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Beginning to See the Light: Pediatric Equine Ophthalmology

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Take Home Message

Most eye problems that present in the first months of life fit into one of three scenarios:
1. Congenital issues that are developmental or acquired during parturition
2. Ocular manifestations of the systemic infectious or non-infectious diseases common to very young horses
3. Traumatic injury

The presentation gives information on recognizing ocular abnormalities that are present at birth and that develop secondary to pediatric illnesses. It gives advice on practical management of ocular trauma commonly encountered in the first year of life.

Ophthalmologic Exam of the Neonate

All foals should receive a full physical examination in the immediate postpartum period. Most practitioners see foals that are nursing normally sometime between 12 and 36 hours of age. This time interval is long enough to assure that colostral ingestion and absorption has occurred so testing for failure of passive transfer of maternal antibodies will be accurate, but also early enough to diagnose common neonatal conditions and intervene if necessary. The post foaling exam involves a physical examination of the dam, the expelled placenta and the foal. During the exam, the foal’s vital signs are measured and the baby is thoroughly scrutinized for congenital conditions such as umbilical or scrotal hernias, angular limb deformities, heart murmurs and bite problems. Ophthalmic examination is a very important part of the process and should be done in a consistent, thorough manner.

A bright penlight or transilluminator as well as a direct ophthalmoscope should be brought into the stall at each neonatal exam. Cooperation is optimized if one person holds the mare and another person holds the foal near the mare’s head. If two assistants are not available, most mares can be turned loose and are usually well behaved as long as they can see the foal directly in front of them. Excessive foal restraint is inappropriate—eye exams are best performed with the foal simply cradled by a calm helper. Rambunctious individuals can be restrained by a firm arm around the chest and a tail hold; sometimes it is helpful to position the foal in a corner or against the stall wall.

The ophthalmic exam begins with a general assessment of facial symmetry and globe position. A transilluminator or penlight examination of the orbit, lids and anterior segment follows. This author cradles the foal’s ventral mandible with one hand and uses the other to hold the ophthalmoscope. All ocular structures are evaluated systematically, with a conscious “check list”...
of layers and regions: Lids, conjunctiva, cornea, anterior chamber, iris and lens. A bright beam is
directed towards the temporal fundus to assess pupillary light reflex (which may be minimal in
neonates). Then the head of the scope is changed to the direct ophthalmoscope. Most newborn
foals are remarkably tolerant of a bright light fundic exam. If the holder is calm and the examiner
can hold the head steady with the non-examining hand, foals often “freeze” their head
movements when the light beam engages the optic disc region. This hypnotic reaction allows a
good screening inspection of the peripapillary area. During the entire examination, the
veterinarian should be keeping a mental score card of findings of the different regions so that the
neonatal exam record captures all findings. Common comments might include notation of
episcleral or retinal hemorrhage, a perfused remnant of the hyaloid artery or entropion of the lids.
If the foal is a member of a breed that is at high risk for eye problems (e.g. Appaloosa, Rocky
Mountain Horse) the neonatal exam is a great opportunity for educating the owner about known
risks that might require screening later in life. If any congenital anomalies have been observed in
the foal, the mare should get a thorough eye exam to see if she has any similar problems. After
the inspection of the eyes is complete the foal should be turned loose and observed. Visual foals
will demonstrate curiosity about their environment and will navigate with confidence. Although
they may be a little clumsy they should not hit walls and should be able to locate their dam’s
udders adeptly.

The neonatal pupil is somewhat round in contour. The globe has a mild ventromedial orientation
at birth that gives neonates a slight downward gaze; the pupil acquires the familiar horizontal
elliptical shape and becomes parallel to the lower lid over the first month of life. Pupillary light
response may be sluggish in the first few days of life but should be brisk after a few days. The
color of the neonatal iris may be a little grayer than the rich chocolate color commonly seen in
adults. Lens suture lines may be very prominent and should not be mistaken for cataracts. The
vitreous should be clear and allow sharp visualization of the fundic structures. Tapetal color is
variable and is correlated with coat color. The most common color is a yellow green but other
colors like blue or green are seen as well. Light colored individuals may have subalbinotic
tapetums that are very pale, revealing the underlying choroidal vasculature. The optic disc is
round to oval in shape and whitish pink to salmon in color. Light gray streaks, representing
bundles of axons traveling to the optic nerve, may be seen in the peripapillary area. Retinal
vessels are small.

Ophthalmic Conditions That May be Present in Neonates

1. Subconjunctival hemorrhage, which is often referred to as episcleral hemorrhage manifests
as patchy streaks of red blood evident on top of the sclera near the limbus. It is a frequent finding
in newborn foals, indicating some bleeding of the vessels that overlay the sclera. The cause of
the bleeding is likely excessive pressure on the conjunctival vessels during the strains of
parturition. The red tinge to the sclera may be alarming to the owner, so the condition should be
pointed out and discussed. It is usually of no clinical significance and will disappear without
treatment within ten days.

2. Entropion, or turning in of the eyelid margin so the haired skin rests against the cornea is a
very common finding in neonates. The author’s practice treats this condition in about 1–2% of
the annual foal crop. Entropion is sometimes seen in normal newborn foals, but it also can occur
as a result of prematurity, dysmaturity or neonatal illness that is accompanied by dehydration or loss of periorbital fat. Any foals that are ill or under veterinary supervision should have the position of the lower eyelid margin checked at least twice a day. It is important that the examiner always takes time to consciously identify both shiny brown palpebral margins. The condition is easy to miss as early on the lid skin is thin enough that the doubling of the thickness near the globe may not be obvious and affected foals may not show any ocular discomfort. Absence of this margin suggests entropion, and the condition can be confirmed by manually everting the turned in tissue. If entropion is identified, the corneas should be stained with fluorescein dye; if ulcers are present appropriate treatment should be initiated.

The author prefers to treat entropion with temporary everting sutures that “baste” the loose lower eyelid skin in a folded contour for a few weeks while the periorbital tissue matures and hydrates. This procedure is simple and quickly performed, but it must be done with the foal in a recumbent position. **CAUTION:** Sedatives must be administered with care as neonatal foals are quite sensitive to the cardiovascular effects of these potent drugs, especially if they are ill. The author advises that the veterinarian kneel below the foal’s neck when inserting the sutures and that an assistant be positioned above the foals head to hold the head still as needles are used near the globe. The “down eye” should be laid on a towel so that the bedding material does not cause a problem. A bleb of 1 ml of local anesthetic should be infiltrated into the subcutaneous lower lid space with a 25 or 23 G needle. Three to four sutures of 4-1 to 1-0 gauge should be placed in the lid well away from the tarsal margin in a vertical mattress pattern that is perpendicular to the lid margin. The sutures are easiest to place in the field if a short (5 1/2”) pair of Olsen Hegar needle holders are used. Closure of these sutures should fold the skin up in a pinched pattern so the lid margin stays everted, but the lids are still fully able to blink and close. No skin should be cut. Care should be taken to place and cut the tags so the sutures have no chance of abrading the corneas. The condition is usually bilateral, so the foal should be flipped over after the first eye is done to repair the other side, with the surgeon and assistant reversing places so the assistant is again above the foal’s head. The author performs the procedure in both eyes on all foals that are sick even if it only presents unilaterally, as the second eye has a strong probability of developing entropion and the stress to the foal is minimized if preventative measures are taken at the time the initial procedure is done. The sutures should be removed in a few weeks—this can usually be done in the standing foal with light sedation. There are no adverse cosmetic effects from the temporary eversion of the lid.

