Brainstem auditory-evoked response in dogs

Wayne J. Wilson, PhD, and Paul C. Mills, BVSc, PhD

Veterinarians, researchers, breeders, and owners all have an interest in the accurate assessment of hearing in dogs.[1] At least 48 breeds of dog, including Dalmatians, Border Collies, Australian Cattle Dogs, and Old English Sheep Dogs, are known or suspected to have hereditary congenital deafness,[2,3] and the prevalence of hereditary deafness in some breeds is substantial. In a study[2] of > 1,000 Dalmatians, 21.6% of the dogs had severe unilateral deafness and 8.1% had severe bilateral deafness. This finding prompted the British Dalmatian Club of Great Britain to recommend auditory assessment for all Dalmatian puppies and breeding stock.[1] Dogs of working breeds also require good hearing and rely on both ears to localize the source of sound. Diagnosis of hearing impairment in these dogs saves time and prevents money from being wasted in expensive training programs.[4]

Evaluation of hearing in dogs in the past was often conducted by measuring behavioral responses to a sound or series of sounds.[5-12] Although such an approach may be used to obtain a psychoacoustic audiogram in which the frequency of the sound is plotted against the minimum intensity required to elicit a behavioral response, results are influenced by the dog's mental state, rate of adaptation to the sound stimulus, and sensitivity to visual and vibratory stimuli.[5-12] As a result, proper training of both the dog and the human tester is required before reliable results can be obtained. These factors make the use of behavioral responses a poor choice for inexperienced testers, young puppies, and clinical patients.[7]

Many veterinarians and researchers have turned to electrodiagnostic testing as an alternative to behavioral testing in assessing auditory function in dogs. The **brainstem auditory-evoked response (BAER)** is one of the most frequently used testing modalities in this respect because the test is objective, reasonably easy to perform, noninvasive, safe, and cost-effective, compared with other objective measures of auditory function. The testing apparatus is portable, and test time is brief. Results are reliable, sensitive, anatomically specific, generally independent of the level of consciousness, and resistant to the influence of drugs and yield a comprehensive index of neurologic status.[13]

This article reviews current knowledge regarding the use of BAER in dogs, including basic principles of technique; effects of nonpathologic subject factors, stimulus factors, and recording factors on the BAER; and common applications of the BAER in dogs. The term BAER will be used in preference to the auditory brainstem response, the brainstem auditory-evoked potential, the brainstem evoked response, and the brainstem evoked potential.[13,14]

Received February 1, 2005.
Accepted March 24, 2005.
From the Schools of Health and Rehabilitation Sciences (Wilson) and Veterinary Science (Mills), University of Queensland, Brisbane, Queensland, 4072 Australia.
Address correspondence to Dr. Wilson.

Features of the BAER

When repetitive sensory stimuli are received, the brain responds with consistent changes in electrical activity. These changes are collectively referred to as evoked responses or evoked potentials.[6] As one of a large family of evoked responses, the BAER represents the mean value of recordings of activity in **cranial nerve (CN)** VIII and the auditory portion of the brainstem in response to externally applied acoustic stimuli.

Modern BAER equipment is typically personal computer–based and can be divided into stimulus components and recording components. The stimulus components include a stimulus generator and earphones. The recording components include recording electrodes and a differential amplifier, signal averager, and display screen.

In modern BAER protocols, dogs are typically positioned in sternal recumbency, with chemical restraint if required. Earphones are positioned over the dog's ears, and needle electrodes are positioned SC in the scalp at the vertex and immediately rostral to the base of each ear. The earphones repeatedly stimulate the test ear with the chosen stimulus and mask the nontest ear with white noise (a broadband signal that contains all frequencies equally and prevents the nontest ear from contributing to the BAER). The needle electrodes repeatedly sample the electrical responses of CN VIII and the auditory portion of the brainstem. These responses are led into a differential amplifier that amplifies the difference in voltage between the vertex and test-ear electrodes. The output of the amplifier is digitized and led into a signal averager. Because unwanted signals (eg, electroencephalographs, ECGs, muscle potentials, and ambient electrical fields) occur randomly while the desired signal (the BAER) is time locked with respect to the stimulus, the unwanted signals eventually sum or average toward a value of 0, whereas the desired signal (the BAER) sums or averages toward its actual value. This process can take from 30 seconds to several minutes depending on the stimulus level and rate and quality of the BAER being recorded. The recording is repeated to ensure replicability of results and to substantiate the biological nature of the recording.[6,13,14] When the BAER is of very low amplitude (as is the case when determining BAER thresholds), its replicability is also compared with that of BAERs recorded in response to a no-sound stimulus (ie, stimulus off). Even a low-amplitude BAER should generate a replicable response to the same sound stimulus, whereas the response recorded to no stimulus should not.

Regardless of species, the typical suprathreshold BAER obtained with this protocol consists of up to 7

positive waves recorded within 10 milliseconds of stimulus onset (it should be noted that the waves are not necessarily positive but simply denote periods when the vertex electrode is electrically positive with respect to the ear electrode). In dogs, the first positive BAER wave begins 1.0 to 1.5 milliseconds after the stimulus onset and each successive wave occurs at 0.5- to 1-millisecond intervals.[6] Each wave has an amplitude (peak-to-trough) ranging from < 1 μV to approximately 6 μV (**Figure 1**). These dimensions will vary, however, depending on the subject, stimulus, and recording variables used.

The labelling of BAER waves in dogs has been a source of confusion. In humans, each positive wave is labelled sequentially with Roman numerals on the basis of a master waveform that contains all 7 waves.[15] In recordings in which any of the waves is absent, each wave retains its label as if all 7 waves were present.[15] In dogs, each positive wave is also labelled sequentially with Roman numerals, but corrections for absent waves have not always been made.[16] Because wave IV is often absent,[16] some authors have incorrectly labelled wave V as wave IV and wave VI as wave V.[17-22] Similar confusion has resulted from bifurcated waves and differences in wave morphologic features that can result from differences in stimulus and recording variables.[16] Confusion regarding wave V may be removed by designating wave V as the positive peak occurring immediately before the deeply negative trough in the second half of the recording.[15]

For most stimulus intensities and rates in dogs, waves I, II, and V have large amplitudes; waves III, IV, and VII have small amplitudes; wave VI is usually present; and wave VII is usually absent.[1,6,19,23-28] Waves III and IV may merge to form a single wave dominated by wave III (the III-IV complex[24,29]), or waves IV and V may merge to form a single wave dominated by wave V.[6,16]

It is important to keep in mind that the BAER is not a real-time representation of true neural activity of the auditory system in response to sound. Rather, it is an artificially created representation of the response of a small subset of neurons (probably onset sensitive and oriented in parallel) in CN VIII and the auditory portion of the brainstem as they respond to a brief sound.[13] As a result, the BAER can only be used as a direct measure of the function of these neurons. The BAER cannot be used as a measure of the behavioral concept that is hearing, because hearing requires the activity of many structures in addition to CN VIII and the auditory portion of the brainstem.

