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Brainstem Auditory Evoked Responses in an Equine Patient Population: Part I – Adult Horses

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Background: Brainstem auditory evoked response has been an underused diagnostic modality in horses as evidenced by few reports on the subject.

Hypothesis/Objectives: To describe BAER findings, common clinical signs, and causes of hearing loss in adult horses.

Animals: Study group, 76 horses; control group, 8 horses.

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. Horses were grouped under disease categories. Descriptive statistics and a posthoc Bonferroni test were performed.

Results: Fifty-seven of 76 horses had BAER deficits. There was no breed or sex predisposition, with the exception of American Paint horses diagnosed with congenital sensorineural deafness. Eighty-six percent (n = 49/57) of the horses were younger than 16 years of age. The most common causes of BAER abnormalities were temporohyoid osteoarthropathy (THO, n = 20/20; abnormalities/total), congenital sensorineural deafness in Paint horses (17/17), multifocal brain disease (13/16), and otitis media/interna (4/4). Auditory loss was bilateral and unilateral in 74% (n = 42/57) and 26% (n = 15/57) of the horses, respectively. The most common causes of bilateral auditory loss were sensorineural deafness, THO, and multifocal brain disease whereas THO and otitis were the most common causes of unilateral deficits.

Conclusions and Clinical Importance: Auditory deficits should be investigated in horses with altered behavior, THO, multifocal brain disease, otitis, and in horses with certain coat and eye color patterns. BAER testing is an objective and noninvasive diagnostic modality to assess auditory function in horses.

Key words: Deafness; Electrophysiology; Equine; Hearing.

Brainstem auditory evoked response (BAER) testing evaluates the integrity of the auditory pathway.[1] The normal BAER consists of a sequence of 5–7 peaks, named as I to VII which occur during the first 10 milliseconds after the onset of a stimulus.[1,2] Each peak is thought to represent the depolarization of specific nuclei along this pathway.[1] Reference values for horses, ponies, and neonatal foals have been reported.[3–7] It is important to note the technique (vertex to mastoid versus vertex to C2 vertebra) and equipment (head phones versus insert ear phones [ie, some models have a length of tubing between the sound source and site of delivery which increases peak latencies]) used for the recording of BAER for the comparison of results with those of reference values.[2,7,8] Further considerations include whether BAER testing is performed under sedation or general anesthesia.[3–5] Abnormalities in the BAER are consistent with hearing loss (deafness), which could be complete or partial and acquired or congenital.[1,9] Failure of conduction of sound waves from the ear canal to the inner ear is termed conductive deafness whereas alterations of the neural structures of the auditory pathway result in sensorineural deafness.[9]

Abbreviations:

BAER	brainstem auditory evoked response
SND	sensorineural deafness
THO	temporohyoid osteoarthropathy
VD	vestibular disease

Despite the ease and noninvasiveness of the procedure, the use of BAER as a diagnostic modality in horses has been limited to a few reports which include congenital sensorineural deafness of American Paint horses, THO, presumed gentamicin toxicity, head trauma, and brain lesions.[6,7,10–20] Furthermore, there is a lack of large scale studies to gain information about hearing loss in horses and determine factors such as breed predisposition, associated clinical signs, and the occurrence of complete versus partial and bilateral versus unilateral hearing loss. The purpose of this study was to review and report BAER results, signalment, clinical signs, causes of hearing loss, and outcome. This study included horses ≥1-year old. Foals were not included because diseases of foals (especially neonatal foals) may differ from those of adult horses.

Materials and Methods

Animals

This retrospective study included all horses of any breed and ≥1 year of age that had a BAER evaluation performed in the Clinical Neurophysiology Laboratory at the William R. Pritchard Veterinary Medical Teaching Hospital of the University of

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California at Davis between 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. BAER data (mean and standard deviation of peak latencies) from 8 healthy adult horses (5–20-year old) of Quarter Horse, Thoroughbred, and Warmblood breeds were used as controls.[7] Horses under study were grouped into disease categories for comparison of BAER data with that of control horses.

