am J Ophthalmol* 1951;**34**:446–447.
71. Cello RM, Lasmanis J. Pseudomonas infection of the eye of the dog resulting from the use of contaminated fluorescein solution. *J Am Vet Med Assoc* 1958;**132**:297–299.
72. Feenstra RP, Tseng SC. Comparison of fluorescein and rose bengal staining. *Ophthalmology* 1992;**99**:605–617.
73. Kim J, Foulks GN. Evaluation of the effect of lissamine green and rose bengal on human corneal epithelial cells. *Cornea* 1999;**18**:328–332.
74. Brooks DE, Andrew SE, Denis H, et al. Rose bengal positive epithelial microerosions as a manifestation of equine keratomycosis. *Vet Ophthalmol* 2000;**3**:83–86.
75. Machado LM, Castro RS, Fontes BM. Staining patterns in dry eye syndrome: rose bengal versus lissamine green. *Cornea* 2009;**28**:732–734.
76. Yoon KC, Im SH, Kim HG, et al. Usefulness of double vital staining with 1% fluorescein and 1% lissamine green in patients with dry eye syndrome. *Cornea* 2011;**30**:972–976.
77. Korb DR, Herman JP, Finnemore VM, et al. An evaluation of the efficacy of fluorescein, rose bengal, lissamine green, and a new dye mixture for ocular surface staining. *Eye Contact Lens* 2008;**34**:61–64.
78. Buss DG, Sharma A, Giuliano EA, et al. Efficacy and safety of mitomycin C as an agent to treat corneal scarring in horses using an *in vitro* model. *Vet Ophthalmol* 2010;**13**:211–218.
79. Buss DG, Giuliano E, Sharma A, et al. Gene delivery in the equine cornea: a novel therapeutic strategy. *Vet Ophthalmol* 2010;**13**:301–306.
80. Knollinger AM, La Croix NC, Barrett PM, Miller PE. Evaluation of a rebound tonometer for measuring intraocular pressure in dogs and horses. *J Am Vet Med Assoc* 2005;**227**:244–248.
81. van der Woerd A, Gilger BC, Wilkie DA, Strauch SM. Effect of auriculopalpebral nerve block and intravenous administration of xylazine on intraocular pressure and corneal thickness in horses. *Am J Vet Res* 1995;**56**:155–158.
82. McClure J, Gelatt K, Manning J. The effect of parenteral acepromazine and xylazine on intraocular pressure in the horse. *Vet Med Small Anim Clin* 1976;**71**:1727–1730.
83. Smith PJ, Gum GG, Whitley RD, Samuelson DA, Brooks DE, Garcia-Sanchez GA. Tonometric and tonographic studies in the normal pony eye. *Equine Vet J Suppl* 1990;**36**–38.
84. Trim CM, Colbern GT, Martin CL. Effect of xylazine and ketamine on intraocular pressure in horses. *Vet Rec* 1985;**117**:442–443.
85. Stine MJ, Michau TM, Williams MK, et al. The effects of intravenous romifidine on intraocular pressure in clinically normal horses and horses with incidental ophthalmic findings. *Vet Ophthalmol* 2014;**17** Suppl 1:134–139.
86. Marzok MA, El-khodery SA, Oheida A. Effect of intravenous administration of romifidine on intraocular pressure in clinically normal horses. *Vet Ophthalmol* 2014;**17** Suppl 1:149–153.

87. Miller PE, Pickett JP, Majors LJ. Evaluation of two applanation tonometers in horses. *Am J Vet Res* 1990;**51**:935–937.
88. Cohen CM, Reinke DA. Equine tonometry. *J Am Vet Med Assoc* 1970;**156**:1884–1887.
89. Komaromy AM, Garg CD, Ying GS, Liu C. Effect of head position on intraocular pressure in horses. *Am J Vet Res* 2006;**67**:1232–1235.
90. Bertolucci C, Giudice E, Fazio F, Piccione G. Circadian intraocular pressure rhythms in athletic horses under different lighting regime. *Chronobiol Int* 2009;**26**:348–358.
91. Gelatt KN, Gum GG. Evaluation of electronic tonometers in the rabbit eye. *Am J Vet Res* 1981;**42**:1778–1781.
92. Gelatt KN, Peiffer RL Jr, Gum GG, Gwin RM, Erickson JL. Evaluation of applanation tonometers for the dog eye. *Invest Ophthalmol Vis Sci* 1977;**16**:963–968.
