Pediatric Auditory Evoked Potentials: Principles and Clinical Practice

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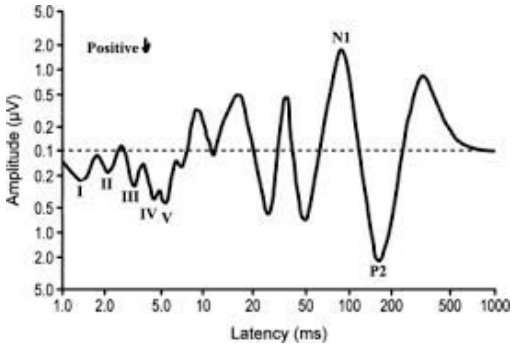
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inventis
Audiology & Balance

Why Acoustic Evoked Potentials Matter in Pediatrics

- AEPs are objective, non-invasive neurophysiological tests
- Critical for the hearing assessment in non-cooperative children
- Gold standard for neonatal hearing screening
- Essential for diagnosing retrocochlear pathology
- Valuable in the intraoperative monitoring
- Help to assess auditory maturation and development
- The webinar contains elements from the graduate course in Audiology, University of Ferrara.



Why Acoustic Evoked Potentials Matter in Pediatrics

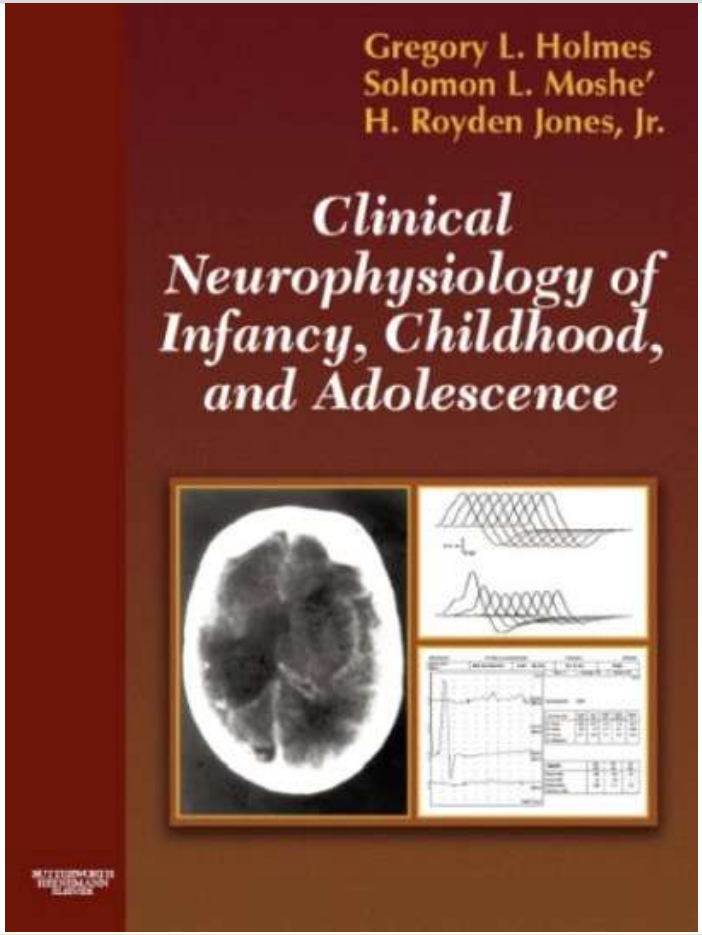
Example: Clinical Decision Making in Pediatric Neurophysiology

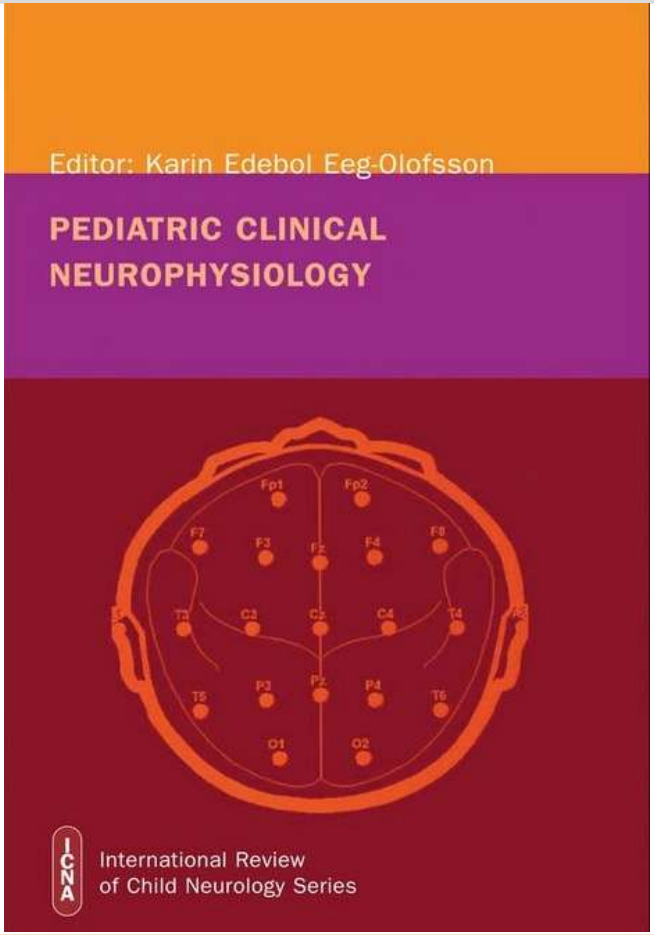
- 1–3 per 1,000 newborns have permanent hearing loss.
- AEPs detect up to 95% of hearing impairments early.
- Early intervention before 6 months optimizes speech and language outcomes
- UNHS programs rely heavily on AEP-based screening (AABR).
- REMEMBER: .....