Brainstem Auditory Evoked Response Testing

Brainstem auditory evoked response testing was done according to laboratory protocols for equine BAER and as described elsewhere.[7] Briefly, because of the duration of the study period, different evoked potentials systems[a–e] were used for recording BAER. Horses were placed in examination stocks and sedated with xylazine hydrochloride at a dosage of 0.3–0.4 mg/kg IV. For most studies, insert earphones[f] were placed deep into the external ear canal (for early recordings, headphones were used). Subcutaneous needle electrodes[g] were placed at the vertex (V), left mastoid (LM), right mastoid (RM), and on the dorsal midline at the level of C2 vertebra (C2) for recording the BAER (Fig 1A). For some evoked potential systems, an additional electrode was placed between the occipital protuberance and C2 to serve as the ground (Z)[7], whereas with others, the contralateral mastoid served this purpose (Fig 1A). Specifications for BAER recording and labeling of peaks were the same as those described elsewhere.[7] Each BAER recording was the average of a minimum of 400 responses over a 10 milliseconds epoch. An alternating (rarefaction plus condensation) broadband click stimulus at 90 dB normal hearing level (nHL) was applied individually to each ear. A simultaneous masking sound was used on the contralateral side with an offset of −30 dB nHL.[7] All BAER studies were done in duplicate and 2 derivations were recorded simultaneously: (1) vertex to ipsilateral mastoid (V-M) and (2) vertex to C2 (V-C2).[7]

Determination of normal versus abnormal BAER was based on the following measurements: Latency and amplitude for peaks I, III, and V measured in milliseconds and microvolts (μV), respectively; interpeak intervals (IPI) for latency among peaks I–III, III–V, and I–V; and, amplitude ratio by dividing peak V by peak I on the vertex to C2 derivation.[7] The study was considered abnormal and categorized as follows: absence of identifiable BAER peaks was consistent with complete hearing loss whereas increased peak latency (prolonged beyond 2 standard deviations [SD] from normal mean values),[21] difficulty identifying peaks (decreased amplitude) or both was suggestive of partial hearing loss.[7,21,22]

Statistical Analysis

Descriptive statistics for age (mean, range) and BAER data (mean, SD) were calculated. Horses were grouped under disease categories for further comparison of data. To compare BAER data from healthy horses to that of diseased horses, a one-way ANOVA was performed; if statistical differences ($P < .05$) were found, a posthoc Bonferroni test was done to determine statistical differences between groups.

Results

Animals

Seventy-six horses had BAER performed during the study period. The horses were of Thoroughbred (n = 22), Quarter Horse (n = 20), American Paint (n = 15), Warmblood (n = 6), Arabian (n = 5), Appaloosa (n = 3), Tennessee Walker (n = 2), Morgan (n = 1), Saddlebred (n = 1), and American Miniature (n = 1) breeds. There were 35 mares, 28 geldings, and 13 intact males. Their ages ranged from 1 to 28 years (mean, 8.5 years; median, 7 years). The most common presenting complaint varied with disease and included altered behavior, such as startling with environmental stimuli (n = 22), leaning to 1 side (n = 22), head tilt (n = 20), undefined gait deficits (n = 20), corneal ulceration (n = 15), and dysphagia (n = 15). Other complaints included headshaking (n = 3), inability to stand (n = 3), seizures (n = 2), and collapse (n = 2). The complaint was not recorded in 15 horses. Suspected hearing loss was reported in 12 horses (10 American Paint and 2 paint Warmblood horses).