Some practitioners treat mild entropion by injecting a small bleb of a viscous drug like procaine penicillin in the subcutaneous space below the lower lid to effect eversion. The author prefers to use sutures because they are guaranteed to be effective as long as the sutures stay in place.

3. Atresia of the Nasolacrimal system can occur but is rarely diagnosed in a neonate as signs of dacryocystitis are very mild in the first few weeks of life. Weanlings or yearlings that present with unilateral or bilateral tearing should have the patency of the nasolacrimal system checked with fluorescein dye. If the nasal opening does not discharge dye then retrograde flushing should be attempted. If the tear drainage system is not operating, further imaging can be done including dacryorhinocystography (radiology of the head with dye infused into any available openings of the system). If abnormalities are detected, referral is indicated for corrective surgery which may
involve opening up the atretic puncta and/or cannulating atretic parts of the system and suturing the cannula in place until a patent duct is present.

4. **Microphthalmos** is a condition where a foal is born with one or two abnormally small globes. In utero, the formation of mammalian eyes is a complex staging of cellular migration involving elegant folding of primarily neural crest tissue in origami like patterns. Problems with genetic coding or toxic influences can alter the intricate development of the globes prior to birth. Some foals with mild microphthalmos are visual and do not have other abnormalities. Other foals have additional ocular anomalies like cataracts or retinal defects; these individuals may be blind. Very rarely, foals are born with tiny, pea like, heavily pigmented stubs of globe tissue that are completely non-functional. There is no treatment for microphthalmos.

5. **Strabismus** is defined as a deviation of the globe. All foals are born with a degree of strabismus—at birth the globe is deviated in a slight ventromedial direction so that the orientation of the pupil is not quite parallel to the lower lid. This physiologic strabismus resolves by about one month of age. However, some foals have a true strabismus that does not improve with maturation. Appaloosas with congenital stationary night blindness may demonstrate hyperopia (globes that deviate upward) or a dorsomedial deviation. Saddlebreds sometimes present with esotropia (crossed eyes). Weanlings with persistent severe strabismus can be referred to veterinary ophthalmologists for evaluation as some cases are amenable to surgical correction using techniques that alter the extraocular muscle insertion.

6. **Dermoids** are benign tumors that are composed of tissue elements not normally found in the area in question. Also called choristomas, they are random anomalies of unknown cause. Congenital corneal, nictitans, conjunctival or eyelid dermoids are occasionally seen in foals. Corneal dermoids appear as masses on the surface of the cornea adjacent to the limbus. Pigmentation is variable and some lesions produce hair or mineralized elements that may irritate adjacent tissue. Foals with conjunctival or corneal dermoids should be referred to a veterinary ophthalmologist for surgical excision. Keratectomy is generally curative, but must be done precisely under general anesthesia with proper instruments and magnification to reduce the risk of perforation or other complications.

7. **Aniridia** is a rare condition where only a rudimentary rim of uveal tissue is present anterior to the lens. Affected foals will show photophobia as they develop, and will appear to have very dark eyes due to the large pupils. No pupil constriction occurs following bright light stimulation, and the corpora nigra are absent. The corneas of these foals may show vascularization and small nodular limbal masses that project very tiny hairs. The zonular processes that suspend the lens are visible through the large pupil. Some aniridic individuals have cataracts that are usually slowly progressive. The condition has no treatment and is thought to be inherited, so affected individuals should not be bred.

8. **Heterochromia iridis** is the term that describes irises that are white or blue or mottled in color. Most foals are born with brown colored irises and dark brown corpora nigra. Heterochromia iridis is common in horses that are paint, spotted, gray, Appaloosa, palomino or chestnut. It is rare in horses that are bay or dark in color. This condition is not pathologic. It is often accompanied by **iridal hypoplasia** or regional thinning of the iris stroma. The weakened
tissue may take on a bulging or wrinkled appearance. A suspicious patch of iris hypoplasia can be studied by administering a mydriatic (thin stroma will wrinkle as the pupil dilates) or by transilluminating with a bright light (the inner darker pigmented layer of iris epithelium will be revealed and often it has a lace like translucency). These simple stall side tests will generally confirm that the region in question is simply thin iris, not iris melanoma, but if there is still any uncertainty, ultrasound imaging of the globe can be done with a microconvex tendon probe. No treatment is indicated. Occasionally there are developmental issues where the optic fissure fails to close and multiple or irregular pupils are present. This condition is called iridal colobomata. Affected eyes can have a bizarre appearance.

9. Persistent Pupillary Membranes (PPMs) are remnants of the tunica vascularis lentis, an embryonic structure. They show up as thin strand of iris tissue. Most often they are web like strands that simply bridge iris stroma, but sometimes the strands extend from the iris to the corneal endothelium or from the iris to the anterior lens capsule. Focal opacity on the cornea or the lens can result from the latter two PPMS. No treatment is indicated.

10. Lens suture lines are visible to a variable extent on both the anterior and posterior axial cortex of foals and are a normal anatomic feature. The anterior pattern is usually in the shape of a Y while the posterior pattern is more variable, often appearing as a sawhorse shape or inverted Y. The suture lines represent the junction of the tips of lens fibers that have grown inwards from the equator. They persist for life. Presence of a very few faint, dust like “dots” in the lens cortex is not cause for concern as these are usually tiny lens imperfections that do not progress.

11. Cataracts are the most common ocular anomaly that has been reported in foals. They are usually bilateral but can be unilateral and may be focal or diffuse. They may be the only ocular abnormality present or they may be associated with many of the other congenital problems described in this paper. If identified, they should be described in terms of location, shape and extent. Descriptive terms of focal opacities in order of anatomic position are: Anterior (in the anterior third of the lens disc), Equatorial, (out near the rim of the lens disc in the peripheral cortex) Nuclear (in the center third of the lens disc associated with the physical nucleus), Lamellar (on the anterior interface between the nucleus and the cortex) and Posterior (within the posterior third of the lens disc). Anterior and posterior cataracts should be further specified as capsular (on the surface of the lens capsule), subcapsular (adjacent to the lens capsule but in the outer cortex) or cortical (in between the capsule and the nucleus), additional terms such as “floriform”, “spoke like”, “punctate”, “cuneiform”, “triangular”, “web like”, etc may be applied to describe the gross appearance of the opacity. If a cataract is described as “incipient” it means that less than 10% of the lens volume is affected. If a cataract is described as “mature” it means it involves the entire lens structure and that no fundic reflection can be seen. Axial cataracts are those that are positioned along the visual axis (center axis) of the lens disc.