Clinical Decision Making in Pediatric Neurophysiology

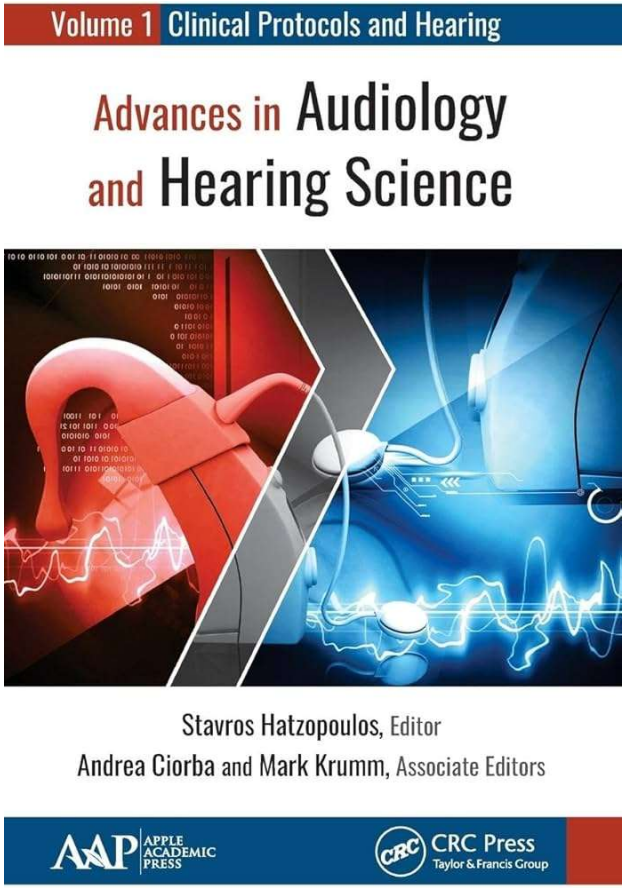
- REMEMBER: AEPs can help us assess the functional integrity of the Auditory pathways, when the pediatric patient is non-verbal, sedated or the previous Audiometric tests are inconclusive.