Physical and neurologic examination showed that the most common clinical signs were those of suspected hearing loss (n = 32; startling when approached, not turning head or ears toward the source of a loud noise), vestibular disease (n = 32; head tilt, nystagmus, positional strabismus, leaning to one side, ataxia), obtundation (n = 19), multiple cranial nerve deficits (n = 16; mastication deficits with or without masticatory muscle atrophy, facial paresis or paralysis with or without vestibular signs, dysphagia, tongue paresis), ataxia not associated with vestibular disease (n = 12), and less common clinical signs such as headshaking (n = 6), recumbency (n = 3), and stupor (n = 1). Diagnostic evaluation consisted of clinical pathology testing (CBC, serum biochemistry, and cerebrospinal fluid cytology and chemistry), otoscopic examination, imaging of the head (radiographs, computed tomography, or both), and in some cases screening for infectious pathogens and microbial culture of tissues such as blood, fluid obtained from the tympanic cavity, and cerebrospinal fluid.

Based on physical and neurologic examination results (neuroanatomic localization) and diagnostic evaluation, horses were grouped into one of the following categories: multifocal brain disorder with or without spinal cord involvement, vestibular disease (without other cranial nerves deficits and not associated with temporohyoid osteoarthropathy [THO]), THO, sensorineural hearing loss (without other neurologic deficits and no evidence of otitis), otitis (externa, media, interna or some combination of these), and other categories such as bilateral spontaneous otoacoustic emissions (SOAEs) and neurologic deficits not localized to the brain or cranial nerves (see Table 1).

Of 16 horses with multifocal brain disease, 5 had infectious encephalomyelitis with or without meningeal involvement. Of these 5 horses, 2 were diagnosed with equine protozoal myeloencephalopathy because of *Sarcocystis neurona* (presumptive diagnosis based on high antibody titers on immunofluorescent antibody test), 1 with *Halicephalobus gingivalis* meningoencephalomyelitis, 1 with *Listeria monocytogenes*, and 1 with mixed bacterial meningoencephalomyelitis. Four of 16 horses