Most cataracts that have significant visual implications can be imaged reasonably well with basic digital photography, and it is advisable to capture pictures of the defect(s) for baseline records. Application of tropicamide for mydriasis will enhance the definition of the extent of the cataract as well as screen for synechia. The literature has several reports of cataracts in certain genetic lines of a variety of breeds that suggest an autosomal dominant mode of inheritance, it is difficult
to give a prognosis for progression with certainty, but focal cataracts that are located in the nuclear region, or associated with the lens suture lines are the least likely to progress.

**Cataract surgery** is a treatment option for diffuse congenital cataracts that cause visual problems. The success rate is about 80% in experienced hands, and success is highest if the foals are operated before they reach 6 months of age. The surgery is done by phacoemulsification, a technique that uses ultra high frequency ultrasound to break up the lens material then suck it out of the capsule with a tiny needle vacuum tube. The surgery should be done under magnification and is best done by an ophthalmologist who has a lot of general experience with horses but also a lot of experience with cataract surgery in small animals. The best candidates are foals that can be handled for frequent post operative treatment with owners that are dedicated to thorough aftercare. The foal must be prescreened for uveitis as intraocular inflammation increases the risk of postoperative complications. Other screening includes verification of an intact pupillary light response and a thorough inspection of the anterior segment. The ophthalmic surgeon might also choose ultrasound imaging to check for retinal detachment and/or administer an ERG to ascertain retinal function, especially if the foal is an Appaloosa. Foals are expected to be far sighted after recovery as intraocular lenses are not commonly inserted into the capsular bag. Operated foals who have recovered without major complications are reported to perform as if their vision is functionally normal.

**12. Lens Luxation** occasionally occurs as a congenital problem if the zonular fibers that normally anchor the lens at the equator do not form properly. The disrupted lens generally opacifies and looks like a rounded quartz disc that is either lying on its convex surface behind the iris in the posterior chamber, or resting in an upright position in the ventral anterior chamber. Posterior lens luxation is not treated. Anterior lens luxation is a concern as it may be associated with pain or glaucoma as the disc mechanically stresses the ventral anterior chamber drainage apparatus.

**13. Hyaloid Artery remnants** are commonly visualized on direct ophthalmoscope exams of neonates. The artery is perfused in a large percentage of horses for a few days after birth and can be seen as a thin red linear structure that bows slightly in the vitreous as it travels from the optic disc area to the center of the back of the lens. These remnants are of no clinical consequence and usually disappear by a week of age. Occasionally, a remnant of the hyaloid artery apparatus or tunica vasculosa lentis (an embryonic membrane that once covered the back of the lens but normally disappears prior to birth) can be seen as a white spot or a series of white linear streaks on the posterior capsule of the lens. This finding can be associated with eventual development of posterior polar cataracts, so these foals should be followed with frequent eye exams.

**14. Coloboma** is a general term that describes defects in the normal ocular tissue, and as such the term can be used to describe congenital defects in any of the ocular structures. **Posterior segment colobomas** are areas that appear as well circumscribed oval, round or irregularly rounded patches of abnormally pale pigmentation in the fundus. They are uncommon, but not rare. They are found most often in the peripapillary non-tapetal region, but they can be noted anywhere in the posterior segment. The margins of colobomas are crisply defined, and careful ophthalmoscopy may reveal slight cupping of their contour below the surrounding normal tissue. They represent areas where there is either no choroid (white in color), minimal choroidal
vasculature (orange white in color) or no retinal pigment epithelium (RPE)(pale gray in color). The latter are the most common anomalies and are also called “window defects”. Window defects of the RPE can be distinguished from chorioretinal scars by the bland, uniform appearance of their depigmentation—in contrast, scars have a hyperpigmented center surrounded by a paler ring of depigmentation and are less smooth at the margins. Window defects of the RPE have no apparent effect on vision and are not thought to be a contraindication for breeding. Large white colobomas above the disc have the most potential to disturb visual function. The accompanying structural cup may be further characterized by ultrasound. Horses with large white or orange white colobomas near the area centralis should not be bred. As fundic exams of excitable neonates are rarely comprehensive, subtle colobomas may be missed. It is a good idea to give all young horses a complete fundic examination when they are mature enough to stand quietly for the procedure. The significance of a coloboma that is noted on a prepurchase exam of a mature horse is easier to interpret if the finding has been documented in the individual at a very young age.

15. Optic nerve hypoplasia is a rare congenital abnormality. Some cases are developmental but others may follow nerve atrophy associated with in utero inflammation. The affected nerve(s) will appear pale and small. Retinal vessels will be absent or hard to see. Affected individuals will have dilated pupils and may be blind or have reduced vision. Nystagmus may be present. No treatment is available.

16. Retinal hemorrhages are a common incidental finding in neonates. They may be punctate or appear as small splash or brush shaped red areas, and will be easiest to see in the tapetal region. They are probably associated with the hypertension during parturition and usually disappear in the first two weeks of life.

17. Retinal dysplasia or congenital retinal detachment are occasionally found in neonates. Dysplasia presents as gray or hyperpigmented streaks or patches of visible fundus tissue. Histologically, the neurosensory retina has been shown to be folded in a rosette or creased pattern in the regions of the dysplasia. Dysplasia may be developmental, or the result of infection in utero. A complete retinal detachment will appear as a floating veil of tissue that resembles wet toilet paper and seems “draped” over the optic disc. The tapetum above the detached tissue may appear hyper-reflective. The affected eye will be blind and may have nystagmus. Other ocular anomalies may be present with both conditions. No treatment is available for retinal dysplasia or detachment.

18. Anterior Segment Dysgenesis (ASD) is a syndrome where several ocular structures fail to develop normally. The syndrome is associated with the silver dapple gene which produces horses that have a chocolate coat color and flaxen mane and tail. ASD has been best described in Rocky Mountain Horses but has also been seen in Miniature horses, Shetland ponies, Kentucky Mountain Saddle Horses, Morgans, Bashkir-Curlies, Narragansett Pacers and Haflingers. The syndrome is inherited as a recessive trait with incomplete penetrance. Some affected horses present with relatively mild ocular abnormalities such as hypoplasia of the corpora nigrans, persistent papillary membranes and iris or ciliary body cysts. These individuals may not show visual challenges and are thought to be heterozygous for the condition. However, other affected horses present with multiple major ocular abnormalities including congenital microphthalmia,
miosis that is nonresponsive to mydriatics, very deep anterior chamber, macrocornea, ciliary body and retinal cysts, cataract, lens luxation and retinal dysplasia or detachment. These individuals may be blind or have severe visual challenges and are thought to be homozygous for the condition.

