Auditory-Evoked Potentials

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Key Learning Points

- Auditory-evoked potentials (AEPs) are most useful to monitor the integrity of the intracranial auditory nerve (cochlear portion of cranial nerve 8).
- The electrocochleogram (ECochG) can provide independent verification of stimulus delivery.
- Waves I and V are most robust on the AEP; wave I originates from the cochlea, which is typically not directly in harm's way. Wave V originates at the level of the inferior colliculus, and ascends to the medial geniculate body.
- Brainstem pathways of the auditory system run predominantly contralateral to the stimulated ear.
- Brainstem auditory potentials are very resistant to the effects of anesthetic agents.

Anatomy of the Auditory System

Sound signals are processed by the auditory system in a sequential manner. First, the acoustic energy of sound is conducted to the cochlea located within the inner ear, where conversion to a coded electrochemical signal takes place. This signal is then transmitted along the auditory pathway via the eighth cranial nerve (CN), brainstem, and midbrain to the primary auditory cortex. Various auditory-evoked potentials (AEPs) can be recorded from all these elements (Fig. 3.1; see also supplemental electronic content, Video 3.1) [1]. The tracings consist of waves with peaks and troughs that correspond to fluctuations in electrical potential. Each wave is either a peak (P = positive deflection) or trough (N = negative deflection) and is further described in terms of both amplitude (peak-to-peak height) and latency (time from stimulus). The waves can be divided based on the time between acoustic stimulus and evoked response into short-, mid-, and long-latency acoustic-evoked potentials. The long-latency acoustic-evoked potentials originate in the association cortex and require cooperation and attention. They are abolished under anesthesia and will, therefore, not be further considered.

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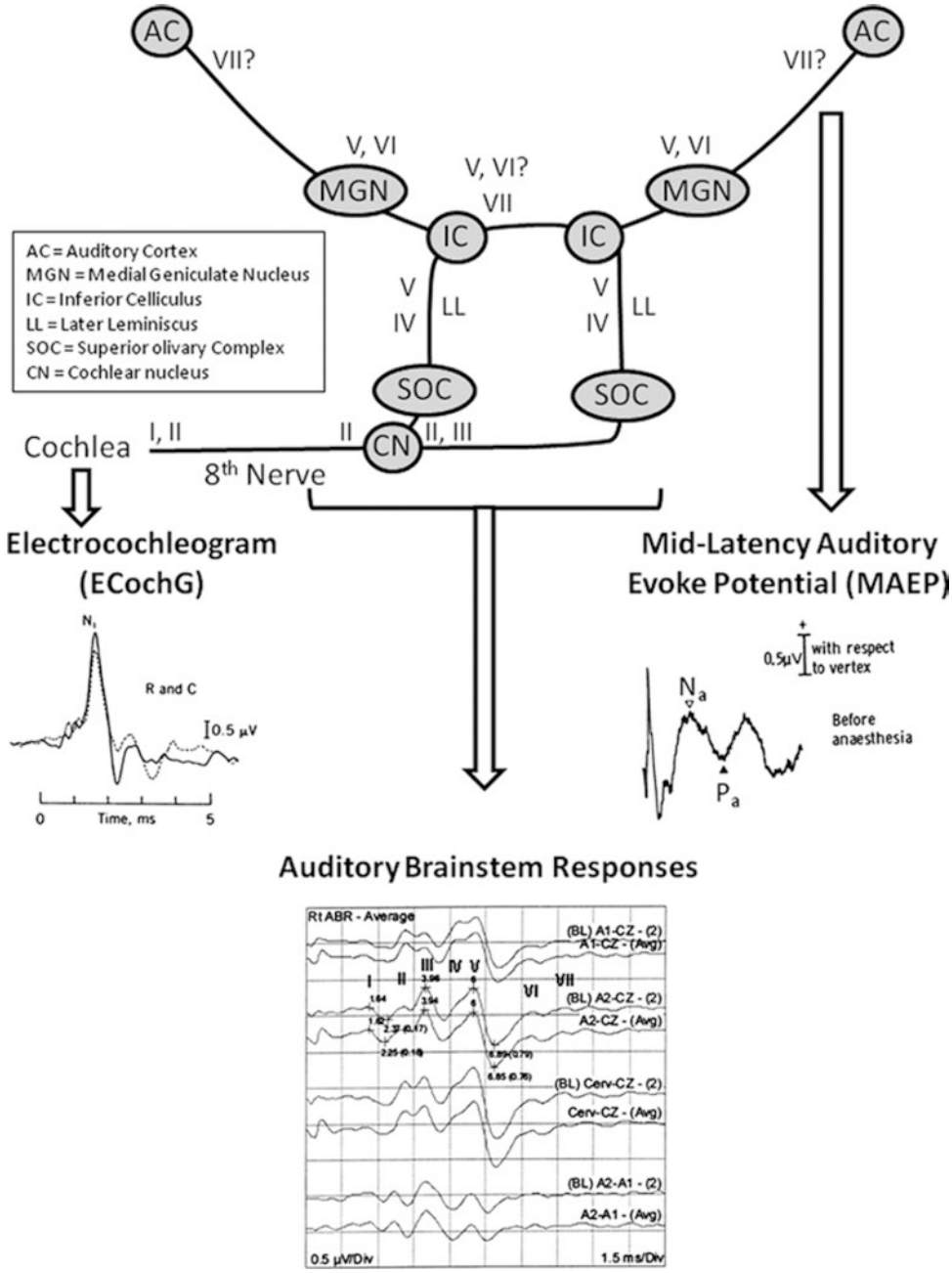


Fig. 3.1 Neural pathway of the auditory system and recordable potentials. Note that auditory input is transmitted to bilateral primary auditory cortices and ascends through brainstem and midbrain partially ipsilateral and partially contralateral to the side of stimulation (for details, see text). The electrocochleogram (ECochG) contains near-field signals from cochlea and distal auditory nerve [49]. The brainstem auditory-evoked potential (BAEP) reflects activity in the entire neural pathway. Note how the morphology of individual waves projects differently in the individual recording channels. In addition to Na and Pa waves reflecting activation of the primary auditory cortex, the mid-latency auditory-evoked potential (MLAEP) contains a distinctive wave V of the auditory brainstem response (ABR) within its first 10 ms (Adapted from Thornton et al. [54]; with permission). Anatomic and radiographic representations of the auditory pathway can be found in the supplemental online materials

Conduction of Auditory Signals from Ear to Cochlea

The ear is subdivided into external, middle, and inner parts. The external ear is composed of the auricle that acts to collect and direct sound through the external auditory meatus toward the tympanic membrane. The tympanic membrane forms the boundary between the external and middle parts of the ear. The tympanic membrane is covered in a very thin squamous epithelial layer externally and by a mucous membrane internally. It moves in response to air vibrations that pass to it through the external auditory meatus. Movements of the tympanic membrane are transmitted by three auditory ossicles, the malleus, incus, and stapes, through the middle ear to the inner ear.

