The neurologic examination of the foal can be easily performed concurrently with the physical examination and provides a more comprehensive assessment of the overall clinical status. The evaluation should include an assessment of behavior, state of consciousness, cranial nerves, posture and postural reactions, segmental (spinal) reflexes, palpation, and gait evaluation. In order to identify abnormalities, it is essential to become familiar with what constitutes normal for the neonatal and developing foal. Author’s address: University of California-Davis, School of Veterinary Medicine, Department of Medicine and Epidemiology, Tupper Hall 2108, One Shields Avenue, Davis, CA 95616; e-mail: mraleman@ucdavis.edu. © 2020 AAEP.

1. Introduction
There are important evolutionary differences between prey and predator animals.1 Horses as a precocious species are born with a more developed brain and fully functional vision and hearing than altricious species, such as humans. Although brain development continues after birth, the cerebellar layers in prey animals, such as the neonatal foal, are already distinct histologically at birth compared to predators’ brains.1 However, cerebellar cell migration continues to occur in the neonatal period (Fig. 1). Cerebellar development and myelination in various parts of the nervous system explain the “bouncy” gait typical of neonatal foals. This bouncy gait becomes less apparent as the foal matures. Neonatal foals are not just little adult horses, and major physiological and neurological differences exist compared to older foals and adult horses.2–5 Therefore, recognition of what is normal according to age is essential. Alertness, response to the environment, and movement are different in utero, during birth, and in extra-uterine life. Foals respond and move in utero but not to the extent that is seen in extra uterine life. In the birth canal, foals are in a drowsy state and become minimally responsive and movement is depressed. During the first few hours of extra uterine life, important milestones must occur for successful functioning and survival of the neonatal foal. This rapid transition is remarkable (Fig. 2). As prey animals, horses must develop a menace response relatively quickly after birth (within 7 to 10 days) compared to predators (several weeks).

Many neonatal diseases present with similar clinical signs, such as lethargy, recumbence, weakness, and reduced or lack of suckle reflex, among others. These signs could be caused by systemic or neurologic disease, or both. Therefore, a complete thorough history must be obtained, followed by a comprehensive physical examination to determine overall health status. Do not forget signalment (breed, sex, and age) because there are disorders that can be seen in specific breeds. A few examples include juvenile epilepsy and lavender foal syndrome in Egyptian Arabian foals. These two disorders are phenotypically and genotypically different.
The principles on how to perform the neurologic examination are the same across species. The neurologic examination in foals can be done concurrently with the physical examination. Neurologic assessment must include the evaluation of behavior, state of consciousness, cranial nerves, posture and postural reactions, segmental (spinal) reflexes, palpation, and gait evaluation. The tools required for the examination are vision (lots of observation), touch (palpation), and listening. The main goal of the neurologic examination is to determine if the foal is normal or abnormal. The second goal of the examination is to determine the anatomical location of the deficits within the nervous system and, finally, to consider possible causes of disease. It is critical to rule out other disorders that might present with similar signs, especially in the neonatal foal (e.g., obtundation, dysphagia, and recumbency weakness).

2. Neurologic Examination in Neonatal Foals
The most important component in performing the neurologic examination in foals is the practitioner’s senses for observation, listening, and palpation; then, a light source to examine the eyes (dazzle reflex [subcortical reflex to bright light] and pupillary light reflexes) and ears; and needle holders, forceps, or a pleximeter to check for segmental (spinal) reflexes. Know what is normal and do more than one examination. Once more, observation is paramount. The neurologic status of the normal neonatal foal goes through a transition from in utero to extra utero life. The APGAR (appearance, pulse, grimace, activity, respiration) score developed for the assessment of neonates in the post-foaling period (1 minute post-foaling) consists of the following variables:

1. Heart rate (normal: regular, ≥60 beats per minute [abnormal if undetectable, irregular, or <60 bpm]).
2. Respiration (normal: regular, ≥60 respirations per minute [abnormal if undetectable, irregular, or <60 rpm]).
3. Mucous membranes (normal: pink; abnormal if blue, bright red, or yellow).
4. Muscle tone (normal: strong enough to be in sternal recumbency).
5. Responsiveness.
   A. Nasal stimulation (expected response: strong grimace, sneeze).
   B. Ear tickle (expected response: head shake).
   C. Back scratch (expected response: attempts to stand).
This evaluation can be repeated at 5 and 15 minutes post-foaling to determine if veterinary intervention is needed. Important milestones include time to sternal recumbency (1–2 minutes), alertness and responsiveness to external (tactile, visual, and auditory) stimuli (within 5 minutes), suckle reflex present (within the first 20 minutes), vocalizing in response to the dam’s nickering (within 30 minutes), time to stand (60 minutes [longer than 2 hours is considered abnormal]), and time to nurse (2 hours [longer than 3 hours is abnormal]) post-birth. The author performs neurologic evaluation concurrently with the physical examination in neonatal foals. Similarly, multiple assessments of the neurologic status can be done concurrently with physical examination. The examination consists of evaluating the neurologic status while the foal is at rest (static) and during movement (dynamic) if ambulatory. The examination could be divided in the following:

1. Behavior and mentation
   A. Behavior (examples of deficits):
      a. Aberrant vocalization (e.g., barking or honking sounds).
      b. Walking compulsively.
      c. Lack of mare bonding.
      d. Sleeping behavior while standing. Neonatal foals must lay down to sleep (drowsy to slow wave sleep and rapid eye movement sleep); this behavior changes as the foal matures.
   B. Mentation (Fig. 3):
      a. Normal, bright, alert, responsive.
      b. Obtunded. This could range from mild to moderate to severe obtundation: from standing quietly to recumbent but responsive to physical stimulus (tactile, auditory, visual). In this state, foals should always respond to non-painful stimulus.
      c. Stuporous. Recumbent, only responsive to profoundly painful stimulus and only while this stimulus is applied.
      d. Comatose. Not responsive at all, including to profoundly painful stimulus.

2. Cranial nerves and responses, reactions, and reflexes.
   A. Afferent, central, and efferent pathways must be intact for proper function.
   B. These can be evaluated individually (I through XII) or grouped into functions (e.g., olfaction [I], vision [II], menace response [II, VII]).

3. Posture (head, neck, trunk, limbs, and tail).
4. Postural reactions (i.e., proprioceptive positioning [foot placement]).
5. Segmental (spinal) reflexes.
   A. Cervicofacial
   B. Thoracolaryngeal
   C. Thoracic and pelvic limb reflexes
   D. Anal and perineal
6. Palpation
   A. Muscle asymmetry
   B. Painful areas
7. Gait evaluation
   A. Consider age of foal (bouncy in neonates)
   B. Consider breed for specific gait
8. Nociception
   A. Evaluate for pain perception (conscious perception = cerebrocortical acknowledgment of pain) only in foals with no apparent voluntary movement.
   B. Reflexive movement while applying a painful stimulus does not equal a conscious response.

The determination of mentation, behavior, and posture can be done as the history is being taken or as the foal is being examined. States of consciousness include bright, alert and responsive, and obtunded.
stuporous, and comatose (Fig. 3). Normal behavior is a bright, alert foal that is responsive to the environment; who has mare attachment; is udder seeking and nursing, curious of the environment; and who sleeps. Head posture in neonatal foals has a “flexed” appearance at the atlanto-occipital joint compared to adults, and their stance is wide-based, which becomes narrower within days of age. The menace response (a learned response) develops at approximately 7–10 days. Foals under 10 days of age are visual but have not learned to blink in response to an approaching or menacing object. However, if a strong light is applied to the eye, the foal is able to blink because this involves a subcortical reflex (dazzle reflex, Fig. 4) already present at birth. Furthermore, cranial nerve reflexes are also present in neonatal foals at the time of birth (e.g., pupillary light reflex). Cranial nerve deficits might be apparent during the initial observation before approaching the patient. As mentioned earlier, auditory function is fully developed in the neonatal foal (Fig. 5).

Palpation is essential to detect areas of apparent pain, local temperature, muscle tone and symmetry, joint extension and flexion, and tail tone among other findings. Tactile stimuli results in brisk exaggerated responses and reactions in normal foals compared to older animals. Segmental (spinal) reflexes that can be evaluated in foals include cervicofacial, cutaneous trunci, biceps, triceps, patellar, gastrocnemius, flexor (withdrawal), anal, and perianal (Fig. 6). In order to assess segmental reflexes involving the thoracic and pelvic limbs, foals must be in lateral recumbency and relaxed. It is not possible to properly evaluate these in the standing foal. Also, if the foal is in lateral recumbency but thrashing or with increased muscle tone or rigidity, evaluation and interpretation of reflexes will be compromised, faulty, inaccurate, or simply not possible to perform. The cross-extensor reflex may or may not be present in the neonatal period. If present, it is not considered abnormal, and as the foal ages, this reflex becomes inapparent. Extensor thrust reflex can also be seen in normal neonatal foals. Neonatal foals have a hypermetric gait that becomes more coordinated by 3 days of age. Effects of systemic disease, orthopedic disease, congenital anomalies, motor deficits (from initiation of movement by the cerebrothalamus [forebrain] all the way to the nerves, neuromuscular junction, and muscle
as the executers), and weakness can result in recumbency. Cutaneous sensation can be evaluated to investigate the presence or absence of sensory function. Nociception (conscious perception of pain) is only evaluated if voluntary motor function is absent or difficult to interpret. It is important to consider congenital or hereditary disorders and other common neonatal diseases that might affect the overall neurologic condition in foals. For examples see Table 1.