93. Kotani T. Which are the optimal tonometers for different animal species? *Anim Eye Res* 1993;**12**:55–61.
94. Dziezyc J, Millichamp NJ, Smith WB. Comparison of applanation tonometers in dogs and horses. *J Am Vet Med Assoc* 1992;**201**:430–433.
95. Gelatt K, Gum G, Mackay E. Estimation of aqueous humor outflow facility by pneumatonography in normal, genetic carrier, and glaucomatous Beagles. *Vet Comp Ophthalmol* 1996;**6**:148–151.
96. Toris CB, Yablonski ME, Wang YL, Hayashi M. Prostaglandin A2 increases uveoscleral outflow and trabecular outflow facility in the cat. *Exp Eye Res* 1995;**61**:649–657.
97. Barnett KC. The ocular fundus of the horse. *Equine Vet J* 1972;**4**:17–20.
98. Crispin SM, Matthews AG, Parker J. The equine fundus. I: Examination, embryology, structure and function. *Equine Vet J Suppl* 1990;**42**–49.
99. Cutler TJ, Brooks DE, Andrew SE, et al. Disease of the equine posterior segment. *Vet Ophthalmol* 2000;**3**:73–82.
100. Matthews AG, Crispin SM, Parker J. The equine fundus. III: Pathological variants. *Equine Vet J Suppl* 1990;**55**–61.
101. Matthews AG, Crispin SM, Parker J. The equine fundus. II: Normal anatomical variants and colobomata. *Equine Vet J Suppl* 1990;**50**–54.
102. Rebhun WC. Retinal and optic nerve diseases. *Vet Clin North Am Equine Pract* 1992;**8**:587–608.
103. Cutler TJ. Ophthalmic findings in the geriatric horse. *Vet Clin North Am Equine Pract* 2002;**18**:545–574.
104. Rubin L. *Atlas of Veterinary Ophthalmoscopy*. Philadelphia: Lea & Febiger; 1974.
105. Barnett K. Principles of ophthalmoscopy. *Vet Med* 1967;**62**:56–57.
106. Murphy CJ, Howland HC. The optics of comparative ophthalmoscopy. *Vision Res* 1987;**27**:599–607.
107. Raffe M. Retrobulbar block in combination with general anesthesia for equine ophthalmic surgery. *Vet Surg* 1986;**15**:139–141.
108. Hewes CA, Keoughan GC, Gutierrez-Nibeyro S. Standing enucleation in the horse: a report of 5 cases. *Can Vet J* 2007;**48**:512–514.
109. Pollock PJ, Russell T, Hughes TK, et al. Transpalpebral eye enucleation in 40 standing horses. *Vet Surg* 2008;**37**:306–309.
110. Oel C, Gerhards H, Gehlen H. Effect of retrobulbar nerve block on heart rate variability during enucleation in horses under general anesthesia. *Vet Ophthalmol* 2014;**17**:170–174.
111. McMullen RJ, Utter ME. Current developments in equine cataract surgery. *Equine Vet J Suppl* 2010;**37**:38–45.
112. Short CE, Rebhun WC. Complications caused by the oculocardiac reflex during anesthesia in a foal. *J Am Vet Med Assoc* 1980;**176**:630–631.
113. Brooks D. Orbit. In: Auer J (ed). *Equine Surgery*. Philadelphia: WB Saunders; 1992:654–666.
114. Gupta S, Sood B, Gulati M, et al. Orbital mass lesions: US-guided fine-needle aspiration biopsy—experience in 37 patients. *Radiology* 1999;**213**:568–572.
115. Hazel SJ, Thrall MA, Severin GA, Lauerma LH Jr, Lavach JD. Laboratory evaluation of aqueous humor in the healthy dog, cat, horse, and cow. *Am J Vet Res* 1985;**46**:657–659.
116. Olin DD. Examination of the aqueous humor as a diagnostic aid in anterior uveitis. *J Am Vet Med Assoc* 1977;**171**:557–559.
