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Brainstem Auditory Evoked Responses in an Equine Patient Population. Part II: Foals

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Background: Reports of the use of brainstem auditory evoked response (BAER) as a diagnostic modality in foals have been limited.

Hypothesis/Objectives: To describe BAER findings and associated causes of hearing loss in foals.

Animals: Study group 18 foals (15 neonatal, 3 nonneonatal), control group (5 neonatal foals).

Methods: Retrospective. BAER records from the Clinical Neurophysiology Laboratory were reviewed from the years of 1982 to 2013. Peak latencies, amplitudes, and interpeak intervals were measured when visible. Clinical data were extracted from the medical records. Foals were grouped under disease categories. Descriptive statistics were performed.

Results: Ten neonatal foals had complete absence of BAER bilaterally and 5 had findings within reference range. Abnormalities were associated with common neonatal disorders such as sepsis, neonatal encephalopathy, neonatal isoerythrolysis, and prematurity. BAER loss also was observed in foals with specific coat color patterns such as completely or mostly white with blue irides or lavender with pale yellow irides. An American Miniature foal with marked facial deformation also lacked BAER bilaterally. One nonneonatal foal with an intracranial abscess had no detectable BAER peaks bilaterally, and 2 older foals, 1 with presumed equine protozoal myeloencephalitis and the other with progressive scoliosis and ataxia, had BAER within normal limits.

Conclusions and Clinical Importance: In neonatal foals, BAER deficits commonly are complete and bilateral, and associated with common neonatal disorders and certain coat and eye color patterns. Sepsis, hypoxia, bilirubin toxicity, and prematurity should be investigated as potential causes of auditory loss in neonatal foals.

Key words: Deafness; Electrophysiology; Equine; Hearing.

Brainstem auditory evoked response (BAER) testing is an objective assessment of auditory function and localization of lesions within the auditory pathway.[1] Postnatal BAER varies among species.[1–7] Results are dependent on whether the species is precocial such as chicken, sheep, cattle, and horses, or altricial such as humans, monkeys, dogs, and cats.[3,4,7–11] In contrast to altricious species, precocious animals are born with fully formed brains, functional hearing, and functional vision.[10,11] Peer-reviewed publications in the English literature about the use of BAER testing in foals are limited to 3 studies.[7,12,13] Of these reports, only 2 include descriptions of BAER in neonatal foals.[7,12] One report described BAER performed in 4 healthy neonatal foals (48–104 hours old) and 6 neonatal foals (14–48 hours old) born to dams grazed on endophyte-infected fescue.[7] There were no differences in BAER peaks, latencies, and peak V/I amplitude ratios between healthy and fescue-exposed neonatal foals.[7] BAER reference values for neonatal foals were provided.[7] The second study investigated auditory function in American Paint horses.[12] This study included 3 neonatal foals diagnosed with lethal white foal syndrome (LWFS) which had absence of all peaks on BAER testing.[12] A different study reported a 2-month-old Appaloosa colt with multifocal brainstem disease presumed to be because of equine protozoal myeloencephalitis.[13] This colt had BAER within adult reference range.[13]

Bilateral hearing loss in human neonatal intensive care units has been reported to range from 2 to 13.4%.[14–16] However, when grouped under disease processes, hearing loss could range from 3 to 57%.[15] There is a lack of information regarding alterations in auditory function, as supported by BAER testing, and associated causes in foals. Furthermore, the effects of sepsis, perinatal hypoxia and marked hyperbilirubinemia on hearing have not been reported in neonatal foals. Therefore, the purpose of this study was to describe BAER findings and associated causes of hearing loss in foals (neonatal and nonneonatal) presented to a referral institution.