3. Neuroanatomical Localization

There are 3 major divisions of the nervous system: brain, spinal cord, and peripheral. These divisions are further divided into various functional areas as follows:

1. Brain
   A. Cerebrothalamic
   B. Brainstem
   C. Cerebellum

2. Spinal cord (spinal cord segments): cervical (C), thoracic (T), lumbar (L), and sacral (S)
   A. C1–C5/C6
   B. C6–C2
   C. T3–L3
   D. L4–S2
   E. S1–S5
   F. Caudal

Precise location of neurologic deficit or pathology caudal to T3 could be challenging to further localize at times if segmental reflexes cannot be examined due to patient cooperation, increased muscle tone, or in larger patients. However, alteration in cutaneous trunci reflex and alterations in skin sensation can be helpful.

3. Peripheral
   A. Nerve rootlets and roots
   B. Ganglia
   C. Nerves (motor and sensory)
   D. Neuromuscular junction
      a. Presynaptic
      b. Synaptic
      c. Postsynaptic (muscle membrane)

When localization to a specific or single area of the functional areas of the nervous system is not possible, consider diffuse or multifocal localization. Do not forget to evaluate the autonomous nervous system: sympathetic, parasympathetic, and intrinsic enteric. Sympathetic denervation of the head, for example, presents as miosis, ptosis, protrusion of the 3rd eyelid with eye lashes pointing down, increased skin temperature and redness apparent in foals with depigmented skin, and sweating of the head due to vasodilation ipsilaterally. Parasympathetic denervation of the head as a single problem is
extremely rare, but if it is present, functions such as salivation and lacrimation would be reduced or absent and mydriasis would be observed. Lesions of the intrinsic enteric system would result in ileus and colic that, if congenital, are incompatible with life.

4. Functional Neuroanatomy
For more detail of functional anatomy, refer to AAEP 2015 proceedings by Aleman.²

5. Localizing the Lesion Based on Functional Anatomy
Cerebrothalamus
One or more signs might be observed, such as behavior alterations (compulsive, bizarre, manic, and vocalization), lack of mare bonding and udder seeking, lack or delayed initiation of movement, central blindness, wide circles ipsilateral to the lesion, seizures, contralateral decreased nociception, and contralateral proprioceptive deficits. Examples of diseases that can cause these deficits include hypoxic/ischemic encephalopathies, metabolic encephalopathies (hyperammonia, sodium disorders, hyperbilirubinemia [neonatal isoerythrolysis], hypoglycemia [neuroglycopenia = low glucose concentration in the brain]), bacterial meningoencephalomyelitis, trauma, and hereditary epilepsies (Egyptian Arabian foals), among others.

Brainstem
Signs that might be observed when there is pathology in this location include an altered state of consciousness or mental status (obtunded, stuporous, and comatose), altered sleep, multiple cranial nerve deficits, and proprioceptive deficits. It is important to note that cranial nerves have nuclei and tracts within the brain (central components). All nerves are always peripheral. A description of cranial nerves is provided below. Vestibular nuclei are located in the caudal brainstem; therefore, diseases affecting the caudal brainstem could cause central vestibular disease (i.e., infection and trauma). Pathologic nystagmus, strabismus, ipsilateral head tilt, ipsilateral leaning to one side, ipsilateral circling (small circles) to one side, and lack of balance and incoordination (vestibular ataxia) are all signs of vestibular disease. Proprioceptive deficits are also observed with central vestibular disease.

Cranial Nerves
Responses, reactions, and reflexes can be evaluated in the standing or recumbent foal. Cranial nerves can be evaluated in order from I to XII or by functional regions. The author prefers functional regions starting with the sense of smell (subjective); all eye functions (menace, palpebral fissure, palpebral reflex, corneal reflex, dazzle reflex, pupillary light reflex, adaptation to light and darkness, eye globe position and retraction, physiologic nystagmus, and tear production); jaw/facial motor, sensation, and symmetry; and eating/drinking (pre-
hension, suction, tongue tone and movement, and gag reflex). For clarification, dazzle reflex is a subcortical reflex that does not involve the brainstem. To check for dazzle and pupillary light reflexes, a strong light source must be used (Fig. 4). This section does not include a full description of the central components in the brainstem involved in cranial nerve function. A brief description of functions associated with cranial nerves is provided below.