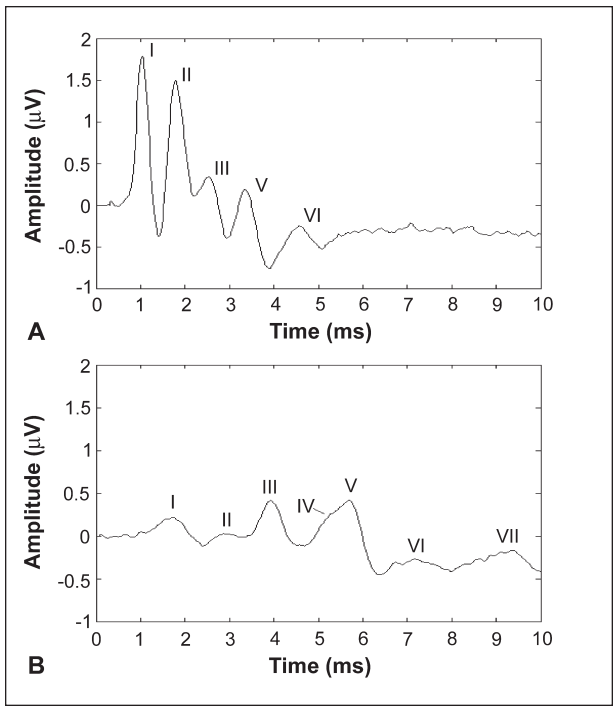

Figure 1—Waveforms (I to VII) of a brainstem auditory-evoked response (BAER) recorded from an adult dog (upper) and an adult human (lower) with normal hearing. A 90-decibels normalized hearing level (dBnHL) click stimulus was given through headphones, and the BAER was recorded by use of a vertex-to-mastoid derivation. Notice that when the vertex-to-mastoid derivation is used, the amplitudes of waves I through V become progressively smaller in dogs but progressively larger in humans.

Neural Generators of the BAER

General features—The few studies investigating the neural generators of the BAER in dogs have been limited to surface recordings obtained after induced lesions[20] or acquired lesions confirmed by necropsy, magnetic resonance imaging, and histologic tissue assessment.[30,31] Results of these studies suggest that waves I to III are generated mostly by CN VIII, the cochlear nucleus and superior olivary complex, whereas waves IV to VII are generated mostly by one or more of the following structures: the nucleus of the lateral lemniscus, the inferior colliculus, or the medial geniculate nucleus.

Investigations of the neural generators of the BAER in other species have made use of direct recordings and induced lesions in experimental animals and of different electrode arrays, clinicopathologic testing, and depth electrode recordings in humans.[13] Despite the reported differences among species in central auditory nervous system anatomic features[32,33] and BAER waveform morphologic features,[6,34] results of these studies represent most of the data that have been reported regarding neural generators of the BAER.

On the basis of early studies[35,36] of BAER abnormalities in cats, it was postulated that each BAER wave could be attributed to a single neural generator: wave I was generated by CN VIII, wave II by the cochlear nucleus, wave III by the superior olivary complex, wave IV by the nucleus of the lateral lemniscus, wave V by the inferior colliculus, wave VI by the medial geniculate nucleus, and wave VII by the thalamocortical radiations. These simple associations are now known to be incorrect. With the exception of waves I and II, each component wave of the BAER is now believed to receive contributions from > 1 anatomic structure.[13,37] Conversely, a single given anatomic structure probably contributes to > 1 BAER wave. Anatomically, there are numerous and varied parallel pathways in the central auditory nervous system for neural transmission between nuclei.[38-40] It is reasonable to expect that nearby structures or those that are activated at or near the same time after the stimulus would contribute in varying amounts to BAER waves. Given that only a subset of neural units in any anatomic region is actually contributing to the BAER,[13,14,41] it can be surmised that the

BAER does not reflect a simple sequential passing of information from 1 relay station to another. Instead, it represents the complexity of different auditory brainstem structures and the variable timing of activity arising from these structures.[42] The result is that the precise anatomic generators of the waves that follow after waves I and II are difficult to determine.

The cochlea has been incorrectly reported as a direct generator of part of the BAER, particularly wave I.[18,26,43] Although the cochlea responds to stimuli with its own evoked potentials (eg, the cochlear microphonic and summating potentials), they occur before wave I of the BAER and are typically considered to be separate from the BAER.[13] However, much of the evidence that appears to rule out the cochlea as the generator of wave I is derived from human BAER recordings. Differences in geometric aspects of the skull lead to suspicions that the volume conduction characteristics of the human skull differ from those of the skulls of other species and may be responsible for some species differences. Dipole modeling in animals could help resolve this issue noninvasively.

Specific features—Almost all data on the specific generators of BAER waves come from studies performed in humans. Wave I is accepted as the far-field representation of the afferent compound action potential of the CN VIII fibers as they leave the cochlea and enter the internal auditory canal.[42,44-57] The negative trough after wave I is thought to reflect activity arising from the region of CN VIII as it exits the internal auditory canal at the cerebellopontine angle.[42] Wave II is likely generated by the proximal portion of CN VIII as it enters the brainstem.[42,58,59] Wave III is thought to arise from the activity of second-order neurons (beyond CN VIII) in or near the cochlear nucleus,[42,44,46-49,58] with the negative trough that follows wave III arising from the trapezoid body.[42] Determining the precise generators of waves IV and V is complicated by 2 factors. The first factor is the close association between wave IV and wave V (commonly resulting in a wave IV-V complex). The second factor is the likelihood of multiple decussations for auditory fibers beyond the cochlear nucleus. Some reports suggest that wave IV originates from the contributions of second- and third-order neurons,[42] mostly in the superior olivary complex of the pons with likely contributions from the cochlear nucleus and nuclei of lateral lemniscal neurons.[58] Earlier suggestions that the nuclei of the lateral lemnisci were generators of wave IV have lost credibility because of the small size of the nuclei and the reduced likelihood of simultaneous neuronal firing in light of the innervation from many different pathways.[33,40] The positive-voltage wave V is likely associated with the termination of lateral lemniscus fibers in the contralateral inferior colliculus. The resulting dendritic potentials in the inferior colliculus are thought to be responsible for the large, broad negative-voltage trough that follows wave V.[44] The identity of the generators of BAER waves VI and VII is even more open to question than that of generators of the earlier waves. Most reports[44,57,60] suggest a thalamic (eg, medial geniculate body) origin, but others[37] attribute those waves to continued synchronous firing of neurons in the inferior colliculus.

Laterality

The laterality of the BAER is another area of debate. Multiple electrode array analysis and correlations between pathologic changes in the brainstem and abnormalities in the BAER suggest that generators of wave components that follow wave I or II are located in brainstem regions contralateral to the stimulated ear.[61] Other clinical evidence suggests there are ipsilateral generators,[62,63] and results of still other studies[13,37] suggest that the BAER waveform is generated simultaneously, but separately, by structures in parallel (ie, by both ipsi- and contralateral) pathways. The issue of BAER laterality remains unresolved.

BAER testing in clinical practice

Accurate recording of the BAER in dogs depends on practical as well as theoretical issues. The practical issues include 3 key areas: nonpathologic subject factors, stimulus factors, and recording factors.

Nonpathologic Subject Factors

Age—Most investigators[6,7,23,27,64,65] report that reliable BAER waveforms can be recorded in dogs after approximately 2 weeks of age, with waves I and II often being the first to appear.[6] Low-amplitude BAER waveforms have been detected in response to high-intensity stimuli (105 and 95 **decibels normalized hearing level** [**dBnHL**]) in Beagle puppies younger than 2 weeks of age (prior to opening of the ear canal), but the difficulties reported in identifying these waveforms preclude their widespread clinical use.[27] Adultlike BAER values are usually obtained by 6 to 8 weeks of age.[6,7,23,27,64,65]

The BAERs recorded in response to click stimuli in dogs at or near 2 weeks of age have delayed absolute wave latencies, prolonged interwave latencies, reduced wave amplitudes, steep wave V latency-intensity functions, and increased thresholds.[6,7,23,27,64,65] Absolute and interwave latencies become shorter and wave amplitudes become larger from approximately 3 to 7 weeks of age,[23,27] with earlier waves stabilizing before later waves.[27] Brainstem auditory-evoked response thresholds to click stimuli also improve rapidly from approximately 100 dBnHL before 2 weeks to approximately 60 to 75 dBnHL shortly after 2 weeks to approximately 0 to 30 dBnHL near 3 weeks of age.[27,65] The improvements in BAER measurements after birth are thought to be associated with the caudal-to-rostral pattern of maturation in the canine CNS,[66] especially increases in neuronal myelination,[13] although differences among breeds have been reported.[64]