Abbreviations:

BAER	brainstem auditory evoked response
CM	congenital malformation
FPT	failure of passive transfer
JIE	juvenile idiopathic epilepsy
LFS	lavender foal syndrome
LWFS	lethal white foal syndrome
MYO	myoclonus
NE	neonatal encephalopathy
NI	neonatal isoerythrolysis
SND	sensorineural deafness

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Materials and Methods

Animals

This retrospective study included foals (neonatal and nonneonatal) of any breed that had BAER performed in the Clinical Neurophysiology Laboratory at the William R. Pritchard Veterinary Medical Teaching Hospital from the University of California at Davis between the years of 1982 and 2013. The BAER database included paper (1982–1998) and electronic (1999–2013) records. Data collected consisted of signalment, presenting complaint, physical and neurologic examination findings, BAER results, clinical or definitive diagnosis, and outcome. Data from 5 healthy neonatal Thoroughbred foals (3 colts, 2 fillies), 12–24 hours old were used as controls for the neonatal group. Published values from others also were used as reference.[7] For nonneonatal foals, BAER results from healthy adult horses were used as reference.[17]

Brainstem Auditory Evoked Response Testing

BAER testing was done according to laboratory protocols for equine BAER as described elsewhere.[17] Briefly, because of the duration of the study period, different evoked potentials systems[a–e] were used for recording BAER. Neonatal foals were evaluated in the intensive care unit and not sedated because of their disease. Nonneonatal foals were evaluated in the stall and sedated with xylazine hydrochloride at a dosage of 0.3–0.4 mg/kg IV. Head phones or earphones[f] were placed next to or deep into the external ear canals, respectively. Subcutaneous needle electrodes[g] were placed at the vertex (V) on midline, left mastoid, right mastoid, dorsal midline at the level of C2 vertebra (C2), and ground (Z) between the occipital protuberance and C2 (Fig 1A).[17] Specifications for BAER recording, and labeling of positive and negative peaks were the same as those described elsewhere.[17] Each BAER recording was the average of a minimum of 400 responses over a 10 ms epoch. An alternating (rarefaction plus condensation) broadband click stimulus at 90 decibels hearing level (dB HL) was applied. Noise masking sound for the contralateral side was done at 60 dB HL.[17] All BAER studies were done in duplicates and 2 derivations for testing were used simultaneously per ear: (1) vertex to ipsilateral mastoid (V-M); and (2) vertex to C2 (V-C2).[17]

Determination of normal versus abnormal BAER was based on the following measurements: Latency and amplitude for peaks I, III, and V measured in milliseconds (ms) and microvolts (μV), respectively; interpeak intervals for latency among peaks I–III, III–V, and I–V; and amplitude ratio determined by dividing peak V by peak I on the vertex to C2 derivation.[17] The recordings were considered abnormal and consistent with hearing loss as follows: Absence of identifiable BAER peaks was most consistent with complete hearing loss whereas increased peak latency (prolonged beyond 2 standard deviations [SD] from normal mean values) and difficulty identifying peaks (decreased amplitude) were suggestive of partial hearing loss.[17–19]

Statistical Analysis

Descriptive statistics (eg, mean, SD, and range) were calculated for the data obtained.

Results

Animals

Eighteen foals had BAER performed during the study period: 15 neonatal foals and 3 nonneonatal foals.

Neonatal Foals. Neonatal foals were Thoroughbred (n = 4), Quarter Horse (n = 3), White Overo (n = 3), Arabian (n = 2), and 1 each of Peruvian Paso, Welsh pony, and American Miniature. Their ages ranged from 12 to 96 hours old at the time of presentation. The most common complaints were marked lethargy, failure to nurse, weakness, and recumbency. Another common clinical sign was seizures. All 3 white overos were presented for apparent severe abdominal pain and distension, and lack of defecation. The diagnostic evaluation consisted of clinical pathology testing (CBC, serum biochemistry, and arterial blood gas analysis), blood culture, thoracic and abdominal ultrasound examination, and otoscopic examination. Eight of 15 foals had sepsis based on a positive blood culture (6/8 foals) or sepsis score >11 (8/8 foals).[20] Microbial organisms isolated by blood culture, or transtracheal wash, or both included *Staphylococcus* sp, *Klebsiella* sp, *Actinobacillus* sp, beta *Streptococcus, Pseudomonas,* and *Escherichia coli*. Atlanto-occipital cerebrospinal fluid centesis was performed in 6 of 8 foals with sepsis, and showed lymphocytic pleocytosis with increased protein concentration. With the exception of 2 foals in this group, those with sepsis had other concurrent problems.