Rocky Mountain Horses, and other horses with chocolate coats and flaxen manes and tails should receive careful scrutiny for ASD in their early months of life, and then be checked annually. Warning signs that would be visible with a penlight would include abnormally small pupils, thin strands of tissue bridging sections of the iris or projecting into the anterior chamber (persistent pupillary membranes), bizarre translucent cysts apparent just behind the iris, lens luxation and cataract. Warning signs that would be apparent on direct or indirect fundic ophthalmoscopy include a reticulated pattern of depigmentation or multiple pigmented, curvilinear streaks that extend toward the optic disc (suggesting retinal dysplasia), or a fuzzy fundic image with a hyper reflective tapetum (suggesting retinal detachment). Suspected retinal detachments can be confirmed by ultrasounding the globe with a 7.5 MHZ transducer either placed on the lid or directly on the cornea. There is no therapy for ASD; affected individuals should not be bred and owners should be cautioned that horses with multiple ocular abnormalities as described may be unsafe or unpredictable in their behavior.

19. Congenital Stationary Night blindness (CSNB) has been documented in many lines of Appaloosas and in some Quarter Horses and other breeds. CSNB is a functional problem in neural transmission between the photoreceptor layers. The condition cannot be diagnosed with a standard ophthalmic examination as there are no associated fundic or anterior segment findings. However, some affected individuals show a star gazing type dorso medial strabismus that may become more apparent as a foal ages. Owners of Appaloosa foals, or QH foals with a history of parental CSNB should be counseled about the condition. Electroretinography can be performed in older individuals to test retinal function in varying light conditions. There is no treatment, and owners should be aware that affected individuals will be greatly reduced in dim light and may have suboptimal acuity even in bright light. The condition is thought to have a hereditary component.

20. Congenital glaucoma is a rare condition that follows goniodysgenesis (abnormal development of the iridocorneal angle that drains aqueous humor) or perinatal uveitis associated with systemic infection. The most prominent sign is generalized corneal edema that gives the cornea a distinctive blue cast. Other signs may include large, fixed pupils, and linear white bands that cross the cornea that represent pressure induced stretching of Descemet’s membrane. The eye will feel tense and tonometry will show elevated intraocular pressure (normal pressure is 17-28mm Hg). Cases can be referred to a veterinary ophthalmologist for evaluation of medical and surgical treatment options, but prognosis for equine glaucoma is guarded. Medical options include topical administration of beta blockers (Timolol Maleate 0.5%) and carbonic anhydrase inhibitors (Dorzolomide), or combinations of the two drugs (Cosopt 0.1%) to lower intraocular pressure. Surgical options include ablation of the ciliary body using freeze therapy or laser photocoagulation, or the implantation of some kind of a shunt system to drain off excess fluid in the aqueous.
Pediatric Ocular Manifestations of Infectious Systemic Diseases

Many infectious conditions that affect horses have ocular manifestations. Those listed here are the infections that are most often diagnosed in foals.

1. **Neonatal Septicemia** is a common complication of prematurity, dysmaturity or foals that experience placental insufficiency or failure of passive transfer. The clinical signs that accompany septicemia are varied and often progress rapidly—affected foals may become severely ill, losing the ability to nurse or stand. The infectious agent(s) involved include a broad number of gram positive and gram negative bacteria: *Salmonella*, *E. coli*, *Streptococci* and *Actinobacillus* are common isolates. The bacteria spread hematogenously and can seed many organs including the eye. The ocular response is the development of uveitis (also referred to as iridocyclitis). The uveitis may be immune mediated (sterile) or infectious (endophthalmitis). Foals with uveitis will present with a green hue to the iris and a very cloudy posterior segment. The green hue represents the influx of inflammatory cells, fibrin and debris through choroidal and iris vessels into the chambers of the eye. White swirls or clots of fibrin resembling partially cooked egg whites may be seen in the anterior chamber and may occlude the pupil. The anterior segment may be very inflamed and may show conjunctival hyperemia, corneal edema and/or deep vascularization and severe miosis. Examination of the chambers of the eye may reveal hypopyon, (purulent material in the anterior chamber) or hyphema (blood in the anterior chamber). The eye may show hypotony (soft eye due to low intraocular pressure). The vitreous may have so much haze and inflammatory debris that the fundic image is not visible.

Systemic treatment of neonatal septicemia should be dictated by the results of blood culture and includes intense supportive care in addition to broad spectrum antibiotic therapy. Treatment of the ocular manifestations will depend on the exact findings, but the condition is potentially blinding and therapy must be aggressive. All eyes with uveitis should be stained with fluorescein dye to check for corneal ulcers. If ulcers are present, they must be treated appropriately (see section below on ocular trauma). If the cornea is intact (no ulcers present) treatment must focus on controlling the intraocular infection and inflammation and saving the pupil. Treatment may include:

a. **Mydriatics:** 1% Atropine is used as the initial mydriatic agent. Some caution on frequency is advised as gastrointestinal tract (GIT) stasis can be a side effect; initial therapy is usually limited to QID then reduced as soon as dilation occurs. GIT motility and manure production should be monitored. If atropine fails to dilate the pupil, 2.5% phenylephrine drops can be added.

b. **Broad spectrum antibiotic with good corneal penetration.** Chloramphenicol and triple antibiotic preparations are good choices. Chloramphenicol is available in ointment form; triple antibiotic preparations are available in ointments or drops.

c. **Topical steroid:** 1% Prednisolone Acetate is the most potent topical steroid, and may be applied in drop form 4x to 6x per day. 0.1% Dexamethasone is also a good choice and is readily available in ointment form. Preparations that include hydrocortisone are poor choices for treating uveitis as they do not penetrate the cornea well.

d. **Topical nonsteroidal:** the use of topical NSAIDS is usually reserved for foals with uveitis complicated by ulceration. Application of 0.1% diclofenac (Voltaren) drops up to QID may be helpful.
e. **Topical osmotic agent**: 5% Na CL (Muro 128) can help clear corneal edema. It is available over the counter as an ointment or drop.

f. **Broad spectrum systemic antibiotics** are the mainstay of therapy of septicemia. Some of these drugs may cross the blood ocular barrier as this barrier is likely compromised in foals with uveitis.

g. **Systemic nonsteroidal agent**: Flunixin meglumine at a dose of 0.5 mg/kg SID or BID is the most effective NSAID to date for uveitis. It carries a risk of gastric ulceration and renal toxicity so foals should receive concurrent gastroprotectants (Omeprazole at 4mg/kg SID) and have their renal function monitored. No studies have been released to date that measure the efficacy of the new Cox 2 inhibitor firocoxib (Equioxx), but this drug may merit investigation.

h. **Topical anticollagenase therapy** using serum, and possibly 0.17% EDTA and/or 5-10% acetylcysteine should be instituted in any foal that has concurrent ulceration (see section below on corneal ulceration).