With respect to the effects of age on the BAER in dogs, a possible increase in the latencies of waves I to IV (although wave V was incorrectly labelled as wave IV) in dogs 10 years of age and older has been reported,[19] but the variability in values made the importance of this finding questionable. Decreased wave I and II amplitudes in 4 dogs 9 to 14 years of age were reported,[29] but those dogs also had impaired peripheral hearing. In another study,[67] there was no change in the

BAER wave latencies in 4 dogs aged 8 to 14 years, but those dogs also had decreased peripheral hearing. In humans, the BAERs in subjects > 60 years of age resemble those of young children.[13]

Head size—Several studies have revealed that the effect of head size on absolute and interwave latencies in dogs is substantial, but other studies have not reported corroborating results. In 1 study, brainstem auditory evoked responses were recorded in response to 60-**decibels hearing level (dBHL)** clicks in 20 dogs (multiple breeds) weighing 2 to 36 kg.[68] The length of the cranium, width of the cranium, nasion-to-external auditory meatus distance, and body weight were positively correlated with wave V and wave I and V-interwave latencies, but not with wave I latency. In a similar study,[69] BAERs were recorded in response to 90-, 80-, and 70-dBnHL clicks in 43 dogs of multiple breeds weighing 4.1 to 50 kg. Cranial and brainstem dimensions were positively correlated with absolute and interwave latencies of BAER waves I to V. This study also concluded that head size accounted for 25% of the variation in BAER latencies.[69] However, in another study,[70] BAERs were recorded to 75-dBnHL clicks in 20 Dalmatians (maximum head width, 123 ± 8 mm) and 20 Jack Russell Terriers (maximum head size, 88 ± 5 mm) and no significant correlation was shown between head size and BAERs. Human studies[13,14] have also found correlations and no correlations between cranial dimensions and BAERs. The best solution may be to obtain normative BAER data that are specific for the breed of dog being assessed.

Sex—In dogs, no significant association between sex and BAER has been reported.[1,25,68-71] However, in humans, there are differences between BEARs in female versus male adults, with females having shorter latency values and larger wave amplitudes than males for the later BAER waves (ie, III, IV, V, and VI).[13] Possible reasons for these findings are that age-matched females have better hearing thresholds, higher body temperatures, shorter nerve lengths because of smaller head size, physiologic and biochemical differences, and hormonal fluctuations from the menstrual cycle.[13]

Body temperature—In 1 study,[19] there was no relationship between the BAER and rectal temperatures from 37° to 39.5°C, but increased latencies were observed for all BAER waves in 2 dogs with rectal temperatures < 36°C. In humans, hypothermia results in increased BAER wave latencies at low body temperatures[72-74] and eventual disappearance of all waves at very low body temperatures (< 14° to 20°C).[74] The effects of hyperthermia on BAERs in dogs or humans have not been well-defined.[13]

Attention and state of arousal—The BAER is not affected by the state of arousal in dogs or humans. There are no differences in BAER waveforms at any stimulus intensity in awake versus natural sleep states,[1,13,16,19,23-26,75,76] and conditions such as narcolepsy and metabolic coma have no substantial impact on BAER latency or amplitude.[13,75,76]

Pharmacologic agents—The BAER in humans is moderately affected by ototoxic drugs (eg, certain antibimicrobials and loop diuretics), antiarrhythmic agents (eg, lidocaine), high doses of certain anticonvulsant drugs (eg, phenytoin), and large doses of alcohol. The BAER is minimally affected or not affected by sedatives, hypnotic drugs, tranquilizers and psychotherapeutic agents (eg, barbiturates, chloral hydrate, benzodiazapines, and morphine), cholinergic agents (eg, carbachol and physostigmine), most anesthetic agents (eg, nitrous oxide, halothane, isoflurane, and ketamine), narcotics (eg, morphine and fentanyl), and neuromuscular blockers (eg, succinylcholine, D-tubocurarine, pancuronium, and metocurine).[13,14]

Many authors have used the BAER to assess the ototoxic effects of drugs of dogs. A complete loss of the BAER was reported[18] after IV administration of neomycin at a dosage of 30 mg/kg/d for 22 days. No changes in the BAER were reported after instillation of chlorhexidine acetate (7 drops of a 0.2% solution daily) into the external auditory canal of dogs with an intact tympanic membrane for 21 days or in those with a perforated tympanic membrane for 21 days,[77] and no changes were reported in BAERs after twice-daily instillation of gentamicin sulfate (7 drops of a solution containing 3 mg of gentamicin/mL) into the external auditory canal of dogs with either an intact tympanic membrane or a perforated membrane, for 21 days.[43] Reversible changes were reported in the BAER waveform after IV administration of kanamycin sulfate at a dose of 100 mg/kg.[21] Certain anesthetic drugs may alter the latency and amplitude of certain BAER waves, whereas others may not. For example, SC administration of acepromazine maleate to dogs at a dose of 0.55 mg/kg has no effect on the BAER.[25] Similarly, other investigators[78] found that IV administration of xylazine (dose, 2 mg/kg) and atropine (0.05 mg/kg) followed by administration of ketamine (15 mg/kg) IM or pentobarbital (10 mg/kg) IV had no effect on the BAER, whereas in another study,[24] IV administration of thiamylal sodium at a dose of 20 mg/kg altered the morphologic characteristics of the BAER. Administration of 3% methoxyflurane (route not stated but inhalation assumed) prolonged all BAER wave latencies except wave I in 1 study.[26]

Muscle-related artifact—Artifact from muscular activity is an important invalidator of BAER recordings.[13,14] The BAER wave components can be completely obscured by excessive muscle-related artifact, which usually originates from musculature of the neck and jaw. The scale of this problem is indicated by the fact that muscle activity is typically measured in millivolts, whereas the neuronal activity in a BAER recording is typically measured in microvolts. As a result of muscle-related artifact, the use of chemical restraint may be needed for the accurate recording of BAERs in some dogs.

Stimulus Factors

Type—The BAER is best generated by very brief or transient stimuli that have nearly instantaneous onset.[13,14] For this reason, a 0.1-millisecond square-wave click is considered the best BAER stimulus for diagnostic or site-of-lesion applications.[13,14] Although this click contains energy in the range of 500 to

4,000 Hz, it effectively stimulates only the 2,000- to 4,000-Hz region of the cochlea in humans. This is because the higher-frequency hair cells (located basally) have already been activated by the time the travelling wave reaches the lower-frequency hair cells (located apically), the leading front of the travelling wave is sharper (more abrupt) for high frequencies than for lower frequencies, and a sharper wave front elicits more synchronous hair-cell firing over a more concentrated portion of the basilar membrane.[39] Different portions of the basilar membrane are also thought to contribute differently to different BAER waves, with wave I appearing to reflect high-frequency (more basal) activity and wave V reflecting midfrequency (more apical) activity.[13,14]

Although the click stimulus is ideal for diagnostic or site-of-lesion applications, its frequency specificity makes it less useful for estimating the threshold of auditory function across the full frequency range of hearing. Most authors recommend the use of tone bursts[13,14] when the area of interest falls into those frequencies. Tone bursts are short-duration tones that attempt to balance the rapid onset of the click with the frequency specificity of the tone. However, this is difficult to achieve, and the BAER wave shape tends to deteriorate, wave latencies tend to increase, and wave amplitudes tend to decrease as the frequency of the tone burst is decreased.[13,14]

In dogs, the click has been the predominant stimulus used. However, the use of tone bursts has been reported,[7,21,22] indicating a potential for clinical use.