The middle ear lies within the petrous portion of the temporal bone. The tympanic cavity lies directly behind the tympanic membrane and shares important anatomical relationships to neighboring structures. Superiorly, the epitympanic recess is separated from the middle cranial fossa by a thin roof of bone, the tegmen tympani. The anterior (carotid) wall separates the carotid canal from the tympanic cavity. The Eustachian tube projects through the anterior wall to connect the middle ear to the nasopharynx. The floor (jugular wall) is formed by a layer of bone that separates the tympanic cavity from the superior bulb of the internal jugular vein. The medial or labyrinthine wall separates the tympanic cavity from the inner ear. The middle ear also connects posterior and superior with the mastoid air cells through the mastoid antrum.

The auditory ossicles form a chain that extends across the tympanic cavity from the tympanic membrane to the oval window (fenestra vestibuli). The malleus (hammer) is attached to the tympanic membrane. Its superior head lies within the epitympanic recess, and its handle is embedded in the tympanic membrane. Movement of the tympanic membrane results in movement of the malleus. The head of the malleus articulates with the incus (anvil). The long process of the incus articulates with the stapes (stirrup). The base of the stapes is attached to the oval window. Typically, most of the sound energy travels via this ossicular chain to the cochlea. If movement of the tympanic membrane or ossicular chain is restricted by fluid or a disease process, a conductive hearing deficit results and the far less effective conduction of sound via bone becomes an important input to the cochlea. Conversely, during bone drilling, bone-conducted noise may overwhelm the cochlea and lead to a temporary hearing deficit.

Two muscles lie within the middle ear and act to prevent excessive movement of the ossicles due to loud noises. The tensor tympani muscle arises from the superior surface of the cartilage forming the auditory tube, the greater wing of the sphenoid bone and the petrous part of the temporal bone. It is innervated by the mandibular division of the trigeminal nerve and inserts on the handle of the malleolus. The tensor tympani pulls on the handle of the malleolus, tenses the tympanic membrane, and thus dampens oscillations of the tympanic membrane. The stapedius muscle arises from the pyramidal eminence on the posterior wall of the tympanic cavity. It inserts on the neck of the stapes. It is innervated by a branch of the facial nerve. The stapedius muscle pulls the stapes posteriorly and tilts the base of the stapes in the oval window. This acts to tighten the stapes and reduce excessive movement.

Neural Components of the Auditory System and Electrical Generators Along the Auditory Pathway

Cochlea: Electrocochleogram

The cochlea converts sound waves into action potentials in the neurons of the cochlear nerve. Sound waves conducted to the oval window propagate in the perilymph of the cochlea. The action of these waves on the spiral organ of Corti generates excitatory synaptic input from the cochlear hair cells, which in turn depolarizes the cochlear end of the auditory nerve. This depolarization leads to the generation of the eighth nerve compound action potential.

The electrical activity within the cochlea can be recorded in the form of an electrocochleogram (ECochG; Fig. 3.1). The ECochG includes the cochlear microphonic, the summation potential, and the eighth nerve compound action potential. The hair

cells generate the cochlear microphonic and summation potential within the cochlea (for details, see section "Electrocochleogram" below). The eighth nerve compound action potential results from depolarization within the distal (cochlear) portion of the auditory nerve axons. It generates wave N1 of the ECochG. It is recorded as a phase negativity in the middle ear or extratympanic recording site. Sounds used to elicit ECochGs may produce more than one volley of action potentials within the auditory nerve, thus producing N1 and N2 (and sometimes N3) components of an eighth nerve compound action potential. The N1 potential corresponds to wave I of the brainstem auditory response discussed below.

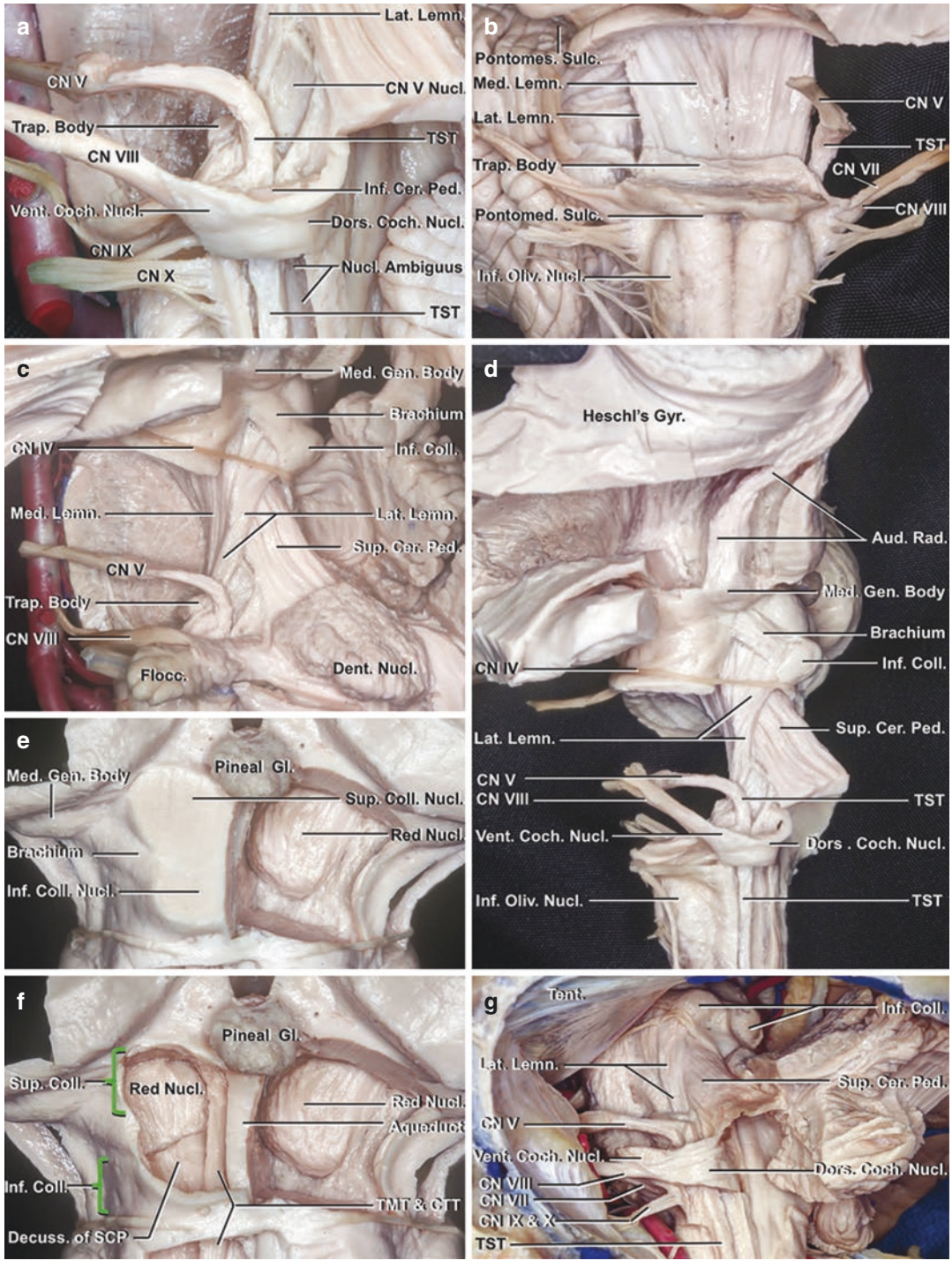
Auditory Pathway from Cochlear Nerve to Midbrain

