Neurophysiologic Intraoperative Monitoring of the Vestibulocochlear Nerve

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Summary: Neurosurgical procedures involving the skull base and structures within can pose a significant risk of damage to the brain stem and cranial nerves. This can have life-threatening consequences and/or result in devastating neurologic deficits. Over the past decade, intraoperative neurophysiology has significantly evolved and currently offers a great tool for live monitoring of the integrity of nervous structures. Thus, dysfunction can be identified early and prompt modification of the surgical management or operating conditions, leads to avoidance of permanent structural damage.

Along these lines, the vestibulocochlear nerve (CN VIII) and, to a greater extent, the auditory pathways as they pass through the brain stem are especially at risk during cerebelopontine angle (CPA), posterior/middle fossa, or brain stem surgery. CN VIII can be damaged by several mechanisms, from vascular compromise to mechanical injury by stretch, compression, dissection, and heat injury. Additionally, cochlea itself can be significantly damaged during temporal bone drilling, by noise, mechanical destruction, or infarction, and because of rupture, occlusion, or vasospasm of the internal auditory artery. CN VIII monitoring can be successfully achieved by live recording of the function of one of its parts, the cochlear or auditory nerve (AN), using the brain stem auditory evoked potentials (BAEPs), electrocochleography (ECochG), and compound nerve action potentials (CNAPs) of the cochlear nerve.

This is a review of these techniques, their principle, applications, methodology, interpretation of the evoked responses, and their change from baseline, within the context of surgical and anesthesia environments, and finally the appropriate management of these changes.

Key Words: Vestibulocochlear nerve, Brainstem, Brainstem auditory evoked potentials, Intraoperative monitoring.

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ANATOMY of CN VIII

Reliable neurophysiologic monitoring of the acoustic nerve (AN) requires a good understanding of its anatomy. A succinct review of the latter can be found below.

The auditory receptors are the hair cells, lying within the organ of Corti, at the level of the cochlea in the internal ear. They synapse with the dendrites of the first neuron of the auditory pathway, which is located in the spiral ganglion and whose axons form the distal part of the cochlear or AN. Its proximal part enters the brain stem in the cerebellopontine angle. The AN ends at the level of the lower pons, in the ventral and dorsal cochlear nuclei, situated on the lateral surface of the inferior cerebellar peduncle. From here, sensory input from each VIII nerve ascends via secondary auditory pathways, most of them crossing contralaterally at several levels within the pons. The next nuclear relay is at the junction between the lower and mid pons, in the superior olivary nucleus. The lateral lemniscus is formed by contralateral > ipsilateral (to the stimulated CN VIII) fibers ascending from this nucleus through the mid and upper portions of the pons, to end at the lower midbrain level, in the inferior colliculus. From here, the auditory tracts continue to the medial geniculate body in the thalamus where they synapse and further project via auditory radiations to the transverse temporal gyrus (see Fig. 1).

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TECHNIQUES

Brain Stem Auditory Evoked Potentials

Principle

Brain stem auditory evoked potentials are short-latency evoked potentials that reflect depolarization of several structures within the auditory pathways because they are traversed by electrical volleys from the cochlea, where they are generated by sound stimulation of the auditory receptors, to the upper pons. Brain stem auditory evoked potentials can be used for detecting neurophysiologic dysfunction of both CN VIII and part of brain stem, from upper medulla to the lower midbrain. As recording is done at distance from their actual generators, through electrodes placed on the scalp, these evoked responses are considered far field potentials.

Neurophysiologic–Neuroanatomic Correlation

Because of the complexity of relays, connections, and pathways in the auditory system, each BAEP waveform is most likely the result of not one but several generators. Additionally, the relatively small size of the brain stem, with only few centimeters between the CPA and the inferior midbrain, makes it quite difficult to pinpoint the exact location of the generation of these far field potentials, recorded at significant distance from their generators.

However, based on the clinicopathologic correlations and associations between different types of recordings (e.g., EcochG and BAEPs), at least 1 main generator per waveform has been consistently identified.

The action potentials are generated within the cochlea by transducing into electrical impulses the mechanical acoustic stimulation of the hair cells.

1. "Wave I" results when the generated electrical volley passes through the distal part of the cochlear part of CN VIII. Thus, damage of the latter, cochlea or simply inappropriate stimulation of the auditory receptors, can result in changes to disappearance of this potential, followed by latency and amplitude changes or disappearance of all subsequent waveforms (Assad et al., 2010; Legatt, 2002).
2. "Wave II" generator has been considered to be the cochlear nucleus. Nevertheless, a potential with similar latency has also been described as arising directly from the proximal

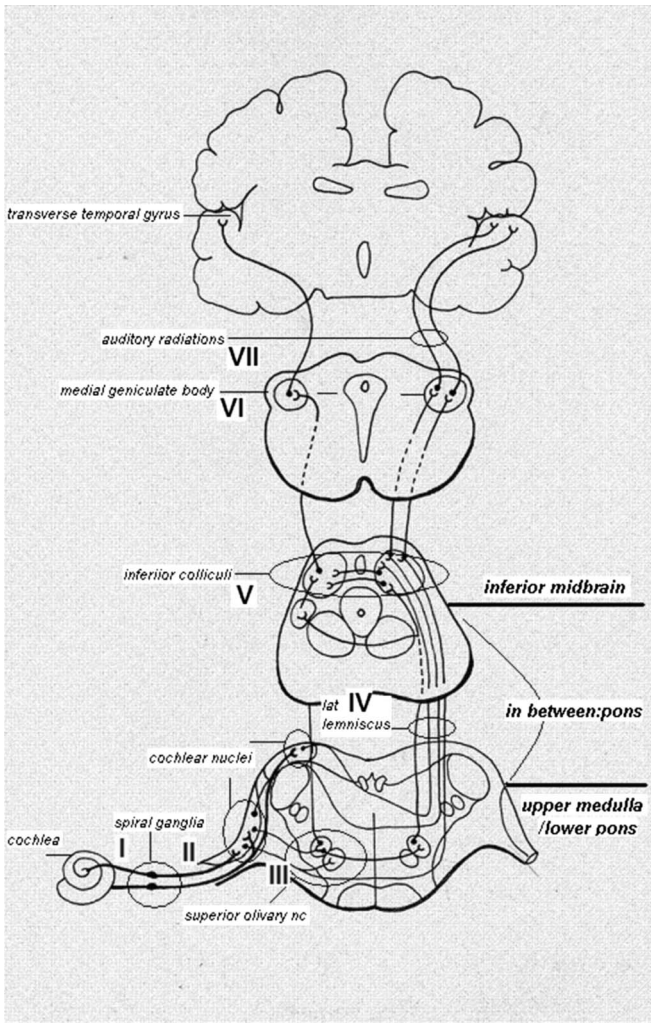

FIG. 1. Auditory pathways and the generators of different BAEP waveforms: distal portion of AN for wave I; proximal portion of AN/cochlear nuclei for wave II; superior olivary complex (junction between lower and mid pons) for wave III; lateral lemniscus (midpons) for wave IV; lateral lemniscus (upper pons) and/or inferior colliculus (inferior midbrain) for wave V; medial geniculate body (thalamus) for wave VI; auditory radiations for wave VII. INTRAOPERATIVE CLINICAL NEUROPHYSIOLOGY by Mirela Simon. Copyright 2010 by DEMOS MEDICAL PUBLISHING LLC. Reproduced with permission of DEMOS MEDICAL PUBLISHING LLC in the format Other book via Copyright Clearance Center.

portion of the nerve, close to the brain stem(Hashimoto et al., 1981; Møller and Jannetta, 1982*a*, 1982*b*; Møller et al., 1994*a*, 1994*b*). Ananthanarayan and Durrant (1991) offered a nice combination between the 2 theories of origin, as they described 2 components of the wave II: IIa, recorded from the proximal portion of the acoustic nerve (AN) and IIb, representing the presynaptic activity of the AN terminating in the cochlear nucleus. Thus, damage to any of the above-mentioned structures (e.g., stretching of the proximal portion of the VIII nerve caused by cerebellar retraction during CPA surgeries) can cause changes in wave II and in those arising proximal to it (i.e., waves III–V, from the brain stem generators). These potentials may be delayed, distorted, or lost. Ultimately, with stretching of the AN, wave I can be affected as well. There have been cases, when the smaller amplitude wave II alone waxed and waned, without further disruption of BAEPs. In most cases, such isolated changes of wave II do not have a clinical significance.