Intensity—The intensity of the stimulus is the key variable when auditory thresholds are being determined with BAERs. Sound intensity is typically measured in units of decibels, and many different types of decibels may be used. For example, **decibels sound pressure level (dBSPL)** is an absolute physical measure of the sound intensity, referenced to 20 micropascals or 10^{-12} W/m^2. The **decibels peak equivalent sound pressure level (dBpeSPL)** is a measure of the intensity of a complex sound (ie, a sound containing > 1 frequency). To calculate dBpeSPL, a simple sound (containing only 1 frequency) is laid over the top of the complex sound so that the simple sound has the same maximum amplitude as the complex sound. The dBSPL of the simple sound is equal to the dBpeSPL of the complex sound. Decibels hearing level is referenced to the threshold of hearing in the young healthy adult human wearing audiologic headphones. This means that 0 dBHL is the quietest sound that a typical young healthy adult human can hear under audiologic headphones, regardless of the frequency of the sound. Technically, dBHL exists only for pure tone sounds; therefore, dBnHL is used to represent the quietest sound that a typical young healthy adult human can hear under audiologic headphones when that sound is not a pure tone. Another useful unit of measure is **decibels sensation level (dBSL)**, which denotes the number of decibels above an individual's threshold. All of these decibel units have been used to correlate information regarding auditory function from animals to humans and from humans to animals.

Stimulus intensity has a profound effect on the BAER waveform.[1,13,14,19,22-28,71,79] As a general principle, absolute BAER wave latencies decrease and amplitudes increase with greater stimulus intensity, but interwave latencies remain more stable. In adult humans, a greater prolongation of wave I latency, compared with wave V latency, is sometimes observed with decreased stimulus intensity, resulting in a net shortening of the wave I-to-wave V interwave latency. The wave III-to-wave-V interwave latency is often less affected by stimulus intensity because it is more independent of wave I and its generators.[13]

As the stimulus intensity decreases to below approximately 50 dBSL in dogs, BAER waves IV, VI, and VII often begin to disappear, (if present initially) followed by waves I, II, and III until only wave V remains (**Figure 2**). The last stimulus intensity at which wave V remains is typically considered to be the BAER threshold and is used to estimate the behavioral hearing threshold.[1,13,19,22-27,71,79] Values reported for mean BAER threshold values in healthy hearing adult dogs for air-conducted click stimuli range from –5 to 15 dBnHL,[1,23,25,80] 24.7 to 28.0 dBHL,[28] and approximately 50 to 56.4 dBpeSPL.[21,22]

Reported values for air-conducted tone burst stimuli include 89.8 dBpeSPL for 500 Hz; 71.3 for 1,000 Hz; 61.2 dBpeSPL for 2,000 Hz; 55.3 dBpeSPL for 4,000 Hz; 67.2 dBpeSPL for 8,000 Hz[22]; and 0.1 to 5.5 dBSPL between 2 and 8 kHz, increasing by 11 dB/octave from 2 to 0.5 kHz and by 20 dB/octave from 8 to 32 kHz.[7] The reported value for bone-conducted click stimuli was –0.8 dBnHL.[71] In general, these values indicate that most auditory stimuli should achieve a BAER threshold < approximately 25 dBnHL in adult dogs with normal hearing.

The physiologic basis of the BAER-stimulus intensity relationship is complex and related to several theories regarding the physiologic basis of generation of BAERs.[81-84] For a click stimulus, the site of generation

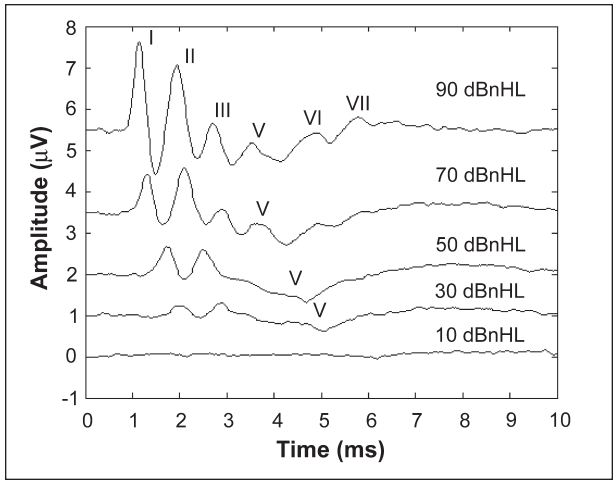

Figure 2—Typical BAER waveforms recorded from an adult dog with normal hearing wearing a headphone and responding to a series of decreasing click stimulus intensities (listed on right side of figure).

of the BAER along the basilar membrane is related, in part, to intensity. High stimulus intensities activate the basilar membrane near the base of the cochlea. The activation site moves progressively toward the apex of the cochlea with decreasing stimulus intensities; at lower intensities, the signal takes longer to get to the apical end of the cochlea. Postsynaptic excitation potentials also reach threshold faster with greater stimulus intensities, therefore decreasing synaptic transmission times. An increase in the number of individual fibers that are firing synchronously and a concomitant increase in the overall volume conduction are thought to cause the increase in wave amplitude observed with increasing stimulus intensity.

It is also important to note that at high stimulus intensities, the stimulus can be transmitted via the skull from the test ear directly to the cochlea of the nontest ear.[13,14] This is referred to as the crossover effect. In humans, crossover may occur with the loss of as little as approximately 40 dB when headphones are being used and approximately 70 dB when insert earphones are being used.[13,14] This means that a 100-dBnHL click stimulus directed to the test ear can cross over to stimulate the cochlea of the nontest ear at 60 dBnHL for headphones or 30 dBnHL for insert phones. To prevent the nontest ear from contributing to the BAER, it must be masked with a noise signal. The noise signal typically used for the BAER is white noise (a signal containing all frequencies at random) at a level at least equal to the signal intensity in the test ear minus the crossover value for the headphones or insert phones.[13,14]

Rate—Stimulus repetition rates up to approximately 20 clicks/s have little effect on the BAER of dogs, humans, or small animals.[6,13,14,24] Above this rate, BAER wave latencies generally increase and amplitudes decrease, although these changes are not the same for each wave component in humans[13,14] and wave latencies remain stable from stimulus rates of 5 to 50 clicks/s in dogs.[6,24] In humans, there is also a direct relationship between maturity of the CNS and the effect of rate on BAER, with higher-rate stimuli exerting greater effects on a less mature CNS.[13,14]

The physiologic basis for the effects of stimulus repetition rate on the BAER appears to be a combination of neural fatigue and adaptation, and incomplete recovery involving hair cell–cochlea nerve junctions and subsequent synaptic transmission.[85,86]

Polarity—Stimulus polarity refers to the direction of pressure change created by the acoustic transducer. A rarefacting stimulus pulls the tympanic membrane toward the transducer, whereas a condensating stimulus pushes the tympanic membrane away from the transducer. An alternating stimulus alternates between the 2 types.

The effects of stimulus polarity on the BAER are not clear. Some authors recommend the use of alternating polarity because it cancels out the stimulus-related electromagnetic and cochlear microphonic artifacts associated with each polarity. The **signal-to-noise ratio (SNR)** is thus improved, and wave I can be more confidently identified on the BAER waveform. Other authors recommend the use of rarefaction polarity because it initially depolarizes the cochlear hair cells, yielding BAER responses of shorter latency and greater amplitude. Condensation polarity stimuli initially induce hyperpolarisation of cochlear hair cells, yielding the opposite effect.[13,14,87]

In support of the use of single rarefaction-polarity stimuli are the differences in latency that are expected between responses to each polarity. When combined into an alternating-polarity response, these differences should increase variability of the BAER response. Conceivably, with alternating-polarity stimuli, out-of-phase responses could be added together to induce responses with reduced amplitude or even an artificially abnormal or absent response in a clinically normal subject.[87] Increased diagnostic sensitivity with the use of rarefacting clicks is also inferred from the higher proportion of abnormal findings detected with the use of this polarity than with condensating stimuli.[13,14]

In dogs, the only studies of stimulus polarity revealed that rarefacting click stimuli elicit better separation of waves III and IV,[6] a greater likelihood of wave IV being present,[88] and shorter latencies for all BAER waves.[16] Contradicting these findings, however, are reports that rarefacting clicks result in longer BAER wave and interwave latencies[88] (although there is concern regarding the true stimulus polarity at the level of the tympanic membrane[16]) and that condensating clicks result in better BAER responses for bone-conducted BAERs.[79]

Transducers—The most common transducers used to record BAERs in dogs are the earphone[6] and insert earphone.[6,28] Although the insert earphone is often favored because it reduces electrical artifact and prevents the external auditory canals from collapsing during testing, it does delay the beginning of the BAER waveform by approximately 0.8 to 1.0 milliseconds, which must be taken into account during waveform analysis.[6,13,14] However, this delay effect was not detected in another study.[28]

A bone conductor transducer may also be used when significant conductive hearing loss is suspected, although appropriate bone conduction normative data must be used at the same time.[71,79,80] In 1 study,[79] investigators found that the optimal site for placement of the bone conductor was over the mastoid process, followed by the mandible and the zygomatic arch, whereas others[71] observed no difference in the BAER obtained by holding the bone vibrator against the head by hand or by application of a 500-g weight.