Auditory Brainstem-Evoked Responses and Cochlear Nerve Compound Action Potential

Neural transmission of auditory signals begins with input from cochlear hair cells into the distal auditory nerve, whose anatomic course puts it at risk of injury during many procedures in the posterior cranial fossa. Once the signals reach the brainstem, they pass through a complex series of relay and processing stations to the midbrain. The signals travel partially ipsilateral to the side of stimulation, but mostly cross over to the contralateral side (see Figs. 3.1 and 3.2).

First-order auditory neuron axons run in the cochlear division of CN VIII from the spiral organ of Corti to the dorsal and ventral cochlear nuclei in the upper medulla. Myelinated dendrites of the auditory nerve pass into and through the spiral ganglia and form a nerve bundle in the internal auditory canal. Both the acoustic and vestibular portions of the auditory nerve pass through the temporal bone alongside the intracranial portion of the facial nerve. Together, they exit the internal auditory canal and travel to the brainstem. At the point of exit from the internal auditory canal, both facial and vestibulocochlear nerves make an acute turn from the anteromedial trajectory of the internal auditory canal within the petrous pyramid of the temporal bone posterolaterally toward the cerebellopontine angle of the brainstem. This acute turn "anchors" the vestibulocochlear nerve and puts it at risk of a stretch-induced neurapraxic injury during retraction of the brainstem, especially if the anat-

Fig. 3.2 Fiber dissection of the central auditory pathway. (**a**) Ventral and dorsal cochlear nuclei. Posterolateral view of the junction of the cranial nerve with the brainstem. The ventral cochlear nucleus is situated on the lateral and dorsal cochlear nucleus on the dorsal surface of the inferior cerebellar peduncle. They are positioned close to the trigeminal spinal tract, facial nucleus, and nucleus ambiguus, which are located ventromedial to the trigeminal spinal tract. The facial nucleus is hidden deep into the cochlear nuclei. (**b**) Anterior view. The ventral pons was removed to expose the medial and lateral lemnisci and the trapezoid body formed by crossing auditory fibers at the level of the lower pons. (**c**) Left lateral view (Fig. 3.2 continued). The lateral lemniscus ascends medial to the intrapontine segment of the trigeminal nerve and lateral to the medial lemniscus and superior cerebellar peduncle to reach the inferior colliculus. (**d**) After reaching the inferior colliculus, auditory information is carried to the medial geniculate body by the brachium of the inferior colliculus, which ascends obliquely on the lateral surface of the midbrain. After reaching the medial geniculate body, the auditory pathway crosses beneath the lentiform nucleus in the sublentiform pathway to reach the auditory cortex on the most anterior transverse temporal gyrus, referred to as Heschl's gyrus. (**e**) Posterior view of the midbrain. The nuclei of the superior and inferior colliculi are located below the surface. The red nucleus is located at a deeper level. (**f**) Further dissection. Structures near the inferior collicular implant, in order from dorsal to ventral, are the oculomotor and trochlear nuclei located just ventral to the aqueduct in the midline, the trigeminal mesencephalic and central tegmental tracts near the midline, decussation of the superior cerebellar peduncle at the level of the inferior colliculus and the red nucleus located between the mid-level of the inferior colliculus, and the lateral wall of the third ventricle. (**g**) Left retrosigmoid view. The left cerebellar hemisphere was removed to expose the dorsolateral brainstem and the ventral and dorsal cochlear nuclei, lateral lemniscus, and inferior colliculus. *Aud* auditory, *Cer* cerebellar, *CN* cranial nerve, *Coch* cochlear, *Coll* colliculus, collicular, *CTT* central tegmental tract, *Decuss* decussation, *Dent* dentate, *Dors* dorsal, *Flocc* flocculus, *Gen* geniculate, *Gl* gland, *Gyr* gyrus, *Inf* inferior, *Lat* lateral, *Lemn* lemniscus, *Med* medial, *Nucl* nucleus, *Oliv* olivary, *Ped* peduncle, *Pontomed* pontomedullary, *Pontomes* pontomesencephalic, *Rad* radiations, *Sulc* sulcus, *Sup* superior, *Tent* tentorium, *TMT* trigeminal mesencephalic tract, *Trap* trapezoid, *TST* trigeminal spinal tract, *Vent* ventral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CN VII
CN IX & X
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omy of the posterior fossa is already disrupted by pathology such as a cerebellopontine angle tumor. This intracranial portion of the cochlear nerve presents an opportunity to record a compound nerve action potential (CNAP) from an electrode placed directly on the nerve in its course from the internal auditory canal to the root entry zone or its vicinity.

Auditory nerve fibers synapse at either the posterior ventral cochlear nucleus or the anterior ventral cochlear nucleus. Fibers that synapse at the posterior ventral cochlear nucleus also have connections with the dorsal cochlear nucleus. From the cochlear nuclei, second-order neurons may follow several pathways or combinations of pathways en route to the inferior colliculus. Most fibers decussate via the trapezoid body and pass up the lateral lemniscus to the contralateral inferior colliculus of the midbrain. Some fibers synapse in either the medial or lateral superior olivary nuclei. Others pass through the ipsilateral lateral lemniscus to the ipsilateral inferior colliculus. All ascending fibers synapse at the inferior colliculus. Third-order neurons from the inferior colliculus ascend to the medial geniculate body at the level of the thalamus on each side. Fourth-order neurons pass through the internal capsule and then form the auditory radiation to the primary auditory cortex. This complex pattern of tracts and relay stations is involved in elementary processing of auditory input, e.g., by extracting directional information about the source of sounds or as the afferent limb of auditory startle responses.