3. "Wave III" is considered to arise in the lower pons, at the level of the superior olivary complex (Møller and Jannetta, 1982*b*). Stretching of the proximal part of CN VIII in the CPA angle (e.g., retraction of the cerebellum) can result in changes to disappearance of wave III, accompanied by disappearance of wave II. Changes of wave III may also occur from a process affecting the lower part of the pons. As with the other potentials, the transmission above this level will be affected, with changes or loss of waves IV and V, with an increase in the absolute latency of wave V and an increase in the I to V interpeak latency and perhaps of III to V interpeak latency, if wave III is still recordable. However, we have seen cases when wave III fluctuated during the case, without associated changes of the other waveform. Such isolated fluctuations may not have a direct clinical significance.
4. "Waves IV and V" are thought to arise in the mid and upper pons/lower midbrain, respectively. This localization is partly the result of observations by several authors (Boller and Jacobson, 1980; Brown et al., 1981; Chiappa, 1982; Epstein et al., 1980; Lev and Sohmer, 1972; Starr and Achor, 1975, 1979; Starr and Hamilton, 1976) that damage to these structures will result in significant changes of IV and V waveforms, to their disappearance. According to some authors (Møller et al., 1994*a*, 1994*b*), the tip of wave V probably generates where the lateral lemniscus terminates in the inferior colliculus. It is generally accepted that wave V is generated in the high pons or low midbrain, at the level of either lateral lemniscus or inferior colliculus. Subsequently, wave IV generation must be in the mid pons, in the lateral lemniscus. The wave V complex will be affected by dysfunction of the auditory pathways distal to or at the level of mid and upper pons or lower midbrain. The associated changes include distorted morphology and decrease in the amplitude of wave V, as well as an increase in its absolute and I to V interpeak latency. In fact, an increase in wave V absolute latency is usually the most common and the earliest sign of dysfunction seen with stretching that occurs during cerebellar retraction in the suboccipital approach of CPA surgery. Most often, wave IV is identified as a small negative peak on the upslope of wave V (giving a notched appearance to the wave V complex) rather than a separate distinct entity. Its configuration is often variable and occasionally it cannot be separated from wave V (a rarefaction polarity stimulus may help in that sense). Thus, wave IV on its own is not considered reliable for monitoring.
5. "Waves VI and VII" are considered to arise at the level of the medial geniculate body and auditory radiations, respectively. However, they are highly variable in morphology and amplitudes and thus not reliable for the use in clinical practice.

In summary, the most useful information in CN VIII neuromonitoring with BAEPs is delivered by waves I and V and to a lesser extent waves II and III because the two latter could vary somewhat during the monitoring and without direct clinical significance. Waves IV, VI, and VII are highly variable and thus have no role in intraoperative neurophysiology.

Methodology

Fig. 2 shows normal BAEP recordings in an anesthetized patient as well as recording and stimulation setup.

Stimulation: technique and troubleshooting

Stimulation involves successful delivery of the acoustic stimuli to the cochlear apparatus. This can be achieved using disposable foam ear tips, available in several sizes. Using the appropriate size is important to ensure a good contact with the auditory canal. These ear foams are connected to an electrical–mechanical transducer by two plastic tubes, one for each ear.

The evoked potentials (EP) machine delivers square electrical pulses to the transducers. These transform the electrical energy into acoustic clicks, which further travel through the connected tubes to reach the auditory receptors within the inner ear. The intensity of the clicks can be easily varied and is measured in peak equivalent sound

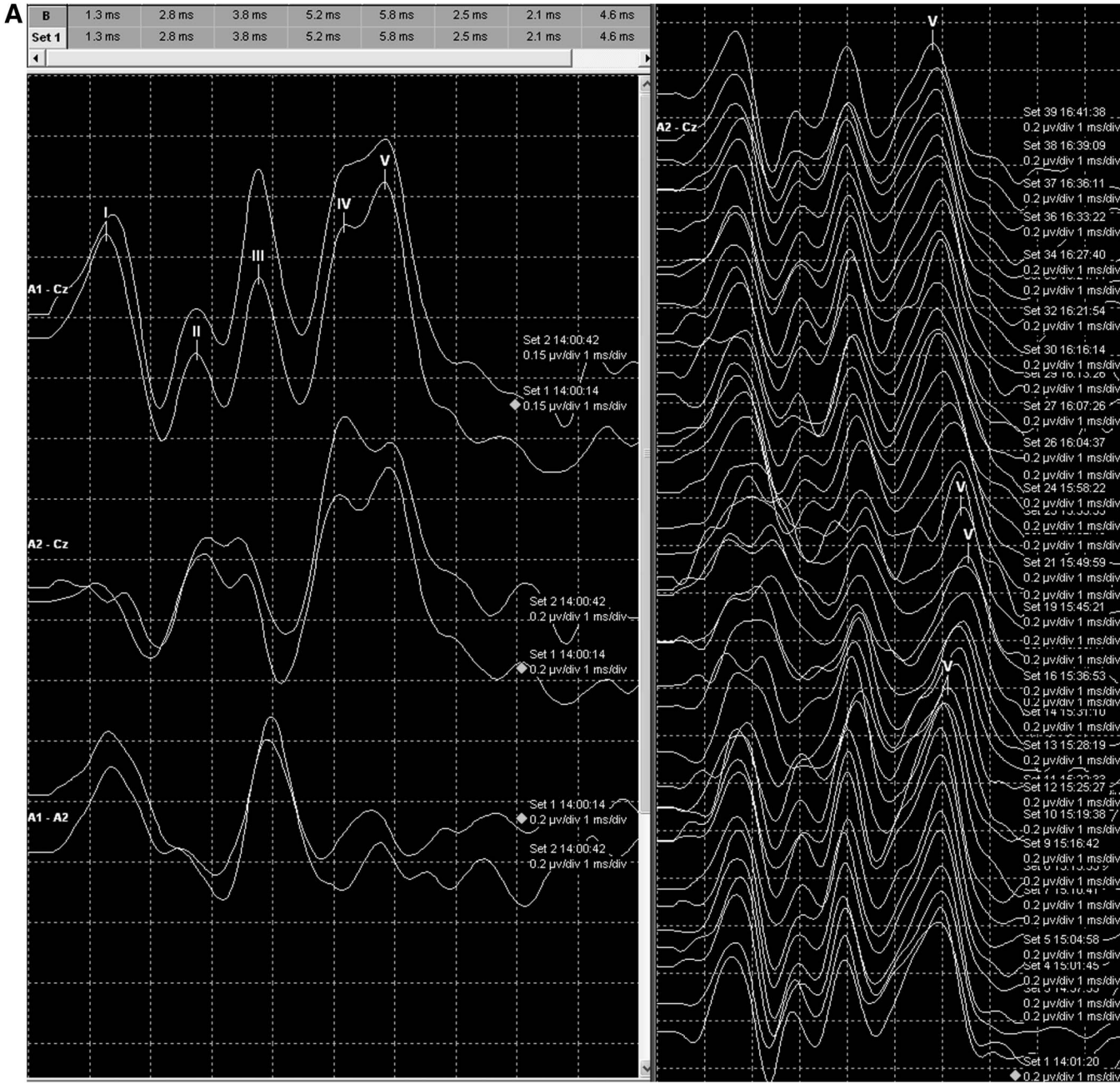

FIG. 2. Normal BAEP recording under total intravenous anesthesia. **A**, Left and right panels showing BAEPs. Notice how recording in different channels emphasizes different waveforms: waves I and III in the Ai-Ac channel; wave IV in the Ac-Cz channel. Ac, contralateral (to the stimulation) ear; Ai, ipsilateral ear. **B**, Software setup for BAEPs: recording electrodes, channels, traces, groups, and stimulators.

FIG. 2. Continued.

pressure level, the standard measure of the loudness of the sound. Stimulus intensity used in the operating room recordings is usually within the 100 to 110 dB range of peak equivalent sound pressure level. Because the sound delivered inevitably stimulates both the ipsilateral ear and the contralateral ear, the latter through bone conduction, it is important to "block" the stimulation of the latter. Otherwise, BAEP responses obtained by bilateral stimulation, even though of enhanced amplitude, will be too nonspecific to allow reliable assessment of one single auditory system, which is ipsilateral to the surgery. Blocking the stimulation of the contralateral auditory system is done by delivering white noise, 40 dB less intense than the stimulus intensity.

To assess the appropriate stimulus intensity to be used and also the expected quality of the BAEPs, hearing thresholds should be obtained before induction in both ears (Assad et al., 2010).

Alternating polarity is preferred at many institutions, as it helps annihilating cochlear microphonics and/or stimulus artifact. By alternating the polarity of the stimulus, its resulting artifact is canceled through the averaging process, while wave I is not. Thus, alternating polarity offers a better signal-to-noise ratio for recording of wave I.

As BAEPs are very small amplitude evoked responses, usually about 1000 to 2000 trials need to be averaged. However, this number can be changed, depending on how robust the signal is and/or how much noise has to be overcome.

The frequency of the acoustic stimuli also varies and is usually between 10 and 40 Hz, with a pulse width of 0.12 milliseconds. As expected, the higher the stimulus frequency, the faster the feedback to the surgeon. However, it is known that high-frequency stimulation adversely affects the morphology and amplitudes of the evoked responses. This effect can be particularly problematic in symptomatic patients, in whom one expects small or distorted baseline BAEPs. For example, we first attempt the stimulation above 20 Hz. If the resulted waveforms are abnormal, we decrease the rate stepwise, until the highest acceptable rate is achieved. Of note, to minimize the 60 Hz electrical noise, which is a common problem for the operating room recordings, we use a number that does not evenly divide 60 Hz (i.e., we avoid multiples of 2, 3, 5, and 10).

If no good baseline BAEPs are obtained, the first question to be answered is whether the stimulation is appropriate (see Fig. 3). All the connections, from the stimulation head box to the tubes and transducers, should be appropriately checked. Also, it is important to ensure correct connections between the stimulation software and hardware.

Appropriate function of the transducers should also be checked. This can be relatively easily achieved even after draping by sliding under and listening with the stethoscope whether the acoustic clicks can be heard (Assad et al., 2010). Additionally, inappropriate stimulation can result from obstruction of the sound propagation, at any level from the transducer to the internal ear.