The bone-conducted BAER has the same morphologic features as its air-conducted counterpart, with a reduction in amplitude and an increase in variation.[6,71,79,80] Differences in skull impedance among dogs are thought to account for the differences.[6] A limiting factor of any bone-conducted stimulus is the large amount of electrical artifact emitted by the bone conductor itself. This artifact often limits bone-conducted BAER recordings to stimulus intensities below approximately 40 dBnHL.[13,14]

Monaural versus binaural stimulation—To the authors' knowledge, only a single investigator[34] has

reported the effects of binaural stimulation on the BAER in dogs. Binaural stimulation caused the amplitudes of BAER waves to increase (but not double), whereas the latencies of BAER waves remained the same. Similar results have been observed in humans.[13,14] The physiologic basis of the binaural effects on BAERs is complex[89,90] and may involve neurons that respond only to binaural stimulation. Because of this complexity, use of binaural stimulation in clinical BAER assessment is rare. A greater understanding of the role of binaurally sensitive neurons, however, would be of value in both dogs[34] and humans.[91,92]

Effects of Acquisition (Recording) Factors

Analysis time—Because a normal suprathreshold BAER in dogs is complete by approximately 8 milliseconds (9 milliseconds with insert earphones), an analysis time of 10 milliseconds is acceptable. For certain pathologic conditions (eg, severe to profound hearing loss) or in newborns, however, a longer analysis time (15 or 20 milliseconds) may be required to ensure that all waves are recorded.

Electrode placement—The ipsilateral electrode array is the conventional array used in dogs and humans. This array places the noninverting (active) electrode on the vertex; the inverting (reference) electrode rostral to the tragus of the test ear; and the ground electrode, for convenience, rostral to the tragus of the nontest ear. Although maximum wave V amplitude is recorded from a vertex site, studies[13,93] of BAER topography indicate that the precise location of the electrode along the midline (ie, the sagittal plane) is not an important factor in the response. Shifting the midline electrode even a short distance laterally from midline, however, has a profound effect on the amplitude of wave I.[93]

Although its use is widely accepted, the conventional ipsilateral electrode array is not the only array used for recording BAERs. Other arrays used include contralateral (noninverting electrode on the vertex and inverting electrode near the nontest ear), vertical (noninverting electrode on the vertex and inverting electrode on the dorsum of the neck overlying C3), and horizontal (noninverting electrode near the nontest ear and inverting electrode near the test ear), all of which result in predictable changes in BAER wave morphologic features. Use of these arrays can be useful in confirming hard-to-identify waves in the ipsilateral recording in dogs[88,93] and humans.[13,14]

Amplifiers—Because of the extremely small amplitude of the BAER waveform, an absolute gain setting of 100,000 to 150,000 is typically used.[6,13,14]

Filters—The aim of BAER filtering is to capture as much of the BAER energy as possible while filtering out as much artifact energy as possible. A wide range of filter settings has been used for obtaining BAERs in dogs and humans, including high-pass filters ranging from 5 to 500 Hz and low-pass filters ranging from 1,000 to 8,000 Hz.[13] High-pass filters pass frequencies higher than their cutoff frequency, whereas low-pass filters pass frequencies lower than their cutoff frequency.[13] In dogs, the BAER contains frequencies ranging from 30 to 1,960 Hz[94]; therefore, it is logical to set the filter settings somewhere in that range. This was supported by a study[95] in which the investigator examined the effects of analogue filter frequency on the BAERs of 7 adult dogs of both sexes that weighed 8.6 to 19.5 kg and found that reducing the low-pass filter setting from 30,000 to 100 Hz caused prolongation of BAER wave latencies and a reduction of BAER wave amplitudes but had little effect on interwave latencies. Increasing the high-pass filter setting from 0.53 to 160 Hz had no effect on any of the BAER waves, except for a decrease in the amplitude of wave 4 (ie, wave V). On the basis of their results, the investigators recommended a low-pass filter setting of 3,000 Hz and a high-pass filter setting ≤ 53 Hz for recording BAERs in dogs. These settings were thought to sufficiently attenuate unwanted high-frequency artifacts and to adequately record both the slow and fast components of the BAER in young and old dogs without substantially affecting the BAER's configuration, wave latencies, or wave amplitudes.

Averaging—With the assistance of amplification, differential averaging, and summation, the minimum number of averages or sweeps required for confident acceptance of a BAER waveform can range from as few as 250 in ideal recording conditions to over 2,000 in less-than-ideal conditions.[6,13,14] The number of averages required is driven by the SNR formula:

$$SNR = (S \times n)/N$$

where S is the signal amplitude, N is the noise amplitude, and n is the number of averages.[13] From this equation, it is apparent that the greater the noise, the more averages will be needed to ensure an acceptable SNR. It has been posited[6] that approximately 1,000 sweeps would give acceptable BAER results in resting or chemically restrained dogs.

Recommended protocols for recording and analyzing the BAER—There are many published protocols for proper recording and analysis of the BAER.[6,13,14] The selection of a protocol to use or modify for use typically depends on the reasons for conducting the BAER. Recommended stimulus and recording parameters for the canine BAER are summarized (**Appendix**).

If the BAER is being evaluated for diagnostic or site-of-lesion reasons, it is typically recorded at suprathreshold intensities ≥ 70 dBnHL. This ensures optimal wave morphologic features and permits accurate assessment of the absolute and interwave latencies of waves I, III, and V (wave amplitudes are not always assessed because they are highly variable). Evaluation of the BAER includes the following assessments:

➤ Waveform morphology: Is there a waveform at all? Are all of the clinically important waves present? If the waves are present, are they sharply defined or is their morphology degraded?

➤ Waveform repeatability: Do repeated traces overlay well? Are the wave latencies similar?

➤ Absolute wave latencies: Are the absolute wave latencies within reference ranges?

➤ Interwave latencies: Are the interwave latencies within reference ranges?

➤ Interaural comparisons: Are the absolute and interwave latencies similar between ears?

➤ Wave amplitudes, if measured: Are values within reference ranges?

If the BAER assessment is being conducted for estimation of the hearing threshold, it is typically recorded at a series of decreasing intensities until the response threshold is obtained.

All of these analyses are dependent on the comparison of an individual dog's BAER results with appropriately matched normative data. Although it would be optimal for each clinic to obtain normative data for its respective animal populations, equipment, and protocols, published normative data are available in the literature. Examples include data for air-conducted click stimuli,[1,19,23-28] air-conducted tone burst stimuli,[7,22] and bone-conducted click stimuli.[71,79] It must be stressed that such data sets can only be used to analyze BAERs obtained in the same dog breeds and with the same stimulus and recording variables as those used to obtain the normative data set.

