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The Art and Science of Equine Neurology – The Forebrain

Robert J. MacKay, BVSc, PhD, Diplomate ACVIM

Author’s address: College of Veterinary Medicine, University of Florida, Gainesville, FL; e-mail: mackayr@vetmed.ufl.edu.

Anatomy and Nomenclature

The forebrain or prosencephalon includes the telencephalon (cerebrum) and diencephalon (thalamus and hypothalamus).1

Functions (Location)2-4

Level of Consciousness (Cortex, Brainstem, Especially Front Half)

One of the critical functions of the reticular formation of the brain stem is activation of the cerebral cortex for the awake state. This component of the formation, known as the ascending reticular activating system (ARAS), is an ill-defined meshwork of cells concentrated in the rostral brain stem that receives afferent input from all parts of the CNS and projects excitatory stimuli cortically. 2 Focal to extensive lesions anywhere in the brain stem may reduce the level of consciousness, whereas cortical injury must be diffuse to cause noticeable obtundation. The ARAS also is involved in the initiation and maintenance of sleep. Abnormalities of production or action of hypothalamic arousal peptides (hypocretins/orexins)5 or imbalances of brain stem neurotransmitters may result in narcolepsy/cataplexy sleep disorders.

Behavior (Limbic System, Temporal Lobes)

Normal behavior requires integration of signals from the entire CNS but principally involves the forebrain. Most important in controlling intrinsic behavior is the limbic system – a connected series of structures in the cerebrum and diencephalon. A minor component is also found in the midbrain. Included are the amygdala, hippocampus, fornix, cingulate gyrus, and septal area. A closely associated region, which is important in primate behavior, is the temporal lobe of the cerebrum. It is thought that behavior based on conditioning and experience (i.e., learning) is controlled by the temporal lobes. Structural, metabolic, or psychological disturbances affecting these areas may result in behavioral abnormalities (i.e., dementia). Dementia can be defined as changes in normal habits, personality, attitude, reaction to the environment, or loss of learned skills. Some of the signs that may be seen include disorientation in a familiar environment, failure to recognize a handler or object, loss of the ability to be led, frequent yawning, headpressing, irritability, unprovoked kicking or biting, compulsive walking or circling, and dramatic changes in eating or drinking habits.

Almost any disturbance of the forebrain potentially can cause dementia. Encephalitis, head trauma, space-occupying lesion, malformation, infarct, and metabolic disorders all are likely to cause changes in behavior. It is likely that structural or metabolic forebrain disease is the cause...
of dementia if other neurologic abnormalities are found by neurologic examination or imaging studies. In the absence of such supportive findings, abnormal behavior such as self-mutilation may have a psychological basis.

Seizures

Seizures are sudden, transient attacks of abnormal motor and/or behavioral activity attributable to paroxysmal depolarization of part to all of the brain. Depolarization occurs either simultaneously throughout the brain or originates from a hyperirritable focus somewhere in the forebrain. Seizures originating from a focus likely will initially have asymmetric clinical signs and there may be additional signs of forebrain disease between seizures that are revealed by neurologic examination. Seizures frequently originate in the frontal (motor) cortex and involve muscle fasciculations and tremors around the head or abnormal movements of the jaws and tongue (“chewing gum fits”). Convulsions characteristic of neonatal encephalopathy often are of this type. In their severest (grand mal) form, seizures manifest as sudden recumbency, with a brief phase of extensor tonus, followed by clonic (“galloping”) movements of the legs, loss of consciousness, and a variety of signs of autonomic discharge (e.g., sweating, urination, defecation, pupillary dilation). Mild motor seizures are often accompanied by behavioral signs such as obtundation, compulsive walking, hyperresponsiveness to stimuli, or other signs of dementia. Seizure foci in the forebrain may occur at sites of previous or current trauma or inflammation. Syndromes of more than multiple seizure episodes without interictal evidence of brain disease often are described as epilepsy.

Perception of Pain (Parietal Cortex, Cranial Nerve V)

Pain sensation is transmitted from the body to the brain in multisynaptic spinothalamic tracts. Signals initiated by stimulation of pain receptors on one side of the body pass through spinothalamic tracts on both sides of the spinal cord. Axons in these tracts course rostrally to terminate in the thalamus. From there, cell bodies project axons to the sensory (somesthetic) cortex for conscious perception of pain or other sensory modalities. It is thought that the somesthetic cortex is located principally in the parietal lobe of the cerebrum. The pathways for pain perception in the head pass through the maxillary, ophthalmic, and mandibular branches of the trigeminal nerve. The central component of this pathway is predominantly contralateral. Unilateral lesions of the sensory parts of the forebrain (thalamus, internal capsule, sensory cortex) thus cause contralateral facial hypalgesia. Because the central components of vision and facial pain perception are close anatomically, it is common to find unilateral facial hypalgesia and blindness (on the same side) in the same horse.