For example, earwax can impede the sound transmission. Inadequate sealing of the external auditory meatus in a watertight manner could result in blood, skin preparation solutions, or irrigation fluids dripping into the ear canal during the surgery and obstructing or modifying the sound transmission. Several authors have offered suggestions on how to avoid these situations: placing cotton or bone wax outside the ear inserted before taping it into place (Legatt, 1991; Little et al., 1983). A similar situation can be encountered during the procedure when the mastoid air cells are opened and their fluid infiltrates into the connecting middle ear.

Along these lines, the length of the tube inserted in the ear is also important. Very flexible tips can kink and block sound

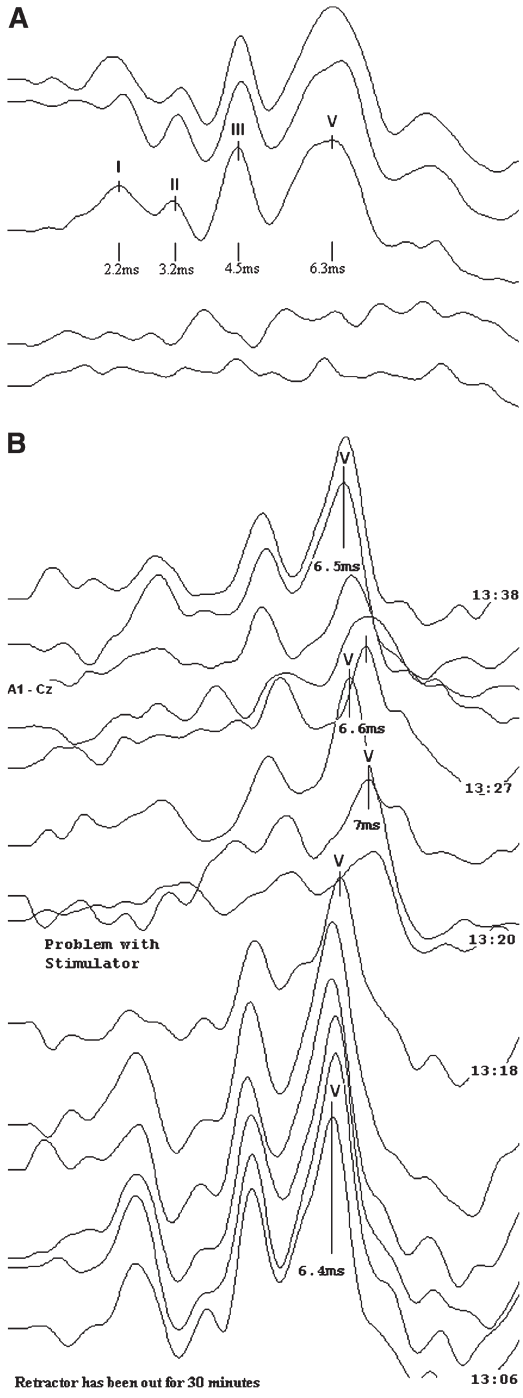

FIG. 3. Two examples of inadequate stimulation. **A**, Failure of the transducer to deliver the stimulation click. **B**, Accidental decrease in stimulus intensity from 110 to 70 dB causes sudden increase in the absolute latency of wave V, with relative preservation of the III to V interpeak latency (13:20). Return of the intensity back to 110 dB is followed by a prompt return to baseline of wave V absolute latency (13:38). INTRAOPERATIVE CLINICAL NEUROPHYSIOLOGY by Mirela Simon. Copyright 2010 by DEMOS MEDICAL PUBLISHING LLC. Reproduced with permission of DEMOS MEDICAL PUBLISHING LLC in the format Other book via Copyright Clearance Center.

transmission. To prevent this, we usually cut these tips shorter. As part of the setup, the air tubes are further taped to the face, to secure their position. However, if the taping is too tight, this can actually deflate the tube and obstruct the sound propagation. We recently had a case, when the BAEPs significantly changed after the setup and before the incision. Once the tape was released at the end of the case, a full prompt recovery of the BAEPs was seen (see Fig. 4).

To assess the appropriateness of stimulus delivery, some authors (Levine et al., 1994) recommended placing a second plastic tube in the ear canal with a microphone at the other end, thus being able to monitor the appropriateness of the stimulus intensity delivered to the internal ear. For example, seepage of fluid in the middle ear will lead to an increase in the pressure of the sound wave because of an increased impedance of the middle ear, and it will be picked up by the microphone. However, a failure to stimulus delivery will result in a decrease in the recorded sound pressure.

We advise that at the end of each case where BAEP changes of uncertain cause occurred, after the drapes are coming out, to check all those elements that could not be checked after the surgery started, such as positioning of the ear pieces, the presence of blood in the auditory canal, kinking/compression of the air tubes, and the like (see Fig. 4).

Recording: technique, recording channels, recorded waveforms, and troubleshooting

As previously mentioned, the evoked responses are recorded in scalp channels. We use as recording electrodes subdermal sterile needles (e.g., 23 mm/27 G, XLTEK). The electrodes are placed at Cz and in the ear lobes, or mastoids (A1 and A2), and at Cz. Alternatively, surface disc electrodes can also be used (e.g., 10-mm gold-plated reusable electrodes, XLTEK). The recording channels are Ac-Cz, Ai-Cz, and Ai-Ac (where Ac and Ai denote the contralateral and the ipsilateral ear positions, respectively).

The first recorded evoked response or wave I is the action potential arising in the distal part of the cochlear nerve and causing the ipsilateral ear/mastoid region to become more electronegative than the contralateral ear or other locations on the scalp; thus, it is seen in Ai-Cz and Ai-Ac channels as an upward, negative peak with an absolute latency of 1.5 to 2 milliseconds. Ai-Ac channel will allow recording a higher amplitude wave I as would rarefaction polarity clicks (Emerson et al., 1982); additionally, the latter were found to also decrease its latency (Maurer, 1985). The following evoked responses, waves II through V, are generated as the electrical volley passes through more proximal levels of the auditory pathways. Their absolute latencies correspond to the time lapsed between the generation of the electrical volley in the cochlea and it reaching the anatomical location of the generator for each of these waveforms. At each of these latencies, all the scalp positions and especially the vertex will be more electropositive than the ipsilateral and contralateral ears. Thus, waves II through V will also be recorded in Ai-Cz, Ac-Cz, and less optimally in Ai-Ac as upward negative peaks. The most prominent negative peak reached after 5.5 milliseconds (with a range of approximately 5.3 and 6.3 milliseconds in the awake patient) is identified as wave V (Chiappa, 1997). The latter (i.e., wave V) is thus best seen in channels Ai-Cz and Ac-Cz, whereas it can be sometimes absent in Ai-Ac. After the negative peak, there is an aftercoming positive, downward deflection, at about 7 milliseconds (Hashimoto et al., 1981).

An increase in wave V amplitude can be obtained by trying condensation polarity clicks and/or by increasing the stimulus intensity. As previously mentioned, wave IV is not always identified. It usually occurs as a small negative peak on the upslope of wave V;

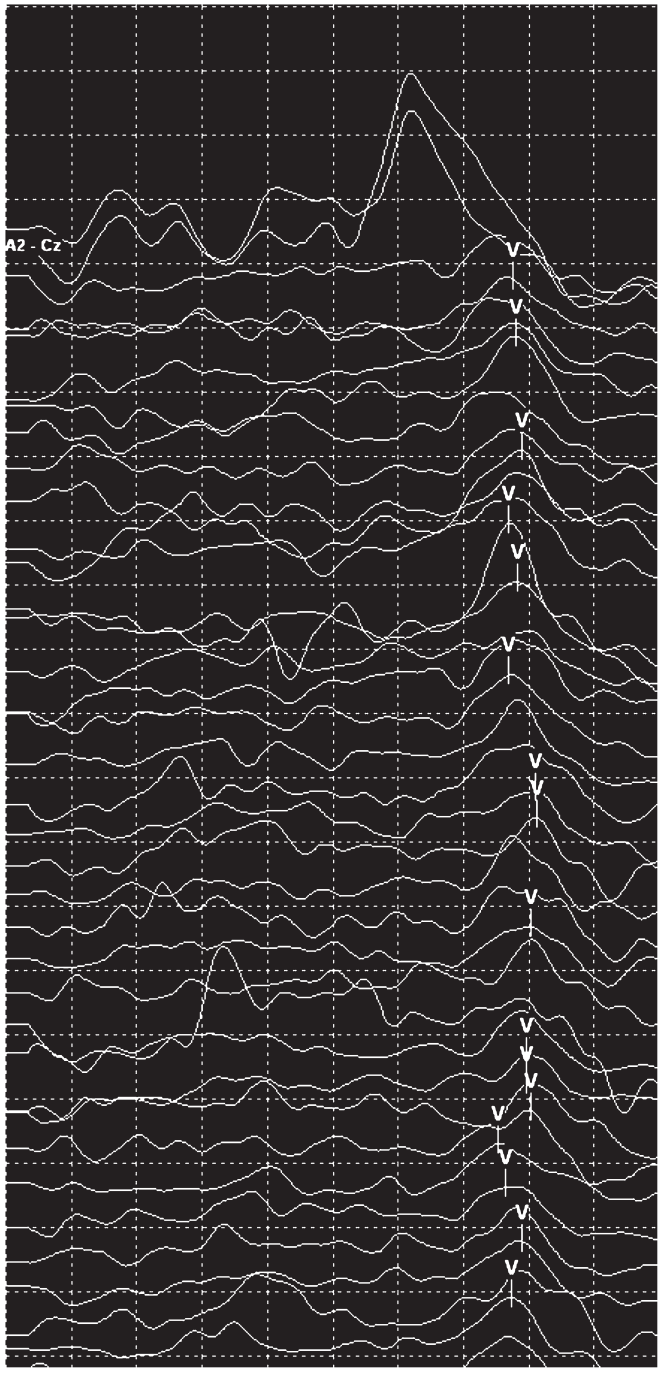

FIG. 4. Right BAEP recordings showing a small wave V with increased absolute latency. At the end of the case, the release of the tape that was securing the position of the air tubes led to sudden improvement of the BAEP amplitudes and latencies (top two trials).