Based on physical examination and initial diagnostic findings, neonatal foals were grouped by disease process as follows: LWFS (n = 3), neonatal encephalopathy (NE, n = 3), failure of passive transfer and sepsis alone (n = 2), prematurity (n = 2, 310, and 312 days gestational age respectively), neonatal isoerythrolysis (NI, n = 1), juvenile idiopathic epilepsy (n = 1), lavender foal syndrome (LFS, n = 1), myoclonus (n = 1), and multiple congenital malformations including facial deformation (n = 1). Of 8 foals with sepsis, 3/8 foals also were diagnosed with NE, 2/8 foals were premature, and 1/8 was an Arabian lavender filly that never stood up and presented with seizures (Table 1). A 4-day-old Thoroughbred colt with NI had a packed cell volume of 9% (reference range, 33.4–45%), serum total bilirubin concentration of 31.8 mg/dL (reference range, 1–3 mg/dL), indirect bilirubin concentration of 23.3 mg/dL (reference range, 0.6–2.6 mg/dL), direct bilirubin concentration of 8.5 mg/dL (reference range, 0.3–0.7 mg/dL), plasma lactate concentration of 8.22 mmol/L (reference range, 1–2 mmol/L), and SpO_2 in the range of 85–92% (reference range, 98–100%) on presentation. This foal was obtunded, dysphagic (lip and tongue incoordination), and had seizures. An electroencephalogram disclosed the presence of epileptiform discharges. The foal was discharged after 20 days of intensive care but remained dysphagic (nasogastric tube in place) and presented again 11 days later with profuse diarrhea and emaciation. The foal died from sepsis and typhlocolitis caused by *Salmonella typhimurium*. Outcome at discharge for all foals is shown in Table 1.

Nonneonatal Foals. Nonneonatal foals consisted of 3 foals: a 2-month-old Appaloosa colt presented with progressive multifocal brainstem disease and a presumptive diagnosis of equine protozoal myeloencephalitis caused

