Diseases of the Brainstem and Cranial Nerves of the Horse: Relevant Examination Techniques and Illustrative Video Segments

Robert J. MacKay, BVSc (Dist), PhD, Diplomate ACVIM

1. Introduction
This lecture focuses on the functions of the portions of the brainstem caudal to the diencephalon. In addition to regulation of many of the homeostatic mechanisms of the body, this part of the brainstem controls consciousness, pupillary diameter, eye movement, facial expression, balance, mastication and swallowing of food, and movement and coordination of the trunk and limbs. Dysfunction of the brainstem and/or cranial nerves therefore manifests in a great variety of ways including reduced consciousness, ataxia, limb weakness, dysphagia, facial paralysis, jaw weakness, nystagmus, and strabismus. Careful neurologic examination in the field can provide accurate localization of brainstem and cranial nerve lesions. Recognition of brainstem/cranial nerve dysfunction is an important step in the processes of diagnosis and treatment.

2. Anatomy and Nomenclature
The brainstem includes the diencephalon, mesencephalon (midbrain), and rhombencephalon (hindbrain). With the exception of the olfactory nerves (I), all cranial nerves are arrayed along the brainstem. The hindbrain is divided into metencephalon (pons and cerebellum) and myelencephalon (medulla oblongata). Because the diencephalon was discussed in the previous lecture under Forebrain Diseases, it will not be covered here.

3. Functions (Location)
Pupillary Light Response, Pupil Size (Midbrain, Cranial Nerves II, III)
In the normal horse, pupil size reflects the balance of sympathetic (dilator) and parasympathetic (constrictor) influences on the smooth muscle of the iris. Preganglionic neurons for sympathetic supply to the head arise in the gray matter of the first four thoracic segments of the spinal cord and subsequently course rostrally in the cervical sympathetic nerve. After synapse in the cranial cervical ganglion adjacent to the guttural pouch, the post-ganglionic sympathetic nerves continue to the smooth muscle of the orbit and act to cause pupillary dilation. Emotional and other influences on sympathetic pupillary tone are governed by hypothalamic centers that act through upper motor neuron (UMN) tracts descending from the midbrain. Interruption of pre- or post-ganglionic sympathetic nerves to the eye causes...
Horner’s syndrome, with miosis of the pupil, ptosis (reflecting hypotonia of the dorsal tarsal (Mueller’s) muscle), and spontaneous sweating and vasodilatation over the side of the face. Parasympathetic preganglionic neurons arise in the midbrain and exit the skull in the oculomotor nerve (III). These neurons synapse behind the eye in the ciliary ganglion. Post-ganglionic neurons pass along the optic nerve to innervate the ciliary muscle and constrictor of the pupil. The afferent part of the pupillary light reflex passes via the optic nerves and optic tracts, past the thalamus, to terminate in the midbrain. There is extensive decussation of these tracts both in the chiasm and midbrain.

Eye Position (Midbrain, Pons, Cranial Nerves III, IV, VI)
From nuclei in the midbrain and pons, the oculomotor, trochlear, and abducens nerves exit the cranial cavity through the orbital fissure and ramify in the periorbital tissues to innervate the muscles of the eye. The oculomotor nerve also supplies the levator palpebrae and pupillary constrictor muscles, and the abducens nerve innervates the retractor bulbi muscle. Lesions in these nerves (or nuclei) cause true strabismus.

Mastication (Pons, Cranial Nerve V)
The lower motor neurons of the trigeminal nerve arise in the pons and pass through the petrous temporal bone in the foramen ovale adjacent to sensory trigeminal neurons and are distributed to the muscles of mastication: masseters, pterygoids, temporals, and rostral digastricus. With unilateral damage to the trigeminal nucleus (or nerve), there is deviation of the lower jaw toward the normal side. By 2 weeks after injury, there is obvious muscular atrophy. Bilateral severe involvement of the trigeminal nuclei (or nerves) causes a dropped jaw, weak jaw tone, slight tongue protrusion, and inability to prehend or chew feed.

Facial Expression and Movement (Medulla, Cranial Nerve VII)
The facial nerves arise from nuclei in the rostral medulla and exit the calvarium with CN VIII via the internal acoustic meatus. The nerve courses through the facial canal in the petrous temporal bone adjacent to the middle ear and emerges through the stylomastoid foramen. The facial nerve is distributed to the muscles of facial expression including those of the ear, eyelid, nose, and lips and the caudal belly of the digastric muscle. With involvement of the nucleus or proximal nerve, there is drooping of the ear and lip, ptosis, collapse of the nostril, and the muzzle is pulled toward the normal side. Saliva often drools from the affected side of the mouth, and the horse has difficulty prehending food, especially grain. There may be exposure keratitis. The facial nerve also contributes parasympathetic neurons to lacrimal glands. The sensory component of the facial nerve contains fibers from the tongue (taste) and middle ear.