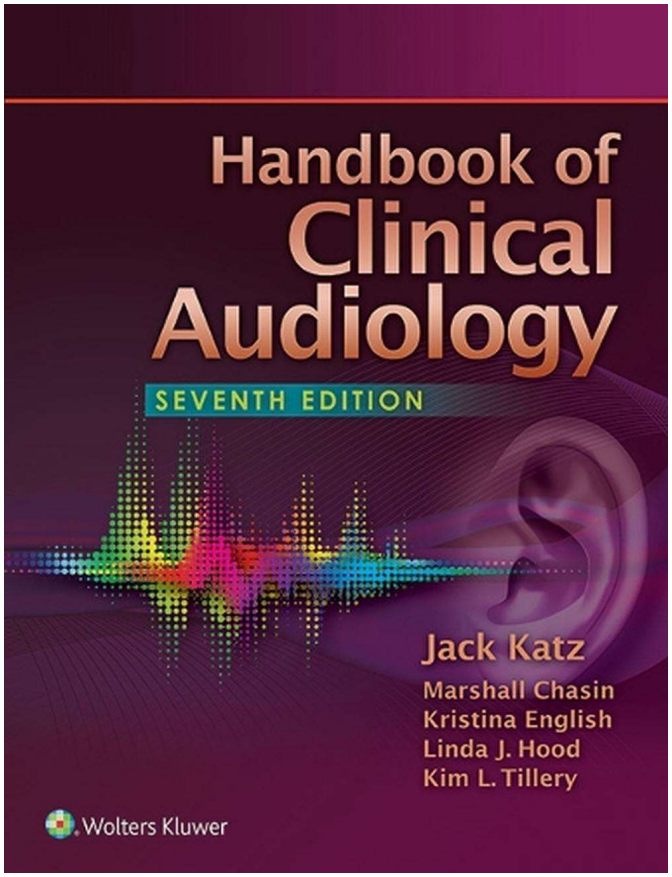
References – to have



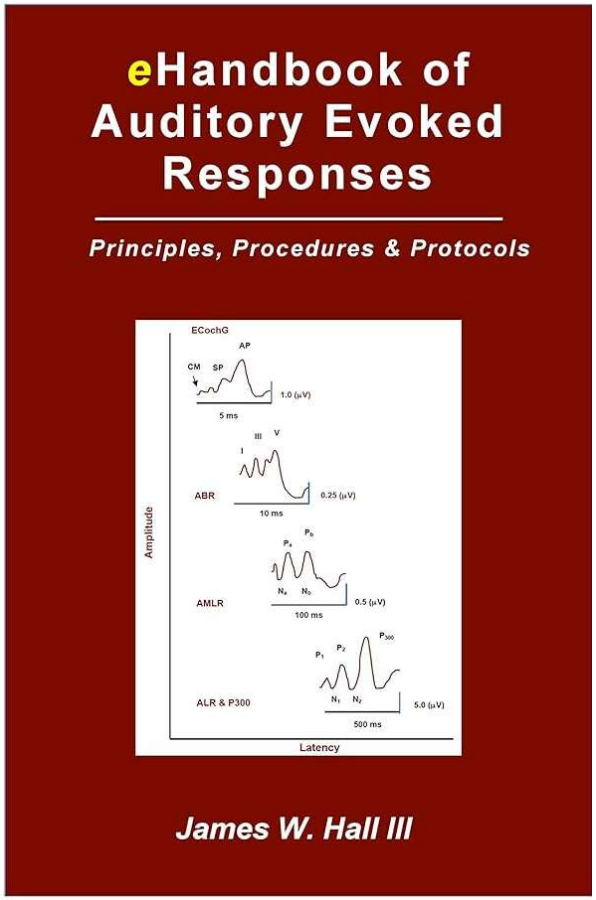


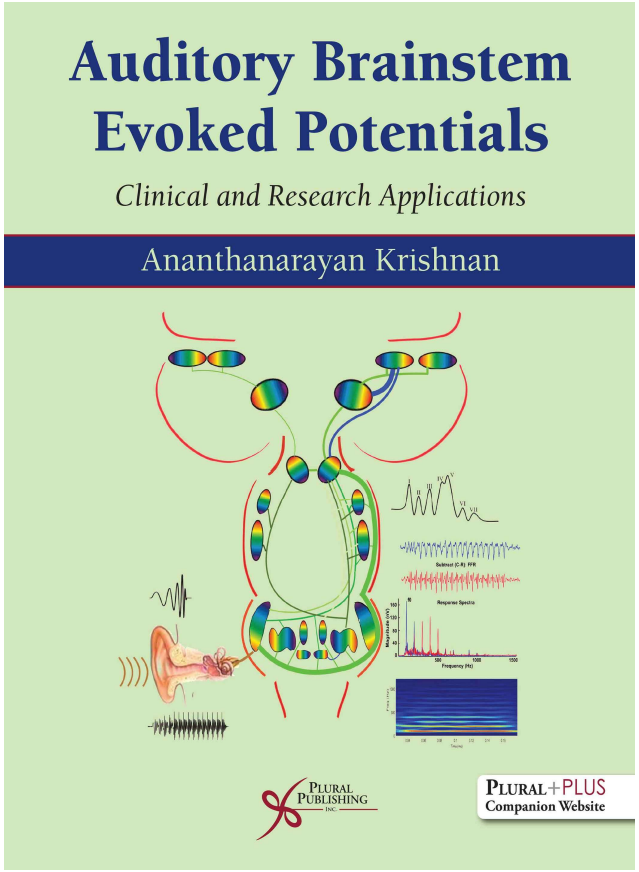
References – AEPs - cont



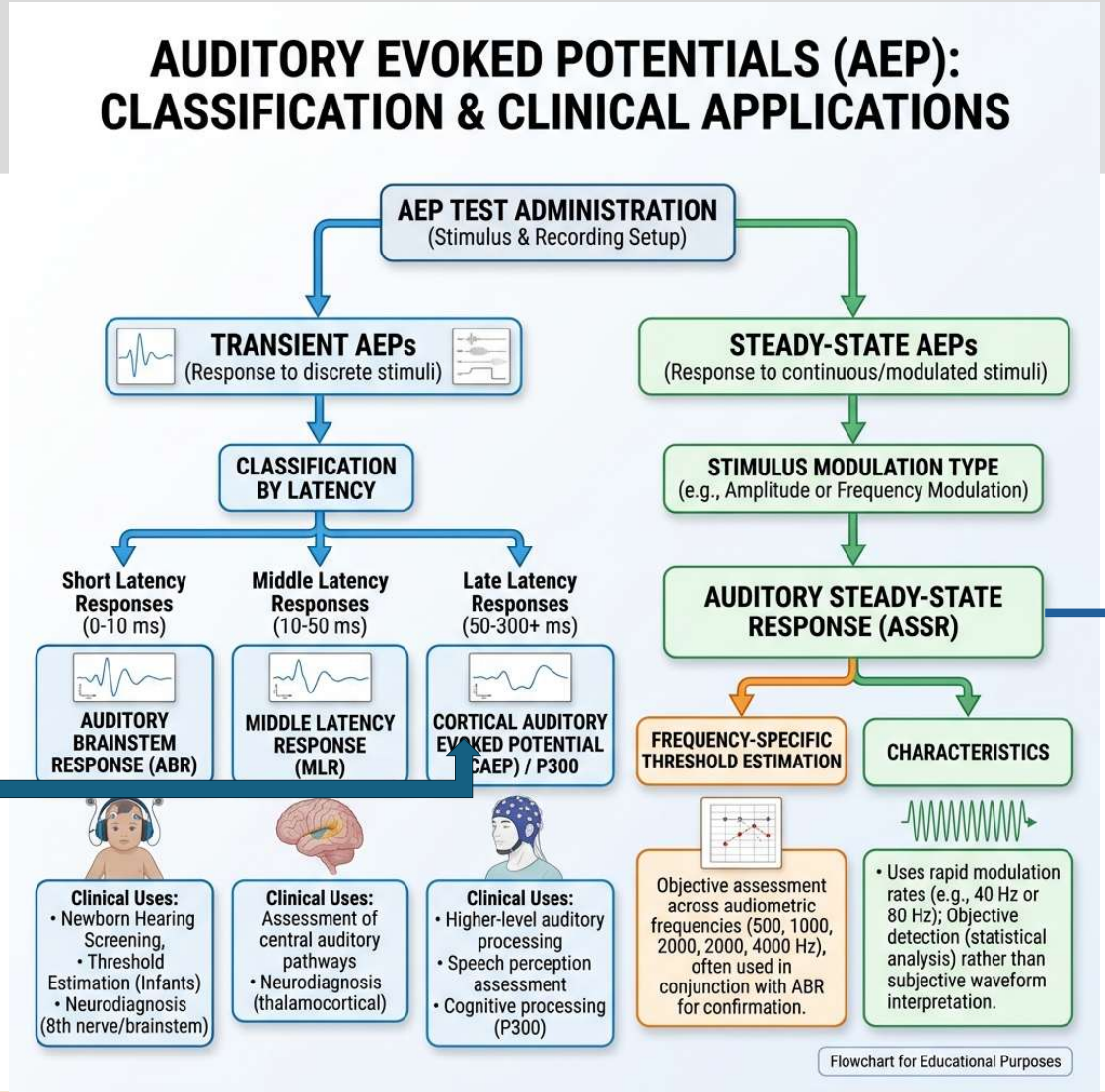


References – ABR - cont





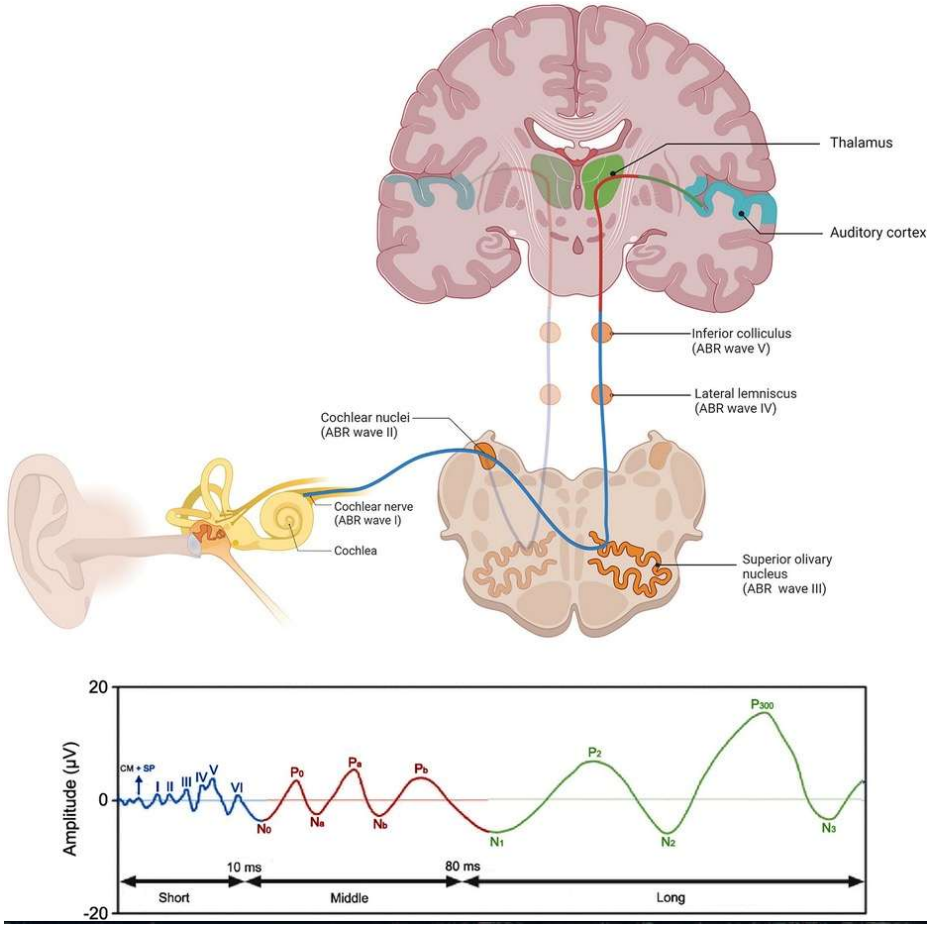
AEPs



Webinar On ACA In late 2026

Webinar On ASSR In 2027

The great Auditory Pathway – Transient AEPs



- The colors in the graph show the Location of the AEP generators.
- The AEPs can have short or long latencies.
- Cortical responses (P100 etc) start from approximately 100 ms.
- The Auditory Brainstem Response is a short latency AEP, within a window of 10-15 ms.
- The majority of the information of this webinar will focus on the ABR.