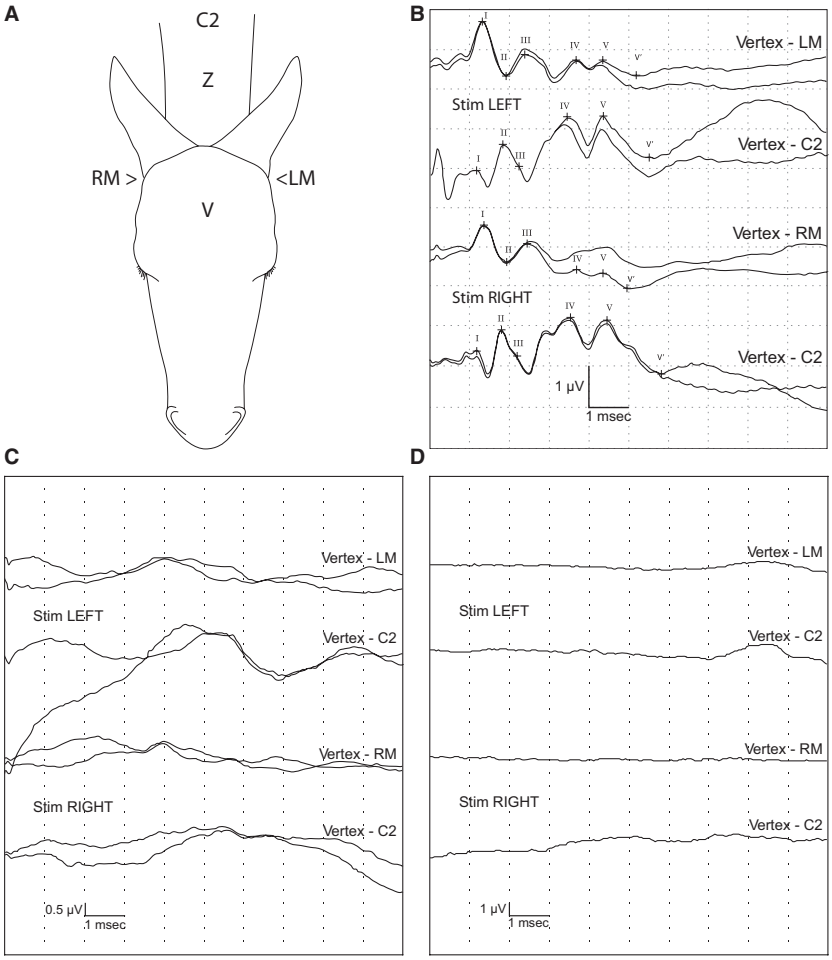

Fig 1. **(A)** BAER electrode placement. Derivations used: vertex to mastoid (left, right) and vertex to C2. V, vertex; RM, right mastoid; LM, left mastoid; C2, level of second cervical vertebra; Z, ground. Ear phones not shown. **(B)** BAER from a healthy neonatal foal. Study done in duplicates (shown), 2 derivations are shown (V-M, V-C2), top 2 tracings correspond to left ear stimulation, bottom 2 tracings correspond to right ear stimulation. Note peaks are labeled. **(C)** BAER from a neonatal foal with neonatal isoerythrolysis. Two derivations are shown, top 2 = left ear, bottom 2 = right ear. Note accurate identification of BAER peaks was not possible. **(D)** BAER from a neonatal foal with lethal white foal syndrome. Two derivations are shown, top 2 = left ear, bottom 2 = right ear. Note absent BAER bilaterally. Only one data set displayed.

by *Sarcocystis neurona* based on Western blot serology testing (the only test available at the time) and favorable response to treatment; a 7-month-old Quarter Horse filly presented for chronic progressive severe scoliosis, ataxia, and possible deafness; and, a 3-month-old Quarter Horse colt with an intracranial extra-axial abscess compressing the brainstem (Table 1). Outcome is shown on Table 1.

Brainstem Auditory Evoked Response Testing

Neonatal Foals. BAER testing was performed within hours to a few days after presentation. Ten neonatal foals had bilaterally absent BAER as follows: LWFS 3/3, NE 2/3, premature 2/2, NI 1/1, LFS 1/1, and 1 American Miniature foal with multiple congenital malformations (Table 1). None of the neonatal foals had unilateral BAER abnormalities, and 5 foals had BAER results within reference range (Table 2; Fig 1B). BAER data from diseased neonatal foals are not provided either because of absent BAER or peaks that were unidentifiable as shown in Figure 1C,D.

Nonneonatal Foals. Two foals had BAER within published reference ranges for adults,[17] and the colt with an intracranial abscess had no detectable BAER peaks bilaterally (Table 1).

Discussion

Hearing deficits occur in foals as identified in this study. Furthermore, complete absence of BAER was identified in neonatal foals with common neonatal disorders such as sepsis, NE caused by hypoxia, prematurity, and NI. Congenital or hereditary disorders such as multiple malformations and particular coat color patterns can be associated with auditory function impairment. Results of this study also emphasize the importance of performing a comprehensive neurologic

Table 1. Hearing loss in foals: neonates (top table), nonneonatal foals (bottom table). Note hearing loss in foals by disease group (bilateral versus unilateral). Column on the right shows number of foals with normal hearing.

Neonatal Foals (N = 15) Disease	Breed	Neurologic Signs (N = 15)	Bilateral (N = 10) BAER	Unilateral (N = 0) BAER	Normal (N = 5) BAER	Outcome
LWFS (N = 3)	White Overo	Ileus	Absent BAER (3)	0	0	EU (3)
NE (N = 3)	TB, QH, Welsh Pony	Obtundation to stupor, seizures, nystagmus, recumbency	Difficult to distinguish (2)	0	1	EU (2) S (1)
FPT/Sepsis (N = 2)	TB, QH	Obtundation, apparent weakness	0	0	2	S (2)
Premature (N = 2)	TB, QH	Stupor, seizures, nystagmus	Difficult to distinguish (2)	0	0	EU (2)
NI (N = 1)	TB	Obtundation, seizures, dysphagia	Difficult to distinguish	0	0	DIED (1)
JIE (N = 1)	Arabian	Seizures	0	0	1	S (1)
LFS (N = 1)	Arabian	Opisthotonus, nystagmus, seizures, tremors, stiffness, inability to stand	Absent BAER	0	0	EU (1)
MYO (N = 1)	Peruvian Paso	Recumbency, tremors, myoclonus	0	0	1	EU (1)
CM (N = 1)	Am Miniature	Obtundation, stiffness, dysmetria	Difficult to distinguish	0	0	EU (1)
Total (N = 15)		15	10	0	5	EU (11) S (4)

