Chapter 2.3

Short-latency auditory evoked potentials

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Introduction

Cochlear and brain-stem auditory evoked potentials include the electrocochleogram (ECOG) and brain-stem auditory evoked potentials (BAEPs). These evoked potentials have come into widespread use for assessment of the clinical state of the cochlea, auditory nerve and middle portion of the brain-stem and for assessment of hearing, particularly in the screening of infants at risk for hearing loss. They provide objective measures for hearing screening for infants when used in conjunction with otoacoustic emissions that quantify sound emissions by healthy hair cells. Cochlear and auditory brainstem potentials are objective, reproducible, and sensitive indicators of many types of cochlear and brainstem disturbances.

Cochlear potentials that can be recorded noninvasively from humans include the cochlear microphonic potentials (CM), the summing potential (SP) and the compound action potential of the auditory nerve (N1). These potentials can be recorded by an electrode placed within the ear canal or even adjacent to the stimulated ear as on the mastoid or ear lobe, allowing their identification in standard BAEP recordings. These cochlear and auditory nerve potentials reflect activities of cochlear hair cell and the portion of auditory nerve within the cochlea and are independent of the subject’s state of arousal or the effects of drugs.

Additional measures of haircell function are afforded by a relatively new physiological measure (Kemp 1990) called otoacoustic emissions (OAEs). OAEs are faint sounds emitted by active mechanical processes in the cochlear outer hair cells, occurring both in the absence of acoustic stimulation (spontaneous OAEs) and in response to acoustic stimuli including clicks (transient OAEs) or two tone combinations (distortion product OAEs). OAEs are recorded with a small sensitive microphone fitted through a small earplug into the external ear canal. The sound stimuli for evoked OAEs are delivered by a small flexible tube connecting the acoustic stimulator to the ear canal behind the small earplug. The equipment for recording OAEs is relatively inexpensive and the recording procedures are relatively brief requiring approximately 5–10 min.

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Brain-stem auditory evoked potentials include a series of 5 or more peaks representing activities of the VIII nerve both distally (wave I, equivalent to N1 in the electrocochleogram) and proximally (wave II) and activities that are presumed to arise in the cochlear nucleus (wave III), superior olive and lemniscal (IV, V) auditory brain-stem.

The ECoG, OAEs, and BAEP can be easily recorded from most patients, including comatose or sedated patients in whom routine audiometry cannot be done. In fact, the best quality records are often obtained with sleep or sedation. ECoG and BAEP can be obtained in the presence of mild or moderate hearing impairment, but may be absent in the presence of severe hearing impairment. OAEs are lost with mild hearing loss (>40 dB) due to cochlear disease.

**Stimulation**

ECoGs and BAEPs are typically evoked by brief “click” stimuli produced by activating an acoustic transducer with monophasic square-wave electrical pulses of short duration (e.g., 100 μs). The resulting stimulus is a series of sound waves lasting several milliseconds. The polarity of the square pulse affects the initial direction of the sound transducer diaphragm as either outward or inward. Initial outward movement (toward eardrum) is called an acoustic “condensation” phase stimulus, whereas initial inward movement is a “rarefaction” stimulus. Occasionally the two stimulus polarities are concatenated, which is referred to as an “alternating” phase stimulus and can be useful in reducing the electrical stimulus artifact or to cancel cochlear microphonics.

In recording auditory evoked potentials rarefaction stimuli are usually chosen because the initial phase of the stimulus displaces the cochlear basilar membrane toward scala vestibuli, leading to eighth nerve excitation. The initial phase of condensation stimuli pushes the basilar membrane away from the scala vestibuli and activation of haircells and VIII nerve is delayed till the subsequent rarefaction phase of the acoustic wave form. Both the morphology and latencies of BAEP components are affected by click polarity requiring knowledge of which stimulus polarity is being employed. The equipment and procedures required for defining the polarity of the initial phase of the acoustic stimulus can be found in the manual of the acoustic equipment used for calibration.