Section 1

Anatomy concepts & a historical perspective of AEPs

Section 1 : Sound Transduction Pathway





SECTION 1: Evoked Potentials (EPs)

- The Evoked Potentials are electrical signals (potentials = differential signals), generated by groups of neurons, in response to a stimulation of a sense organ (ear, eye etc.).
- These small signals are usually acquired by surface electrodes; they are amplified and then averaged, in order to estimate a coherent response.

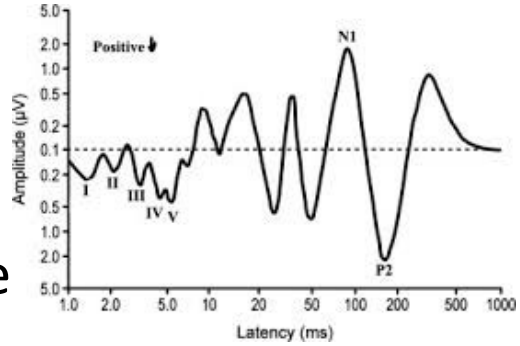
SECTION 1: Evoked Potentials (EPs)

1. Electrocochleography (ECochG). A technique used to measure electrical potentials generated in the inner ear (cochlea) and the auditory nerve in response to sound.
2. Auditory Brainstem Response (ABR), also referenced as Brainstem Auditory Evoked Response (BAER): this is the most widely used AEP in pediatrics. It measures the neural activity from the auditory nerve up through the Brainstem. The automated version AABR is used globally in Neonatal Hearing Screening
3. Auditory Steady-State Response (ASSR).An objective test that uses continuous, amplitude- and frequency-modulated tones to evoke a response. It is often used in conjunction with the ABR.

SECTION 1: Evoked Potentials (EPs)

4. Middle Latency Response (MLR). Occurs after the ABR and before the CAEP, reflecting activity in the thalamus and primary auditory cortex. It is not used very often in clinical practise.

5. Cortical Auditory Evoked Potentials (CAEP). Also known as Late Latency Responses (LLR), these measure the responses from the auditory cortex. Unlike ABRs, CAEPs are highly dependent on the patient's state of arousal (the patient must be awake)



SECTION 1: Evoked Potentials (EPs)

AEP Type	Primary Pediatric Use	Patient State Required
aABR	Newborn hearing screening (Pass/Refer)	Sleeping / Quiet
ABR	Threshold estimation, ANSD diagnosis	Sleeping (natural or sedated)
ASSR	Frequency-specific threshold estimation (severe/profound)	Sleeping (natural or sedated)
ECochG	Cochlear function, ANSD diagnosis	Sleeping (often sedated/surgical)
MLR	Central auditory processing (older children)	Awake and quiet
CAEP	Hearing device validation, cortical maturation	Awake and alert

2027 webinar

Detailed Coverage in **another 2026 Webinar**

Section 1 : How AEPs are Generated & Recorded

- Far-field recording of synchronous neural activity
- Electrode impedance requirements (<3 kΩ; interelectrode <2 kΩ)
- Signal-to-noise ratio (SNR) and averaging principles
- Differential amplification and common mode (ground)
- Signal averaging to extract AEP from background EEG noise

Section 1 : Latencies & generators of AEPs

AEP Type	Latency	Generator	Clinical Use
ECochG	<2 ms	Cochlea/AN	ANSD
ABR	1–10 ms	Brainstem	Threshold, neuro
AMLR	10–50 ms	Thalamus/Cortex	Cortical processing
ALR (CAEP)	50–300 ms	Cortex	Cortical thresholds
P300	~300 ms	Cortex	Cognitive function

Remember :

ANSD = Auditory Neuropathy & Spectrum Disorder

AMLR = Auditory Middle Latency Response

ALR = Auditory Late Response (P100)

While both are evoked potentials, the AMLR (10–50 ms) bridges the gap between brainstem (ABR) and cortical responses, whereas the ALR (60–500 ms) is a strictly cortical response reflecting higher-order processing.

Section 1A

Auditory Brainstem Response

ABR Nomenclature

ABR: Auditory Brainstem Response (Audiology version)