Balance and Equilibrium (Medulla, Cranial Nerve VIII), Hearing (Medulla, Cranial Nerve VIII, Forebrain)
The vestibular system is responsible for orientation of the horse relative to gravity. The receptor is in the bony labyrinth of the inner ear. The membranous labyrinth includes 3 semicircular ducts containing endolymph that connect to vestibular nerve endings at the cristae ampullares. Vestibular neurons pass centrally through the internal acoustic meatus to penetrate the rostral medulla and terminate in 4 vestibular nuclei. These nuclei have numerous projections to the nuclei controlling extraocular muscles, the cerebellum, and the spinal cord. The vestibular system controls the conjugate movements of the eyes during movement of the head through extensive connections with the nuclei of cranial nerves III, IV, and VI. Vestibular-cerebellar pathways pass through the caudal cerebellar peduncle. These pathways function to smoothly coordinate the movements of the eyeballs, trunk, and limbs with those of the head. Vestibulospinal tracts descend ipsilaterally to synapse on LMN and facilitate extensor muscles of the limbs while inhibiting flexor muscles. Some vestibulospinal tracts cross and reduce extensor tonus in contralateral limbs.

Unilateral disease involving the peripheral part of the vestibular system causes asymmetric ataxia with preservation of strength. The poll rotates toward the side of the lesion, and the head and neck may be turned toward the lesion. The body leans, falls, or rolls toward the side of the lesion, and the horse may stagger in tight circles. Because there is some visual compensation for vestibular ataxia, blindfolding exacerbates the signs. In horses with central vestibular disease, head tilt may be either toward or away from the side of the lesion. The latter presentation is known as paradoxical central vestibular disease and usually follows involvement of vestibular connections within the cerebellum. Unilateral vestibular disease often causes spontaneous or positional nystagmus, and physiological (vestibular) nystagmus may be absent or abnormal when the head is moved toward the side of the lesion. In peripheral disease, the nystagmus is always horizontal, rotatory, or arc-shaped, with the fast phase away from the lesion. With central involvement of the vestibular system, nystagmus also may be vertical. Typically, the eye on the affected side rotates ventrally in the orbit, whereas the eye on the normal side rotates dorsally (especially when the head is extended). This abnormal eye position is termed vestibular strabismus. Bilateral vestibular disease is characterized by severe symmetric ataxia and wide, sweeping movements of the head from side to side. Neurons of the cochlear division of cranial nerve VIII pass from receptors in the middle/inner ear to auditory centers in the midbrain.
and thalamus. A variety of local reflexes are initiated by stimulation of the cochlear nerve. In addition, there is projection of conscious pathways for hearing from the thalamus to the cortex (temporal lobe?). Deafness is congenital in some “splashed white” blue-eyed horses of several different breeds; otherwise, deafness is rarely recognized in horses.

Taste (Cranial Nerves VII, IX, X, Medulla, Forebrain)
Taste buds are found on the surface of the tongue and also in the soft palate, pharynx, lips, and cheeks. Sensory gustatory innervation is provided by the facial nerve (rostral two thirds of the tongue), glossopharyngeal nerve (caudal one third of the tongue), and vagus (pharynx and palate). General sensory innervation to the rostral two thirds of the tongue is provided by the trigeminal nerve, which probably also contributes gustatory information. Perception of taste involves the forebrain, including the limbic system. Deficiencies in the sense of taste are very difficult to detect by clinical testing.

Movement of Pharynx and Larynx (Cranial Nerves IX, X, XI, Medulla)
Motor innervation of the larynx and pharynx originates in neurons in the nucleus ambiguus, a fusiform structure extending the length of the medulla. This nucleus provides axons for the glossopharyngeal, vagus, and spinal accessory (internal branch) nerve roots. These roots form nerves that innervate the soft palate, pharynx, larynx, and cranial esophagus via the pharyngeal plexus and cranial and recurrent laryngeal nerves. The vagus nerve provides most of the motor fibers to pharyngeal muscles, and the glossopharyngeal muscle is the principal sensory nerve for the caudal one third of the tongue and the pharynx. The nucleus ambiguus is continued in the spinal cord, as the nucleus of the external branch of the spinal accessory nerve (innervation of trapezius and parts of brachiocephalicus and sternocephaelicus). The facial and hypoglossal nerves innervate several of the muscles that control movements of the hyoid apparatus; therefore, impaired movement of the hyoid apparatus caused by paralysis of these nerves could affect movements of the larynx and pharynx. Clinically, such effects are minor in horses at rest but can be revealed by intense exercise. Unilateral (hemiplegia) or bilateral vagal dysfunction causes pharyngeal paralysis, which interferes with swallowing and manifests as signs of dysphagia: coughing and gagging during eating with return of saliva, feed, and water through the nostrils and mouth. Bilateral anesthesia of the proximal glossopharyngeal nerves does not cause dysphagia. With unilateral laryngeal hemiplegia, there is exercise-induced respiratory stridor and aspiration of feed into the trachea. In horses with bilateral laryngeal paralysis, inspiratory stridor occurs at rest, and there is aspiration pneumonia.