Foals that are on an intensive regime of therapy may need to have a **subpalpebral lavage tube (SPL)** in place to facilitate frequent application of topical therapy. There is a section below with comments on SPL usage in foals.

If the foal has a large fibrin clot in one or both anterior chambers, injection of **Tissue Plasminogen Activator (TPA)** into the aqueous may be extremely beneficial. Dr. Dennis Brooks at the University of Florida has a supply of frozen individual 100 microgram (0.2 ml) doses of TPA that he will sell to practitioners at a very reasonable cost. The injection technique is simple but must be done with adequate chemical and physical restraint and delicate technique. The foal should be sedated carefully or given short term general anesthesia and placed in lateral recumbency with the target eye up. It is helpful if several assistants are present to aid with physical restraint as the head must be perfectly still, and the lid will have to be raised to provide access to the limbus. An auriculopalpebral and supraorbital nerve block are performed. The chosen target spot on the limbus is numbed by application of tetracaine or proparacaine on a Q tip. The same region of the limbus is prepped by swabbing it with Q tips soaked in a solution of 2.5% betadine (0.5 ml of Betadyne solution mixed with 20 ml of sterile saline). A 27 G needle is placed on the tuberculin syringe that contains the TPA and the arm of the needle is bent at about a 30 degree angle to facilitate entry into the anterior chamber. The limbus is entered with the 27G needle attached to the TPA syringe and great care is taken to avoid puncturing the iris. The 0.2 ml dose of TPA (ideal dose is 50 to 150 micrograms of TPA per eye) is injected in to the anterior chamber and the needle is immediately withdrawn. Resolution of the fibrin clot is usually dramatic and rapid.

**NOTE!** TPA should NOT be used if intraocular hemorrhage has occurred in the previous 48 hours. However, it can be effective when injected up to two weeks AFTER the episode of hemorrhage.

Clients who own foals with septicemia complicated by uveitis should receive careful counseling. The septicemia itself is life threatening. However, the uveitis is also sight threatening and foals that survive septicemia but end up with a lot of intraocular scarring may have some visual
2. **Rhodococcus Equi** is a pyogranulomatous disease caused by infection with the gram positive pleomorphic bacteria of the same name. Infection is thought to occur soon after birth, but clinical disease rarely manifests before several months of age. The disease may be endemic on some farms, especially those with dirt pastures. The organism causes large multifocal abscesses in the lung and sometimes the abdominal cavity. Clinical signs include fever, severe neutrophilic leukocytosis and elevated fibrinogen levels, cough, weight loss and dyspnea. Foals with abdominal abscesses may show diarrhea or peritonitis. The disease is sometimes accompanied by an immune mediated polyarthropathy with pronounced joint effusion.

Ocular signs of *R. Equi* may include hyphema and marked uveitis. Foals may or may not show signs of photophobia or ocular pain but if uveitis is present variable degrees of corneal edema, aqueous flare, hypopyon, miosis, and vitritis will be found on ocular exam. Exudation of fibrin into the anterior chamber may be dramatic and require treatment with TPA (see previous section on septicemia). Treatment of the uveitis is as described above for neonatal septicemia. In some cases the systemic disease may be cured but the associated uveitis may leave permanent changes in the eyes and some compromise of vision.

Treatment of *R. Equi* requires expensive combination antibiotic therapy as well as supportive care. Foals are usually treated with either azithromycin, clarithromycin or erythromycin combined with rifampin for several weeks. Mortality from the disease is a significant source of foal loss on some endemic farms.

3. **Septic meningoencephalitis** can follow hematogenous spread of numerous neonatal infectious agents. Clinical signs include stiffness of the neck, photophobia and hyperesthesia. Progression may be rapid and ocular signs may include blindness, nystagmus, elevated nictitans, cranial nerve deficits, coma, seizures and death. Treatment may be difficult due to the blood brain barrier.

4. **Botulism** or “**shaker foal syndrome**” is a disease caused by a neurotoxin produced by the anaerobic bacteria *Clostridium botulinum*. The condition has been reported frequently in central Kentucky and many foals born in this region undergo vaccination for the disease. The prevailing signs are weakness, recumbency and muscle tremors. Ocular examination may reveal neurotoxic influence on the muscles in and around the eye, manifested as enophthalmos from retractor bulbi spasm, upper lid ptosis, mydriasis and sluggish papillary light responses. The disease has a high mortality rate. Treatment revolves around supportive care and administration of botulinum antitoxin.

5. **Adenovirus infections** can invade the respiratory tracts of foals with **combined immunodeficiency** or the gastrointestinal tract of foals with **rotavirus diarrhea**. If the individual is immunocompromised, the virus can cause a profound panuveitis. It also causes necrosis of conjunctival epithelium with characteristic intranuclear inclusions in the cells.
6. **West Nile Virus** has caused many cases of encephalitis in horses and foals in the United States since the first cases were seen in New York in 2001. The virus is spread by mosquitoes. Fever, muscle fasciculations, weakness and ataxia are the most common systemic signs. Ocular signs may include facial nerve paralysis or other cranial nerve deficits and sudden blindness. Diagnosis is by IgM capture of serum or CSF and titer serology. Treatment is mainly supportive and the mortality rate is about 30%.

7. **Strangles** is an infection of the upper respiratory tract and lymph nodes around the head with the gram positive cocci *Streptococcus equi*, var. *equi*. Presenting clinical signs in foals and horses usually include high fever, nasal discharge and swelling/abscessation of the submandibular and/or retropharyngeal lymph nodes. Affected individuals usually show a mucopurulent ocular discharge with conjunctival hyperemia. Occasionally horses or foals with strangles will present with panophthalmitis and chorioretinitis. Treatment of strangles is controversial: some cases are managed with long term administration of antibiotics and some are managed without antibiotics. Complications can include later development of purpura hemorrhagica as well as development of a carrier state with organisms periodically shed from the gulleral pouch. A fundic examination should be scheduled after the horse recovers as many horses will exhibit permanent chorioretinal scarring as a footprint of chorioretinitis associated with the streptococcal infection.