The separation of stimulus artifact from the cochlear receptor responses is essential. A temporal separation can be obtained by introducing an acoustic delay between activation of the acoustic transducer and the appearance of the sound at the ear drum. This can be accomplished by placing the transducer at a distance from the ear canal and conducting the emitted sound to the subject’s ear canal by a tube. For example, a 30 cm length of coupling tube introduces a 1 ms delay between stimulus artifact and the arrival of sound at the ear canal. Consequently, there will be a 1 ms delay between stimulus artifact and the physiological response of the cochlea.

Clicks are usually presented 10–70 times/s. At fast rates, data can be collected more quickly, but the neural components in both the ECoG and BAEPs will be attenuated and delayed. Cochlear haircell potentials are not affected by stimulus rates. For neurological assessments rates of approximately 11/s are commonly chosen whereas for assessment of hearing, rates between 20 and 40 are usually used. For neurological assessments, the effect of increasing stimulus rate from slow to fast rate may be indicative of specific impairments affecting brainstem transmission. The stimulus rates selected should not be exact factors divisible into 50 or 60 Hz, because such rates would predispose to inclusion of power line noise in the average.

Stimuli are often delivered at about 70 decibels (dB) above normal hearing threshold. There are several different decibels scales in common clinical use. It is important to understand the difference between these intensity scales.

1. **Hearing level** (dB NHL, dB HL, dB nHL). This scale refers to the intensity in decibels (dB) of intensity compared to the threshold of hearing in a group of normal subjects tested in a quiet environment. Zero on this scale is defined as the threshold intensity at which an average normal subject can just perceive the stimulus 50% of the time.
(2) **Sensation level (dB SL).** This is the scale on which zero is defined as the intensity at which the individual patient can just barely appreciate the stimulus. This may be very different from 0 dB HL. Patients with hearing loss may have their personal 0 dB SL found at very high intensities such as 70 dB nHL.

(3) **The physical definition (dB peSPL, peak equivalent sound pressure level).** Physical measurement of sound pressure levels uses as the 0 dB reference level a pressure of 20 micropascals (μPa), which equals 0.0002 dyne/cm² (2 × 10⁻⁵ N/m²). The 0 dB peSPL of a typical click turns out to be approximately equal to −30 dB nHL.

(4) **Individual equipment scales.** Each particular piece of equipment has dial settings in dB units. Although the original intention of the manufacturer was to make these equivalent to dB nHL, many individual pieces of equipment actually differ from that standard. An individual user should know the relationship between his/her equipment and the nHL scale.

For testing of hearing, a latency-intensity test can be performed. In this test, the stimulus is reduced to specific lower intensities, such as 65, 50, 40 and 30 dB nHL. At each intensity, a BAEP trace is run. Waves V can be detected on each of these and changes in the Wave V latency reflect primarily cochlear functions, as described below. Some users prefer to use just 2 stimulus-intensity steps, e.g., 70 dB and 30 dB nHL as a screening procedure to assess cochlear function. The use of procedures for hearing evaluation will be reviewed in a subsequent portion of this report.

Contralateral white noise masking is often employed. It is most often useful when there is an asymmetry of hearing between the ears. The contralateral white noise serves as a “masker” and interferes with acoustic crossover through air and bone conduction of the click stimulus from the side with impaired hearing to the unstimulated side with better hearing. The contralateral white noise masking is usually set at 40 dB below the intensity of the click presented to the ear being tested. Such low contralateral stimulus levels are unlikely to activate neural systems leading to efferent suppression of cochlear function, which to date has been demonstrated for OAEs and only with higher intensities of contralateral stimulation.

**Recordings**

The ECoG can be recorded from the ear canal using an electrode resting on the tympanic membrane (see Stypulkowski and Staller 1987 for details of the electrode), referenced to a surface electrode on the contralateral earlobe or mastoid. Before the tympanic membrane electrode is placed, the integrity of the tympanic membrane must be verified otoscopically and the ear canal thoroughly cleansed of deposits to assure good electrical contact of the electrode. Once in place, the electrode is stabilized by placing a compressed foam earplug through which a sound delivery tube enables stimulus presentation. When the foam earplug expands it secures the electrode and sound delivery tube in the ear canal.