117. Gilger BC, Salmon JH, Yi NY, et al. Role of bacteria in the pathogenesis of recurrent uveitis in horses from the southeastern USA. *Am J Vet Res* 2008;**69**:1329–1335.
118. Glaze MB, McGuire TC, Schmidt GM, Leid RW. Immunoglobulin levels in tears and aqueous humor of horses before and after diethylcarbamazine (DEC) therapy. *Vet Immunol Immunopathol* 1984;**7**:185–198.
119. Halliwell RE, Brim TA, Hines MT, Wolf D, White FH. Studies on equine recurrent uveitis. II: The role of infection with *Leptospira interrogans* serovar pomona. *Curr Eye Res* 1985;**4**:1033–1040.
120. Halliwell RE, Hines MT. Studies on equine recurrent uveitis. I: Levels of immunoglobulin and albumin in the aqueous humor of horses with and without intraocular disease. *Curr Eye Res* 1985;**4**:1023–1031.
121. Wurster U, Riese K, Hoffmann K. Enzyme activities and protein concentration in the intraocular fluids of ten mammals. *Acta Ophthalmol (Copenh)* 1982;**60**:729–741.
122. Miller TL, Willis AM, Wilkie DA, Hoshaw-Woodard S, Stanley JR. Description of ciliary body anatomy and identification of sites for transscleral cyclophotocoagulation in the equine eye. *Vet Ophthalmol* 2001;**4**:183–190.
123. Gemensky-Metzler AJ, Wilkie DA, Weisbrode SE, Kuhn SE. The location of sites and effect of semiconductor diode trans-scleral cyclophotocoagulation on the buphthalmic equine globe. *Vet Ophthalmol* 2014;**17** Suppl 1:107–116.
124. McLaughlin BG, McLaughlin PS. Equine vitreous humor chemical concentrations: correlation with serum concentrations, and postmortem changes with time and temperature. *Can J Vet Res* 1988;**52**:476–480.
125. Wollanke B, Rohrbach BW, Gerhards H. Serum and vitreous humor antibody titers in and isolation of *Leptospira interrogans* from horses with recurrent uveitis. *J Am Vet Med Assoc* 2001;**219**:795–800.
126. Kaps S, Richter M, Spiess BM. Corneal esthesiometry in the healthy horse. *Vet Ophthalmol* 2003;**6**:151–155.
127. Welihozkiy A, Bedenice D, Price LL, et al. Measurement of corneal sensitivity in 20 ophthalmologically normal alpacas. *Vet Ophthalmol* 2011;**14**:333–336.
128. Boberg-Ans J. Experience in clinical examination of corneal sensitivity; corneal sensitivity and the naso-lacrimal reflex after retrobulbar anaesthesia. *Br J Ophthalmol* 1955;**39**:705–726.
129. Kolstad A. Corneal sensitivity by low temperatures. *Acta Ophthalmol* 1970;**48**:789–793.
130. Millodot M. Do blue-eyed people have more sensitive corneas than brown-eyed people? *Nature* 1975;**151**–152.
131. Millodot M. The influence of pregnancy on the sensitivity of the cornea. *Br J Ophthalmol* 1977;**61**:646–649.

132. Millodot M. The influence of age on the sensitivity of the cornea. *Invest Ophthalmol Vis Sci* 1977;**16**:240–242.
133. Murphy CJ, Marfurt CF, McDermott A, et al. Spontaneous chronic corneal epithelial defects (SCCED) in dogs: clinical features, innervation, and effect of topical SP, with or without IGF-1. *Invest Ophthalmol Vis Sci* 2001;**42**:2252–2261.
134. Weigt AK, Herring IP, Marfurt CF, Pickett JP, Duncan RB Jr, Ward DL. Effects of cyclophotocoagulation with a neodymium:yttrium-aluminum-garnet laser on corneal sensitivity, intraocular pressure, aqueous tear production, and corneal nerve morphology in eyes of dogs. *Am J Vet Res* 2002;**63**:906–915.
135. Gilger BC, Whitley RD, McLaughlin SA, Wright JC, Drane JW. Canine corneal thickness measured by ultrasonic pachymetry. *Am J Vet Res* 1991;**52**:1570–1572.
136. Gilger BC, Wright JC, Whitley RD, McLaughlin SA. Corneal thickness measured by ultrasonic pachymetry in cats. *Am J Vet Res* 1993;**54**:228–230.