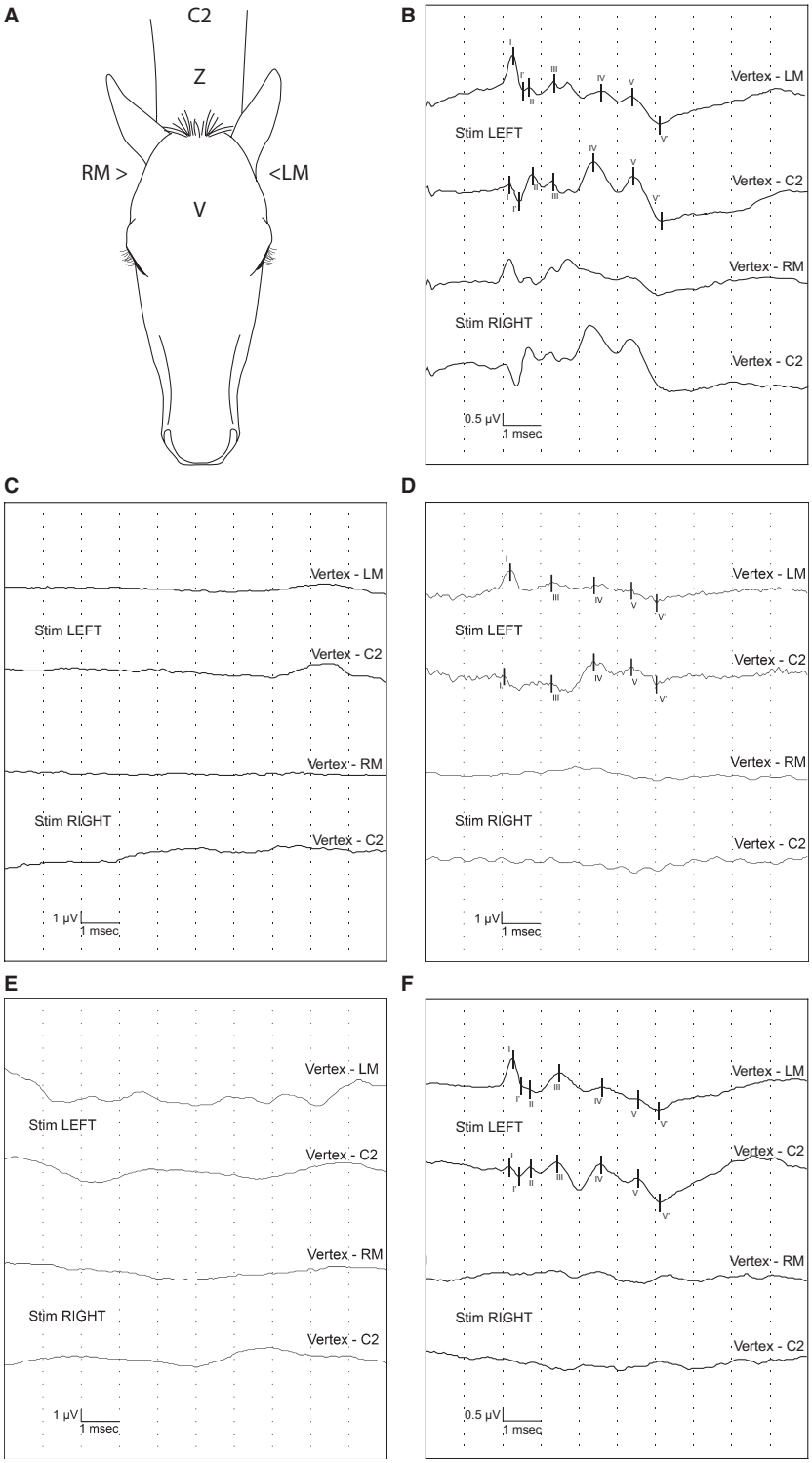

Fig 1. (**A**) BAER electrode placement. Derivations used: vertex to mastoid (left, right) and vertex to C2. V, vertex; RM, right mastoid; LM, left mastoid; C2, level of second cervical vertebra; and Z, ground. Ear phones not shown. (**B**) Normal BAER in healthy horse.[7] BAER peaks and troughs labeled (I, I', II, III, IV, V, V'). Top 2 tracings = stimulation of left ear, bottom 2 tracings = stimulation of right ear; LM, left mastoid; C2, level of second cervical vertebra, bar as indicated. (**C**) Horse with congenital sensorineural deafness. No BAER peaks in either ear. Top 2 tracings = left ear (V-LM, V-C2), bottom 2 tracings = right ear (V-RM, V-C2). (**D**) Horse with multifocal brain disease. Peak ratio V/I was less than 1.5 on the left ear, and there was no BAER on the right ear. Top 2 tracings = left ear (V-LM, V-C2), bottom 2 tracings = right ear (V-RM, V-C2). (**E**) Horse with multifocal brain disease. Bilateral BAER loss: difficult to identify peaks on the left ear (therefore not marked) and none on the right ear. Top 2 tracings = left ear (V-LM, V-C2), bottom 2 tracings = right ear (V-RM, V-C2). (**F**). BAER from a horse with THO. Top 2 tracings = left ear (V-LM, V-C2), bottom 2 tracings = right ear (V-RM, V-C2).

Table 1. Signalment and clinical diagnosis by disease group.