**Pediatric Ocular Manifestations of Non-Infectious Disease**

1. **Neonatal isoerythrolysis** is a disease seen only in young foals who nurse or are fed colostrum that contains antibodies against the foal’s red blood cells. Clinical signs may develop within 12 to 36 hours after nursing and include weakness, lethargy, poor suckling, tachypnea and tachycardia. The signs are all related to the hemolytic anemia which can be life threatening. Ocular signs include pale conjunctival membranes that progresses to icterus. Intense scleral icterus may be a key symptom that aids in the diagnosis of the condition. In some cases, conjunctival, episcleral and intraocular hemorrhages occur. Treatment revolves around blood transfusions and supportive care. Subsequent foals from the same mare should be muzzled at birth and tube fed colostrums from a source that does not have alloantibodies to the foal’s red blood cells.

2. **Hypoxic ischemic encephalopathy (HIE)** may follow dystocia, red bag delivery or placental insufficiency. Affected foals may require extensive supportive care as they may be unable to nurse or stand. Ocular manifestations may include small retinal hemorrhages, optic disc hemorrhages, corneal edema and pupil asymmetry. The condition may predispose to septicemia (see above).

3. **Vitiligo** is a skin condition that affects more areas than the eye. Also known as “Juvenile Arabian Leukoderma” or “Arabian Pinky syndrome”, vitiligo is a skin disease where focal areas of the body become depigmented in a mottled pattern. The eyelids are often markedly affected. The condition is seen in many gray Arabian horses, and has also been reported in Andalusians and Saddlebreds. The depigmentation can start as early as 6 months of age but also has been reported as late as 24 months. It may become more pronounced in pregnancy in mature affected individuals. It is thought to have both an autoimmune and a hereditary basis.
Pediatric Eye Trauma

1. Periorbital trauma can occur if a foal struggles after getting its head trapped in a confined space like a trailer or slotted hay feeder. Orbital trauma can also result from general roughhousing when foals play together or are kicked by older horses. Both self induced and external trauma can result in sinus fractures, orbital fractures, cranial nerve damage and blunt trauma to the globes. Sinus and orbital fractures place the foal at risk for septic meningoencephalitis and orbital infection as well as permanent strabismus if the orbital cone heals with a contour that deviates the position of the globe or the action of the extraocular muscles. Sometimes referral for surgery is appropriate if orbital or sinus fracture has occurred. Blunt trauma to the globes can be blinding, especially if intraocular hemorrhage or ocular rupture occurs.

All foals who have suffered head trauma should be evaluated immediately and treated aggressively. Sinus fractures should be treated as open fractures even if the skin is intact, as organisms may gain access to the sinus spaces from the nasal passages; long term antibiotic therapy is appropriate. Traumatic uveitis and physical ocular damage should be treated aggressively with mydriatics and anti-inflammatories. The owner should be warned that blunt trauma can cause rupture of the lens capsule with release of lens proteins into the eye. As these proteins are usually not subject to immune surveillance, release of lens material can trigger an autoimmune reaction with cataract development as a later complication. Follow up on orbital and periorbital trauma should be thorough to assure inflammation has been treated and long term sequellae like strabismus or cataract have been documented.

2. Concussive head and neck trauma can result from training accidents or collisions, especially if the foal rears and falls over backwards. Bleeding from the ear canals is a poor prognostic sign and may precede sudden death. Sequellae can include long term CNS problems if traumatic brain injury or cervical fracture occurs. First aid should center around intense supportive care and anti-inflammatory therapy, but if major damage has occurred to the optic chiasm or visual processing centers, bilateral blindness may result.

3. Eyelid lacerations may occur if the lid margin becomes hooked on some kind of stationary object and the foal pulls away. A common source of this trauma is the J shaped end of bucket handles that are usually abundant in the foal’s environment. Practitioners should counsel owners to tape these J hooks shut with duct tape or electrical tape as a preventative measure.

Eyelid lacerations can heal well with prompt, careful surgical attention. Important surgical principles include minimal debridement, careful apposition of the torn pedicle, use of 4-0 absorbable suture for all layers of the repair and closure of the tarsal plate using a figure of eight suture pattern that keeps suture tags from abrading the cornea. Repair under general anesthesia is optimal but often not available in the field.

A few “practice tips” for eyelid repair in the field: If the repair must be done standing, it is very helpful to create a “surgery table” by stacking two or three bales of hay or shavings on top of each other, covering them with a clean towel or blanket, and resting the sedated foal’s chin on
the top bale. Administering accurate sedation to a foal or weanling may be tricky: be prepared that the foal may not remain standing if the dose is excessive! Sometimes it is helpful to create additional support for the sedated foal by pushing a bale under the belly and/or making sure there is a wall for the baby to lean against. Ophthalmic instruments may prove too delicate for field use, but the use of a very short (5 1/2") Olsen Hegar needle holder and a small pair of forceps for the repair is handy. Be careful if a two-layer closure is needed—the inner layer of sutures MUST not abrade the cornea. Single layer closures with meticulous deep bites are sometimes the safest repairs to attempt in the field. Temporary tarsorrhaphies may stabilize the tarsal plate in severe tears. The globe should be checked for uveitis and the cornea should be stained to screen for ulcers. Systemic and topical antibiotics as well as the judicious use of NSAIDs will aid healing. Even though the material is absorbable, 4-0 suture knots that are used to close skin should be removed in 10-14 days. Cosmetic and functional results are often excellent if the initial repair was done carefully.

4. **Superficial uncomplicated corneal ulcers** can occur in foals just as in horses. However, foals may not show the pain, tearing and blepharospasm that accompany superficial ulceration in a mature horse, so diagnosis is not always prompt.

Shallow ulcers will take up fluorescein stain. Initial treatment should include debridement of the lesion with cotton swabs. Culture and cytology may be indicated, depending on the extent and age of the lesion. Therapy should include:

a. **A mydriatic** (1% Atropine, TID to start, with the dose tapered down as dilation occurs),
b. **Broad spectrum antibiotics** (chloramphenicol or triple antibiotic 4-6x per day),
c. **An anticollagenase** (topical serum, 4-6x per day).
d. **Systemic NSAID**: Flunixin meglumine (Banamine) 0.5mg/kg SID

Cooperative, otherwise healthy foals can often be treated at home using topical commercial ointments and serum that is administered from a sterile dropper bottle or Tb syringe. Ulcers should always be rechecked in a few days to assure that healing has occurred. Non healing ulcers and progressive ulcers will require considerably more aggressive therapy as described in sections 5 and 6 below.

5. **Deep or melting corneal ulcers** are a serious problem for foals. Deep ulcers will take up fluorescein stain but if part of the defect has reached Descemet’s membrane, there will be a corresponding gap in stain adherence. If a lesion has become a desmetocoele, the tissue that is keeping the eye from perforating is extremely thin, (less than 20 microns), and the eye is in danger of rupturing.