Tongue Movement (Cranial Nerve XII, Medulla)
Neurons of the hypoglossal nerve originate in the hypoglossal nucleus in the caudal aspect of the medulla and emerge from the medulla as a horizontal row of rootlets, which combine to form the nerve as it enters the hypoglossal foramen. After emerging from this foramen, the hypoglossal nerve runs forward and ventrally in association with the gullet pouch and stylohyoid bone to innervate the genioglossus and muscles of the tongue. Interruption of the hypoglossal pathways causes hemiparesis of the tongue, evident as deviation of the apex of the tongue away from the affected side. Within 1 to 2 weeks, atrophy of the tongue becomes noticeable.

Regulation and Smoothing of UMN Activity (Cerebellum)
The cerebellum sits in the caudal fossa of the skull and is separated from the cerebral hemispheres by the tentorium cerebelli. It is divided into the foliunculodular lobe and the much larger body of the cerebellum. The cerebellar body consists of a median region, the vermis, and 2 lateral cerebellar hemispheres. Connections with the rest of the CNS are via 3 peduncles: Efferent connections pass through the rostral peduncle and afferent pathways enter the cerebellum via the middle and caudal peduncles. The cerebellum regulates and smoothes motor activity initiated by the UMN system. It also acts to maintain equilibrium and appropriate body posture during rest and motion. Propricoceptive information is gathered via afferent connections from the spinal cord (spinocerebellar and cuneocerebellar tracts) and vestibular system and is notified of UMN activity via extensive connections with brainstem UMN nuclei (including the olivary nucleus). Efferent cerebellar neurons project to vestibular nuclei and other parts of the brainstem, including the thalamus. There is virtually no projection of cerebellar efferents into the spinal cord. Cerebellar disease is usually diffuse and manifests as symmetric ataxia without weakness. There is defective regulation of the rate, range, and force of movement. Limbs may appear spastic, with excessive (hypermetric) or inadequate (hypometric) flexion during protration. Signs are most obvious when there is a change in the force or direction of voluntary movement. At rest, the body may sway, laterally or backward and forward, and there may be coarse head bobbing or tremor that is exacerbated by voluntary movement, such as reaching the head out for food. Extensor muscle tone is increased and limb reflexes may be hyperactive. With diffuse cerebellar cortical disease, the menace response is absent, although vision is normal.

4. Clinical Examination of Brainstem Function
Note that all parts of the CNS contributing to a particular neurologic function are covered in this section.2,4,5
Orientation and Coordination of the Head
Evaluate the orientation of the head from directly in front. Any head “tilt” is described from the patient’s perspective; thus, if the poll is rotated to the horse’s left (i.e., clockwise from the examiner’s point of view), the abnormality is described as a left head tilt. Carefully blindfold the horse and observe the effect on head position. Blindfolding removes visual input to head position and exacerbates abnormalities caused by vestibular disease. Observe the head and neck from the side. Persistent horizontal or low position of the head may indicate neurologic or muscular weakness of the neck, whereas extended head position may be found in horses with upper cervical vertebral problems or guttural pouch disease. Offer feed or a treat to the horse and observe the way in which the horse moves its head in response. Horses with cerebellar disease often make jerky or bobbing movements of the head as they move toward the offered feed.

Muscles of Mastication
If the mouth hangs open and the tongue protrudes, there probably is bilateral paresis of the muscles that close the jaw (temporalis, masseter, and pterygoid). Grasp the upper and lower jaws at the level of the interdental space and attempt to pull the lower jaw downward. The jaws pull apart easily in horses with bilateral paresis of the masticatory muscles. Tuck the forelock behind one of the ears and compare the temporalis muscles from in front of the horse. Turn the head from side to side and observe and palpate the masseter muscles. The pterygoid muscles and cranial belly of the digastricus muscle are located on the medial side of the mandible, so they are not readily palpable. Peel back the upper and lower lips and examine the alignment of the upper and lower jaws. With acute unilateral paresis of the masticatory muscles, even before atrophy is apparent, the lower jaw may be deviated toward the normal side.