Ac-Cz derivation may help with separation of waves IV and V and does using rarefaction polarity click (Emerson et al., 1982; Maurer et al., 1980; Maurer, 1985). At approximately half distance between waves I and V, wave III is recorded as a negative peak in all derivations.

While all the above suggestions can lead to overcoming a difficult recording, the most informative basis of troubleshooting remains the characteristics of these evoked responses. They are short latency and small amplitude potentials; thus, the recording parameters should include a short time base and high sensitivity. In direct relationship to their small size, obtaining a reliable signal-to-noise ratio is many times challenging, particularly in symptomatic patients. However, even in such cases, it may still be successfully achieved by decreasing the overall environmental noise. For example, recording through the scalp electrodes can be contaminated by a significant amount of muscle artifact. Thus, inducing and maintaining a neuromuscular block can be very helpful in such circumstances. However, this may become problematic later on, when EMG activity is used to monitor and map other motor cranial nerves (e.g., CN VII).

Electrical noise, such as 60 Hz, is a common nuisance for the intraoperative neurophysiologic recordings. Besides choosing an optimal stimulus frequency (see Stimulation: Technique and Troubleshooting), using the 60 Hz notch may also help. Finally, electrical devices in the operating room may be the actual source of electrical noise, such as light sources, venodyne boots, the bed, and the microscope itself. We have had several instances when there was an obvious association between the movement of the microscope in the field and an increase in the electrical noise. Thus, if during an essential part of the surgery, the recorded waveforms are lost in the presence of significant noise, it is worth probably unplugging nonessential electrical devices, temporarily moving the microscope away from the surgical table, etc.

The period of opening and incision is notorious for a significant amount of electrical noise, produced by the bovie. We found it useful to pause the averaging during bovie, thus minimizing the amount of noise introduced in our recordings.

Also, a significant amount of acoustic noise is encountered during drilling in the temporal bone, resulting in waxing and waning of the recorded BAEPs and their disappearance, especially of wave I. Because real damage to the cochlea and distal part of the CN VIII can occur during this time, it is important to ask the surgeon to intermittently stop drilling, so that one can average reliable responses.

Use of cavitron ultrasonic aspirator (CUSA) aspirator can also introduce a significant amount of noise in the recording (see Fig. 5). If that is the case and because this surgical step does have a high potential for real injury, as tumor is removed by aspiration, it is worthwhile asking the surgeon to allow time for averaging.

Several troubleshooting ideas for reducing the amount of noise in BAEPs have been described in the literature (Aravabhumi et al., 1987), such as braiding the wires of the recording electrodes, changing grounds or applying extragrounds, and switching off the BAEP recording machine during the use of bipolar cautery device. Averaging should be paused during ultrasonic surgical aspirator use, and high or mismatched recording electrode impedances should be addressed (Legatt, 1991, 1995).

As the recorded BAEPs have a short latency, besides using a small time base, it is also important to minimize the stimulus artifact, the latter being synchronous with the activation of the transducer. Thus, the longer the distance between the transducer and earlobe or mastoid recording, the smaller the amplitude of the stimulus artifact and the better the separation between wave I and stimulus artifact, as the absolute latency of the former increases (Legatt, 2002). Additionally, as already mentioned in Stimulation: Technique and Troubleshooting, alternating polarity clicks also help in canceling this artifact.

As an alternative to recording far-field BAEPs via surface scalp electrodes, Møller et al. (1994*a*, 1994*b*) proposed recording

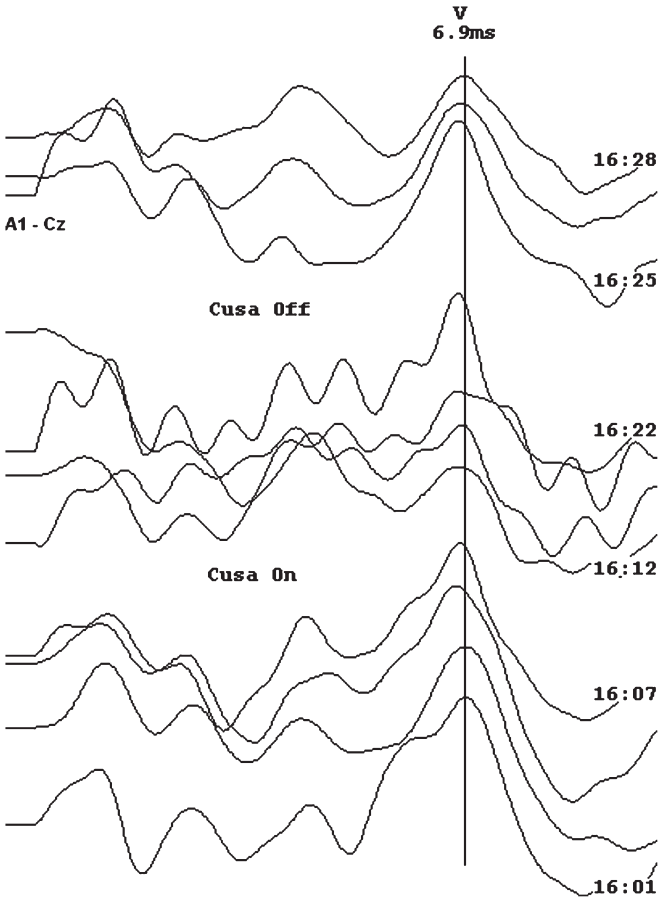

FIG. 5. Left BAEP monitoring during CPA tumor resection. The use of CUSA (sets from 16:12 to 16:22) introduces significant noise in the recordings, which adversely affects the BAEP recordings. Pausing the CUSA allows averaging of reliable evoked responses (sets from 16:25 to 16:28). INTRAOPERATIVE CLINICAL NEUROPHYSIOLOGY by Mirela Simon. Copyright 2010 by DEMOS MEDICAL PUBLISHING LLC. Reproduced with permission of DEMOS MEDICAL PUBLISHING LLC in the format Other book via Copyright Clearance Center.

evoked responses directly from the cochlear nucleus (thus the equivalent of wave II) by placing an electrode in the lateral recess of the fourth ventricle. Given the close proximity to the generator, the amplitude of the evoked responses is much higher, thus less time is spent in averaging and interpretable responses can be obtained in less than half a minute. Also, unlike the recording electrode for CNAPs, which may be in the surgeon's way (see Compound Nerve Action Potential: Advantages and Disadvantages), recording from the lateral recess of the fourth ventricle does not interfere with the tumor removal while it gives information about the entire intracranial portion of CN VIII, including the part proximal to the tumor.

Neurophysiologic Changes in BAEPs

As with any neurophysiologic tests, interpretation of changes from baseline should start with technical troubleshooting of stimulation and recording techniques, as specified above. Only after technical causes are ruled out, true neurophysiologic causes can be considered.

Related to systemic factors

Neurophysiologic changes of BAEPs can occur unrelated to surgical maneuvers. Hypotensive episodes, especially if prolonged and/or associated with significant blood loss or anemia, and hypothermia are just a few such example.

For example, significant hypothermia can be encountered during posterior fossa surgery. Additionally, mild systemic hypothermia can be the direct result of anesthesia; however, more frequently local hypothermia is encountered and directly related to irrigation of the surgical field or simply during opening of the dura (the latter though may also cause a change in the environment around the AN, also resulting in "benign" BAEP changes).

Hypothermia causes an increase in the absolute latency of wave I and of the interpeak intervals. The latency increases per 1°C decrease in temperature, being more significant at lower temperatures. For example, at 26°C, the absolute latencies are double their values at 37°C, while at temperatures higher than 26°C, there is an approximately 7% increase in latency per 1°C drop in temperature; hysteresis has been described during rewarming, meaning that the change in latencies per 1°C increase does not match the change in latency per 1°C decrease (Markand et al., 1990).

Changes in BAEP amplitudes have also been described, more so of the later components. There is a general consensus that above 25°C and below 30°C, there is an initial increase in amplitudes (Markand et al., 1987; Rodriguez et al., 1995), followed by a decrease in amplitudes to disappearance at lower temperatures. However, there is disagreement as far as what is the cutoff for recording waveforms: 20°C (Markand et al., 1987), whereas other authors described recordable waveforms at this temperature (Rodriguez et al., 1995) and even at lower (14°C) temperatures (Rosenblum et al., 1985).

Additionally, changes in anesthetic regimen should be considered (Janssen et al., 1991), although the latter have been found to have minimal effect on the BAEPs. Under the anesthetics effect, an increase in absolute latencies of the evoked responses could be seen, however, with relative preservation of their interpeak latencies, as well as of their amplitudes and morphologies (Drummond et al., 1987; Dubois et al., 1982; Lloyd–Thomas et al., 1990; Manninen et al., 1985; Newlon et al., 1983; Purdie and Cullen, 1993; Sanders et al., 1979; Sloan, 1998; Thornton et al., 1983).

Some reversal of neurophysiologic changes will occur with correcting the cause (e.g., an increase in blood pressure and blood transfusion, irrigation of the surgical field with warm saline). Even when persistent, just identifying their etiology, will help in detecting and interpreting the significant injurious changes.