Activity in the neural pathways from the cochlea to the midbrain can be recorded non-invasively in the form of auditory brainstem responses (ABRs, sometimes also called brainstem auditory-evoked responses [BAERs] or brainstem auditory-evoked potentials [BAEPs]; see Fig. 3.1) or invasively after exposure as compound action potentials from the cochlear nerve. ABR peak are labeled I through VII. As with other sensory-evoked potentials, the wave amplitude, absolute latencies, and interpeak latencies are evaluated to assess the integrity of the auditory system. The purported generators for these peaks are shown in Fig. 3.1 and are summarized in Table 3.1. Although some researchers have postulated that each peak corresponds to one generator, it appears that most ABRs are a result of the summation of inputs from multiple generators [2–4]. The pattern of connections in the auditory system contributes to this complexity, as ascending fibers both cross and bypass relay nuclei [4–6]. Nonetheless, the information in Figs. 3.1 and 3.2 and Table 3.1 can be used to help localize the site of an injury. While functional deficits can often be localized when injury or ischemia occurs, the complexity of the system may sometimes lead to changes in ABRs with no change in function [7, 8].

Table 3.1 Purported neural generators of ABR peaks[a]

Peak	Generator
I	Acoustic nerve (extracranial)
II	Acoustic nerve (intracranial); cochlear nuclei
III	Cochlear nuclei
IV	Lateral lemniscus; superior olivary complex
V	Inferior colliculus; contralateral lateral lemniscus
VI	Medial geniculate nuclei
VII	Thalamocortical radiations

[a]Peaks I, III, and V are considered the most useful for intraoperative neuromonitoring of ABRs. Most peaks are likely a result of the summation of inputs from multiple generators. While not all of these generators have been proven, the designations are clinically useful because they point out the approximate location of an injury. *ABR* auditory brainstem response

Wave I of the ABR arises from action potentials in the most distal portion of the myelinated auditory nerve [9]. Wave I of the ABR is equivalent to N1 of the ECochG [10]. Wave I is a near-field potential, recorded in the vicinity of the ipsilateral stimulated ear. It represents the peripheral potential of this sensory modality. Loss of wave I may indicate damage to the inner ear but may also be caused by technical problems in the delivery of acoustic stimuli to the ipsilateral ear. When wave I is absent, ABRs cannot be used to make inferences about the integrity of the brainstem. Wave II of the ABR occurs at approximately the same latency as N1 of the compound action potential of the proximal auditory nerve to the cochlear nucleus. It occurs on the ipsilateral side. Wave III predominantly originates in the caudal pontine tegmentum and region of the superior olivary complex. Near-field activity in the ipsilateral cochlear nucleus corresponds with wave III [11]. Ascending projections are bilateral, so wave III may receive input from both the ipsilateral and contralateral ear. Scalp-recorded wave III has been recorded at the same time as near-field activity in the cochlear nucleus [12]. Other recordings from the area of the

cochlear nucleus in the lateral recess of the fourth ventricle indicate activity that coincides with wave IIIn (the negative peak between III and IV) [13, 14]. The auditory nerve may continue to be active during generation of the scalp-recorded waves III and IV [4]. Waves IV and V are often fused into a IV–V complex and their generators appear to be near each other. Wave IV appears to reflect activity in ascending auditory fibers within the dorsal and rostral pons, caudal to the inferior colliculus with input from both ipsilateral and contralateral sides. Wave V appears to predominantly reflect activity at the level of the inferior colliculus, perhaps including activity in the rostral portion of the lateral lemniscus as well as activity in the contralateral lateral lemniscus as it terminates on the inferior colliculus [4, 11]. Waves VI and VII are inconsistent and variable; therefore, they are not routinely monitored [15]. Most intraoperative neuromonitoring utilizes only waves I, III, and V to guide the intraoperative course [6, 16, 17].

Primary Auditory Cortex: Mid-Latency Auditory-Evoked Potentials

Tracts carrying auditory information project from the medial geniculate body to the cortex and other brain areas by multiple routes [18]. The medial geniculate body and cortex are linked by two main routes. The first pathway from the ventral medial geniculate body carries only auditory input and follows a sublenticular route through the internal capsule to Heschl's gyrus in the superior temporal lobe. The second pathway from the medial geniculate body projects into the inferior portion of the internal capsule and carries mixed auditory, somatic, and possibly visual sensory input. Auditory fibers from the medial geniculate body also project to the caudate nucleus, the putamen, and the globus pallidus.

Intrahemispheric and interhemispheric connections occur within the primary auditory cortex. Multisynaptic pathways likely exist in the middle and posterior areas of the superior temporal gyrus. Fibers also extend from the superior temporal gyrus to the insula and frontal operculum. The arcuate fasciculus provides auditory input from the auditory cortical areas in the temporal lobes to the frontal lobes. Wernicke's area in the temporal lobe and Broca's area in the frontal lobe receive auditory information via the arcuate fasciculus. Auditory input also passes to the hippocampus and occipital regions of the brain. Although these areas and pathways are not anatomically defined, they provide auditory input to memory and visual association areas. Auditory information passes between the two hemispheres through the corpus callosum, the primary connection between the left and right hemispheres. The transcallosal auditory pathway begins at the auditory cortex and passes posteriorly and superiorly around the lateral ventricles.

Electrical phenomena associated with activation of the auditory cortex can be recorded in the form of mid-latency auditory-evoked potentials (MLAEPs, see Fig. 3.1). MLAEPs are observed 10–60 ms after an auditory stimulus [19]. They appear to arise from the ventral portion of the medial geniculate body and primary auditory cortex in the primary pathway and non-primary pathways in the auditory thalamocortical projection [20, 21].

MLAEPs consist of four deflections, labeled Na, Pa, Nb, and Pb. Na and Pa latencies are between 10 and 25 ms and 22 and 40 ms, respectively [19]. Nb latency is at 40 ms and Pb is at 40–60 ms. Magnetoencephalographic and intracerebral recordings suggest that the Na/Pa complex is generated in the posteromedial part of the first transverse gyrus. MLAEPs correlate well with wakefulness during general anesthesia when using desflurane or propofol [22] and are associated with awakening from anesthesia following verbal command [23]. MLAEPs may be abnormal in neurologic diseases such as dementia, Parkinson's disease, multiple sclerosis, and myotonic dystrophy [24–33].

Vascular Supply of Auditory Pathway Structures

The cochlea receives its blood supply from the internal auditory artery, which is usually a branch of the anterior inferior cerebellar artery. The internal auditory artery is quite small in diameter and passes through the internal auditory canal along with the eighth nerve [34]. Damage to this artery

will cause cochlear ischemia or infarction. Cochlear ischemia from obstruction or disruption of the internal auditory artery may affect the ECochG and wave I of the ABRs resulting in the loss of all subsequent components [35]. This may occur during tumor resection and lead to postoperative deafness [36]. These effects may be reversible if perfusion is restored within 15 min [36].