Cochlear potentials can also be recorded from the ear canal, using a ball-tipped wire secured against the canal wall, or from the mastoid or earlobe, using a standard surface disc electrode, referenced to a disc electrode on the contralateral earlobe or mastoid. The amplitude of cochlear potentials decreases as the distance between the cochlea and the recording electrode increases, with a differential effect on high compared to low frequency potentials.

In routine clinical application, a typical band-pass for recording all components of ECoG is 3–3000 Hz, stimulus rate is around 10/s, and the number of responses averaged for each trace is between 1000–2000. To avoid waveform distortions and to enhance latency resolution, sampling rate should not be lower than 20 kHz (50 μs/ address or less).

Recording electrodes for BAEP recording should be placed at both ear lobes and at site Cz at the scalp vertex. In each case, an approximately 1 cm diameter disk electrode should be securely fastened to the recording site and the electrical contact impedance should be kept below 5000 Ω. The recording electrode on the ipsilateral ear may be designated Ai, and on the contralateral ear Ac.

The recording montage suggested is:
Channel 1: Cz-Ai, the ipsilateral channel,
Channel 2: Cz-Ac, the contralateral channel.

In each case negative electrical potentials at the ear electrodes should be displayed as upgoing deflections or peaks.

Some users prefer 1 or 2 additional recording channels. Recording Ai-Ac can sometimes help clarify wave I. Recording from Cz to an occipital or neck reference will result in higher amplitudes of waves IV/V compared to ear reference recordings.

The filter high-pass (lower frequency limit allowed through the recording system) is often set at 100 Hz and the low-pass (higher limit of the frequencies passed and amplified through the recording system) at 3000 Hz. These settings will attenuate waves IV/V and some users prefer to set the high-pass as low as 10–30 Hz, which will not adversely affect the amplitude of waves IV–V. Use of a narrow band 50 or 60 Hz filter does not usually interfere with BAEP recordings. Recordings are made during the 10 ms after the stimulus in most settings. For neonates, premature infants and for latency-intensity testing, 12–20 ms analysis time is usually chosen in place of 10 ms. Approximately 2000 trials are required to reduce the background EEG noise and obtain useful BAEP recordings. However, the actual number may vary widely depending on factors that introduce ‘noisy’ recordings such as patient movements, high electrode impedance, and close proximity to other recording equipment. Two or three separate repetitions are run and superimposed to ascertain that reproducible records are obtained. Latency values measured on the separate repetitions should agree with each other within 0.10 ms or less. Amplitude values should agree with each other to within ±10%.

Computational, rather than ‘eye-ball’, methods for determining whether a response is present and for assessing its quality include signal detection and statistical measures. Some of these methods have been implemented in commercially available recording systems. The simplest of these methods is the (±) method (Schimmel 1967) in which in addition to averaging, the single trials are alternately added and subtracted resulting in cancellation of the constant, time-locked signal and survival of the residual noise. Comparing the waveforms of the average and of the (±) waveforms allows estimation of the signal to noise ratio in the average. A statistical measure of the likelihood of a signal in a waveform is based of the F ratio of the variance between points along the waveform relative to the variance at a single point in time across the trials in the average. The probability of a signal in the waveform is computed from the F value, and hence the name of the method, F of a single-point ($F_{sp}$, Don et al. 1984). Because peak V is the largest component and the last to disappear close to threshold, its peak is the single point for which $F_{sp}$ calculation is usually made. However, in BAEP the exact latency for which $F_{sp}$ is calculated makes little difference.

Principal peaks and their identification

In ECoG (Fig. 1) the cochlear compound action potential in response to alternating polarity, high

![ECoG Diagram](image)

Fig. 1. ECoG to rarefaction (top) and to alternating polarity (bottom) clicks. Note the cochlear microphonic potentials (CM) preceding the action potential (N1) in response to the rarefaction clicks, and their cancellation in response to alternating polarity clicks, exposing the summating potential (SP).
intensity clicks is recorded as a major negative peak of a few microvolts, called \( N_1 \), at approximately 1.5 ms after stimulus onset in the ear canal. \( N_1 \) is followed by a negativity called \( N_2 \), at about 2.5 ms. The Summating Potential (SP) preceding \( N_1 \) is a negative step-like deflection from baseline, best identified with alternating polarity stimuli, and is obscured by the Cochlear Microphonic (CM) when using one polarity of stimulation.