Related to injurious surgical maneuvers

Once technical and systemic factors are ruled out, BAEP changes can be considered to be related to the surgical maneuvers. Following are key portions of specific surgeries when such changes are more likely to occur.

Early drilling in the squamous part of the temporal bone and in the internal auditory canal (IAC) has been described (Gouveris and Mann, 2009) as one of the surgical steps most commonly involved in significant changes in both BAEPs and ECochG and one of the most common reasons of sudden loss of all BAEPs in CPA tumor surgery (Nadol et al., 1987).

This step is part of the acoustic schwannoma surgery, during translabyrinthine and middle fossa approach and characterized by a significant amount of ambient noise, reflected in a decrease in the signal-to-noise ratio, as described above. However, the latter can also be affected by a decrease in signal caused by direct injury of the

cochlear apparatus or of the distal portion of the AN, by direct physical and noise-induced damage during drilling and by vascular compromise. The first situation can be encountered when the membranous labyrinth is accidentally entered during drilling, with the loss of all BAEPs and deafness (Legatt, 2002). Second, noise itself can be injurious to the auditory receptors. The third situation is directly related to damage of the labyrinthine artery, also known as auditory or internal auditory artery, a long branch of either the anterior inferior cerebellar artery (in more than 90% of the cases) or basilar (in less than 10 % of the cases). This artery that supplies the inner ear and accompanies CN VIII through the internal acoustic meatus. Thus, it is quite vulnerable to damage during drilling in the IAC or during the nerve dissection and resection of the intracanalicular portion of the tumor. The result will be, in the best scenario, vasospasm and, in the worst scenario compression, coagulation or rupture, thus resulting in several degrees of vascular compromise, from temporary ischemia to infarction of the cochlea and/or distal portion of AN. Vascular compromise should always be considered in case of sudden loss of wave I with subsequent disappearance to all waveforms. Because of its anatomical trajectory, Ojemann (2001) has recommended to try to preserve any vessel that is entering the internal auditory meatus.

Because increased noise can mask real signal loss, whenever BAEP changes are encountered during this step, it is recommended to ask the surgeon to pause the drilling until appropriate averaging is done, in the absence of ambient noise.

Cerebellar retraction (see Fig. 6A) frequently causes neurophysiologic dysfunction of AN nerve, reflected in BAEP changes. Several mechanisms have been proposed as cause such as compression of the small vessels supplying CN VIII, resulting in nerve ischemia, compression of the cochlear nuclei, and mechanical stretching of CN VIII (Nishihara et al., 1986). While the abruptness of resulting BAEP changes will then depend on the cause, the most common finding remains a progressive increase in the latency of wave V (see Fig. 6B). Sato et al. (2009) found a strong linear relationship between the increase in wave V latency and the increase in the waves I to III interpeak latency. Most often, the release of the cerebellar traction will be followed by prompt recovery of these parameters, even in cases of total loss of wave V (see Fig. 6B). However, we have encountered exceptions, when wave V began recovering only to suddenly disappear again raising the question of an irreversible vascular compromise (see below). Several authors have proposed different latency thresholds that should prompt traction release and beyond which irreversible damage occurs (see BAEP Predictors of Postoperative Hearing Outcome). In general, we warn the surgeon when wave V starts moving out, again when the increase in latency reaches 0.5 milliseconds and next when it reaches 1 millisecond. Depending on the preoperative state of CN VIII, the type of surgery, and the surgical requirements, a judgment call is made as of when to release the traction.

Concomitant changes in wave I have also been described. Polo et al. (2004) described a bifid wave I. Sato et al. (2009) referred to the appearance of a wave I, between waves I and IIa (see Fig. 7), as an early sign neurophysiologic dysfunction caused by stretching of AN at its most vulnerable part, near the porus acusticus internus, where the central myelin is replaced by peripheral myelin (i.e., Obersteiner–Redlich zone). Matthies and Samii (1997) found that traction of the cochlear nerve to the medial direction results in amplitude reduction or loss of wave I, while pulling the nerve to the lateral direction results in amplitude reduction or loss of wave III.

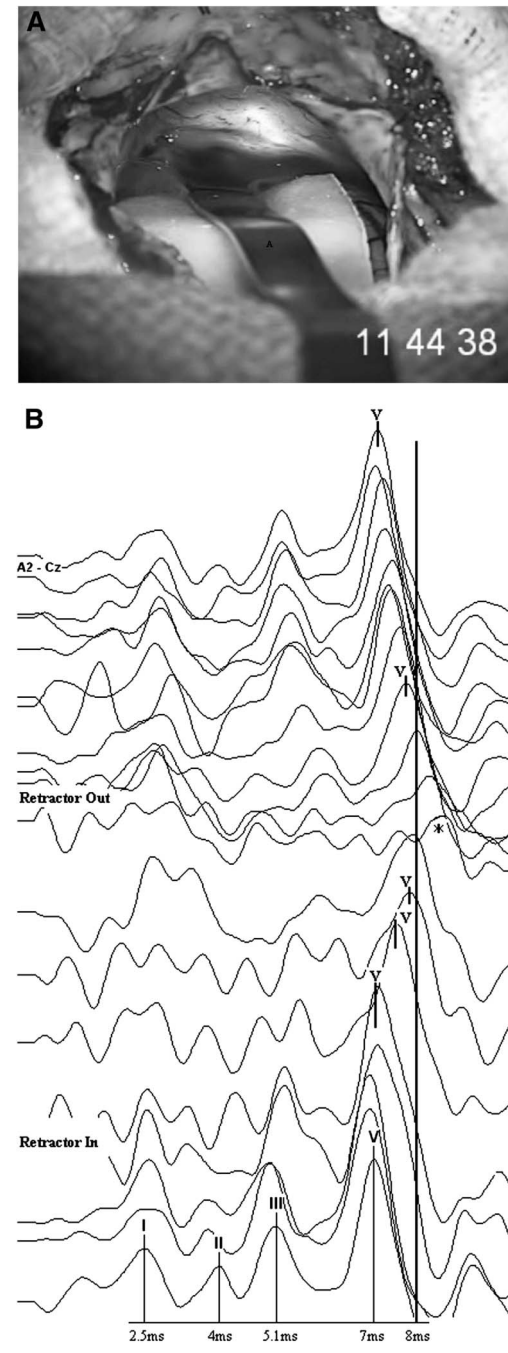

FIG. 6. Right BAEP monitoring during microvascular decompression for trigeminal neuralgia. Retraction of the cerebellum causes impaired conduction in CN VIII. A, Retractor (labeled A) on the cerebellum. B, The cerebellar retraction stretches the AN. This translates in a marked worsening of the amplitude and morphology of wave V and in an increase in its absolute latency of more than 1 millisecond (trial marked *). The immediate release of the cerebellar traction is promptly followed by recovery of all the waveforms. INTRAOPERATIVE CLINICAL NEUROPHYSIOLOGY by Mirela Simon. Copyright 2010 by DEMOS MEDICAL PUBLISHING LLC. Reproduced with permission of DEMOS MEDICAL PUBLISHING LLC in the format Other book via Copyright Clearance Center.

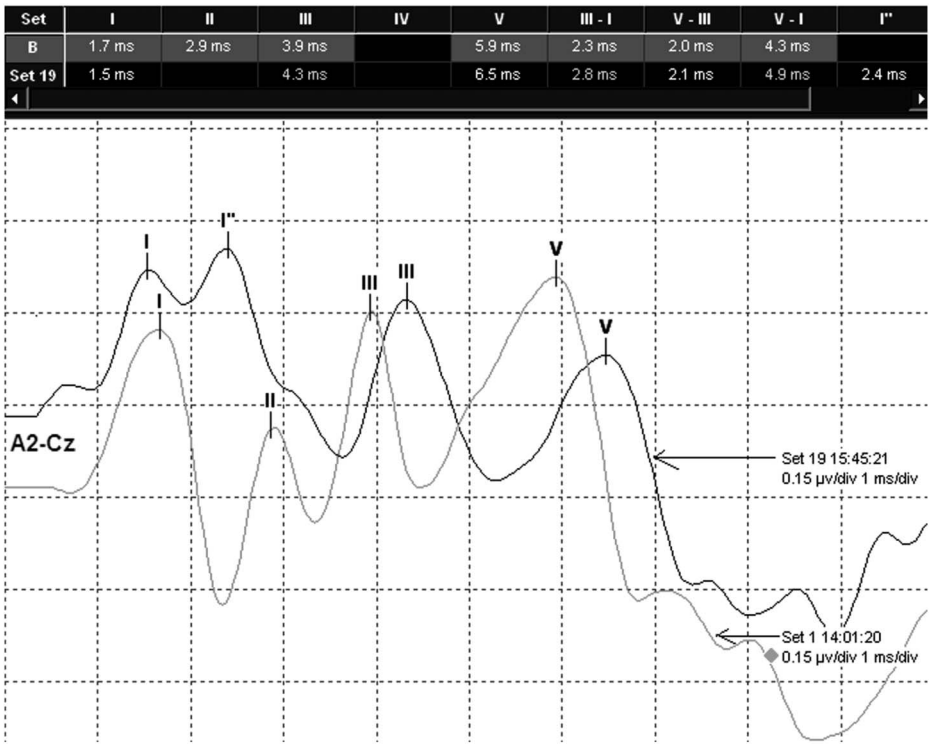

FIG. 7. Cerebellum retraction in CPA surgery is stretching the AN. Besides an increase in wave V absolute latency (of more than 0.6 milliseconds), this causes the appearance of wave I between waves I and II as a sign of impaired conduction.