The brainstem (medulla, pons, and midbrain) receives the bulk of its blood supply from the vertebrobasilar system [37]. Except for the labyrinthine branch and early branches of the vertebral arteries, all other branches supply the brainstem and medulla. In principle, conducting vessels run along the brainstem surface, whereas the nuclei within the brainstem and the fiber tracts are supplied by perforating vessels. The vertebral arteries supply the medulla. The paramedian branches of the basilar artery supply medial pontine structures. Short circumferential arteries supply the anterolateral pons. Long circumferential branches of the basilar artery run laterally over the anterior surface of the pons and anastomose with branches of the anterior inferior cerebellar artery. The inferior colliculus receives blood from the anterior inferior cerebellar artery (caudally) with some reinforcement rostrally from the superior cerebellar artery. Quadrigeminal arteries arise from branches of the basilar artery and also supply the inferior colliculus.

The medial geniculate nucleus lies in the dorsal thalamus and receives its blood supply from posterolateral arteries (thalamogeniculate), which arise from the posterior cerebral artery. The primary auditory cortex, which lies in the superior temporal lobe, is supplied by branches of the middle cerebral artery and, therefore, by the anterior cerebral circulation. Interhemispheric fibers that connect the left and right auditory cortices pass through the posterior corpus callosum, which receives its blood supply from the pericallosal artery, a branch of the anterior cerebral artery [18, 38].

ABRs may change during posterior fossa surgery because of ischemia or infarction from clipping or compression of arteries that perfuse the brainstem auditory pathways [8]. Patients who experience major changes in ABRs that persist to the end of the operation almost always have new postoperative neurologic deficits [39, 40]. Changes in waveforms almost always reflect anatomical deficits. For example, damage below the mesencephalon will affect wave V, and wave III may or may not be spared depending on the location of the lesion. Wave III would be lost if the lesion is caudal to or at the superior olivary complex. However, wave I would remain intact. Interruption of the blood supply proximal to the internal auditory artery may affect wave I. For example, during posterior fossa vascular surgery, damage to the vertebrobasilar system, which supplies the anterior inferior cerebellar artery and the internal auditory artery, could cause ischemic cochlear damage and loss of all waveforms.

Techniques for Recording Auditory-Evoked Potentials

Auditory-evoked potentials can be recorded from all neuronal structures that contribute to the auditory system [41]. The first potentials generated in response to sound come from the cochlea. They can be recorded in the form of the ECochG. Because the cochlea lies well protected in the temporal bone, direct damage during surgery is typically either not a concern or planned as part of the surgical access, such as in the translabyrinthine approach to the posterior fossa. Therefore, monitoring of the ECochG is not widely used. From the cochlea, potentials are carried along the auditory nerve and the brainstem to the primary auditory cortex and further to association areas of the cortex. MLAEPs reflect activation of the primary auditory cortex occurring 10–50 ms after acoustic stimulation. MLAEPs are also sensitive to the effects of general anesthetics and are therefore not used for intraoperative monitoring of the integrity of the auditory pathway. On the contrary, because of their sensitivity to anesthetics, they have been used to monitor cortical anesthetic drug effect to help assess "anesthetic depth." Their performance as a monitor of anesthetic depth is comparable to that of other monitors relying on the processed electroencephalogram (EEG) [42].

The use of MLAEPs-guided depth of anesthesia and its impact on the incidence of postoperative delirium and postoperative cognitive dysfunction has been evaluated in recent trials [43, 44]. Recording MLAEPs requires both stimulation and recording and is therefore technically more elaborate than the setup for processed frontal EEG. This fact has hampered commercial exploitation and clinical acceptance of MLAEPs as a monitoring technique during anesthesia.

Short-latency potentials occur less than 10 ms after an acoustic stimulus and originate in the acoustic nerve and brainstem. They are typically referred to as auditory brainstem responses (ABRs); sometimes also described as BAERs or BAEPs. An anesthesiologist is most likely to encounter intraoperative use of auditory-evoked potentials in the form of ABRs.

The recording of auditory-evoked responses presents significant technical challenges because the signals originate from anatomical structures that are far removed from the site of electrode placement on the head's surface. Because of this distance between purported anatomical generator and recording electrode, these types of responses are called far-field responses. Their amplitude is small, typically less than 0.5 μV, compared to the EEG and the electrocardiogram, which are a 100 and 1000 times larger, respectively. Because of their small amplitude, auditory-evoked potentials cannot be seen on continuous recordings of electrical activity. Instead, they require signal averaging of the responses to 500–2000 acoustic stimuli.

A few centers use direct recording from the cochlear nerve. The CNAP is a near-field technique where the electrode is placed proximal to the neural generators of the ABR at the nerve entry zone of the brainstem. This method overcomes the need to average responses. Since this method requires direct access, concerns with the use of CNAPs include the lack of familiarity among surgeons and the monitoring teams, the need for specialized electrodes, the difficulty of "anchoring" the electrode to achieve stable recordings, and concerns about placing an electrode on a portion of the nerve that is most vulnerable to mechanical injury, i.e., its root entry zone to the nerve; a further drawback is that the method is "blind" to the risk of stretch injury to the cochlear nerve during initial exposure. Nonetheless, some centers have reported improved hearing preservation over ABRs alone in microvascular decompression and in surgeries for small acoustic neuromas [45, 46].

Stimulation

Acoustic stimuli are presented intraoperatively as "clicks" of 100-μs duration. The clicks comprise a broad spectrum of tone frequencies and thus activate much of the cochlea. This nearly simultaneous activation of the cochlea triggers a synchronized volley of action potentials in the acoustic nerve, which in turn can be recorded as well-defined peaks in the auditory-evoked response. Clicks can be delivered in three different "polarities"—rarefaction, condensation, or alternating (Fig. 3.3). The description of the click polarity refers to the initial movement of the tympanic membrane away from, toward, or alternating between away and toward the stimulator. In practice, either an alternating polarity is used to cancel out the stimulus artifact or the polarity that results in the clearest response. In rare instances, bone conduction can be used to deliver the acoustic stimulus.

The intensity/volume of the click stimulus can be based on the results of preoperative determinations of hearing thresholds. A stimulus intensity of 70 dB above normal hearing level (70 dB nHL) normally yields maximal auditory responses. In the absence of a preoperative audiogram, 90–95 dB is frequently used, particularly in the presence of a preoperative hearing deficit. Note that a decrease in the stimulus intensity as it is delivered to the cochlea reduces the amplitude of the auditory-evoked potentials (see Fig. 3.3). Such a reduction can result from a dislodged stimulator, fluid in the middle ear (e.g., from breached mastoid air cells), accumulation of nitrous oxide in the face of a blocked Eustachian tube, or damage to the auditory pathway [4].