Nonneonatal Foals (N = 3) Disease	Signalment	Neurologic Signs (N = 3)	Bilateral (N = 1) BAER	Unilateral (N = 0) BAER	Normal (N = 2) BAER	Outcome
EPM suspect (N = 1)	2 m Appaloosa Colt	Obtundation, multiple cranial nerves deficits	NA	NA	1	S
Brain Abscess (N = 1)	3 m QH Colt	Stupor, seizures, multiple cranial nerves deficits	1	NA	NA	EU
Scoliosis (N = 1)	7 m QH Filly	Ataxia, asymmetrical tetraparesis, dysmetria, muscle atrophy	NA	NA	1	EU
Total (N = 3)		3	1	0	2	EU (2) S (1)

LWFS, lethal white foal syndrome; NE, neonatal encephalopathy; FPT, failure of passive transfer; NI, neonatal isoerythrolysis; JIE, juvenile idiopathic epilepsy; LFS, lavender foal syndrome; MYO, myoclonus of Peruvian Paso; CM, congenital malformations; difficult to distinguish, difficult to distinguish BAER peaks; S, survived; EU, euthanasia; m, age in months; NA, not applicable.

examination in foals, particularly in neonatal foals presenting to the intensive care unit because many neonatal disorders have similar signs.[21] Alterations in behavior and states of consciousness, absent or weak suckle reflex, weakness, and recumbency are common clinical signs.[21] Assessing the level of hearing based on behavioral response would likely be difficult in critically ill neonatal foals because of the state of their disease. Furthermore, the effects of sepsis, hypoxia, or marked hyperbilirubinemia on hearing have not been described or evaluated in foals. As the first step in the investigation of hearing loss by BAER studies, reference ranges must be established in healthy animals according to age, technique, and derivation used.[1,22] Other important considerations include breed and sex, which were not evaluated in this study because of the low numbers of foals.[23]

This study also provided reference values for BAER testing in neonatal foals using 2 different derivations: vertex to mastoid and vertex to C2. There is a single BAER study in which reference values from 4 neonatal foals were reported using the vertex to mastoid derivation.[7] In addition, BAER thresholds were investigated from 10 to 90 dB sound pressure level (SPL).[7] Distinct peaks (I–III, V) were seen in 4 healthy and all 6 fescue-exposed neonatal foals at stimulus intensities of 70 and 90 dB SPL.[7] Peaks also were visible at 50 dB SPL in some foals, and, at lower stimuli, the majority of foals did not have identifiable peaks.[7] When tested at stimulus levels of 90 dB SPL, most of the BAER latencies in foals were within 3 SD of the reported values for adult horses tested at the same stimulus intensities.[7] Therefore, the authors concluded that peak latencies in neonatal foals can be compared to those of adult horses.[7,24,25] Our findings concur with these observations. However, BAER thresholds might be necessary to detect more subtle alterations in auditory function.

Eight of 15 neonatal foals had sepsis. Of these, 6 foals had other concurrent problems which included

Table 2. BAER reference values for neonatal foals recorded after stimulating at 90 dB HL with head phones.

	Latency (ms)						Amp (μV)
Neonates	I	III	V	I–III	III–V	I–V	V/I
V-M							
Controls (N = 5) 90 dB HL	1.36 (0.05)	2.31 (0.36)	4.41 (0.09)	0.95 (0.26)	2.10 (0.25)	3.06 (0.07)	NA
Steiss 1991 (N = 4) 90 dB SPL	1.44 (0.05)	3.77 (0.26)	4.64 (0.17)	NA	NA	NA	0.56 (0.21)
V-C2							
Controls (N = 5) 90 dB HL	1.2 (0.05)	2.19 (0.26)	4.56 (0.18)	0.98 (0.24)	2.53 (0.54)	3.36 (0.13)	3.31 (1.2)

Note reference values were obtained using different measurements of sound level (HL versus SPL).