Current applications of the BAER in dogs

As a site-of-lesion diagnostic tool—Although the BAER is most suited for detection of neural lesions in CN VIII and the auditory portion of the brainstem, it can be used indirectly to form conclusions about the likelihood of conductive lesions in the outer and middle ears, sensory lesions in the inner ear, and neural lesions higher in the CNS than the auditory portion of the brainstem.[6,13,14]

Conductive lesions in the outer and middle ear most often affect wave I of the BAER, although a normal wave I does not rule out a conductive lesion. Because wave I represents CN VIII activity, conductive lesions can delay the onset of this wave and possibly reduce its amplitude. This can affect the remaining BAER waves, but because the source of the delay is caudal to the generators of the BAER, the BAER interwave latencies remain within reference ranges. In other words, once the BAER activity begins, it continues through the neuroaxis without interference. Conductive lesions of the outer and middle ear can therefore have the same effect on the BAER as a reduction in the stimulus level.[6,13,14]

Sensory lesions in the inner ear can have effects on the BAER that are similar (although not identical) to those of conductive lesions. At low stimulus intensities, sensory lesions can delay absolute wave latencies and reduce wave amplitudes yet leave interwave latencies mostly unchanged. At high stimulus intensities, however, there is sound recruitment by the hair cells in the cochlea and the BAER may return to normal limits. Complete loss of inner ear function may result in the complete loss of the BAER despite the absence of a retrocochlear neural lesion.[6,13,14]

Neural lesions of CN VIII and the auditory brainstem affect the BAER in different ways, depending on the site of the lesion. If the lesion is on CN VIII, BAER waves may be absent, delayed, or reduced in amplitude. If the lesion is between CN VIII and the caudal region of the auditory brainstem, then wave I may appear normal but waves II and those after it may be absent, delayed, or reduced in amplitude (often creating a delayed interwave latency between the earlier, but not the later, BAER waves). If the lesion is in the rostral region of the auditory brainstem, the early BAER waves may appear normal but the later waves (especially wave V) may be absent, delayed, or reduced in amplitude (often creating a delayed interwave latency between the later, but not the earlier, BAER waves). The key finding that differentiates a neural lesion in CN VIII or the auditory portion of the brainstem from a lesion in the outer, middle, or inner ear brainstem is a prolonged interwave latency.[6,13,14]

Although lesions caudal to CN VIII and the auditory brainstem should not affect the BAER, the finding of a normal BAER as part of a larger diagnostic evaluation can be used to infer the likelihood of such a lesion by way of a process of elimination. The sites of conductive,[3,4,6,80,96,97] sensory,[2-4,6,18,21,29,43,77,98] and neural[30,31] lesions have been evaluated by use of BAER testing in dogs.

As a tool for estimating hearing threshold—Hearing threshold estimation is the most frequently published use for the BAER in dogs.[1,6,21-23,25,71,80] By determining the threshold of the BAER response, the auditory function of the dog up to the level of the high brainstem can be estimated. This can be done by use of a range of stimulus types and frequencies (such as clicks and frequency-specific tone bursts) and a range of stimulus transducers (such as headphones and bone conductors), allowing both conductive and sensorineural threshold patterns to be determined across a range of frequencies.

Despite the frequent use of BAERs for determining the auditory threshold as cited in the veterinary literature pertaining to dogs, the technique is probably more widely used clinically as a threshold screening tool for the diagnosis of inherited and pigment-associated deafness. With this test, the dog's BAER is recorded in response to a single stimulus (typically a click) at a single intensity. If a BAER response results, the dog passes the test, whereas if no BAER response results, the dog fails the test. The main advantage of this approach is convenience; it can often be performed without anesthesia or sound-insulated facilities such as are required for full, accurate BAER-threshold assessment. The main disadvantage of the approach is that it is only a screening tool: only limited information is yielded because the BAER is derived from a single stimulus-intensity combination.

The BAER remains a useful tool for the assessment of auditory function in dogs. As our knowledge of the BAER increases, use of the technique as a measure of auditory function in dogs will continue.

References

1. Shiu JN, Munro KJ, Cox CL. Normative auditory brainstem response data for hearing threshold and neuro-otological diagnosis in the dog. *J Small Anim Pract* 1997;38:103–107.

2. Strain GM, Kearney MT, Gignac IJ, et al. Brainstem auditory-evoked potential assessment of congenital deafness in Dalmatians: associations with phenotypic markers. *J Vet Intern Med* 1992;6:175–182.

3. Strain GM. Aetiology, prevalence and diagnosis of deafness in dogs and cats. *Br Vet J* 1996;152:17–36.

4. Munro KJ, Cox CL. Investigation of hearing impairment in Cavalier King Charles spaniels using auditory brainstem response audiometry. *J Small Anim Pract* 1997;38:2–5.

5. Rose WR. Audiology-2: pure-tone audiometry. *Vet Med Small Anim Clin* 1977;72:422–431.

6. Sims MH. Electrodiagnostic evaluation of auditory function. *Vet Clin North Am Small Anim Pract* 1988;18:913–944.

7. Poncelet LC, Coppens AG, Deltenre PF. Audiograms estimated from brainstem tone-evoked potentials in dogs from 10 days to 1.5 months of age. *J Vet Intern Med* 2002;16:674–679.

8. Rose WR. Audiology-3: interpretation of audiograms (air-conduction testing). *Vet Med Small Anim Clin* 1977;72:624–629.

9. Rose WR. Small animal oncology. Audiology-4: interpretation of audiograms (bone-conduction testing). *Vet Med Small Anim Clin* 1977;72:863–867.

10. Rose WR. Audiology-5: conditioned pure-tone audiometry. *Vet Med Small Anim Clin* 1977;72:1090–1096.

11. Rose WR. Audiology-6: natural-sound test. *Vet Med Small Anim Clin* 1977;72:1165–1169.

12. Rose WR. Audiology-7: additional hearing tests. *Vet Med Small Anim Clin* 1977;72:1295–1298.

13. Hall JW. *Handbook of auditory evoked responses*. Boston: Allyn & Bacon, 1992;3–419.

14. Hood LJ. *Clinical applications of the auditory brainstem response*. San Diego: Singular Publishing Group, 1998;11–122.

15. American Encephalographical Society. Guidelines for clinical evoked potential studies. *J Clin Neurophysiol* 1984;1:3–53.

16. Kawasaki Y, Inada S. Peaks of brainstem auditory evoked potentials in dogs. *Vet Res Commun* 1994;18:383–396.

17. Barry SJ, Barry SD. Surface-recorded auditory brainstem responses in the dog. *J Aud Res* 1980;20:249–252.

18. Morgan JL, Coulter DB, Marshall AE, et al. Effects of neomycin on the waveform of auditory-evoked brain stem potentials in dogs. *Am J Vet Res* 1980;41:1077–1081.

19. Bodenhamer RD, Hunter JF, Luttgen PJ. Brain stem auditory-evoked responses in the dog. *Am J Vet Res* 1985;46:1787–1792.

20. Uzuka Y, Fukaki M, Wada M. A study on the generator of auditory evoked responses in dogs. *Jpn J Electroencephalogr Electromyogr* 1992;20:233–240.

21. Uzuka Y, Furuta T, Yamaoka M, et al. Threshold changes in auditory brainstem response (ABR) due to the administration of kanamycin in dogs. *Exp Anim* 1996;45:325–331.

22. Uzuka Y, Fukaki M, Hara Y, et al. Brainstem auditory evoked responses elicited by tone-burst stimuli in clinically normal dogs. *J Vet Intern Med* 1998;12:22–25.

23. Kay R, Palmer AC, Taylor PM. Hearing in the dog as assessed by auditory brainstem evoked potentials. *Vet Rec* 1984;114:81–84.

24. Sims MH, Moore RE. Auditory-evoked response in the clinically normal dog: early latency components. *Am J Vet Res* 1984;45:2019–2027.