BAEP consists of between 5 and 8 vertex positive peaks, generally labeled with Roman numerals. The principal peaks I–V are the main peaks of clinical interest. These are shown in Fig. 2. The several succeeding peaks VI–VIII are quite variable and not used clinically.

The troughs immediately following each peak are designated by the same numeral followed by a prime-mark. For example, the trough after V is labeled \( V' \). Identification of components is helped by the rules listed below, although they do not apply to every case. For example, when other abnormalities exist, the typical shapes of a particular component may be altered.

Wave I: This wave is generated by the portion of the auditory nerve within the cochlea and is the far-field reflection of the N1 component of the ECog. It is a prominent initial negative-going peak in the ipsilateral ear recording channel and is markedly attenuated, reversed in polarity or absent from the contralateral ear recording channel. Patients who have preserved cochlear functions should have a preserved wave I. Wave I is often present as the only BAEP component in brain death.

Wave II: This wave may be generated by the proximal auditory nerve near or at the cochlear nucleus and can be preserved in brainstem death. Wave II is poorly defined in some adults and most neonates. It sometimes appears as a small peak between waves I and III. It is often more prominent on the contralateral channel recording, where it has a slightly prolonged latency compared to the ipsilateral channel, sometimes fusing with wave III into an M-shaped II–III complex.

Wave III: This wave is probably generated by the lower pons as the pathway travels from the cochlear nucleus through the trapezoidal body and superior olives. The fiber tracts and/or nuclei that are most responsible for generating this potential are unknown and may be multiple.

Wave III is followed by a prominent \( III' \) trough. In the contralateral channel wave III often appears smaller and earlier than in the ipsilateral channel because its similar polarity and amplitude at the vertex and contralateral ear is diminished by the differential recording.

Waves IV and V: Contributions to these two potentials probably include generators in the upper pons or lower midbrain, in the lateral lemniscus but not the inferior colliculus proper. There are conflicting reports about whether these peaks are generated in the ipsilateral or contralateral brainstem, but the preponderance of evidence favors a contralateral brainstem generator site for wave V.

Waves IV and V may fuse together forming an IV–V complex in the ipsilateral recording channel. This complex can vary between: (a) two peaks which are close but still visibly separate, and (b) a single peak which is completely fused. There are also various intermediate stages of trapezoidal-

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**Fig. 2.** The principal five BAEP peaks are identified by numerals I–V. Both ipsilateral and contralateral ear channels are shown, both recorded with a vertex reference. The peaks seen here are typical for an adult patient.
shaped figures which represent the partial fusion of the two peaks. In the contralateral recording channel the IV and V peaks tend to be separated more from each other with wave IV being slightly earlier and wave V being slightly later than in the ipsilateral channel. Comparison of ipsilateral and contralateral records can be helpful for distinguishing which peak or shoulder to score as wave V. Wave V is generally followed by a large V' trough. Sometimes wave VI appears before the bottom of this trough and can be confused with a wave V. Such IV–V–VI fusion tends to occur more often when the recording high-pass is set at 10 or 30 Hz.

The typical IV–V complex has the shape of a somewhat inflated triangle or trapezoid. The base of this complex should be more than 1.5 ms across. A peak narrower than this is usually a wave IV appearing alone. When doubt as to the identity of wave V exists, the examiner should change some of the test parameters in order to help the identification process. For instance, wave V is a robust peak that is present at low intensities of stimulation (often even despite high frequency hearing impairment, or other types of peripheral auditory changes). Wave V', and especially the V' trough, are often the last deflections to disappear when the stimulus intensity is gradually decreased to threshold. Click polarity may also help distinguish peak V from IV as the two are more distinct in response to rarefaction clicks. This effect may, however, be confounded by some cochlear pathologies which may diminish peak V. There are several traditional ways to help identify wave V. First, the typical splitting of the IV–V complex in a contralateral recording. Second, the preservation of wave V at lower stimulus intensities and high stimulus rates. Third, the increase in the IV/V complex when recording from Cz to C7 compared to Cz to Ai or Ac recordings.