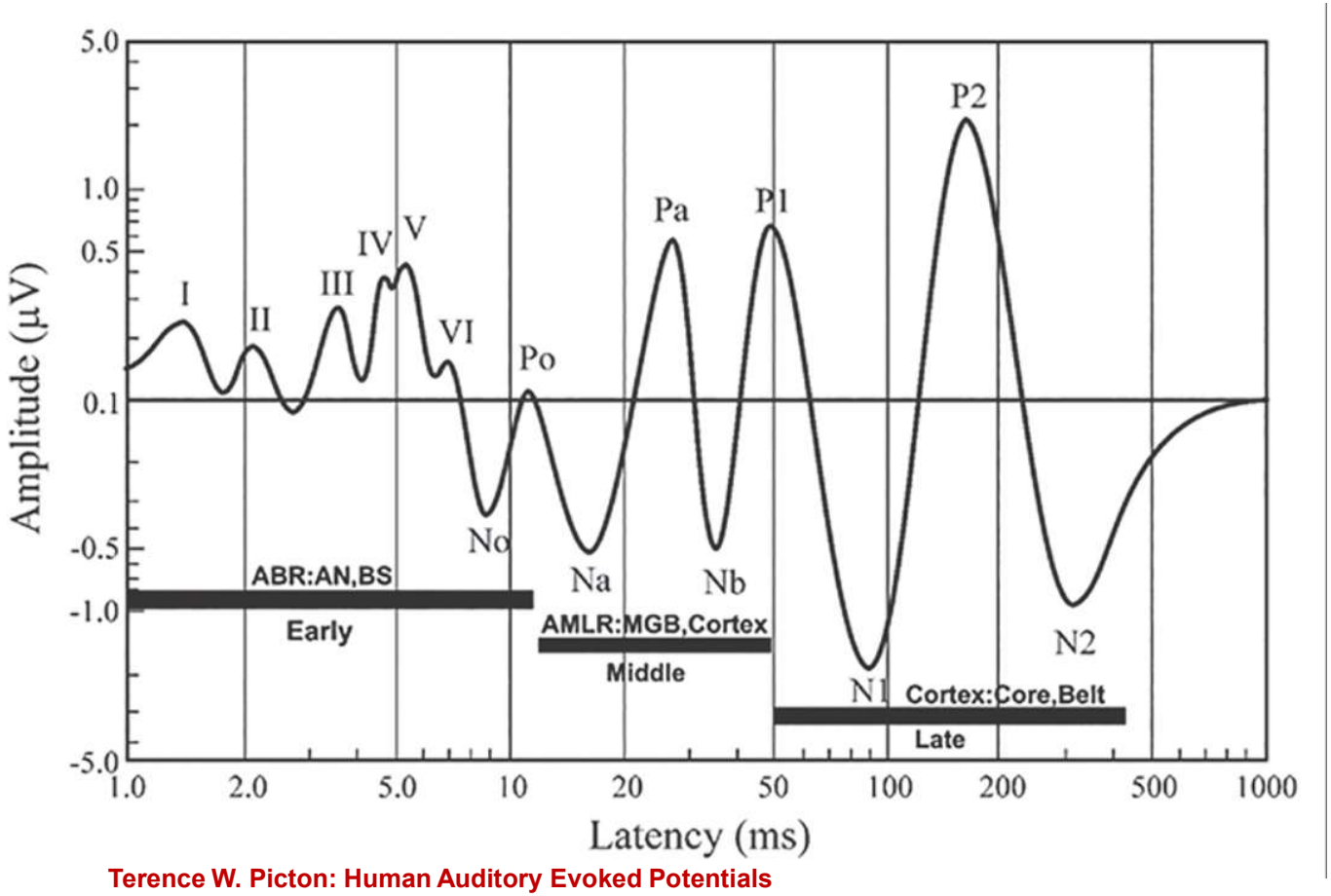
BAER: Brainstem Auditory Evoked Response (Neuroscience version).

BAEPs: Brainstem Auditory Evoked Potentials (Neuroscience)

Section 1 : What Happens upon stimulation

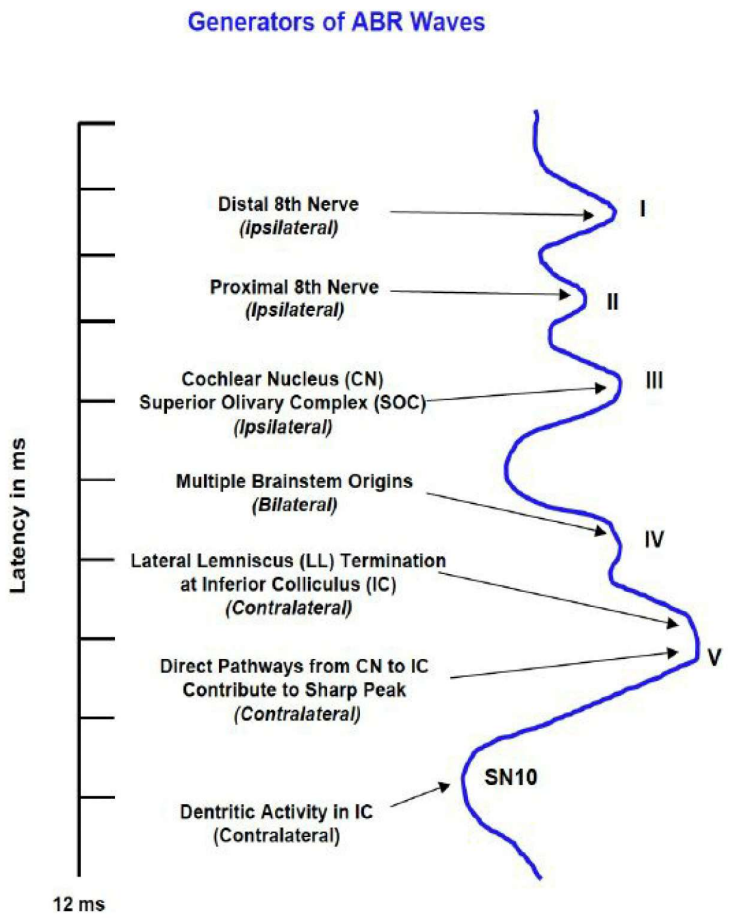
- The ABR evoked with highly transient stimuli (clicks) is used for rapid, broadband assessment of neurological integrity. It cannot be used to define hearing sensitivity or cochlear status at audiometric frequencies or for specific frequency regions.
- Tone bursts and chirps are necessary in adult / pediatric applications of the ABR.