Horses (N = 76)	Breed	Sex			Age (Years)	Signs		
Group	Most Common	Female	Gelding	Stallion	Mean (Range)	Most Common	Clinical Diagnosis	Outcome
THO (N = 20)	QH (10)	10	8	2	14 (9–19)	Vestibular, facial paresis/ paralysis, ulcerative keratitis	Temporohyoid osteoarthropathy	S (16) EU (4)
SND (N = 17)	American Paint Overo (15)	9	6	2	4 (1–8)	Behavior, startle, suspected hearing loss	Sensorineural deafness associated to coat color	S (17)
Brain (N = 16)	NP	8	6	2	9 (1–27)	Obtundation, multiple cranial nerve deficits, tetraparesis, proprioceptive deficits	Infectious (+/- meningo) encephalomyelitis (5), intracranial masses (4), Undetermined (4), TBI (3)	S (4) EU (12)
VD (N = 11)	TB (8)	4	4	3	7 (1–28)	Vestibular	Undetermined (9), TBI (1), suspected EPM (1)	S (10) U (1)
Otitis (N = 4)	NP	2	1	1	11 (3–20)	Headshaking, head tilt	Otitis externa/media/ interna	S (1) EU (3)
Others (N = 8)	NP	2	3	3	5 (1–10)	Variable	Spinal cord disease (3), altered behavior (3), spontaneous otoacoustic emissions (1), idiopathic headshaking (1)	S (8)
Total (N = 76)	NA	35	28	13	8.5 (1–28)	Startle, vestibular	NA	S (55) EU (19) U (2)

THO, temporohyoid osteoarthropathy; SND, sensorineural deafness; brain, multifocal brain disease; VD, vestibular disease (not associated with other BS deficits or THO); others, various disorders, TBI, traumatic brain injury; EPM, equine protozoal myeloencephalopathy; NP, no predisposition; NA, not applicable; S, survivors; EU, euthanasia; U, unknown.

had intracranial masses: 2 had melanoma in the brainstem, 1 had a large glioblastoma multiforme in the piriform lobe with severe compression of the brain, and 1 had an abscess in the brainstem and otitis media and interna. Three of 16 horses suffered from traumatic brain injury, had sustained skull fractures, and had hematomas compressing the brainstem. In 4 horses, the cause of the multifocal brainstem deficits was unknown. The outcome at discharge for all 76 horses is shown in Table 1.

Brainstem Auditory Evoked Response Testing

Based on BAER testing, 57 horses had hearing deficits and 19 horses had BAER findings within reference range (Fig 1B). Hearing deficits were bilateral in 42 and unilateral in 15 horses (Table 2). Complete bilateral hearing loss was found in 19 horses of which 15 were American Paint horses (Fig 1C) and 2 were paint Warmbloods with white faces and heterochromic irides. The 2 remaining horses lacking BAER bilaterally were 1 horse with chronic severe bilateral otitis and 1 American Miniature horse with facial deformation. Bilateral partial hearing deficits were seen in 23 horses: 11 horses were diagnosed with multifocal brain disease (Fig 1D, E), 10 with THO, 1 with vestibular disease not associated with THO, and 1 with undetermined etiology. The common causes of unilateral hearing loss were THO (n = 10, Fig 1F), otitis media and interna (n = 3), and multifocal brainstem disease (n = 2). See Table 2.

For statistical purposes, only abnormal BAER results from disease groups were used for comparison with control horses. Statistical differences in both derivations (V-M, V-C2) were found for latency of waves I, III, and V between controls and horses with THO but not between controls and horses with multifocal brain disease (Table 3). Statistical differences were found for amplitude ratios between horses with THO and multifocal brain disease but not when each group was compared with controls (Table 3). Because all 17 horses with SND were completely deaf and 10 of 11

Table 2. Findings by disease group based on BAER testing.

Horses (N = 76)	Bilateral (N = 42)			Unilateral (N = 15)		
Group	Complete	Partial	C/p	R	L	Normal (N = 19)
THO (N = 20)	0	0	10	7	3	0
SND (N = 17)	17	0	0	0	0	0
Brain (N = 16)	0	9	2	1	1	3
VD (N = 11)	0	1	0	0	0	10
Otitis (N = 4)	1	0	0	2	1	0
Others (N = 8)	1	0	1	0	0	6
Total (N = 76)	19	10	13	10	5	19

Complete, bilateral loss of all BAER peaks; partial, bilateral partial hearing loss; C/P, bilateral hearing loss with total absence of BAER peaks in 1 ear; R, right ear; L, left ear.

horses with vestibular disease had BAER within reference values, no comparisons of these groups with control horses were made. Comparison of data from control horses with that of the remaining groups was not possible either because of small group size (horses with otitis [n = 4] which had no detectable BAER in the affected ear) or miscellaneous disorders not suitable for comparison.