25. Marshall AE. Brain stem auditory-evoked response of the nonanesthetized dog. *Am J Vet Res* 1985;46:966–973.

26. Myers LJ, Redding RW, Wilson S. Reference values of the brainstem auditory evoked response of methoxyflurane anesthetized and unanesthetized dogs. *Vet Res Commun* 1985;9:289–294.

27. Strain GM, Tedford BL, Jackson RM. Postnatal development of the brain stem auditory-evoked potential in dogs. *Am J Vet Res* 1991;52:410–415.

28. Venker-van Haagen AJ, Siemelink RJ, Smoorenburg GF. Auditory brainstem responses in the normal beagle. *Vet Q* 1989;11:129–137.

29. Knowles KE, Cash WC, Blauch BS. Auditory-evoked responses of dogs with different hearing abilities. *Can J Vet Res* 1988;52:394–397.

30. Fischer A, Obermaier G. Brainstem auditory-evoked potentials and neuropathologic correlates in 26 dogs with brain tumors. *J Vet Intern Med* 1994;8:363–369.

31. Steiss JE, Cox NR, Hathcock JT. Brain stem auditory-evoked response abnormalities in 14 dogs with confirmed central nervous system lesions. *J Vet Intern Med* 1994;8:293–298.

32. Moore JK. The human auditory brainstem: a comparative review. *Hear Res* 1987;29:1–32.

33. Moore JK. The human auditory brainstem response as a generator of auditory evoked potentials. *Hear Res* 1987;29:33–43.

34. Huang CM. A comparative study of the brain stem auditory response in mammals. *Brain Res* 1980;184:215–219.

35. Buchwald JS, Huang C. Far-field acoustic response: origins in the cat. *Science* 1975;189:382–384.

36. Lev A, Sohmer H. Sources of averaged neural responses recorded in animal and human subjects during cochlear audiometry (electro-cochleogram). *Arch Klin Exp Ohren Nasen Kehlkopfheilkd* 1972;201:79–90.

37. Møller AR. Neural generators of the brainstem auditory evoked potentials. *Sem Hear* 1998;19:11–27.

38. Goldberg JM. Physiological studies of auditory nuclei of the pons. In: Ades HW, Keidel WD, Neff WD, eds. *Auditory system*. New York: Springer-Verlag, 1974;109–144.

39. Pickles JO. *An introduction to the physiology of hearing*. London, New York: Academic Press Inc, 1988;34–48.

40. Helfert RH, Aschoff A. Superior olivary complex and nucleus of the lateral lemniscus. In: Ehret G, ed. *The central auditory system*. New York: Oxford University Press, 1997;193–258.

41. Dumitru D. *Electrodiagnostic medicine*. Philadelphia: Hanley & Belfus, 1995;29–64.

42. Scherg M, von Cramon D. A new interpretation of the generators of BAEP waves I-V: results of a spatio-temporal dipole model. *Electroencephalogr Clin Neurophysiol* 1985;62:290–299.

43. Strain GM, Merchant SR, Neer TM, et al. Ototoxicity assessment of a gentamicin sulfate otic preparation in dogs. *Am J Vet Res* 1995;56:532–538.

44. Hashimoto I, Ishiyama Y, Yoshimoto T, et al. Brain-stem auditory-evoked potentials recorded directly from human brain-stem and thalamus. *Brain* 1981;104:841–859.

45. Møller AR, Jannetta PJ. Compound action potentials recorded intracranially from the auditory nerve in man. *Exp Neurol* 1981;74:862–874.

46. Møller AR, Jannetta PJ. Evoked potentials from the inferior colliculus in man. *Electroencephalogr Clin Neurophysiol* 1982;53:612–620.

47. Møller AR, Jannetta PJ. Auditory evoked potentials recorded from the cochlear nucleus and its vicinity in man. *J Neurosurg* 1983;59:1013–1018.

48. Møller AR, Jannetta PJ. Interpretation of brainstem auditory evoked potentials: results from intracranial recordings in humans. *Scand Audiol* 1983;12:125–133.

49. Møller AR, Jannetta PJ. Monitoring auditory functions during cranial nerve microvascular decompression operations by direct recording from the eighth nerve. *J Neurosurg* 1983;59:493–499.

50. Møller AR, Jannetta P, Bennett M, et al. Intracranially recorded responses from the human auditory nerve: new insights into the origin of brain stem evoked potentials (BSEPs). *Electroencephalogr Clin Neurophysiol* 1981;52:18–27.

51. Møller AR, Jannetta P, Moller MB. Intracranially recorded auditory nerve response in man. New interpretations of BSER. *Arch Otolaryngol* 1982;108:77–82.

52. Grandori F. Field analysis of auditory evoked brainstem potentials. *Hear Res* 1986;21:51–58.

53. Ino T, Mizoi K. Vector analysis of auditory brainstem response (BSR) in human beings. *Arch Otolaryngol* 1980;226:55–62.

54. Sohmer H, Feinmesser M, Szabo G. Sources of electrocochleographic responses as studied in patients with brain damage. *Electroencephalogr Clin Neurophysiol* 1974;37:663–669.

55. Starr A, Achor J. Auditory brain stem responses in neurological disease. *Arch Neurol* 1975;32:761–768.

56. Starr A, Hamilton AE. Correlation between confirmed sites of neurological lesions and abnormalities of far-field auditory brainstem responses. *Electroencephalogr Clin Neurophysiol* 1976;41:595–608.

57. Stockard JJ, Rossiter VS. Clinical and pathologic correlates of brain stem auditory response abnormalities. *Neurology* 1977;27:316–325.

58. Møller AR, Jannetta PJ, Sekhar LN. Contributions from the auditory nerve to the brain-stem auditory evoked potentials (BAEPs): results of intracranial recording in man. *Electroencephalogr Clin Neurophysiol* 1988;71:198–211.

59. Spoendlin H, Schrott A. Analysis of the human auditory nerve. *Hear Res* 1989;43:25–38.

60. Arezzo J, Pickoff A, Vaughan HG Jr. The sources and intracerebral distribution of auditory evoked potentials in the alert rhesus monkey. *Brain Res* 1975;90:57–73.

61. Prasher DK. Alternative hypotheses concerning the sources of the human brain stem auditory evoked potentials. *Scand Audiol* 1981;10:63–64.

62. Musiek FE, Geurkink NA. Auditory brain-stem response and central auditory test findings for patients with brainstem lesions: a preliminary report. *Laryngoscope* 1982;92:891–900.

63. Oh SJ, Kuba T, Soyer A, et al. Lateralization of brainstem lesions by brainstem auditory evoked potentials. *Neurology* 1981;31:14–18.

64. Poncelet L, Coppens A, Deltenre P. Brainstem auditory evoked potential wave V latency-intensity function in normal Dalmatian and Beagle puppies. *J Vet Intern Med* 2000;14:424–428.

65. Poncelet LC, Coppens AG, Meuris SI, et al. Maturation of the auditory system in clinically normal puppies as reflected by the brain stem auditory-evoked potential wave V latency-intensity curve and rarefaction-condensation differential potentials. *Am J Vet Res* 2000;61:1343–1348.

66. Latshaw WK. *Veterinary developmental anatomy: a clinically oriented approach*. Philadelphia: BC Decker, 1987;249–274.

67. Knowles K, Blauch B, Leipold H, et al. Reduction of spiral ganglion neurons in the aging canine with hearing loss. *Zentralbl Veterinarmed [A]* 1989;36:188–199.

68. Pook HA, Steiss JE. Correlation of brain stem auditory-evoked responses with cranium size and body weight of dogs. *Am J Vet Res* 1990;51:1779–1783.

69. Meij BP, Venker van Haagen AJ, van den Brom WE. Relationship between latency of brainstem auditory-evoked potentials and head size in dogs. *Vet Q* 1992;14:121–126.

70. Munro KJ, Shiu JN, Cox CL. The effect of head size on the auditory brainstem response for two breeds of dog. *Br J Audiol* 1997;31:309–314.