Normal limits and the clinical correlation of changes

Important measures derived from the ECochG waveform include N₁ latency (from stimulus onset) and amplitude (between the transition from SP to N₁ and the negative peak of N₃), as well as SP duration (between its onset and transition to N₁) and amplitude (between its onset and most negative point). Cochlear microphonic amplitudes can be defined as occurring before the SP and measured as the peak difference between the largest amplitude waves evoked by condensation and rarefaction clicks.

Electrocochleography is used to monitor cochlear function intraoperatively, to help distinguish amongst cochlear disorders, and to identify VIII nerve compound action potentials for the determination of central auditory transmission time when surface recording of wave I is unreliable due to a peripheral hearing loss. The definition of cochlear microphonics when the auditory nerve action potential (N₁ or wave I) is absent is consistent with disordered auditory nerve functions. Electrocochleography is also used in intraoperative monitoring of brain-stem and/or temporal bone procedures to help identify if cochlear damage were to occur.

Typical adult BAEP measures are provided in Table 1.

There are five principle measures used to assess BAEPs.

First, I–V interpeak interval: This is the primary feature for most BAEP interpretations. It represents conduction from distal eighth nerve through pons and into the midbrain. It can be slowed in a variety of disorders, including focal damage (demyelination, ischemia, tumors), or diffuse lesions (degenerative disorders, post-hypoxic damage, etc.) anywhere along the auditory pathway between the generators for wave I (distal VIII nerve) and wave V (upper pons).

A typical upper limit for the I–V interpeak interval is 4.5 ms. That limit is slightly lower for young women and slightly higher for men above the age of 70. Normal right-left asymmetries for the I–V interpeak intervals should be at most 0.5 ms. For full-term infants, the I–V interpeak interval should be less than 5.4 ms.

High-tone hearing loss with relative preservation of low-frequency hearing (sloping audiogram of more than 30 dB/octave around 2000 Hz) may produce a paradoxical shortening of the I–V
interval to as little as 3.6 ms. The basis for this finding is that wave I with such hearing losses is generated by the low frequency end of the cochlea, resulting in a longer latency than is customary when high frequency cochlear activity dominates the contribution to wave I. In contrast, wave V latency can be preserved in the normal range in low frequency hearing loss because the few remaining high-frequency cochlear inputs diverge within the brainstem and activate a sufficient portion of the brainstem pathway to produce a normal latency wave V. Consequently, knowledge of the audiogram is helpful in insuring that a normal I–V interpeak interval represents a normal conduction along the central auditory pathway. Conversely, a delayed I–V interval is unlikely to be due to cochlear defects and represents altered central conduction delays in the brainstem.

Second, I–III interpeak interval: This interpeak interval represents conduction from the cochlear portion of the eighth nerve across the subarachnoid space, into the core of the lower pons. This I–III portion of the pathway is susceptible to a tumor, inflammation or other disorder specifically affecting the proximal portion of the eighth nerve or the pontomedullary junction where the eighth nerve enters the brainstem, or impairments in the lower pons around the trapezoidal body. Acoustic neuromas or other cerebellopontine angle tumors can cause a delay at the juncture. Infarction can cause an interruption or a delay here too, although the classic Wallenberg syndrome is usually too caudal to affect this segment. Inflammation in the subarachnoid space can also increase this I–III interpeak interval (subarachnoid hemorrhage, meningitis, and Guillain–Barré syndrome).

The upper normal limit for the I–III interpeak interval is about 2.5 ms. The acceptable right/left asymmetry of this interval is less than 0.5 ms.

Third, III–V interval: This interval reflects conduction from the lower to the upper pons and tegmentum. There is not yet complete agreement on whether this III–V interpeak interval represents conduction along the brainstem ipsilateral or contralateral to the stimulated ear. The preponderance of evidence favors a contralateral brainstem site.

The typical upper limit of normalcy for a III–V interpeak interval is about 2.4 ms. A right-left asymmetry for these intervals should be less than 0.5 ms. An excessively long III–V interpeak interval is not considered abnormal unless either the I–V interval or the V/I amplitude ratio is also abnormal.