Controls, UCD healthy foals; Steiss 1991, Study by Steiss in neonatal foals using 90 dB SPL (reference values not available for the V-C2 derivation)[7]; NA, not available or not applicable.

NE because of hypoxic ischemia (n = 3), prematurity (n = 2), and LFS (n = 1). Cerebrospinal fluid cytology showed inflammation in 6 foals but was not consistent with septic meningitis. However, sampling at early stages of disease might play a role in cytologic findings.[26] Sepsis and meningitis have been reported to be causes of hearing loss in newborn infants.[15,16] In a review of neurodevelopmental dysfunction in newborn infants, hearing loss was detected in 10 and 12% of newborns with sepsis and meningitis, respectively.[15,16] Two neonatal foals diagnosed with sepsis alone had BAER within reference ranges. Five of 6 foals with sepsis and other concurrent problems had no identifiable BAER bilaterally.

Two of 3 foals with NE that suffered from hypoxic ischemia in the perinatal period had no identifiable BAER. The remaining foal had normal BAER. Neurologic disabilities after intrapartum hypoxic ischemia in newborn infants include cerebral palsy, learning and memory disabilities, epilepsy, attention disorders, and visual and hearing impairment.[27] Some neonatologists believe that hypoxia damages the hair cells of the cochlea,[14] others have reported that the prevalence of hearing impairment in newborn infants with NE because of hypoxia is not substantially higher than in those without NE.[28] However, results from a recent review of 27 studies reported hearing loss in 9% (n = 291/2,708) of newborn infants with hypoxic ischemic encephalopathy.[15] Because of severity of disease and lack of response to intensive care, 2 foals were euthanized. The third foal with normal BAER findings was discharged from the hospital.

Two premature foals in this study were found to have absent BAER bilaterally. However, these foals also were septic, and whether prematurity or sepsis was the cause of hearing loss was unknown. It is also unknown at what stage of maturation in utero the auditory system becomes fully functional in foals. Birth weight in newborn infants is an indicator of biologic maturity.[29] Two of 3 infants with low birth weight are premature.[29] Extremely premature babies (<32 weeks of gestation) or infants with very low birth weight (<1,500 g) regardless of gestational age are considered a population at higher risk for sensorineural hearing loss.[29] The prevalence of childhood sensorineural hearing loss among premature newborn infants from several studies was 7%.[15]

A 4-day-old Thoroughbred colt with NI and marked hyperbilirubinemia (31.8 mg/dL) had unidentifiable BAER peaks in both ears. Bilirubin encephalopathy, more commonly known as kernicterus, is an important cause of neurologic dysfunction and permanent brain damage in newborn infants.[30] In a study of 72 foals with NI, 18 foals did not survive.[31] Of these, 9 foals had severe neurologic signs (2 died, 7 euthanized).[31] Histologic evaluation of the brain was consistent with kernicterus and hepatic encephalopathy in 6 and 1 neonatal foals, respectively, and no lesions were observed in 2 foals.[31] None of the survivors displayed neurologic signs.[31] The globus pallidus, subthalamic nucleus, auditory pathway, and oculomotor brainstem nuclei are most vulnerable to bilirubin toxicity in newborn infants.[30] Other areas affected by bilirubin include the cerebellar Purkinje cell layer and hippocampus.[30] The affected areas in foals with NI comprise the cerebral cortex, hippocampus, and cerebellar Purkinje layer.[31] In the foal in our study, no histologic lesions were identified in the brain. However, the precise sites of histologic evaluation were not specified in the report. Therefore, inappropriate selection of samples could have resulted in failure to identify lesions. Foals with kernicterus had reported total serum bilirubin concentrations that ranged from 19 to 41 mg/dL.[31] Furthermore, the authors concluded that foals with total bilirubin concentrations ≥27 mg/dL were 17 times more likely to develop kernicterus than foals with lower concentrations.[31] In newborn infants with bilirubin encephalopathy, motor, auditory, and cognitive dysfunction are the most common permanent abnormalities.[30] In the acute stages, lethargy, ophthalmoplegia, high pitched vocalization, opisthotonus, and seizures are the most common signs of kernicterus.[30] Although neurologic signs vary depending on whether bilirubin toxicity is acute or chronic, one of the cardinal signs is auditory impairment, which can range from subtle to complete lack of hearing.[30,32] This study reports for the first time auditory dysfunction in a colt with marked hyperbilirubinemia. However, hypoxia