Discussion

This study highlights the importance of performing BAER examination as part of the diagnostic evaluation in horses with suspected hearing loss manifested by altered behavior (startling easily, lack of responsiveness to loud sounds, or difficulty training), otitis (head shaking, head or ear rubbing, head tilt), cranial nerve deficits as in horses with THO (mainly CN VII, VIII), peripheral or central vestibular disease, and diffuse or multifocal brain disease. Furthermore, examination of hearing in horses with certain coat and eye color patterns is warranted. Failure of embryonic melanocyte migration from the neural crest to the stria vascularis of the cochlea resulting in hair cell degeneration in the organ of Corti has been associated with particular phenotypes.[23] The most common causes of BAER abnormalities in this study were THO, congenital sensorineural deafness, multifocal brain disease, and otitis. The most common diagnosis associated with euthanasia in this study was multifocal brain disease. The majority of horses with other disorders survived to discharge.

Table 3. BAER in healthy and diseased horses.

	Latency (milliseconds)						Amp (μV)
Horses	I	III	V	I–III	III–V	I–V	V/I
V-M 90 dB HL							
Controls (N = 8)	2.21 (0.06)[a]	3.61 (0.17)[a]	5.36 (0.16)[a]	1.40 (0.15)[a]	1.74 (0.15)[a]	3.14 (0.12)[a]	NA
THO (N = 20)	2.62 (0.59)[b]	4.01 (0.52)[b]	5.93 (0.54)[b]	1.39 (0.26)[b]	1.92 (0.11)[a]	3.31 (0.22)[a]	NA
Brain (N = 16)	2.35 (0.19)[a,b]	3.79 (0.15)[a,b]	5.8 (0.59)[a,b]	1.5 (0.15)[a]	1.98 (0.63)[a]	3.48 (0.64)[a]	NA
VD (N = 11)	WNL except for 1 horse						
SND (N = 17)	No BAER	No BAER	No BAER	No BAER	No BAER	No BAER	No BAER
Otitis (N = 4)	No BAER in affected ear						
Others (N = 8)	Variable not shown						
V-C2 90 dB HL							
Controls (N = 8)	2.11 (0.06)[a]	3.62 (0.17)[a]	5.38 (0.12)[a]	1.45 (0.14)[a]	1.74 (0.17)[a]	3.22 (0.10)[a]	2.75 (1.46)[a,b]
THO (N = 20)	2.63 (0.68)[b]	4.01 (0.53)[b]	5.91 (0.51)[b]	1.38 (0.27)[a]	1.9 (0.11)[a]	3.28 (0.23)[a]	4.37 (2.42)[a]
Brain (N = 16)	2.24 (0.11)[a,b]	3.81 (0.17)[a,b]	5.65 (0.75)[a,b]	1.56 (0.13)[a]	1.98 (0.63)[a]	3.55 (0.59)[a]	1.60 (0.55)[b]
VD (N = 11)	WNL except for 1 horse						
SND (N = 17)	No BAER	No BAER	No BAER	No BAER	No BAER	No BAER	No BAER
Otitis (N = 4)	No BAER in affected ear						
Others (N = 8)	Variable not shown						

V-M, vertex to mastoid; V-C2, vertex to level of C2 vertebra; N, number of horses; WNL, within normal limits; No BAER, no identifiable BAER (absent). Data are presented as mean (SD). Different superscripts between groups represent significant difference, and same superscripts represent no statistical difference. Note, ear inserts add 0.9 milliseconds to latencies (I, III, V).