71. Munro KJ, Paul B, Cox CL. Normative auditory brainstem response data for bone conduction in the dog. *J Small Anim Pract* 1997;38:353–356.

72. Marshall NK, Donchin E. Circadian variations in the latency of brainstem responses and its relation to body temperature. *Science* 1981;212:356–358.

73. Hall JW. Auditory brainstem response audiometry. In: Jerger JF, ed. *Hearing disorders in adults*. San Diego: College-Hill Press, 1984;3–55.

74. Rosenblum SM, Ruth RA, Gal TJ. Brainstem auditory evoked potential monitoring during profound hypothermia and circulatory arrest. *Ann Otol Rhinol Laryngol* 1995;94:281–283.

75. Sohmer H, Gafni M, Chisin R. Auditory nerve and brain stem responses. Comparison in awake and unconscious subjects. *Arch Neurol* 1978;35:228–230.

76. Osterhammel PA, Shallop JK, Terkildsen K. The effect of sleep on the auditory brainstem response (ABR) and the middle latency response (MLR). *Scand Audiol* 1985;14:47–50.

77. Merchant SR, Neer TM, Tedford BL, et al. Ototoxicity assessment of a chlorhexidine otic preparation in dogs. *Prog Vet Neurol* 1992;4:72–75.

78. Tokuriki M, Matsunami K, Uzuka Y. Relative effects of xylazine-atropine, xylazine-atropine-ketamine, and xylazine-atropine-pentobarbital combinations and time-course effects of the latter two combinations on brain stem auditory-evoked potentials in dogs. *Am J Vet Res* 1990;51:97–102.

79. Strain GM, Green KD, Twedt AC, et al. Brain stem auditory evoked potentials from bone stimulation in dogs. *Am J Vet Res* 1993;54:1817–1821.

80. Wolschrijn CF, Venker van Haagen AJ, van den Brom WE. Comparison of air- and bone-conducted brain stem auditory evoked responses in young dogs and dogs with bilateral ear canal obstruction. *Vet Q* 1997;19:158–162.

81. Davis H. Principles of electric response audiometry. *Ann Otol Rhinol Laryngol* 1976;28(suppl 85):1–96.

82. Ozdamar O, Dallos P. Input-output functions of cochlear whole-nerve action potentials: interpretation in terms of one population of neurons. *J Acoust Soc Am* 1976;59:143–147.

83. Møller AR. Latency in the ascending auditory pathway determined using continuous sounds: comparison between transient and envelope latency. *Brain Res* 1981;207:184–188.

84. Klein AJ. Properties of the brain-stem response slow-wave component. II. Frequency specificity. *Arch Otolaryngol* 1983;109:74–78.

85. Pratt H, Sohmer H. Intensity and rate functions of cochlear and brainstem evoked responses to click stimuli in man. *Arch Otorhinolaryngol* 1976;212:85–92.

86. Suzuki T, Kobayashi K, Takagi N. Effects of stimulus repetition rate on slow and fast components of auditory brain-stem responses. *Electroencephalogr Clin Neurophysiol* 1986;65:150–156.

87. Borg E, Lofqvist L. Auditory brainstem response (ABR) to rarefaction and condensation clicks in normal and abnormal ears. *Scand Audiol* 1982;11:227–235.

88. Moore MP, Fisher DJ, Gavin PR, et al. Effects of electrode position and stimulus polarity on brain stem auditory evoked responses in dogs. *Prog Vet Neurol* 1990;1:461–472.

89. Jones SJ, Van der Poel JC. Binaural interaction in the brainstem auditory evoked potential: evidence for a delay line coincidence detection mechanism. *Electroencephalogr Clin Neurophysiol* 1990;77:214–224.

90. Rawool VW, Ballachanda BB. Homo- and anti-phasic stimulation in ABR. *Scand Audiol* 1990;19:9–15.

91. Polyakov A, Pratt H. The effect of binaural masking noise disparity on human auditory brainstem binaural interaction components. *Audiology* 1998;37:17–26.

92. Brantberg K, Fransson PA, Hansson H, et al. Measures of the binaural interaction component in human auditory brainstem response using objective detection criteria. *Scand Audiol* 1999; 28:15–26.

93. Holliday TA, Te Selle ME. Brain stem auditory-evoked potentials of dogs: wave forms and effects of recording electrode positions. *Am J Vet Res* 1985;46:845–851.

94. Kawasaki Y, Inada S. Power spectral analysis and digital filtration of brain stem auditory evoked potentials in dogs. *Am J Vet Res* 1993;54:1822–1826.

95. Kawasaki Y, Inada S. Effects of analog filtering on brain stem auditory-evoked potentials in dogs. *Am J Vet Res* 1992; 53:1096–1100.

96. Steiss JE, Boosinger TR, Wright JC, et al. Healing of experimentally perforated tympanic membranes demonstrated by electrodiagnostic testing and histopathology. *J Am Anim Hosp Assoc* 1992;28:307–310.

97. Eger CE, Lindsay P. Effects of otitis on hearing in dogs characterised by brainstem auditory evoked response testing. *J Small Anim Pract* 1997;38:380–386.

98. Sims MH, Shull-Selcer E. Electrodiagnostic evaluation of deafness in two English Setter littermates. *J Am Vet Med Assoc* 1985;187:398–404.

Appendix appears on the next page

Appendix
Summary of nonpathologic, stimulus, and recording factors used in recording the brainstem auditory evoked response (BAER) in dogs.

Variable	Effect	Recommendation
Nonpathologic factors		
Age	Reliable BAERs from 2 weeks Adultlike BAERs by 6 to 8 weeks	Age-appropriate normative data required, especially for dogs < 6 to 8 weeks of age
Head size	Thought to affect the BAER	Correction for head size should be considered
Sex	No known effect	No correction for sex
Body temperature	No effect from 37°–39.5°C	Correction factors needed for body temperatures < 37° and > 39.5°C
Attention and state of arousal	No effect	No corrections needed
Pharmacologic agents	Affected by ototoxic drugs	No corrections needed
Muscle artifact	Substantially affects the BAER	Chemical restraint recommended if required
Stimulus factors		
Type	Many stimulus types available	0.1-ms square-wave click recommended for site-of-lesion and threshold screening Frequency-specific tone bursts recommended for threshold assessment
Intensity	BAER profoundly affected by stimulus intensity	Suprathreshold intensities (≥ 70 dBnHL) for site-of-lesion assessments Decreasing stimulus intensities for threshold assessments Appropriate masking of nontest ear as required
Rate	BAER affected by stimulus rates > 20 clicks/s	Stimulus rates < 20 clicks/s for site-of-lesion assessments Stimulus rates > 20 clicks/s but < 40 clicks/s for threshold assessments
Polarity	BAER minimally affected by stimulus polarity	Rarefacting stimulus recommended, but alternating stimulus can be used if electrical artifact is a problem
Transducers	Many transducers can be used for BAER	Insert earphones avoid ear canal collapse, but headphones are often more accessible Bone conductor needed if outer or middle ear involvement is suspected
Monaural vs binaural stimulation	Binaural stimulation affects BAER	Monaural stimulation
Recording factors		
Analysis time	BAER complete by approximately 8 ms	10.24-ms analysis time 15.36-ms analysis time for newborns or when hearing loss is profound
Electrode placement	BAER is affected by electrode placement	Ipsilateral array: noninverting electrode (active) on vertex (Cz), inverting electrode (reference) rostral to tragus of test ear, ground electrode rostral to tragus of nontest ear
Amplifiers	High gain required	100,000–150,000 absolute gain
Filters	BAER affected by filter settings	Low-pass filter setting of 3,000 Hz High-pass filter setting of ≤ 53 Hz
Averaging	Substantial averaging is required to record the BAER	About 1,000 sweeps

dBnHL = Decibels normalized hearing level.