Facial Tone
Examine the head carefully for symmetry of facial expression, particularly with respect to the ears, eyes, and muzzle. With complete unilateral facial paralysis, there is drooping of the ear, upper eyelid (ptosis), and lower lip and immobility, narrowing, and lengthening of the affected external nare. The muzzle is deviated away from the affected side, and saliva may drool from the mouth. Any to all of these components can be affected separately. Next, evaluate facial nerve function by testing “flick” reflexes on each side of the face. Each of these reflexes requires intact trigeminal sensory branches, central connections in the hindbrain, as well as functioning facial nerves. To test these reflexes, touch in turn the commissure of the lips, the medial and lateral canthi of the eye, the supraorbital fossa, and the ear. Appropriate responses are retraction of the commissure of the lip, blinking of the eye, and flick of the ear, respectively. Test facial innervation of lacrimal glands by performing Schirmer tear tests.

Size of Pupils and Pupillary Light Reflex
Stand in front of the horse while holding the noseband of the halter and swing the light back and forth from one side to the other to obliquely and briefly illuminate each eye without causing constriction of the pupils. Unequal pupillary size is termed anisocoria, a constricted pupil is miotic, and a dilated pupil is mydriatic. From this examination, determine whether or not the pupils are of equal size and if the diameter of each pupil is appropriate for the conditions. In this way, refine the diagnosis of anisocoria to miosis or mydriasis affecting a single eye. Move closer to the horse and again swing the light from one eye to the other to elicit pupillary light reflexes (without dazzle reflexes). Next, aim the light at the skin below one eye. Redirect the beam directly into the eye. This strong light should elicit both a dazzle reflex in the ipsilateral eye and pupillary light reflexes in both eyes. The dazzle reflex is an avoidance reaction to bright light. There is blinking, retraction of the eyelid, and movement of the head away from the light. A normal pupillary light reflex is immediate constriction of the pupils of both eyes in response to light directed into one eye. If the direct (i.e., ipsilateral) pupillary light reflexes are normal on both sides, no further testing is necessary. If one is abnormal, then consensual (indirect) reflexes should be tested. To perform the consensual reflex, watch the pupil in one eye while an assistant shines the light into the opposite eye.

Position and Movement of the Eyeballs
While continuing to stand in front of the horse, observe the position and size of the pupils while the head is held level (i.e., a line through the center of each eyeball is parallel to the ground). While keeping the head level, lift the chin slowly. The eyeballs should remain stationary while the chin moves upward; thus, the eyes rotate ventrally relative to the long axis of the head. In horses with vestibular disease, abnormal eye positions are exaggerated by this maneuver. If the pupils are in abnormal positions, try to position the head in such a way (usually by rotation) that the pupils are normally oriented relative to the transverse axis of the head. For example, a horse with vestibular disease often has ventral deviation of the eyeball on the side of the lesion and dorsal deviation on the opposite side. Eye position can be normalized relative to the axis of the head simply by rotating the head in the direction of the ventrally deviated eye. The abnormal position of the eyes in horses with vestibular disease is termed vestibular strabismus. True strabismus is eye deviation that cannot be corrected by repositioning the head and usually reflects an anatomic anomaly or dysfunction of nerves to the extraocular muscles. Further assess abducens nerve function.
by performing a modified corneal reflex. Hold the eyelids closed and, through the eyelid, push the eyeball medially. The normal response to this maneuver is retraction (adduction) of the eyeball. Move the horse’s head in a horizontal arc from side to side and observe the movements of the eyeballs. Signs of physiologic nystagmus should normally be elicited—namely, a series of horizontal movements of the eyeball consisting of a rapid phase in the direction of head movement followed by a slow phase in the opposite direction. Each fast phase is accompanied by an eye blink. Physiologic nystagmus is normal and should be distinguished from eye movements characteristic of vestibular disease: spontaneous nystagmus, which occurs when the head is stationary and in a neutral position, and positional nystagmus, which only occurs when the head is moved to certain positions. In horses with asymmetric vestibular disease, physiologic nystagmus often is abnormal or absent when the head is moved toward the side of the lesion.

Swallowing

It is difficult to assess competence for swallowing during a physical examination. On the basis of history and observation, note whether feed, water, or saliva return through the nose, especially when the horse eats or drinks. Pass a nasogastric tube into the pharynx and assess effectiveness of swallowing movements as the horse attempts to move the tube into the esophagus.

Tongue

Pull the jaws slightly apart and observe the movements of the unrestrained tongue. With unilateral weakness, the tongue curls toward the normal side. Grasp the tongue from one side after inserting the hand through the interdental space. Note resistance of the tongue to being stretched and look for atrophy and muscular fasciculations. Gently pinch the side of the tongue with a hemostat and look for reflex retraction. Pull the tongue out one side of the mouth, release it, and look for retraction of the tongue back into the mouth. In normal horses, one or two chewing movements occur as the tongue is quickly retracted.

References