Fourth, V/I amplitude ratio: Absolute amplitudes of BAEP peaks vary widely among normal subjects. In addition, several technical factors influence the absolute amplitudes of the BAEP peaks. To reduce this normal intersubject variability, a ratio of amplitudes is usually calculated. For this ratio, the amplitude of the IV–V complex is divided by the amplitude of wave I. The IV–V complex is measured from the highest point of the complex to the trough of the V' peak. When V is completely separated from IV, the V amplitude is used in place of the IV–V amplitude. When wave VI is found part way down the descending slope of wave V, then the amplitude is measured to the trough following wave VI (VI'). The wave I amplitude is measured from the top of the highest part of wave I to the bottom of the trough of I'. If wave II is riding on the descending slope of wave I, then wave I amplitude is measured to the succeeding trough of wave II'.

The amplitude ratio should be between 50% and 300%. These numbers vary between laboratories, and they are especially affected by filter setting changes. When the V/I is less than 50%, then the IV–V peaks are too small. In that case, suspicion is raised of some central impairment which has diminished the amplitude of the IV–V even if it may have not increased the I–V interpeak interval. This is a useful criterion for abnormality, especially when the IV–V peaks are so low that they are difficult to distinguish from background noise. Then the record may be interpreted as abnormal because of such low amplitude central peaks, even if the latencies cannot be precisely defined. For full-term infants, the lower limit for V/I is 30%.

When the V/I amplitude ratio is greater than 300%, wave I is usually considered to be too small. This raises the suspicion of some peripheral hearing impairment, especially of a high frequency or a sensorineural hearing loss.

Fifth, presence of waves I through V: The waves
I–V are all seen in most normal individuals. Occasional normal subjects have a wave IV that is so merged into a IV–V complex that it cannot be clearly distinguished as a separate peak, unless extra traces are run at different stimulus rates, polarities and intensities. Such a merging of wave IV into a IV–V complex is considered to be a normal variant. Wave II may be absent in some normal subjects and many newborns. Thus absence of waves II and IV is not considered abnormal.

When all of the waves I–V are absent the BAEP is abnormal, a technical problem must first be ruled out. The absence of all BAEP waves occurs in profound hearing loss, disordered auditory nerve function within the cochlea, and brain death. These conditions should be clinically distinguishable. The presence of just wave I localizes the disorder to the brain-stem and has been seen in tumors, brain death, and demyelination for instance. The presence of just waves I and III is compatible with pathology at the upper pons.

Waves VI, VII, and VIII can normally be present or absent or asymmetrical in latency or amplitude, without any known clinical correlation.

Three additional measures of the BAEP include the following.

First, absolute latency measurements: The absolute latency measurement of waves I, III and V can be of clinical value. This is particularly so when some peaks are absent. For example, the absolute latency of wave V can be compared against normal limits when there are no waves I–IV. The absolute latency of wave V is normally less than 6.4 ms. The right–left asymmetry of the wave V absolute latency is normally 0.5 ms or less. Absence of waves I–IV with delayed wave V may be due to a hearing loss.

The absolute latency of wave I can be used as part of the assessment of hearing. Wave I is often seen around 1.75 ms, but may be seen up to 2.2 ms in some neurologically normal subjects. The right–left asymmetry of wave I absolute latencies is normally 0.3 ms or less. Wave I latency delays or asymmetry suggest a hearing impairment, rather than brainstem dysfunction.

Second, BAEP threshold: When screening for hearing impairment, latency-intensity curves and threshold evaluations can be useful. Stimulus intensity is gradually reduced to determine the threshold of peaks. The waves gradually decrease in amplitude and increase in latency as intensity decreases. Eventually only wave V persists, and usually the V′ trough is the very last deflection to disappear. The threshold is considered to be the intensity at which the last peak or trough is barely seen. It is important in threshold evaluations that the background noise be kept to a minimum, usually by having the patient asleep at the time of testing. An objective threshold determination may be based on statistical response criteria and quality controls such as the $F_{sp}$ or (+) method.