- **Olfaction (smell):** CN I, subjective evaluation and interpretation.
- **Menace response:** CN II and VII, cerebral cortex, and cerebellum.
- **Palpebral fissure:** CN III, VII, sympathetic innervation.
- **Palpebral reflex:** CN V and VII (tap gently the medial and lateral parts of the eyelids separately, the normal result is blinking).
- **Trigeminal facial reaction/reflex:** CN V and VII (touch face, nasal mucosa, and inner pinnae).
- **Mastication:** CN V motor part (mandibular branch). Examine in older foals.
- **Facial and nasal sensation:** CN V (VII for inner pinnae).
- **Pupillary light reflexes (direct and indirect):** CN II and III.
- **Corneal reflex:** CN V, VI, and VII (avoid if the horse has corneal disease or risk of contamination).
- **Eye globe position:** CN III, IV, and VI (also contribution of VIII, rule out extraocular muscle disease or retrobulbar or periocular mass). Upon head elevation, foals will have mild ventral strabismus which is considered normal.
- **Eye globe retraction:** CN VI.
- **Tear production:** CN VII (Schirmer’s test on both eyes, compare).
- **Physiological nystagmus:** CN VIII vestibular. Both eyes should move with the direction of the head movement in a synchronized manner.
- **Pathologic nystagmus, occurring at rest or when the head is held in a certain position, is an indication of vestibular disease (central or peripheral).**
- **Audition:** CN VIII cochlear (Fig. 5, auditory function is fully developed in neonates and similar to the adult horse).
- **Gag reflex:** IX, X, XI, XII (observe swallowing).
- **Cervical spinal musculature:** CN XI.
- **Tongue tone and movement:** CN XII.

**Cerebellum**

Common signs of cerebellar pathology include intention tremors (tremors upon intended movement), hypermetria of all limbs (more pronounced in the thoracic limbs), ataxia, and plus or minus menace deficits. Examples of diseases include cerebellar abiotrophy in Arabian horses and cerebellar hypoplasia. Dandy-walker syndrome is a congenital malformation consisting of partial or complete absence of the cerebellar vermis, resulting in the aforementioned signs. Cerebellar cysts have also been seen by this author.

**Spinal Cord**

Deficits, such as general proprioceptive ataxia, paresis, and upper motor neuron (UMN) or lower motor neuron (LMN) signs, depending on location within specific spinal cord segments, will be observed. Sensory deficits include general proprioceptive ataxia (manifested as incoordination and body sway side to side) and proprioceptive deficits. General proprioceptive ataxia is commonly known as spinal ataxia. If ataxia is noted, rule out other causes of ataxia, such as cerebellar or vestibular. To determine the “type” of ataxia, look at the rest of the neurologic status of the foal. Does the foal have cerebellar (hypermetria, intention tremors, and lack of menace response) or vestibular (pathologic nystagmus, head tilt, body lean, and circling in the direction of the head tilt) signs? If the answer is yes to one of these, then you can answer the question.

Motor deficits include paresis (i.e., paraparesis, hemiparesis, and tetraparesis) or paralysis (i.e., paraplegia, hemiplegia, and tetraplegia), UMN or LMN signs, dysmetria, hypermetria (UMN) or hy-pometria (LMN), and decreased muscle tone and weakness (more profound with LMN). Segmental reflexes might be decreased or absent if LMN are involved. Sensory and/or motor deficits will be observed depending on the location of injury within the spinal cord. For example, compressive myelopathies present with a combination of sensory and motor deficits, whereas neuroaxonal dystrophies primarily affects sensory tracts within the spinal cord in addition to brainstem nuclei.

**Peripheral Nervous System**

This includes all areas of the nervous system that are outside the brain and spinal cord. Note that all nerves are always peripheral (cranial nerves and spinal nerves). Therefore, it is redundant to say peripheral nerves. Cranial nerve deficits will be those pertaining to specific nerves (described above). Similarly, specific gait deficits will be observed depending which nerves are affected in the thoracic and pelvic limbs. The brachial and lumbosacral plexuses are also part of the peripheral nervous system (outside the spinal cord).