Tumor dissection and removal is one of the surgical steps most commonly associated with significant BAEPs, EcochG, and CNAPs changes (Gouveris and Mann, 2009). During this step, several mechanisms can be responsible for CN VIII damage: mechanical compression, transection, and stretching as well as vascular and thermal injury of the nerve. The two latter can be caused by the use of cautery, because of either the generated heat or coagulation and vessel thrombosis. Brain stem auditory evoked potential changes can involve both latency and amplitude changes, of all waveforms, from I to II, with subsequent involvement of III and V, as well as I to III and I to V interpeak latencies. When such changes occur, the first action should be arresting the dissection or the injurious maneuver (Legatt, 2002), irrigating with warm saline and increasing the blood pressure**.** Interestingly, it is believed that even the changes seen after tumor removal are likely directly related to the nerve injury that has occurred during the dissection and resection process. During dissection and tumor removal, traction on the nerve can also occur. This may cause its injury at the distal, cochlear end, with direct impact on the fragile nerve endings, which can be avulsed. Their damage will result in loss of wave I and the subsequent waveforms. Thus, nerve traction should be applied, whenever possible toward, rather than away from the cochlea (Sekiya and Møller, 1987). Additionally, the use of aspirators, such as CUSA, can remove not only tumor fragments but also nerve fragments. Given the increased noise caused by these devices, which can also decrease the signal-to-noise ratio, it is important to ask the surgeon to intermittently turn off the aspirator, thus allowing appropriate averaging and appreciation of real changes (see Recording: Technique, Recording Channels, Recorded Waveforms and Troubleshooting and Fig. 5).

Vasospasm of the arteries can arise during different steps of the surgery, and it may involve different vessels. It is important to recognize it as if captured in time and acted upon will result in total reversal of the neurophysiologic changes.

Given the nature of the surgical procedure, with manipulation of vascular structures situated in close proximity to CN VIII, this situation is more so common during microvascular decompressions (see Microvascular Decompression of CN V, CN VII, or CN VIII). During such procedures, we have encountered sudden changes in BAEPs on a few occasions, several minutes after the Teflon piece had been placed between the nerve and the vascular loop. No signs of mechanical damage of the CN VIII could be identified by visual inspection. However, irrigation with papaverine and warm saline irrigation did reverse these changes, indicating as that likely mechanism was arterial vasospasm caused by manipulation of the irritative vascular structures. Unfortunately, in other cases, irreversible sudden loss of wave I and subsequent waveforms was encountered (see Fig. 8).

Occasionally, a pale ischemic CN VIII can be appreciated by visual inspection, with reversal of both neurophysiologic and anatomic changes with papaverine and warm saline (Nadol et al., 1987).

Interestingly, Bischoff et al. (2008) concluded that, in general, reversible intraoperative changes in BAEPs are likely to be caused by disturbed microcirculation of AN, and thus, it is these cases who will benefit from postoperative vasoactive treatment consisting of hydroxyethyl starch and nimodipine. Additionally, they concluded that an abrupt or irreversible progressive loss of BAEPs would be more indicative of a mechanical injury of the nerve fibers as the etiology. However, it is important to specify that regardless of the presence or absence of mechanical injury of the nerve fibers, a catastrophic ischemic injury (e.g., disruption of the labyrinthine artery) will also show abrupt and irreversible loss of the BAEPs.

Dura closure can result in compression of the auditory pathways. This may result in postoperative hearing loss (Møller and Møller, 1989). As a consequence, the BAEP monitoring should be continued during closure.

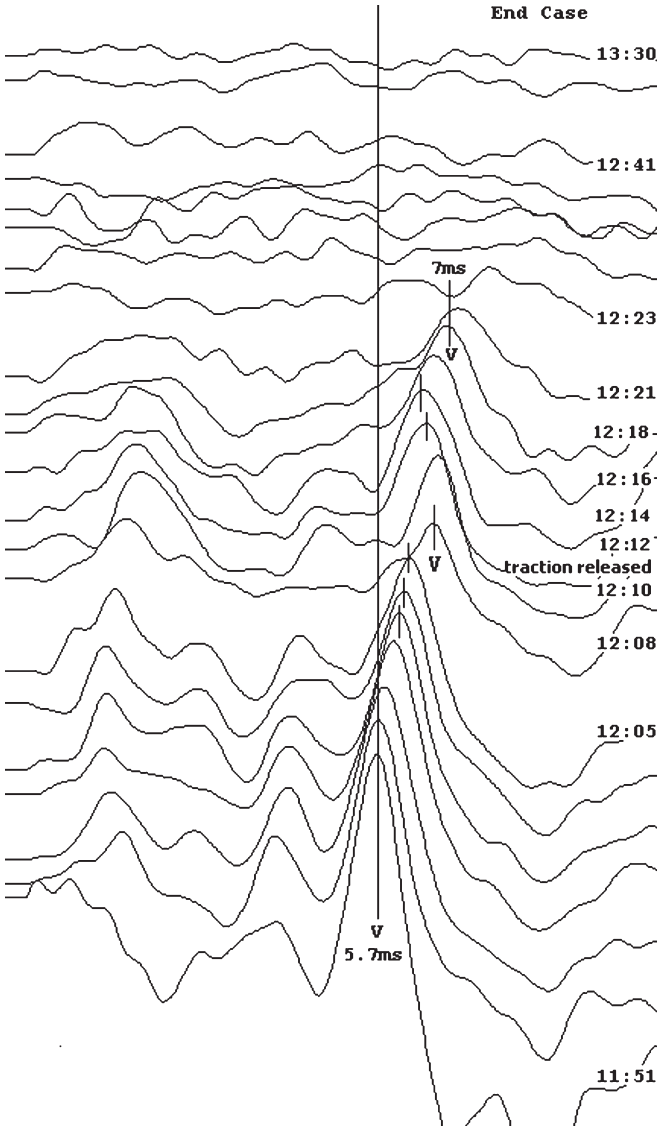

FIG. 8. Microvascular decompression for trigeminal neuralgia. An initial progressive increase in wave V absolute latency caused by cerebellar retraction is seen until 12:10, when the retractor is removed. This is followed by an improvement in wave V latency until 12:16. However, afterward, wave V latency suddenly worsens once again to total disappearance at 12:23. After this time, there is total loss of all waveforms. The Teflon piece that had been placed between CN V and the compressing vessel is removed, and the surgical field is irrigated with papaverine and warm saline. Despite all these measures, no BAEP recovery is seen. The patient will wake up with hearing deficit. The mechanism of injury is likely cochlear or AN infarction. INTRAOPERATIVE CLINICAL NEUROPHYSIOLOGY by Mirela Simon. Copyright 2010 by DEMOS MEDICAL PUBLISHING LLC. Reproduced with permission of DEMOS MEDICAL PUBLISHING LLC in the format Other book via Copyright Clearance Center.

BAEP Predictors of Postoperative Hearing Outcome

James and Husain (2005) showed that different warning levels for BAEP changes should be applied for different types of surgery. For CPA surgeries other than tumor resection, hearing loss occurs only when there is loss of wave V, while in CPA tumor surgery, much smaller changes in wave V latency (less than 1 millisecond) and amplitudes (less than 50% from baseline) can result in hearing loss. Sugiyama et al. (1989) also found that in microvascular decompression, for CN VII and CN VIII decompression, an increase in wave V latency is not a good predictor for postoperative hearing deficit and that only permanent disappearance of wave V predicts postoperative hearing loss. Similarly, Nadol et al. (1992) found that the persistence of wave V at the end of surgery is highly predictive of preserved hearing. More so, Kemink et al. (1990) and Watanabe et al. (1989) found that even in cases of acoustic neuroma resection, a robust predictor of hearing loss remains the total loss of wave V, whereas the persistence of wave V or recovery of this waveform after its total loss was predictive of successful hearing preservation regardless of latency increases. Kemink et al. (1990) have suggested that an optimal postoperative outcome can be achieved by stopping the tumor dissection when the changes in wave V latency are greater than 2 milliseconds. Other authors (Gouveris and Mann, 2009) concluded that a permanent decrease of 75% or more in the BAEP amplitudes are associated with postoperative hearing loss, whereas recovery of such changes carries a better hearing outcome.

Along these lines, Schramm et al. (1988) found that a decrease in the amplitudes of BAEPs of more than 50 % or a total disappearance of 1 or more waves has a significant correlation with a postoperative decrease in hearing. Thus, he suggested that during BAEP monitoring, more attention should be given to amplitude reduction than to increases in latencies and that the surgeon should be informed when decrease in amplitudes of more than 50% occurs.

To decrease the risk of postoperative hearing deficit, some authors proposed that each traction on nervous structures should last less than 5 minutes, with at least 2-minute intervals between 2 tractions, and also that the pressure applied to cerebellum should be less than 20 mm Hg (Nishihara et al., 1986).

Overall, Abramson et al. (1985) found that intraoperative BAEP monitoring is useful in predicting the postoperative hearing outcome, yet the biggest predictor remained the tumor size. Along the same lines, other authors (Harper et al., 1992; Slavit et al., 1991) concluded that BAEP monitoring was associated with higher rate of hearing preservation in the resection of small acoustic neuromas.