Smell (Olfactory Bulbs, Cranial Nerve I)

Olfactory nerves pass through the cribriform plate and into the olfactory bulbs of the cerebrum. Information on smell is relayed through the thalamus to centers in the unconscious (limbic system) and conscious cerebrum.

Vision (Thalamus, Occipital Cortex, Cranial Nerve II)
In horses, 80-90% of optic nerve fibers from one eye cross at the optic chiasm and 80% of fibers in the optic tracts synapse at the lateral geniculate nucleus in the thalamus. The remainder course to the midbrain to function in the pupillary light reflex. Neurons in the lateral geniculate nucleus project via the internal capsule to the visual cortex in the occipital lobe of the cerebrum. This area is caudal in the cerebrum, immediately rostral to the tentorium cerebelli and caudal to the parietal cortex. The pathway from the eye to the contralateral visual cortex via the optic nerve, optic tract, lateral geniculate nucleus, and internal capsule must be intact for normal vision. Lesions in the pathway caudal to the chiasm result in blindness in the opposite eye and lesions rostral to the chiasm affect vision in the ipsilateral eye. Damage to the cortex should not affect the pupillary reflex pathway. Visual perception is evaluated by obstacle tests (with and without blindfolding of one eye) and by the menace response. The menace response requires the central visual pathway just described plus normal facial nerve function. Integrity of the cerebellar cortex is also needed, although it is not known if the pathway that mediates this response actually passes through the cerebellum. It is important to note that the menace response does not develop in foals until they are 1-2 weeks old.

**Upper Motor Neuron System - Voluntary Movement (Cerebrum, Brainstem)**

The UMN system is responsible for the initiation of voluntary movement and regulation of posture through support against gravity. The latter is effected in part by modulation of the anti-gravity myotactic reflexes of the limbs. The component of the UMN system originating in the hypothalamus is responsible for the control of muscular activity associated with visceral functions (respiratory, cardiovascular, urinary). Direct cortical influence over motor activity in the horse via the **pyramidal system** is largely limited to fine control of the muzzle and lips. The much more important **extrapyramidal system** is a multisynaptic pathway from the brain to the lower motor neurons of the spinal cord and brain stem. The frontal and parietal lobes of the cerebrum, basal nuclei, diencephalon, midbrain, and hindbrain all contribute to the UMN system. UMN, particularly those originating in the midbrain, generally are inhibitory to myotactic reflexes. Damage to the neurons or axons of these tracts in the caudal midbrain, hindbrain, or spinal cord, increases extensor tonus and may result in limb spasticity (“stiffness”). With UMN injury there also is limb weakness, hyperactive extensor reflexes, and crossed extensor reflexes, all in the ipsilateral limbs. Movement disorders are characteristic of UMN disease in the rostral brainstem. Abnormal involuntary movements include dystonia (writhing movements of the muscles of the head and spine), ballism (violent flailing of a limb), chorea (repetitive jerky movements of different muscle groups), and myoclonus (repetitive movements of a single muscle group). Nigropallidal encephalomalacia is a disease of this type in horses that is caused by the chronic consumption of yellowstar thistle or Russian knapweed plants. There is dystonia and rigidity of the muscles of the head resulting in lip retraction, tongue protrusion, and inability to prehend food. More generalized extrapyramidal syndromes in horses reportedly are associated with reactions to fluphenazine (an antipsychotic sedative drug) and metaclopramide (a prokinetic agent).

**Clinical Examination of Forebrain Function**

Note that all parts of the CNS contributing to a particular neurologic function are covered in this section. For example, both vestibular and forebrain influences on head position are discussed.
Alertness

Assess level of consciousness or alertness on a continuous scale from normal (i.e., bright and alert) to comatose. Progressive levels of obtundation are termed lethargy, stupor, semi-coma, and coma. With lethargy, there is a somewhat blank facial expression with slight drooping of the ears and eyelids, sluggish responsiveness to stimuli, and reduced voluntary activity. It should be noted that some conditions cause horses to lose facial expression while maintaining normal alertness. Botulism is a disease of this type. A stuporous horse stands in one place with the head held low and responds only to strong stimuli. Stuporous horses that are recumbent are defined as semi-comatose while recumbent horses that do not respond to any stimulus are comatose. Some horses with reduced alertness appear irritable and anxious, walk compulsively, or otherwise interact abnormally with handlers. An obtunded horse showing such behavior may be termed delirious. It is very unlikely that a horse with narcolepsy will have a sleep attack while being examined; thus, assessment of possible narcolepsy usually rests on description by the owner or video recordings of an attack. Occasionally, neonatal foals will collapse during examination and briefly appear comatose. These typically are benign episodes of cataplexy and probably are an exaggerated version of the collapse response by which a normal foal can be cast into recumbency.

Obtundation reflects damage in the rostral brain stem or diffusely in the cerebrum. Dementia is a sign of forebrain.

Limb ataxia/weakness and signs of cranial nerve dysfunction are common in horses with obtundation secondary to brain stem disease.