Third, latency-intensity slope: A latency-intensity curve can be established by graphing the V latency across several stimulus intensities. The slope of the change in wave V latency can be calculated in microseconds per decibel (µs/dB). The slope of the wave V latencies can be compared graphically to a standard set of normal points. Alternatively, the overall slope can be compared numerically to standard normal limits for the slope. For the latter calculation, a slope is usually considered normal if less than 50–55 µs/dB between stimulus intensities of 30 dB and 70 dB nHL.

Conductive hearing impairment usually manifests in prolonged peak latencies, beginning with I, an elevated threshold but a relatively normal slope. Sensorineural hearing impairment usually shows an elevated threshold and a steep slope and a low amplitude or absent wave I. Sometimes a sensorineural impairment will have relatively normal amplitudes and latencies of waves III–V at high and intermediate stimulus intensities, but an abrupt loss or delay of all waves below a critical intermediate stimulus intensity value.

**Patient related factors**

ECOg and BAEP are relatively insensitive to many patient-related factors that affect middle- and long-latency evoked potentials. Medications have very little effect on these potentials, and subjects, particularly children are often sedated in order to improve the quality of the tracings. Anes-
### TABLE 1

**TYPICAL ADULT NORMATIVE UPPER LIMITS FOR BAEP MEASURES TO 10/s, 70 dB NHL CLICKS**

<table>
<thead>
<tr>
<th></th>
<th>Mean</th>
<th>Upper limit</th>
<th>Interaural difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Absolute latencies</td>
<td>I</td>
<td>1.75</td>
<td>2.2</td>
</tr>
<tr>
<td></td>
<td>II</td>
<td>2.8</td>
<td>3.3</td>
</tr>
<tr>
<td></td>
<td>III</td>
<td>3.9</td>
<td>4.5</td>
</tr>
<tr>
<td></td>
<td>IV</td>
<td>5.1</td>
<td>5.9</td>
</tr>
<tr>
<td></td>
<td>V</td>
<td>5.7</td>
<td>6.4</td>
</tr>
<tr>
<td>Interpeak latency difference</td>
<td>V–I</td>
<td>4.0</td>
<td>4.5</td>
</tr>
<tr>
<td></td>
<td>III–I</td>
<td>2.1</td>
<td>2.5</td>
</tr>
<tr>
<td></td>
<td>V–III</td>
<td>1.9</td>
<td>2.4</td>
</tr>
<tr>
<td>Amplitude ratio</td>
<td>V/I</td>
<td>1.3</td>
<td>Lower limit 0.5</td>
</tr>
</tbody>
</table>

Values are representative typical values and should not be used as norms for any particular setting. The norms for each laboratory should be calculated based on records from subjects recorded on the same system in the same setting. Neonates and children may have different normative values. Latency measures are in milliseconds, interaural differences are in the same units as their monaural counterparts in the same line, amplitude ratios are in decimal fractions.

Therapeutic agents and high doses of barbiturate drugs can produce slight increases in the wave V latency. Decreases in temperature can also produce small increases in the latencies, including the I–V interpeak interval. Sleep itself does not alter the potentials.

Age does have a distinct effect on the expected latencies. Under 1 years of age, BAEP normal values are age dependent. For premature infants and neonates, the expected BAEP latency values change almost weekly. Gender also influences the expected BAEP latencies for adult testing, with female I–V interpeak intervals approximately 0.1 ms shorter than for males. Older adults also have slightly longer I–V interpeak intervals, averaging 0.10–0.15 ms longer than for young adults.

Hearing impairment can alter the BAEP, and consequently the test is also used for assessing hearing function. Prior to beginning testing, the examiner should check the external ear canal with an otoscope to assure that the canal is not blocked by cerumen. For all patients, the interpreter should have available some information about the patient’s hearing. Such information may be the results of routine audiometric pure-tone threshold testing. If that is not available, the examiner should test the patient’s subjective hearing at least briefly before beginning the BAEP recordings. A minimum amount of testing would be the subjective thresholds for clicks and for 1000 and 4000 Hz tones, for each ear separately. A BAEP latency–intensity assessment is appropriate when the subjective testing cannot be done, e.g., for children or for comatose or uncooperative patients. This hearing assessment information should be taken into account when interpreting alterations or abnormalities in the high-intensity BAEP results.

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### References