It is always appropriate to take a culture sample from a deep ulcer. Cytology should also be performed as long as the act of obtaining the cells does not risk rupture of the cornea. Culture is best done by either swabbing the lesion with a Dacron swab and applying the swab sample to a culture plate or a container of transport media, or by scraping the lesion with the blunt end of a sterile scalpel blade and then dropping the blade into thioglycollate broth. Cytology samples are obtained by dripping topical anesthetic on the cornea then scraping the edges of the lesion with the blunt end of a sterile scalpel blade. The debrided cells are then smeared from the blade onto
glass slides for staining and analysis. Cytology can be performed in veterinary clinic laboratories after the slides have been stained with Dif Quik and Gram stain. Alternatively they can be submitted to a reference laboratory.

Many deep corneal ulcers in young horses turn out to be sterile, but they still require aggressive anti-infective and anti-collagenase therapy. Deep ulcers in foals (both sterile and infected) often become melting ulcers. The appearance of a melting ulcer is dramatic—a lip of opaque tissue becomes rubbery to gelatinous in consistency and may appear to “drip” off the edge of the lesion. Melting occurs when collagenolytic activity exceeds the mechanisms that are working to repair the defect. Therapy is thus directed to not only treat/prevent infection, but also to try to mitigate collagenolysis that is mediated by host neutrophils, infectious factors, MMPs and other cytokines in the tear film. Deep or aggressively melting ulcers are best referred to an ophthalmologist for intensive care and monitoring. Therapy involves multiple topical agents and is often administered around the clock. It may include a combination of the following medications:

a. **Mydriatics:** 1% Atropine is usually used initially, and 2.5% phenylephrine may be added if dilation is not occurring. Frequency is 4x per day initially.
b. **Aggressive anticollegenase therapy:** Serum from the foal or the dam may be applied HOURLY; always at least 6x per day. Other strategies may include topical instillation of 0.17% EDTA (which can be prepared by filling a 5 or 10 ml EDTA tube (half full with sterile water), or 5-10% solutions of acetylcyteine. Frequency is 8-24x per day depending on severity of the melting.
c. **Broad spectrum antibiotic therapy:** Choice of drug is dependent on culture results. Agents commonly used include triple antibiotic preparations, tobramycin, cefazolin, ciprofloxacin or gentocin. Frequency is 8-12x per day.
d. **Antifungal therapy:** May not be necessary in some parts of the country but is critical in southeastern states with high incidence of keratomycosis. Often 1% miconazole is used as well as 3.3% natamycin. Other agents that can be tried include 1% itraconazole, 1% voriconazole, 5% Betadyne/saline solution and 1% silver sulfadiazene crÈme. Frequency of liquid antifungals is 6-8x per day. Frequency of topical crÈmes or ointments is 2-4x per day.
e. **Osmotic agent:** Topical 5% NaCl (Muro 128) may be applied 3-4x per day.
f. **Systemic NSAID:** Flunixin meglamine (Banamine) at 0.5 mg/kg, IV or orally, SID.
g. **Gastroprotectant:** Omeprazole, 4 mg/kg, orally, SID.

Surgical therapy is often used to complement medical therapy. Surgical therapy ranges from simple standing procedures (amputation of malacic tissue or serial debridement of the ulcer bed every 3-4 days) to general anesthesia procedures like keratectomy or the application of a conjunctival or amnion graft. Therapy for deep ulcers often continues for several weeks but frequency can be reduced once signs of healing occur.

6. **Severe corneal trauma with iris prolapse.** Foals can present with trauma that perforates the globe and results in iris prolapse. Traumatic corneal infections can also progress to the point of corneal rupture with secondary iris prolapse. The globe will take on an altered contour with the iris prolapse appearing as a reddish black tongue of tissue projecting from a thickened, opaque cornea. Surrounding tissue may be very malacic and intraocular hemorrhage may be present.
These cases carry a guarded prognosis. There are a few university specialty centers that will treat these cases surgically, performing amputation of the prolapsed tissue and **penetrating keratoplasty** (full thickness corneal transplants) to attempt to save the globe. Tectonic support for the injured eye may include construction of conjunctival or amnionic grafts as well as temporary tarsorrhaphy. The surgery and associated aftercare are expensive and not widely available so many foals that present with iris prolapse undergo enucleation.

**7. Persistent corneal erosions** are a common complication of foals that are in neonatal intensive care units as a result of prematurity, dysmaturity or septicemia. Clinicians must screen sick foals daily for the development of corneal ulcers because affected foals may not show the typical signs of pain, photophobia or lacrimation. The corneal defect is often shallow. The cornea may exhibit decreased sensation and fail to vascularize and the tear film may be deficient.

It is important to perform a close inspection of the eye to make sure that there is not a foreign body or stray hair that is causing the persistent erosion, and to perform a culture and cytology of cells that are debrided from the lesion to determine whether there is infection of the defect. If no infection or outward cause is found, therapy to try to encourage healing is multipronged and may include a combination of the following:

a. Frequent debridement of the defect using Q tips to roll away loose epithelium. (every 3-4 days)
b. Mydriatics to dilate the pupil (as needed)
c. Frequent application of a broad spectrum antibiotic that does not interfere with corneal epithelialization. Triple antibiotic is a good choice. (4-6x/day). Gentamicin should be avoided. Ophthalmic ointment may retard corneal healing.
d. Topical administration of 5% sodium chloride (Muro 128) in liquid or ointment form, (3-4x per day)
e. Frequent topical administration of serum, either autologous or homologous. The dam would be a good source of homologous serum. (5-8x/day)
f. Topical 1% Hyaluronic Acid solution, or topical polysulfated glycosaminoglycan (Adequan) mixed in artificial tears to a concentration of 50 mg/ml (3 x per day)

Cases that are refractive to treatment may benefit from a grid or punctate keratotomy but the clinician must be CERTAIN that infection is not playing a role before attempting these procedures. Grid procedures should ONLY be done on superficial lesions that have undergone culture, cytology and have failed to heal after less invasive attempts at therapy. Referral for thermal cautery or superficial keratectomy can be tried if no other method works. Sometimes healing can be facilitated by the use of contact lenses, nictitans flaps or temporary tarsorrhaphies.

**Use of Subpalpebral Lavage Tubes in Foals**

Corneal lacerations, deep ulcers, melting ulcers, ulcers with keratomalacia or iris prolapse and cases of severe uveitis are all conditions that require intense topical therapy. Subpalpebral lavage tubes (SPL tubes) may be a necessary treatment tool for these conditions for several reasons:

1. Young foals may be fractious to handle and difficult to treat effectively
Severely traumatized globes may be at risk for rupture, and may not tolerate close handling for topical treatment.

Many necessary topicals are only available in liquid format—it is difficult to administer a precise dosage into the tear film without an SPL.