Behavior

Abnormalities of behavior are termed dementia. Behavior is assessed from history and general observation in the course of the examination. Typical abnormal behaviors resulting from CNS disease include self-mutilation, head-pressing, compulsive walking (often in a circle), yawning, aggression (including unprovoked biting or kicking), timidity, loss of affinity of a foal for its dam, and loss of learned behaviors and skills. Inability or refusal of a trained horse to follow when being led is a form of dementia that may be obvious during neurologic examination. Compulsive turning of the head and neck in one direction, blindness, and seizures are other signs of forebrain dysfunction that may be seen in demented horses.

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. Repeatedly straighten and release the horse’s neck and head and observe whether or not the head returns to one side. Asymmetric disease of the vestibular system causes the head to tilt and turn, whereas asymmetric cerebral disease may cause the head and neck to turn without tilting. Carefully blindfold the horse and observe the effect on head position. Blindfolding removes visual input to
head position and exacerbates abnormalities caused by either vestibular or cerebral 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.

Head tilt suggests involvement of the vestibular labyrinth, nerve, or root peripherally, or the vestibular nuclei and connections in the medulla oblongata or cerebellum centrally. It has also been suggested that lesions in the proximal cervical nerves may cause signs suggestive of vestibular disease. If the head is turned without tilting, involvement of the cerebrum is likely. Coarse or fine head bobbing, especially intentional, indicates diffuse cerebellar dysfunction.

Because of the close association of cranial nerves VII and VIII, facial paralysis is often seen in horses with vestibular disease. With peripheral vestibular disease, there often is spontaneous horizontal or arc-shaped nystagmus with the fast phase directed away from the side of the lesion. In horses with involvement of the central components of the vestibular system, there may be horizontal or vertical nystagmus. Damage to adjacent structures in the hindbrain may additionally cause obtundation, other signs of cranial nerve dysfunction such as dysphagia and masseter atrophy, and weakness/ataxia of the limbs on the side of the lesion. Horses with cerebellar cortical disease may lack menace responses despite having normal vision.

**Facial Sensation**

While standing in front of the horse, place the palm of the right hand on the muzzle, work the thumb into the right nostril and touch the nasal septum on that side. In mirror-image fashion, use the left thumb to stimulate the left side of the septum. The normal response to this noxious stimulus is vigorous movement of the head away from the side of the stimulus. Compare the intensity of responses on each side. If there is obvious asymmetry of response, test cutaneous sensation at multiple locations over the face by pinching the skin with hemostats. The pathway for recognition of this noxious stimulus is input through the sensory division of the trigeminal nerve to the pons and thence to the somesthetic area of the contralateral cortex. If flick reflexes are normal, a reduced or absent response to a noxious stimulus suggests involvement of the contralateral forebrain, probably at the level of the parietal cortex. In contrast, hypalgesia and hyporeflexia of flick reflexes of part to all of one side of the head indicates involvement of the trigeminal nerve or branches. With hemifacial hypalgesia because of forebrain disease, cortical blindness, obtundation, and abnormal behaviors may also be seen. Peripheral lesions of the trigeminal nerve may involve motor fibers and result in weakness and atrophy of masticatory muscles.

**Menace Response**

Test menace responses while standing in front of the horse. To do this, use the palm of the hand to make a threatening gesture toward the eye. Test from both temporal and nasal directions on each side. Stimulate the horse just before each menace gesture by tapping the skin below the eye.
For safety reasons, always hold the noseband of the halter with one hand while the other is used for testing. A normal menace response is blinking of the eye, sometimes accompanied by evasive movement of the head and neck. Compare carefully the intensity of the menace responses elicited from each side. In this regard, menace should be considered a quantitative response. Unlike simple reflexes, which are assessed as present or absent, a menace response can be considered abnormal if it is less vigorous than that elicited from the opposite (normal) side. The menace response can be interrupted anywhere in its pathway from the eye via the optic nerve to the contralateral optic tract, diencephalon, internal capsule and visual cortex. From the visual cortex, the menace response pathway continues to the facial nucleus and nerve on the side being tested and receives essential input from the cerebellum. If there is no other sign of cerebellar involvement, and facial nerve function and pupillary light reflexes are intact, the lesion is contralateral, central to the optic chiasm, and likely in the forebrain. In normal neonates and in older horses with diffuse cerebellar cortical disease, there is no menace response but the horse can see. In these settings, vigorous threatening gestures toward the eye may cause evasive movements of the head without blinking of the eye. There may be other signs of asymmetric cerebral disease such as compulsive walking in circles, dementia, head and neck turn, obtundation, seizures, and facial hypalgesia on the side opposite the lesion (i.e., the same side as the defective menace). If the site of the lesion causing a menace deficit is the optic nerve or fundus of the eye, the pupillary light reflex on the same side should also be abnormal. Head-bobbing, dysmetria, and ataxia are expected in horses with defective menace responses resulting from cerebellar dysfunction. If eyelid paralysis is preventing the menace response, the palpebral (eyelid flick) reflex should also be abnormal and there may be additional signs of facial paralysis.

References