Section 1 : The Overall AEP Responses



Terence W. Picton: Human Auditory Evoked Potentials

Section 1 : The ABR Generators (over-simplified)



Wave	Generator	Anatomical Location
Wave I	Distal CN VIII	Auditory nerve (cochlear end)
Wave II	Proximal CN VIII	Auditory nerve (brainstem entry)
Wave III	Cochlear Nucleus	Pontomedullary junction
Wave IV	Superior Olivary Complex	Lower pons
Wave V	Lateral Lemniscus / Inferior Colliculus	Upper pons/midbrain

James Hall III : Handbook of Auditory Evoked Responses

Section 1 : What Happens upon a SPECIFIC stimulation

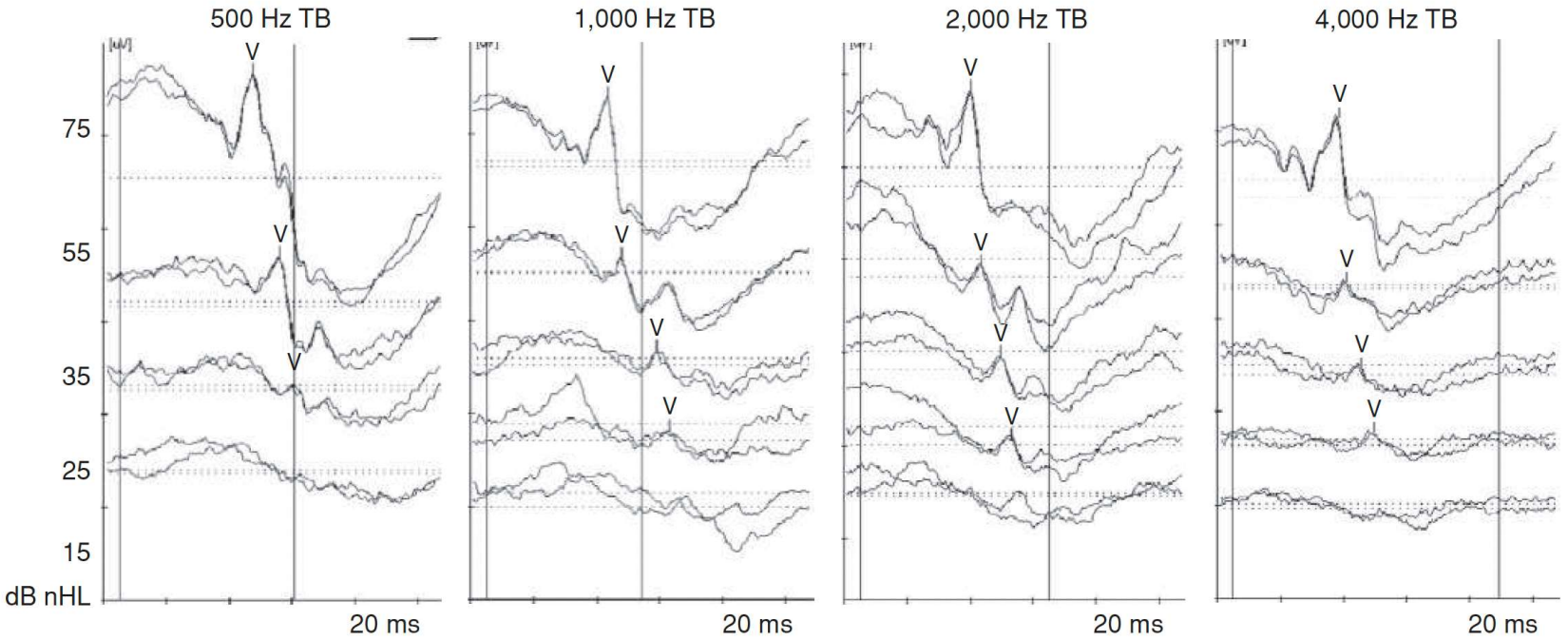


FIGURE 14.2 ABRs to tone bursts centered at 500, 1,000, 2,000, and 4,000 Hz recorded from an individual with normal hearing. The latencies of Wave V at 75 dB nHL are 8.53 ms for a 500-Hz tone burst, 7.70 ms for a 1,000-Hz tone burst, 7.03 ms for a 2,000-Hz tone burst, and 6.53 ms for a 4,000-Hz tone burst, demonstrating the latency decrease as center frequency of the tone burst increases.