137. Choi CY, Youm DJ, Kim MJ, Tchah H. Changes in central corneal thickness of preserved corneas over time measured using anterior segment optical coherence tomography. *Cornea* 2009;**28**:536–540.
138. Pirie CG, Alario AF, Barysaukas CM, et al. Manual corneal thickness measurements of healthy equine eyes using a portable spectral-domain optical coherence tomography device. *Equine Vet J* 2014;**46**:631–634.
139. Ling T. Osmotically induced central and peripheral corneal swelling in the cat. *Am J Optom Physiol Opt* 1987;**64**:674–677.
140. Andrew SE, Ramsey DT, Hauptman JG, Brooks DE. Density of corneal endothelial cells and corneal thickness in eyes of euthanized horses. *Am J Vet Res* 2001;**62**:479–482.
141. Plummer CE, Ramsey DT, Hauptman JG. Assessment of corneal thickness, intraocular pressure, optical corneal diameter, and axial globe dimensions in Miniature horses. *Am J Vet Res* 2003;**64**:661–665.
142. Pinto NI, Gilger BC. Spectral-domain optical coherence tomography evaluation of the cornea, retina, and optic nerve in normal horses. *Vet Ophthalmol* 2014;**17** Suppl 1:140–148.
143. Herbig LE, Eule JC. Central corneal thickness measurements and ultrasonographic study of the growing equine eye. *Vet Ophthalmol* 2015;**6**:462–471.
144. Mochal CA, Miller WW, Cooley AJ, et al. Ocular findings in Quarter horses with hereditary equine regional dermal asthenia. *J Am Vet Med Assoc* 2010;**237**:304–310.
145. Badial PR, Cisneros-Alvarez LE, Breandao CVS, et al. Ocular dimensions, corneal thickness, and corneal curvature in Quarter horses with hereditary equine regional dermal asthenia. *Vet Ophthalmol* 2015;**18**:385–392.
146. Gum GG. Electrophysiology in veterinary ophthalmology. *Vet Clin North Am Small Anim Pract* 1980;**10**:437–454.
147. Komaromy AM, Andrew SE, Sapp HL Jr, Brooks DE, Dawson WW. Flash electroretinography in standing horses using the DTL microfiber electrode. *Vet Ophthalmol* 2003;**6**:27–33.
148. Gilger BC, Malok E, Stewart T, et al. Long-term effect on the equine eye of an intravitreal device used for sustained release of cyclosporine A. *Vet Ophthalmol* 2000;**3**:105–110.
149. Nunnery C, Pickett JP, Zimmerman KL. Congenital stationary night blindness in a Thoroughbred and a Paso Fino. *Vet Ophthalmol* 2005;**8**:415–419.
150. Riis RC, Jackson C, Rebhun W, et al. Ocular manifestations of equine motor neuron disease. *Equine Vet J* 1999;**31**:99–110.
151. Sandmeyer LS, Breaux CB, Archer S, Grahn BH. Clinical and electroretinographic characteristics of congenital stationary night blindness in the Appaloosa and the association with the leopard complex. *Vet Ophthalmol* 2007;**10**:368–375.
152. Strobel BW, Wilkie DA, Gilger BC. Retinal detachment in horses: 40 cases (1998–2005). *Vet Ophthalmol* 2007;**10**:380–385.
153. Sandmeyer LS, Bellone RR, Archer S, et al. Congenital stationary night blindness with the leopard complex in the Miniature horse. *Vet Ophthalmol* 2012;**15**:18–22.
154. Finno CJ, Aleman M, Ofri R, et al. Electrophysiological studies in American Quarter horses with neuronaxonal dystrophy. *Vet Ophthalmol* 2012;**15** Suppl 2:3–7.
155. Allbaugh RA, Ben-Shlomo G, Whitley RD. Electroretinogram evaluation of equine eyes with extensive ‘bullet-hole’ fundic lesions. *Vet Ophthalmol* 2014;**17** Suppl 1:129–133.
156. Church ML, Norman JC. Electroretinogram responses of the normal Thoroughbred horse sedated with detomidine hydrochloride. *Vet Ophthalmol* 2012;**15** Suppl 2:77–83.