Depending on the structures at risk during surgery, stimuli are typically presented unilaterally even though rostral parts of the auditory pathway

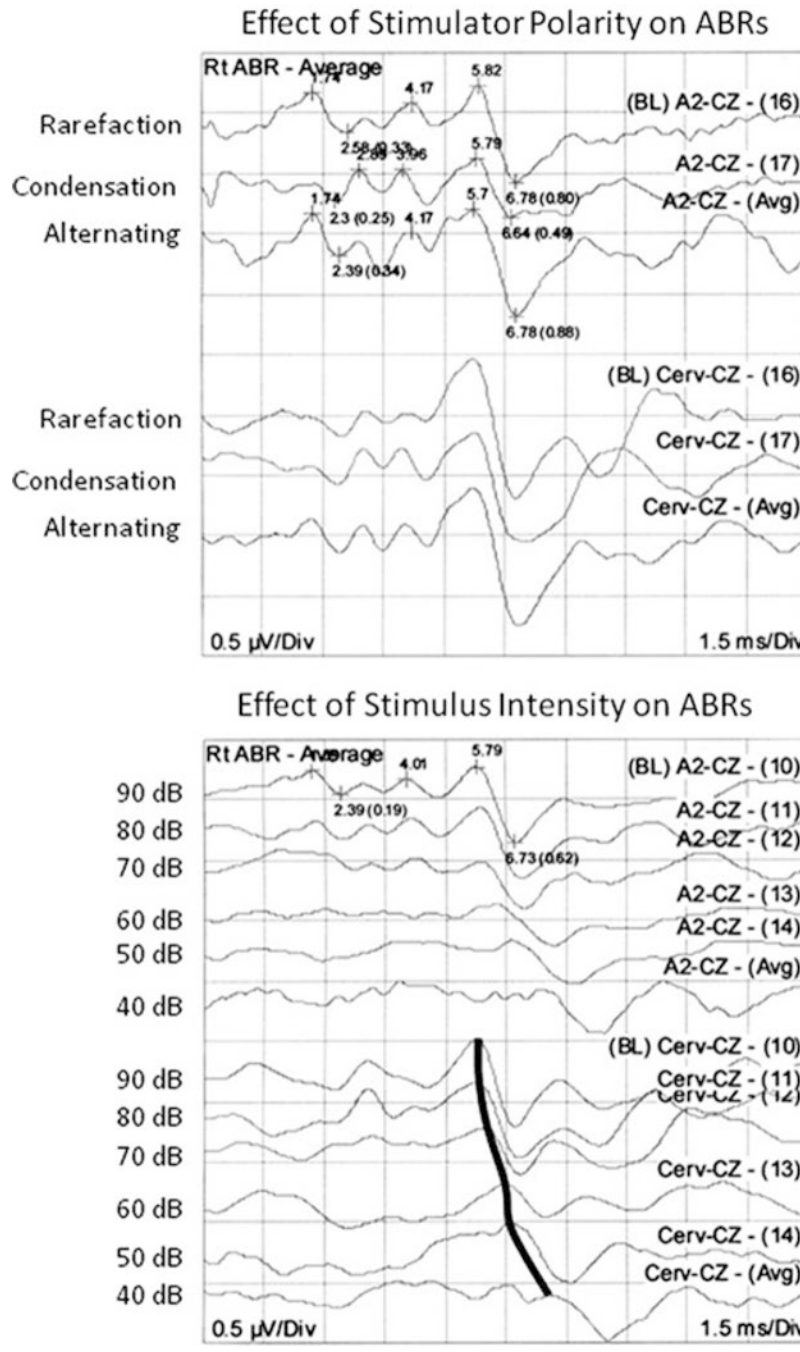

Fig. 3.3 Effect of click polarity and stimulus intensity on brainstem auditory-evoked potentials. The top panel shows ABRs recorded in response to three different click polarities. Note that click polarity has notable effects on the early ABR in the A2-Cz channel, because it contains information from the ECochG. The *bottom* panel shows the effect of a stepwise decrease in stimulus intensity on the ABR. Note that wave I is lost at stimulus intensities less than 80 dB, suggesting a problem with sound delivery, whereas the desynchronization of waves III and V at low-stimulus intensities are indistinguishable from changes caused by damage to the auditory system

proceed largely crossed over to the contralateral side, but also to a lesser extent uncrossed (see above). Unilateral presentation of stimuli allows diagnosis of lesions to the cochlea and distal auditory nerve ipsilateral to the side of stimulation. If unilateral stimulation is used, bone conduction to the contralateral ear is typically "blocked" by masking. Masking refers to the continuous administration of white noise to the non-stimulated ear typically at intensities 30 dB less than the click stimulus. An alternative way of stimulation is the use of interleaved stimuli alternating between right and left ears but sorted into separate averaged recordings for left and right ear

stimulation. Such interleaved stimulation does not allow for masking, but the small decrement in sensitivity is made up by the fact that both sides are monitored continuously. Bilateral stimulation is sometimes used to record MLAEPs.

Because auditory responses up to the level of the brainstem occur within 10 ms of stimulation, stimuli can be presented as frequently as 30–50 times per second (30–50 Hz). If there is a preexisting hearing deficit such as that caused by a large acoustic neuroma, slower stimulation at 10–15 Hz may be necessary. Stimulus rates should never be equal to or a divisor of the line frequency of 60 Hz, because signal averaging will then tend to augment the electromagnetic interference from the line frequency rather than canceling it out.

Physically, the stimuli can be presented either with earphones or with foam ear inserts connected by a tube to stimulators placed at a short distance (i.e., <10 cm) from the ear. Earphones are used less frequently, because they put a source of electromagnetic interference close to the generators of the auditory response. Ear inserts are less bulky and do not contribute to noise. The acoustic transmission of sound from the stimulator through the tube insert to the tympanic membrane delays auditory responses by less than 1 ms.

Electrocochleogram

Recordings of the ECochG require placement of a primary electrode close to the cochlea. During middle ear surgery, such an electrode can be placed on the promontory or the round window. A non-invasive recording is possible from the external ear canal, which is preferred over a more distal mastoid electrode [47]. The secondary or reference electrode can be at the contralateral ear or at Cz. The filter bandpass is set to 5000–3000 Hz. Stimulation parameters are typically the same as those described above, even though the two cochlear potentials depend on stimulus duration and are more pronounced, when longer stimuli are used [48]. The typical time base used for ECochGs is 10 ms or less.

Three potentials characterize the ECochG (Fig. 3.3). In sequence of activation, they are the cochlear microphonic, the summating potential, and the N1 potential. Based on the sequence of steps that translate auditory input into nerve impulses, the cochlear microphonic and summating potential originate in the hair cells of the organ of Corti and the N1 potential originates in the distal auditory nerve. The cochlear microphonic is an AC voltage that mirrors the waveform of the acoustic stimulus. Therefore, it can be minimized by stimulating with alternating polarity and can be augmented by subtraction of traces recorded with alternating polarity (Fig. 3.3) [49]. In contrast, the summating potential is a DC current thought to reflect the fact that transduction of the stimulus by the hair cells does not occur uniformly and at the same time throughout the cochlea. Therefore, the summating potential is increased in patients with inner ear diseases such as Meniere's disease that further distort the transduction of acoustic stimuli in the inner ear [49]. With the short clicks typically used for stimulation, the summating potential is reflected as a "shoulder" on the much larger N1 potential. The final potential recorded in the ECochG is the N1 potential, which reflects activation of the distal auditory nerve and thus the same physiologic phenomenon as wave I of the ABR. Because it is a near-field potential, it is large in amplitude and requires fewer averages than a full ABR. Those laboratories that use ECochG for intraoperative monitoring typically focus on the N1 potential to quickly ascertain successful stimulation of the auditory system similar to the more familiar Erb's point recording used with somatosensory-evoked potentials (SSEPs).