Zappulla et al. (1984) also described a new technique for the objective quantitative assessment of the BAEPs based on the variance of the phase angle between small group averages. This component synchrony measure corroborated standard peak latency and amplitude of BAEP changes during surgical manipulation.

Electrocochleography

Principle

Another method used for cochlear nerve monitoring is transtympanic and extratympanic ECochG. The method implies direct recording of the cochlear action potentials (CAP) and can be done via electrodes placed in close proximity to cochlea.

Advantages and Disadvantages

Advantage of ECochG over BAEPs is that it offers higher amplitude potentials as a reflection of the function of cochlea and the very origin of the AN. Thus, it provides a good alternative for

monitoring their function in cases when BAEPs are nonrecordable or unreliable; especially, EcochG offers a great alternative for recording the most peripheral potentials, as equivalents of wave I, which is, in general, even more difficult to record via surface electrodes placed at distance, on the scalp and earlobes (Lambert and Ruth, 1988). Along these lines, EcochG has proven very useful particularly during "noisy" steps of the surgery, such as during electrocautery and drilling (Schlake et al., 2001). Additionally, the better signal-to-noise ratio offers faster responses than BAEPs. Certain types of procedures that may result exclusively in the damage of the inner ear/cochlea and can benefit better from ECochG monitoring than BAEPs, such as in middle ear reconstructive surgeries (Wazen, 1994).

However, "pure" disconnection of the cochlea from the brain stem, observed as a preserved ECochG but absent distal BAEPs, can confound this technique; postoperative deafness is often the result (Schlake et al., 1999). Similar results were found by Symon et al. (1988), showing that there was no consistent correlation between the preservation of CAP recorded on transtympanic EcochG and the preservation of postoperative hearing. Also, EcochG recordings can be significantly affected by the increased noise introduced by drilling (Torrens et al., 1994).

Methodology

Recording: Technique and Troubleshooting

Transtympanic electrocorticography requires insertion of an electrode through the tympanic membrane into the promontory bone. The recorded waveform is a near-field CAP that is the equivalent of wave I of the BAEP. This potential will be directly affected when cochlea itself is damaged. However, indirect damage of these most distal structures of the auditory pathways could still occur during manipulations of the more proximal parts of the AN because of stretching of the fragile nerve terminals in the organ of Corti or ischemia of the labyrinthine structures. Additional to CAP, EcochG also directly records cochlear microphonics.

Extratympanic ECochG has also been described in the literature (Mullatti et al., 1999; Winzenburg et al., 1993). It does not require a myringotomy and may provide similar results. As expected, the latencies of the CAPs obtained via the two approaches are identical, whereas there may be large amplitude differences (Winzenburg et al., 1993). For this type of recording, Lambert and Ruth (1988) proposed a small, reticulated foam plug offering a large surface recording area, with lower impedance and improved signal-to-noise ratio and more stability during manipulations in the surgical field.

Stimulation: Technique and Troubleshooting

Same as for BAEPs: tone bursts condensation, rarefaction, and alternating polarity; the latter will get rid of the cochlear microphonics.

EcochG Predictors of Postoperative Hearing Outcome

There are many reports in the literature showing a good correlation between changes in CAP as recorded by EcochG and postoperative hearing deficits, even in the absence of direct cochlear damage (Attias et al., 2008; Battista et al., 2000; Colletti and Fiorino, 1998; Lambert and Ruth, 1988). Levine et al. (1994) reported that disappearance of these potentials for 15 minutes or more is a good predictor of postoperative hearing loss. Gouveris and Mann (2009) concluded that a permanent decrease of 75% or more in the CAP amplitudes are associated with postoperative hearing loss, while just a temporary decrease likely associates with a more favorable outcome. However, other authors (Sabin et al., 1987) did not find a good correlation between preservation of CAPs and postoperative preservation of hearing. This is to be expected, as damage of the more proximal portions of ANs may not translate in distal nerve dysfunction.

Compound Nerve Action Potential

Principle

Cochlear CNAPs can be recorded directly from the AN through an electrode positioned by the surgeon proximal to the site of surgery and directly on the nerve and thus used for continuous live monitoring of the function of the latter.

Advantages and Disadvantages

As with the CAP recorded by ECochG, these evoked responses have higher amplitudes than the BAEP counterparts and thus are more advantageous for use in noisy environments. For the same reasons, they may be more useful in monitoring symptomatic patients who have poor BEAPs at baseline. Additionally, because of a much better signal-to-noise ratio, they require minimal averaging, assuring a faster feedback to the surgeon than that offered by BAEPs (Yamakami et al., 2009; Yingling and Gardi, 1992). Compound nerve action potentials were also described to be more robust than BAEPs during the dissection procedures (Yamakami et al., 2009). Møller and Jannetta (1983) in fact recommended the use of both BAEPs and CNAPs during microvascular decompression for cranial nerve dysfunction. Taniguchi et al. (1992) also found it useful to concomitantly use both methods for CN VIII monitoring during acoustic neurinoma resection. Compound nerve action potentials can be used for mapping cochlear nerve fibers during CPA surgeries (Colletti and Fiorino, 1993).

The main disadvantage comes from the technique itself (see below) because it requires careful placement and steady maintenance of the recording electrode directly on the nerve. Its position could shift with movements in the field during surgical manipulation, resulting in false-positive changes of the recorded potentials. Additionally, the cochlear nerve is a delicate structure and the application of the electrode itself could be potentially traumatic.

The electrode could be " in the way" of the surgeon (Yingling and Gardi, 1992), and the procedure works best for small tumors, which leaves enough space close to the brain stem for the placement of the recording electrode. Finally, placement of the electrode requires appropriate exposure of the nerve, which may be achieved only after some degree of dissection is performed; this itself could cause nerve injury.

Methodology

Recording: Technique and Troubleshooting

Recording of the CNAPs is done via an electrode that is placed on the nerve itself. Yamakami et al. (2009) presents a newly designed electrode for such recordings in the removal of small acoustic neuroma via retrosigmoid approach. The electrode consists of a tuft of cotton attached to a flexible wire that offers a more steady position on the cisternal portion of the nerve, offering a better contact and more reliable recordings. The electrode is placed between the nerve tumor and its root entry in the brain stem. Others (Jackson and Roberson, 2000) described the recording electrode as placed distal to the tumor, in the extradural portion of the nerve. However, this positioning can give false-negative results because the distally recorded CNAPs will not change if the damage involved the more proximal portions of CN VIII. Thus, to avoid such situations, the CNAPs need to be recorded proximal to the lesion to be resected

(Møller, 1996; Møller et al., 1982). This type of recording is most easily achieved during the removal of small and more distal acoustic neuromas, which may not obstruct the view in the CPA angle and allow the placement of the electrode proximal to the tumor.

The CNAP represents the sum of all the nerve action potentials arising in the nerve fibers constructing the cochlear nerve. More so, the CNAPs recordings were found by many authors to be more robust than BAEPs during the dissection procedures (Yamakami et al., 2009). It is therefore recommended that in surgeries where BAEPs are not reliable, CNAPs recording should be tried. Bipolar recordings of the cochlear nerve CNAPs were found to be very sensitive and selective in correct identification of the AN during acoustic neuroma removal: repeated recordings from the peritumoral structures facilitated the identification of cochlear nerve fibers.

Whenever sudden changes in the recording CNAPs are encountered, one should first rule out displacement of the recording electrode.

Stimulation Technique

Stimulation of the cochlear nerve is done as described for BAEP monitoring.

Neurophysiologic Changes in CNAPs

While systemic factors have little or no effect on CNAPs and technical challenges were discussed previously, the same "injurious surgical maneuvers" that can cause BAEP changes can also result in CNAP disturbances.

1. Drilling in the temporal bone and IAC is characterized by "noisy" recordings and thus, as in the case of ECochG, CNAPs can be more advantageous than BAEP monitoring. However, real changes of CNAPs can occur during this phase and have the same significance as changes in EcochG or wave I disappearance on BAEP recordings (see also Early Drilling in the Squamous Part of the Temporal Bone and in the IAC).
2. Cerebellar retraction may or may not cause CNAP changes, depending on the direction of the traction, with only traction of the nerve medially being responsible for such changes (Matthies and Samii, 1997). Thus, it is only when traction of the AN results in stress of its distal part or cochlea, CNAPs will show changes (see Neurophysiologic–Neuroanatomic Correlation).
3. Tumor dissection and removal can result in significant damage of CN VIII, reflected in CNAP changes.

Separation of CN VIII from CN VII, dissection of tumor from the CN VIII, intracapsular tumor debulking, and tumor resection have been associated with changes in the CNAP amplitudes. It has been recommended to use sharp (rather than blunt) tumor debulking and dissection of the tumor from the nerves, using microscissors. Blind tumor dissection near the fundus of the IAC can be dangerous. To avoid thermal injury, it is also recommended not to use bipolar coagulation for hemostasis in the IAC. As already mentioned, to avoid false-negative results, the recording electrode needs to be positioned proximal to the tumoral process.

The types of CNAP changes have also shown correlation with certain types of injuries. Abrupt loss of CNAPs is most likely associated with a vascular injury. Along these lines, Rowed et al. (1988) reported sudden loss of CNAPs after manipulation of the AN nerve and coagulation of small vessel of the tumor capsule. A gradual loss, stepwise decrease in the amplitude with increase in the latencies, is usually associated with a mechanical injury. Compression of the nerve can result in a desynchronization of the individual nerve action potentials causing loss or decreased amplitude in CNAP. Conversely, decompression of the nerve is sometimes followed by an improvement in the CNAPs amplitudes.