157. Bilge M, Bingle A, Seneviratne KN, Whitteridge D. A map of the visual cortex in the cat. *J Physiol* 1967;**191**:116P–118P.
158. Yanase J, Ogawa H. Effects of halothane and sevoflurane on the electroretinogram of dogs. *Am J Vet Res* 1997;**58**:904–909.
159. Ben-Shlomo G, Plummer C, Barrie K, et al. Characterization of the normal dark adaptation curve of the horse. *Vet Ophthalmol* 2012;**15**:42–45.
160. Francois J, Wouters L, Victoria-Troncoso V, de Rouck A, van Gerven A. Morphometric and electrophysiologic study of the photoreceptors in the horse. *Ophthalmologica* 1980;**181**:340–349.
161. Witzel DA, Smith EL, Wilson RD, Aguirre GD. Congenital stationary night blindness: an animal model. *Invest Ophthalmol Vis Sci* 1978;**17**:788–795.
162. Ramsey DT, Hauptman JG, Petersen-Jones SM. Corneal thickness, intraocular pressure, and optical corneal diameter in Rocky Mountain horses with cornea globosa or clinically normal corneas. *Am J Vet Res* 1999;**60**:1317–1321.
163. Wouters L, de Moor A, Moens Y. Rod and cone components in the electroretinogram of the horse. *Zentralbl Veterinarmed A* 1980;**27**:330–338.
164. Farrall H, Handscombe M. Equine vision. *Equine Vet J* 1999;**31**:354–355.
165. Farrall H, Handscombe MC. Follow-up report of a case of surgical aphakia with an analysis of equine visual function. *Equine Vet J Suppl* 1990;**91–93**.
166. Harman AM, Moore S, Hoskins R, Keller P. Horse vision and an explanation for the visual behaviour originally explained by the ‘ramp retina’. *Equine Vet J* 1999;**31**:384–390.
167. Knill LM, Eagleton RD, Harver E. Physical optics of the equine eye. *Am J Vet Res* 1977;**38**:735–737.
168. Roberts SM. Equine vision and optics. *Vet Clin North Am Equine Pract* 1992;**8**:451–457.
169. Sivak J, Allen D. An evaluation of the ramp retina in the horse eye. *Vision Res* 1975;**15**:1353–1356.
170. Davidson M. Clinical retinoscopy for the veterinary ophthalmologist. *Vet Comp Ophthalmol* 1997;**7**:128–137.
171. Murphy CJ, Zadnik K, Mannis MJ. Myopia and refractive error in dogs. *Invest Ophthalmol Vis Sci* 1992;**33**:2459–2463.
172. McMullen RJ, Davidson MG, Gilger BC. The effect of 1% tropicamide-induced mydriasis and cycloplegia on spherical refraction of the adult horse. *Vet Ophthalmol* 2014;**17**:120–125.

173. Miller P. A limited review of feline and equine vision. *Proceedings of the American College of Veterinary Ophthalmologists Conference*, 1996.
174. McMullen R. Refractive state of the normal equine eye. Personal communication, 2009.
175. Grinninger P, Skalicky M, Nell B. Evaluation of healthy equine eyes by use of retinoscopy, keratometry, and ultrasonographic biometry. *Am J Vet Res* 2010;**71**:677–681.
176. Bracun A, Ellis AD, Hall C. A retinoscopic survey of 333 horses and ponies in the UK. *Vet Ophthalmol* 2014;**17** Suppl 1:90–96.
177. McMullen R, Davidson M, Salmon JH, Campbell N, Gilger BC. In vitro and in vivo evaluation of 30 D and 25 D equine intraocular lens implant. *Am J Vet Res* 2010;**71**:809–816.
178. Millichamp N, Dziezyc J. *Cataract Surgery in Horses*. Fort Lauderdale: Association for Research in Vision and Ophthalmology; 1996:S763.
179. Townsend WM, Jacobi S, Bartoe JT. Phacoemulsification and implantation of foldable +14 diopter intraocular lenses in five mature horses. *Equine Vet J* 2012;**44**:238–243.
180. Harrington JT, McMullen RJ, Clode AB, et al. Phacoemulsification and +14 diopter intraocular lens placement in a Saddlebred foal. *Vet Ophthalmol* 2013;**16**:140–148.