Compound Nerve Action Potentials from the Cochlear Nerve

An electrode placed directly on the cochlear nerve can record a CNAP with an amplitude that is an order of magnitude larger than an ABR and follows each acoustic stimulus. Such a near-field potential provides immediate monitoring of the neural conduction of the auditory nerve [50]. During posterior fossa craniotomy surgery, surgical manipulation of the auditory nerve like stretching can affect the conduction velocity,

which will be reflected as increased latency of the negative peak of the CNAP. While partial conduction block may result in the decreased amplitude of the negative peak of the CNAP, which will result in an increase in the initial positive peak, a total conduction block will result in the complete absence of the negative peak, and then the CNAP will show as a single positive deflection (known as a cut end potential) [50]. Since manipulation in the surgical field or, in case of a vestibular schwannoma, of the vestibulocochlear nerve itself risks dislodgment of the electrode, this technique has not gained widespread acceptance. Recordings from the edge of the surgical field, e.g., from the cochlear nucleus by placing the electrode through the foramen of Luschka, result in a far-field potential that is similar in size to the ABR, but retain the advantage of updating with each stimulus (Fig. 3.4) [51].

Brainstem Auditory-Evoked Potentials

A normal ABR recording is characterized by at least three clearly identifiable waves/peaks. Although an ABR recording is classically described as containing seven waves (see Fig. 3.1), a minimum of waves I, III, and V should be present for the signal to be useful for intraoperative monitoring. A typical montage includes electrodes on the ipsilateral ear and one at the vertex, i.e., A1-Cz and A2-Cz for left and right sides, respectively. Additional channels can be used to aid in the identification of individual peaks (see Fig. 3.1). Specifically, a cervical midline electrode referenced to Cz sometimes aids in identification of wave V, whereas an A1-A2 or A2-A1 channel can aid in identification of wave I if the ECochG is not monitored separately. Except for wave I, all potentials recorded as ABRs originate from structures deep within the head and are considered far-field potentials. Therefore, wave I shows up best in channels that contain an electrode near the ipsilateral ear. Subsequent waves, in contrast, can be activated by stimulation from either side and contain limited information that allows assignment of changes to either the right or left side. In a baseline recording, it is important to clearly identify wave I and compare it with that from the contralateral ear. Clear identification of wave I guards against situations when the stimulators for the right and left sides have mistakenly been reversed. Furthermore, the presence of wave I assures delivery of an adequate stimulus and thus allows identification of unilateral damage to the ipsilateral auditory nerve.

The typical time base for ABRs is 10 ms although the stimulus delay imposed by ear insert stimulators and the presence of hearing loss sometimes requires a longer recording period. The bandpass is set from 100 or 150 to 3000 Hz and a notch filter for the 60-Hz line frequency can be added.

MLAEPs

Auditory-evoked potentials beyond the brainstem are recorded either for research purposes or to measure anesthetic drug effect [52, 53]. Examples of late potentials include the event-related potential P300, which occurs about 300 ms after an appropriate stimulus, or the mismatch negativity that occurs late after an oddball sequence of stimuli. Both reflect elements of higher-order processing and are absent under anesthesia. Latency and amplitude of early peaks of the MLAEP correlate with anesthetic drug effect (see Fig. 3.3) [54]. Two commercial monitors of anesthetic drug effect have been developed based on MLAEP technology, although neither is widely used [55]. Stimulation for MLAEPs can be done as described above, although stimulus durations up to 500 μs and simultaneous stimulation of both ears are described. The typical band pass is set to 15–250 Hz. The stimulation rate needs to be less than 10 Hz because the epoch is at least 50 ms. The montage can be mastoid—Cz, especially under anesthesia—but can also be a midline montage of a cervical electrode referenced to Cz or Fz. The benefit of using a midline montage is that it avoids recording the postauricular muscle response [52]. The postauricular muscle response is an involuntary muscle reflex in response to loud acoustic stimuli. Its amplitude may exceed