(because of profound anemia) may also have been a factor. In addition to BAER abnormalities in this colt, other signs such as obtundation, seizures, and dysphagia (because of incoordination of lips and tongue) could be attributed to bilirubin toxicity.[30] The basal nuclei, which include the globus pallidus, affect motor activity of the lips and tongue, functions that this colt did not regain.[33]

Hereditary and congenital disorders in this study comprised 3 neonatal foals with LWFS, 1 Arabian filly with LFS, and 1 American Miniature colt with multiple congenital malformations including a facial deformation that occluded the internal and external acoustic meatus. Hereditary and congenital disorders have been reported to be a major cause of comorbidities in newborn infants.[16] These comorbidities include hearing loss.[16] Congenital sensorineural deafness associated with a genetic mutation in the endothelin B receptor (*EDNBR*) gene was identified in 3 white overo neonatal foals in this study.[12,34,35] This receptor plays an essential role in neural crest development.[34–36] Melanocytes are derived from the neural crest and migrate to the skin and inner ear.[36] Therefore, alterations in this receptor can result in pigmentation and hearing impairment.[35] Horses with certain color patterns can be carriers of the mutation, such as paints with extensive white areas and white faces and heterochromic or blue irides.[12] The foal with LFS also was septic, and whether absence of BAER was associated with the coat color dilution, sepsis, or both was unclear. However, hearing loss has not been reported in foals with LFS, but warrants investigation.[37] The disorder is caused by a myosin 5A (*MYO5A*) genetic mutation with an autosomal recessive mode of inheritance in Egyptian Arabian horses.[38] Affected foals are homozygous for the mutation.[38] *MYO5A* gene mutation results in a frame shift and premature termination of transcription of the secretory vesicle-specific binding domain of the protein involved in vesicle traffic.[38] This loss of vesicle traffic can interfere with the normal function of melanocytes and neurons.[38]

In conclusion, foals are born with a functional auditory system comparable to that of adult horses when tested at 90 dB HL. Brain insults in the perinatal period such as those associated with sepsis, hypoxic ischemia, and marked hyperbilirubinemia should be considered as potential causes of hearing loss in neonatal foals. Prematurity could result in a nonfunctional auditory system similar to that reported in premature infants.[16] Furthermore, neonatal foals are commonly presented with a combination of problems possibly increasing the risk of hearing impairment. Congenital or inherited disorders such as those associated with multiple malformations and coat color patterns also should be considered as potential associations with hearing loss. Subjective evaluation and interpretation of hearing in the critically ill neonatal foal can be difficult and inaccurate. BAER testing should be considered in patients with suspected neurologic disease, especially if signs localize to the brain (ie, multifocal brainstem disease), vestibular system (peripheral or central), ear (outer, middle, or inner), or some combination of these. Similar to adult horses, BAER also is indicated in foals with altered behavior, such as being easily startled, particularly if these signs occur in combination with certain coat and eye color patterns. As reported horses and other species, potentially ototoxic drugs can result in hearing impairment and should be avoided or used cautiously in foals.[16,39] BAER is a noninvasive and objective diagnostic technique to assess hearing in foals with neurologic disease and suspected hearing deficits. Prospective studies to investigate auditory function in diseased foals, especially neonates, are needed. Such studies will aid in the understanding of disease processes, provide useful information to the owners with regard to special training needs and precautions because of auditory impairment, and help owners make financial decisions.

Footnotes

[a] DANTEC Electronics; Inc, Allendale, NJ
[b] TecaEP40/ST10; Teca Corp, Pleasantville, NY
[c] Nihon Kohden Neuropack; Neuro Medical Equipment, Inc, Arlington, TX
[d] Viking IVD; Nicolet Biomedical Inc, Madison, WI
[e] VikingQuest; Nicolet Biomedical Inc
[f] TIP 300; Nicolet Biomedical, Inc
[g] FE-2; Grass/Astro-Med Inc, West Warwick, RI

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