Underestimation of the number of hearing abnormalities within the hospital's equine patient population likely occurred with this study, given that only horses that underwent BAER testing were included. Medical records were screened using the words "deafness" and "hearing loss", and 4 additional horses were identified: 2 American Paint horses and 2 Quarter Horses with multifocal brainstem disease but BAER had not been performed. Subjective evaluation of hearing (response to sound) as part of the neurologic examination was seldom recorded in the medical records. This could have resulted in failure to identify more horses with hearing deficits. Furthermore, the fact that horses respond to loud sound does not rule out hearing deficits, such as partial loss or unilateral deficits, as described previously in horses with THO.[7] Appropriate localization of sound requires intact bilateral hearing.[24] To assess hearing subjectively, it is important to determine if the animal is capable not only of responding but also of orienting toward the source of the sound. Clinicians' approach toward the examination of horses with neurologic disease also could have resulted in underestimation of hearing loss. In this study, BAER testing occurred more often when one of the authors (MA) was the primary or consulting clinician on the case.

Because the most popular breeds seen at our institution include Quarter Horses, QH-related breeds, and Thoroughbreds, an overall breed predisposition was not observed in this study. However, when these horses were grouped under disease categories, American Paint horses (15 of 15) were overrepresented in the group of congenital sensorineural deafness (15 of 17 horses). These American Paint horses had splashed white frame coat patterns with extensive head and limb white markings, and blue or heterochromic irides. These horses were tested and found to have the endothelin B receptor (*EDNBR*) mutation associated with overo lethal white foal syndrome.[12,25,26] This gene plays an essential role in neural crest development.[23] Melanocytes are derived from the neural crest and migrate to the skin and inner ear.[23] Therefore, alteration in the migration, differentiation, or function of melanocytes results in pigment alterations and deafness.[23] The remaining 2 horses were Warmblood breeds with a paint coat color, white markings of the face, and heterochromic irides. These horses were not tested for the genetic mutation. A study performed at our institution concluded that congenital sensorineural deafness may exist in some but not all Paint horses with particular coat color patterns such as splashed white overo, frame overo, tovero, and overo-blend[12] particularly those with extensive white markings on the face and limbs and those with heterochromic or completely blue eyes.[12] Such horses should be tested for the *EDNBR* mutation.[12]

Although a predisposition for sex or age group was not observed, most horses were young adults at the time of BAER testing (mean 8.5; median, 7-year old). However, some of these horses had signs compatible with hearing loss at an earlier age. Examples of these included American Paint horses with congenital sensorineural deafness and horses with chronic THO. Congenital sensorineural deafness was identified in all American Paint and suspected in paint Warmblood horses. BAER in these horses was characterized by the lack of identifiable peaks in either ear consistent with complete loss of function. Horses diagnosed with THO were older (mean, 14 years) than other groups. The effects of aging on hearing in the horse have not been fully evaluated.[8,14] However, partial hearing loss was reported in 4 horses, 17–22 years of age.[14] Of 57 horses with hearing deficits, 14% (n = 8) were >15 years of age. Of these 8 horses, 5 horses had THO, 2 had chronic bilateral otitis, and 1 had vestibular disease of undetermined cause. Therefore, the effects of aging on BAER could not be evaluated in this study because of the presence of disease that could result in deafness. Whether partial bilateral hearing deficits in a 28-year-old horse with vestibular disease were associated with the underlying cause of disease or were the result of aging (presbycusis) could not be determined.

The most common signs in horses with hearing loss varied by disease and according to whether complete or not or partial deafness was present. For example, altered behavior and being startled easily was more commonly reported in horses with complete bilateral hearing loss independent of disease. Unilateral peripheral vestibular disease, facial nerve deficits, and corneal ulceration were the most common signs in horses with THO, as previously reported.[7] Horses with multifocal brain disease had signs consisting of altered state of consciousness and multiple cranial nerve deficits. Hearing deficits were more difficult to detect in horses with altered state of consciousness (decreased or absent response to various stimuli including sound). Head tilt, shaking, and rubbing were the most common signs in horses with otitis.