**Neuromuscular System**

The neuromuscular system has central (LMNs) and peripheral (nerve rootlets, roots, ganglia, nerves, and neuromuscular junction) components. Neuromuscular disorders can be diffuse or focal. Signs of diffuse neuromuscular disease include generalized weakness, difficulty supporting weight, paresis or
paralysis, muscle fasciculations, and a tendency to become recumbent. Dysphagia, dysphonia, laryngeal collapse, and dyspnea could be clinical manifestations of neuromuscular disease. Segmental (spinal) reflexes can be decreased or absent in neuromuscular disease. An example of a common diffuse neuromuscular disorder in foals is botulism. Electrolyte derangements can affect the function of nerves and neuromuscular junction. Ionized calcium and magnesium are particularly important for proper neurotransmission. Note that alterations in pH affect the binding of calcium and magnesium to proteins, resulting in a decrease or increase of available ionized calcium and magnesium. It is important to practice caution with the use of drugs and fluids that could further alter neuromuscular function if neuromuscular disease is present. For instance, hypermagnesemia can cause similar signs to botulism. Therefore, fluids containing magnesium might not be the ideal type of therapy for foals with botulism or other causes of neuromuscular weakness. Examples of drugs that might contribute to or exacerbate neuromuscular dysfunction include procaine penicillin, lidocaine, tetracyclines, aminoglycosides, erythromycin, and metronidazole, among others. Focal LMN disease or neuropathies lead to specific signs pertaining to the region affected, such as specific gait deficits, decreased muscle tone, decreased or absent tendon reflexes, and focal muscle atrophy.

Neuromuscular Disease of Critical Illness

Acquired neuromuscular dysfunction in the critical care setting is a recognized problem in human medicine, with an estimated prevalence of 46% in critical patients. Acquired disorders include critical illness myopathy, critical illness polyneuropathy, or a combination of both. These disorders are associated with sepsis, systemic inflammatory response syndrome, multi-organ dysfunction, and/or prolonged mechanical ventilation. The clinical hallmark of these is weakness. Weakness is a common clinical sign in diseased neonatal foals, and recognition of acquired neuromuscular disease might be challenging. An increased risk of acquired neuromuscular disease has been associated with hyperglycemia and mechanical ventilation for over 7 days. Dysregulation of calcium and magnesium concentrations might contribute to neuromuscular weakness. Such dysregulation has been reported in foals with septicemia, endotoxemia, and gastrointestinal disease.

6. What Is Next?

Once it is determined that the foal has a neurologic problem alone or concurrent with other illness, a diagnostic work up can be performed. The diagnostic approach should be tailored to the specific problem of the foal and what is going to provide more information or guidance for diagnosis, therapy, and prognosis in a low-risk and cost-effective way. It is not necessary to run all the tests available, and risks and benefits of certain diagnostic aids must be considered (e.g., procedures that require anesthesia may be contraindicated in severely compromised foals due to neuromuscular disease). A full description and indication of diagnostic modalities are beyond the scope of this text. A brief description is provided.

Full blood work (complete blood count, chemistry panel, blood gases, and pH), and urinalysis should be part of a minimum data base collection in critically ill foals. Although myopathies will not be discussed here, for the purpose of clarification, muscle enzymes within reference values do not rule out a myopathic disorder (e.g., hyperkalemic periodic paralysis disease and myotonia congenita). Neuromuscular disorders on which muscle enzymes may be elevated include ionophores, organophosphate toxicity, and those associated with tick infestation. Electrolyte analysis must also include ionized calcium (Ca++) and magnesium (Mg++) because they are physiologically active ions essential for neuromuscular homeostasis and function. Cerebrospinal fluid collection for cytology, biochemical panel, serology, neural biomarkers, PCR, and antimicrobial culture can be performed. Collection of cerebrospinal fluid must be performed closer to the lesion to increase the chances of finding abnormalities. Meningitis or meningencephalomyelitis in neonatal foals is usually caused by pathogens causing sepsis. Imaging such as radiography and ultrasonography can be easily performed in the field. Other imaging modalities include computed tomography and magnetic resonance that are available in some referral institutions. Electrophysiology, such as electroencephalography, brainstem auditory evoked responses, visual evoked potentials, retinography, repetitive nerve stimulation, and electromyography have been proven essential in the understanding of neurologic and neuromuscular disease. However, these techniques are only available in academic institutions.

Acknowledgments

Declaration of Ethics

The Author has adhered to the Principles of Veterinary Medical Ethics of the AVMA.

Conflict of Interest

The Author has no conflicts of interest.

References