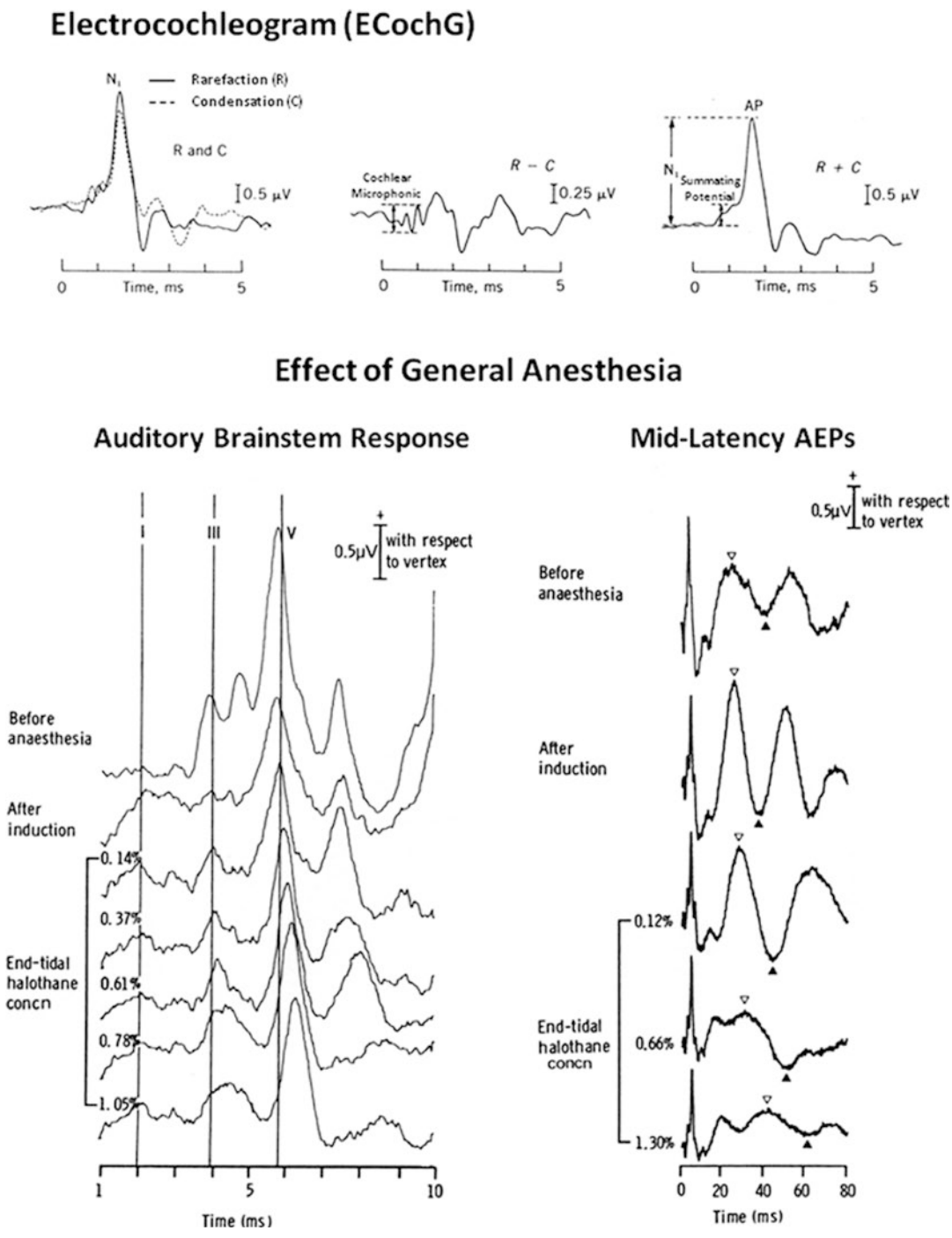

Fig. 3.4 ECochG and effects of anesthesia on brainstem and MLAEPs. The ECochG contains a prominent wave N1 that coincides with activation of the distal auditory nerve. N1 is larger than a typical ABR and therefore easier to record than wave I of the ABR. Electrical activity within the cochlea is reflected in the cochlear microphonic and summating potential. Subtraction of ECochG traces in response to rarefaction and condensation clicks emphasizes the cochlear microphonic (middle panel), whereas addition emphasizes the summating potential. Anesthesia with halothane differentially affects ABRs and MLAEPs. Whereas the latency of wave V of the ABR increases by less than 1 ms and the amplitude is unaffected (left panel), the mid-latency response nearly vanishes at high concentrations of halothane [49, 54]

that of the MLAEP and its latency of 15–20 ms coincides with the early peaks of the MLAEP. Furthermore, because it is triggered by the acoustic stimulus, signal averaging will not remove it.

Anesthetic and Physiologic Considerations for Monitoring of Auditory Brainstem Responses

Auditory brainstem responses are very resistant to the effects of general anesthetic agents (see Fig. 3.3) [54]. Therefore, no modification of the anesthetic approach to a patient is needed because of ABR monitoring. The small increases in latency caused by anesthetic agents are not clinically significant and are easily distinguished from changes in ABRs caused by technical and physiologic factors.

Sometimes technical problems cause gradual or abrupt changes in ABRs intraoperatively in the absence of physical damage to the auditory pathway (see also Chap. 29, "ENT and Anterior Neck Surgery," Tables 29.2 and 29.3). Diminished input can occur abruptly, e.g., by kinking the tube of an ear insert on the down ear in a lateral or park bench position, or gradually, e.g., by fluid accumulation in the middle ear. Conduction in the middle ear diminishes when fluid enters the middle ear either in the form of irrigation fluid, e.g., during drilling in the mastoid bone, or as blood from any of the anatomic structures in the vicinity. Accumulation of nitrous oxide in the face of a blocked Eustachian tube can also decrease conduction in the middle ear. Note that the changes caused by diminishing acoustic input look very similar to progressive damage of the auditory pathway if changes to wave I are not assessed (see Fig. 3.2). Finally, drilling of bone causes noise of an intensity that overwhelms the cochlea, prevents recording of ABRs during drilling, and alters ABRs recorded shortly after the cessation of drilling.

Physiologic factors that affect the entire ABR are interruption or vasospasm of the cochlear artery or avulsion of fascicles of the distal auditory nerve within the inner auditory canal. Both diminish or abolish wave I and all subsequent waves of the ABR and result in diminished hearing or deafness, respectively.

The intracranial portion of the auditory nerve can be affected by traction on either nerve or brainstem resulting in an increase in the latency between waves I and III. This change occurs only ipsilateral to the side of the injury. The degree of desynchronization of wave III reflects the severity of the insult, and frequently the rate of change of the potential is inversely related to reversibility, i.e., a signal that changes profoundly and rapidly is less likely to recover [56]. Similar changes can be caused by cold irrigation or heat from the cautery or drying of the auditory nerve [57, 58]. Application of papaverine to relieve vasospasm [59] or aggressive attempts to fill the subarachnoid space with irrigation fluid prior to dural closure may lead to ABR changes [60].

Damage to the brainstem either in the form of direct trauma or through compromise of blood supply or blood flow will be reflected in ABRs to the extent that the auditory pathway is involved. While persistent ABR changes nearly always predict brainstem dysfunction, damage to the brainstem may still occur even though ABRs remain unchanged. Many centers use ABRs together with other modalities such as SSEPs and motor-evoked potentials (MEPs) to monitor the integrity of the brainstem. Again, persistent changes in monitored signals typically predict new postoperative deficits, but unchanged signals do not rule out the potential for injury to the brainstem. That is because the monitored pathways only cover a small part of the cross-sectional area of the brainstem. Thus, multimodality monitoring has good specificity, but limited sensitivity for assessing brainstem integrity.

ABR Alarm Criteria

Typical alarm criteria for BAEP have been reported as a persistent decrease in the amplitude of wave V by more than 50% and/or a persistent absolute increase in latency of wave V greater than 0.5 ms. Typically, changes in more than two consecutive averaged trials are considered "persistent changes" [61, 62]. In other reports, wave V latency increases greater than 1 ms represented

a high risk for intraoperative auditory nerve damage [63]. Increased latency of wave III during cerebellar retraction during microvascular decompression surgery has been reported to be associated with other changes in the ABR and postoperative hearing loss [64]. Since the typical site of injury involves stretching of the intracranial cochlear nerve, latency changes in waves III and V can be approached similarly. Conversely, a total loss of the ABR should be approached differently, since both the peripheral, cochlear signal of wave I and the subsequent signals are lost. A loss can present a technical failure, e.g., failure to deliver a stimulus due to a dislodged or kinked insert; failure of sound conduction, e.g., blood in the middle ear; or a vascular injury due to stretch or spasm of the labyrinthine artery, which travels along the vestibulocochlear nerve. Persistent loss of the ABR portends postoperative hearing loss on the affected side [65, 66].

As for other modalities, the alarm criteria should take clinical context into account. A healthy auditory system, e.g., during a microvascular decompression, will tolerate more severe changes to the ABR than an auditory system already compromised by a cerebellopontine angle tumor. Likewise, the threat to the auditory system and by extension the interpretation of the cause for an alert will vary with the stage of the operation.

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