The most common causes of abnormal BAER findings included THO (n = 20/20), congenital sensorineural deafness in horses with paint coat color (American Paint horses [n = 15/17], Warmblood [n = 2/17]), multifocal brain disease (n = 13/16), and otitis media or interna (n = 4/4). Bilateral BAER deficits were more common in horses with congenital sensorineural deafness (n = 17/17), THO (n = 10/20), and multifocal brain disease (n = 11/16). BAER studies in horses with THO, congenital sensorineural deafness of American Paint horses, and a single case of glioblastoma multiforme have been described by the authors.[7,12,19] Causes of multifocal brain disease with concurrent BAER abnormalities identified in this study included melanoma, glioblastoma multiforme, intracranial abscess, suspected equine protozoal myeloencephalitis, meningoencephalomyelitis because of *H. gingivalis* and mixed bacterial growth involving the inner ear, and traumatic brain injury. The most common cause of unilateral BAER deficit was THO (n = 10/20). Other causes of unilateral loss included otitis media or interna (n = 3/4) and multifocal brain disease (n = 2/16). Horses with vestibular disease and no other neurologic deficits had normal

BAER findings. The exception was a single 28-year-old horse in which the etiology remained undetermined.

Other causes of hearing deficits included an American Miniature horse with congenital malformations including facial deformation that had complete loss of BAER bilaterally. This horse had no visible external or internal acoustic meatus. Bone conduction testing was not performed but, theoretically, the presence of a response on this test would be consistent with a severe conduction loss with sparing of the sensorineural pathway. A different horse had partial bilateral hearing loss which the owner suspected to have developed acutely. The cause was unknown. A horse with spontaneous otoacoustic emissions had a BAER within reference range. Spontaneous otoacoustic emissions are low intensity nonevoked emissions (sound not triggered by external stimuli) generated at the cochlea by the biomechanical activity of the outer hair cells.[27] Spontaneous otoacoustic emissions have been reported in humans, dogs, cats, guinea pigs, and a pony.[17,27–29] Similar to our horse, the single reported pony also had normal BAER, and the cause of the spontaneous otoacoustic emissions was undetermined.[17]

Criteria for distinguishing normal versus abnormal BAER included absence of BAER peaks, absence of peaks following I or II, and abnormal prolongation of interpeak intervals (I–III, III–V, and I–V).[9] These criteria serve to localize lesions in the cochlear nerve, cranial brainstem, and caudal brainstem.[9] Horses with THO had prolonged latency of peaks I through V, but the interpeak intervals were within reference range. These findings supported a cochlear nerve abnormality as shown in a previous study.[7] Horses with multifocal brain disease had peak latencies I through V within reference range but prolonged interpeak intervals, which supported a brainstem lesion. In further support, the peak V/I ratio in the vertex to C2 derivation was lower (1.6) in these horses than in controls (2.75) or in horses with a peripheral lesion (eg, THO, 4.37). In dogs, low V/I peak ratio ($\leq$0.5) in the vertex to mastoid derivation has been associated with a brainstem lesion.[2] Determination of BAER thresholds also is useful to detect subtle hearing loss. Thresholds were not analyzed in this study because they were not routinely performed.

In conclusion, hearing deficits should be investigated in horses with altered behavior (eg, being easily startled), THO, multifocal brainstem disease, and otitis. Patients with intracranial masses, traumatic brain injury, and infectious causes of brain disease also may have alterations in auditory function. Horses with certain coat patterns and eye color should be screened for congenital sensorineural deafness. Facial deformities, the application of ototoxic drugs, and the normal aging process may be associated with auditory dysfunction. Mild hearing deficits such as those resulting from partial or complete deafness in 1 ear may be easily missed on physical examination. BAER testing is an objective, safe, noninvasive, and easy to perform diagnostic modality for the assessment of the auditory pathway in horses.

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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