CNAP Predictors of Postoperative Hearing Outcome

Yamakami et al. (2009) showed that CNAPs preservation had a 100% positive predictive value and negative predictive value for serviceable hearing, while Colletti et al. (1998) concluded that the permanent loss of CNAPs predicts postoperative hearing loss with 100% sensitivity and specificity. Rowed et al. (1988) found that the presence of CNAPs at the end of the surgery is positively correlated with postoperative hearing preservation.

Møller and Jannetta (1983) concluded that adding CNAPs to BAEP recordings during microvascular decompression for cranial nerve dysfunction will further decrease the risk of postoperative hearing loss.

During vestibular neurectomy for vertigo release, a change in the CNAP latencies of 0.3 milliseconds or less without changes in the morphology was a good predictor for an excellent postoperative hearing outcome at 1 month (Silverstein et al., 1985).

Nedzelski et al. (1994) found that a click threshold shift of 20 dB or less was usually predictive of preserved postoperative serviceable hearing, while an increase in the threshold by 30 to 60 dB resulted in loss of it.

Distortion Product Otoacoustic Emissions

Principle

These faint low-level sounds are thought to be the result of the movement of hair cells as a function of the difference between two presented frequencies. Morawski et al. (2004) found their monitoring potentially useful, but no direct comparison with other types of monitoring (e.g., the ECoG) was done in humans. In animal studies, they have been found to be very sensitive to ischemia of the inner ear, and some authors consider them a promising adjunct to neuromonitoring of CN VIII (Telischi et al., 1995).

Methodology

They are elicited by cochlear stimulation with a bitonal stimulus complex, and they are recorded and measured with the aid of a minimicrophone in the external auditory canal.

Laser Doppler Measurements

Principle

Animal research done on guinea pig models (Levine et al., 1993) indicates that Laser Doppler monitoring during acoustic neuroma surgery is a sensitive tool for early identification (earlier than with EcochG) of decrease in the cochlear blood flow. In these models, compression of the CN VIII resulted in abrupt drop of the Laser Doppler measurements with their quick recovery once their decompression was released. Interestingly, there was a delay in the recovery of the evoked responses simultaneously recorded in ECochG, lagging behind the recovery of the Doppler signals.

CLINICAL APPLICATIONS

As previously mentioned, BAEP utility in neurophysiologic intraoperative monitoring results from its ability to monitor the functions of the auditory pathways from the cochlear nerve and their corresponding brain stem structures, up to the lower midbrain. Electrocochleography gives a sensitive alternative or complement BAEPs in assessing the cochlear function and that of the distal portion of CN VIII. Compound nerve action potentials are a good monitoring tool for the AN up to the nerve root entry in the brain stem.

CPA Tumor Removal

There are several types of approaches during CN VIII tumor removal, each with their own advantages and risks. Understanding the surgical approach used in each case is essential for early recognition and correct interpretation of neurophysiologic changes seen during monitoring.

Translabyrinthine Approach

This approach is through the mastoid and semicircular canals to the IAC. This approach works best for small intracanalicullar tumors because it gives an excellent view of the tumor in the internal canal and provides direct exposure of the tumor without the need to push aside any brain tissues. It also permits easier identification of the facial nerve and thus minimizes the risk of facial paresis (Sterkers et al., 1994). However, total deafness and loss of vestibular apparatus are major problems with this approach.

Middle Fossa Approach

This approach has a great record for preserving hearing for ANs below 2 cm, which is why it is preferred by surgeons who have extensive training in AN surgery.

Again, this procedure works best for small tumors, confined mostly in the inner auditory canal. With the middle fossa approach, the IAC is opened superiorly. The facial nerve is visualized first, on top of the bundle of nerves running through the inner auditory canal. Next, it is the vestibular portion of CN VIII, from which the tumor usually grows. Beneath it is the cochlear portion of CN VIII, the AN. Because the approach opens the inner auditory canal from the top, the surgeon must go around the facial nerve to get to the AN. Thus, this approach may increase the risk of CN VII damage. Additionally, because retraction of the temporal lobe is necessary for exposure, memory or language transient deficits may be encountered postoperatively.

Retrosigmoid or Suboccipital Approach

This approach is most beneficial when the tumor is located outside the IAC and adjacent to the brain stem. This approach has been found by several authors as decreasing the risk of postoperative hearing deficit or facial paresis. However, this approach requires retraction of the cerebellum during which pulling on the CN VIII can occur and result in dysfunction. Thus, BAEP monitoring is successfully used to "guide" the traction on CN VIII.

Based on the evaluation of 335 cases, Harner et al. (1990) found this approach, combined with intraoperative electrophysiologic monitoring in acoustic neuromas removal, to be most beneficial because it (1) provides wide access to the tumor, (2) works for any tumor size, and (3) offers the potential to preserve facial and auditory function in all cases as well as the possibility to change the surgical technique avoiding thus labyrinthine damage that can often happen during the translabyrinthine approach.

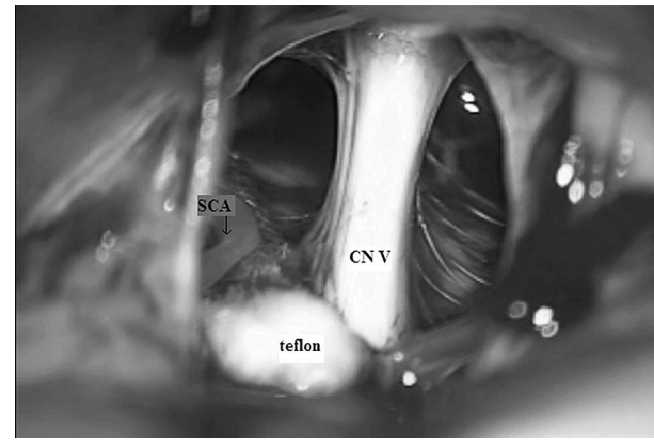

FIG. 9. Microvascular decompression in trigeminal neuralgia: placement of Teflon material between CN V and superior cerebellar artery (SCA).

Microvascular Decompression of CN V, CN VII, or CN VIII

These surgical procedures are usually performed under neurophysiologic monitoring. Trigeminal neuralgia and hemifacial spasm are usually caused by mechanical compression with irritation of CN V and CN VII, respectively, by a vascular structure. Similarly, disabling positional vertigo can be caused by vascular compression of CN VIII, in which case microvascular decompression can be attempted. The surgical procedure consists of the identification of such vessel and of its separation from the nerve, by the placement of Teflon material between the 2 structures (see Fig. 9).

Base Skull Surgeries

CNVIII monitoring can be successfully added to monitoring of other cranial nerves during base skull surgeries.

Middle Ear and Inner Ear Surgeries

These procedures are associated with an increased risk of damage of auditory apparatus and thus they can benefit from monitoring of the cochlea and of the distal part of CN VIII.

Brain stem Surgeries

Procedures that could injure the brain stem more diffusely, such as brain stem tumor removal or vascular surgery of the posterior fossa or suboccipital decompression, could benefit from BAEP monitoring.

In particular, BAEPs are a good tool to monitor the pons. Injuries will occur through similar mechanisms: mechanical, thermal, and vascular injuries. Injury of the lower pons (including cochlear nucleus and superior olivary complex) will result in disappearance of wave II, with equal delay in waves III and V to total loss. Instead, wave I is unchanged or even increased because of the disinhibition of the cochlea or because of the interruption of the inhibitory pathways (e.g., the olivocochlear bundle or descending brain stem auditory pathways (Legatt, 1999)).

TABLE 1. Neurophysiologic Monitoring of the Auditory Pathways: Surgical Applications

Nervous Structures Monitored	Neurophysiologic Tests Used	Surgical Procedure
CN VIII	EcochG (cochlea and distal CN VIII)	Intracanalicular acoustic neuroma removal
		Middle ear and inner ear surgery
	CNAPs (CN VIII distal to the recording site)	CPA tumor removal (e.g., acoustic neuroma, meningioma)
		Microvascular decompression CN V (for trigeminal neuralgia)
	BAEPs (entire CN VIII)	Microvascular decompression CN VII (for hemifacial spasm)
		Microvascular decompression CN VIII (for positional vertigo)
		Vestibular neurectomy (for Meniere disease)
Brain stem	BAEPs (auditory pathways from lower pons to the inferior midbrain/inferior colliculi)	CPA tumors
		Skull base surgery
		Vascular surgeries of the posterior circulation
		Suboccipital decompression (e.g., chiari malformation, C1/C2 vertebral processes)

Injury of the mid to upper pons results in the preservation of waves I, II, and III and a delay in wave V or its total loss. Injury of the auditory pathways rostral to the mesencephalon most likely will not result in BAEP changes**.**

Table 1 offers a list with surgical procedures that could benefit from BAEPs, EcochG, and CNAPs monitoring.

CONCLUSIONS

Neurophysiologic monitoring of CN VIII can be successfully used during a variety of surgical procedures that could cause its damage and can be done with the aid of several tests. While BAEPs offer an overall assessment of the auditory pathways and thus not only of CN VIII but also of the brain stem up to the inferior midbrain, EcochG and CNAPs are very sensitive and more reliable in assessing the function of the cochlea and the distal part of the nerve.

Accurate and useful interpretation of changes seen from baselines cannot be done without solid understanding the principles, methodology, and neurophysiologic–neuroanatomic correlations of the neurophysiologic techniques involved. It also requires the knowledge of technical troubleshooting and of effects of systemic factors on the recorded evoked responses.

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