The amount of effort required to treat serious cases around the clock is intense; SPL tubes make the therapy as easy as possible for the caretakers.

SPL tubes that are commonly used in foals and young horses are made by Mila International (www.milaint.com, 1-888-645-2468). The kits are sold in two different lengths but just one diameter of tubing. Both kit tube lengths will be too long for foals and weanlings, but the tubing can easily be cut to fit the patient neck length. Recently Mila has modified their tube kits so that the needle that introduces the tubing is “swedged on” to the treatment tube—there is no needle lumen, so the tubing is pulled through the lid at insertion rather than dragged through the bore of the needle.

Prior to tube insertion, the mane of the foal should be braided so that the SPL tubing can be threaded through the braids. If the foal is very young there will not be enough mane to braid, so small “pig tails” can be made by gathering tufts of mane and securing the middle of the tufts with rubber bands. Tube insertion may be accomplished standing in large foals that are several months old, but small foals should be placed in lateral recumbency. Size, temperament and accompanying systemic conditions will dictate whether the insertion is done with the foal heavily sedated or under very short term general anesthesia. Actual insertion only takes a few seconds, but there are risks of having such a large needle in close proximity to the globe.

The skin over the intended exit site above the lid fornix should be infiltrated with local anesthetic. Several drops of topical anesthetic should be squirted onto the corneal surface and also applied to the intended needle entry site in the conjunctiva over the fornix with a Q tip. The SPL needle should be guided into the desired tissue region by carefully sliding it along a gloved finger that is touching the intended entry site. Tentative probing will determine if local anesthesia is sufficient. The needle should then be pushed completely through the lid skin and then long tubing should be pulled through the lid. Care should be taken to assure that the treatment footplate is oriented to be parallel with the slanted angle of the fornix. The needle should remain attached to the SPL tubing until the tubing has been threaded through the braids or mane tufts. The best pattern is to bring the needle under the braid/tuft, then thread it through the center of the section adjacent to the neck crest, then pass the tubing over the remainder of the braid before going “under” the next braid down the neck. The tubing should be woven all the way to the base of the neck near the withers. A 20G 1 inch IV catheter should be carefully threaded into the injection end and the system should be closed by threading a catheter injection cap on the tubing. The injection assembly (tubing, 20 G catheter and catheter cap) should be taped to a short section of tongue depressor with adhesive tape and secured to one of the braids or tufts of mane. White adhesive tape should be used to construct “butterfly wings” to secure the tubing to the face. The wings can be affixed to the skin with non-absorbable suture of 2-0 or 1-0 diameter.

Injection of medication is accomplished by infusing 0.2 ml of drug into the cap and up the tube and then “chasing” it with a quantity of air that will deliver the drug into the tears. Often 1.5 ml
of air is a sufficient amount to get the drug up the shortened foal tube. The air should be drawn into a 3 ml syringe and injected SLOWLY though a 25G needle. Opinions vary on whether topical products lose efficacy if they are injected one after another in short order. Many treatment regimens advise waiting 5 minutes between medications, and this is a prudent approach. Medications should be drawn up in Tb syringes and injected through 25G needles. It is helpful to color code the syringes with tape and keep each kind of medication syringe in a separate cup so that a complicated drug regimen can be followed. It is also helpful to create a spreadsheet with boxes or circles that indicate treatment times of various medications. Checking off the boxes as treatments are completed will assure compliance and good communication between a team of treatment technicians.

Properly managed SPL tubes can remain in place for a month or more. Catheter caps should be replaced every 2-3 days and the white butterfly wings that secure the tubing to the face should be repaired if the sutures break. The tube should be checked daily. There is a label on the tubing near the foot plate that says “Mila”—this should be identified as well above the exit hole in the lid to assure that the tube footplate is snug in the palpebral conjunctiva and is not migrating towards the cornea.

A hard cup hood should be placed over the SPL tube and injured eye. Jorgensen (www.jorvet.com, 1-800-525-5614) sells an “Eye Saver” protective hood that comes in “left” and “right” models in several foal sizes. The plastic cup should be perforated with several drilled holes so it resembles a whiffle ball for optimum air circulation. The terrycloth “tear catchers” that snap into the inner aspect of the cup should be changed and washed daily, and a new gauze pad should be snapped across the hood lumen as well.

Most foals tolerate injection of SPL meds readily. However protective dams with very young foals may show aggression towards foal handlers who come into the stall often to treat their baby! For safety’s sake, always position the foal near the mare’s head so that she knows what you are doing, and you are aware of her reactions. Foals that are undergoing the stress of frequent treatment and examination are at risk for gastric ulcers so they should be on daily gastroprotectant medication and should be monitored for colic and diarrhea.

**Table 1. Breed Related Eye Disease Reported in the Horse**


Note that some of these conditions may only manifest in mature horses, but awareness of breed risks is useful for owner counseling at the neonatal examination.

1 **APPALOOSAS**: Congenital stationary night blindness, uveitis, congenital and acquired cataracts, glaucoma, lens luxation, optic disc colobomas, squamous cell carcinomas.
2 ARABIANS: Congenital cataracts, vitiligo, atropine sensitivity, CID with susceptibility to adenovirus

3 BELGIAN DRAFTS: Aniridia and secondary cataracts. Squamous cell carcinomas.

4 MORGANS: Bilateral nuclear non progressive cataracts.

5 QUARTER HORSE: Congenital cataracts, entropion, some lines have congenital stationary night blindness

6 THOROUGHBREDS: Congenital cataracts, microphthalmos with multiple ocular anomalies, retinal dysplasia/detachment, entropion

7 COLOR DILUTE BREEDS: Iridal hypoplasia, heterochromia iridus, squamous cell carcinoma

8 STANDARDBREDS: Retinal detachment, congenital stationary night blindness

9 PASO FINOS: Congenital stationary night blindness, glaucoma

10 AMERICAN SADDLEBRED: Cataract, esotropia, aggressive keratomycosis

11 WARMBLOOD BREEDS: Glaucoma, ERU

12 ROCKY MOUNTAIN HORSE: Anterior segment dysgenesis; findings may include congenital miosis, corpora and iris hypoplasia, macrocornea, ciliary body cysts, cataract, lens luxation, retinal dysplasia/detachment

13 MINIATURE HORSES: Upper lid entropion

14 CONNEMARA PONIES: Anterior segment dysgenesis

15 MULES: Aggressive sarcoid, misplaced nasolacrimal puncta

16 HAFLINGERS: Squamous cell carcinomas

17 LIPIZZANERS: Sarcoid resistant but often have aggressive melanomas

18 PERCHERONS: Melanomas

Useful References

The following is a list of recent comprehensive reviews and reference texts that the reader may find useful for further information.