Linda Hood –Handbook of Clinical Audiology – Chapter 14

Section 2

The Developmental CLOCK

Section 2 : The "Developmental" Clock

- The Cochlear functionally is mature by ~26 weeks of gestation
- The Myelination of auditory pathways continues until ~18–24 months
- The Synaptic refinement is ongoing through childhood
- The ABR latencies progressively shorten with maturation of Wave V, which reaches adult values by ~18–24 months
- Wave I reaches adult values earlier (~3–6 months)

Section 2 : ABR maturation within various age groups

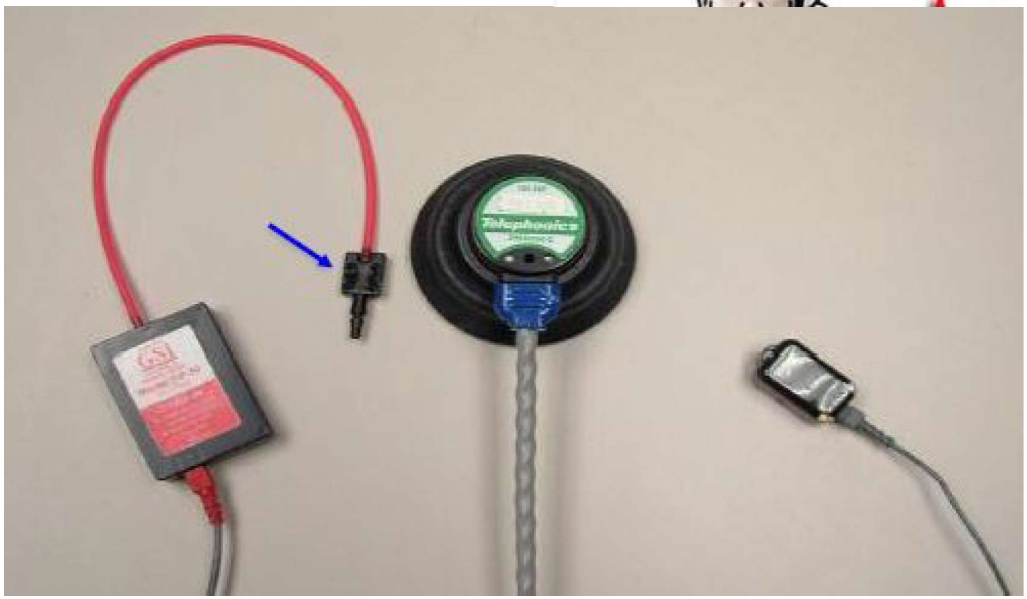
Age	Wave I (ms)	Wave III (ms)	Wave V (ms)	I–V IPL (ms)
28 wk GA	~2.0	~5.5	~8.0	~6.0
34 wk GA	~1.9	~5.0	~7.5	~5.6
Term (40 wk)	~1.7	~4.5	~7.0	~5.2
3 months	~1.6	~4.2	~6.5	~4.8
6 months	~1.6	~4.0	~6.2	~4.5
12 months	~1.6	~3.9	~6.0	~4.3
18–24 months	~1.5	~3.8	~5.8	~4.1
Adult	~1.5	~3.7	~5.6	~4.0

Section 3: FOCUS on ABR

Stimulus parameters, acquisition parameters and Electode-montage in Infants

Section 3 : ABR Transducer types



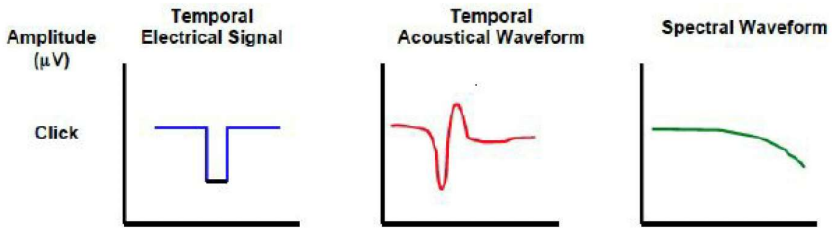


Left to Right:
Insert, disc electrode,
bone conduction oscillator.

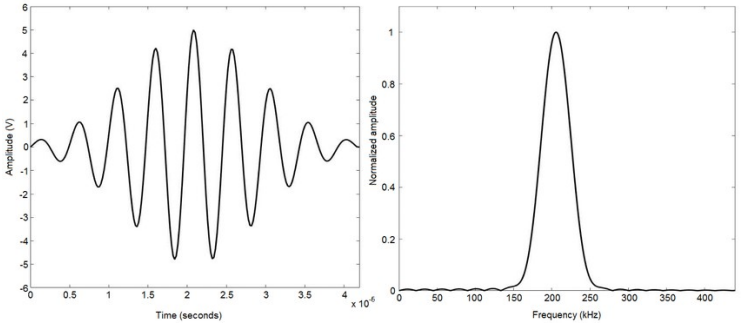
For the ABR acquisition we use an insert for the acoustic stimulation and disc electrodes for the signal recording.

Section 3 : Common Stimuli (air conduction)

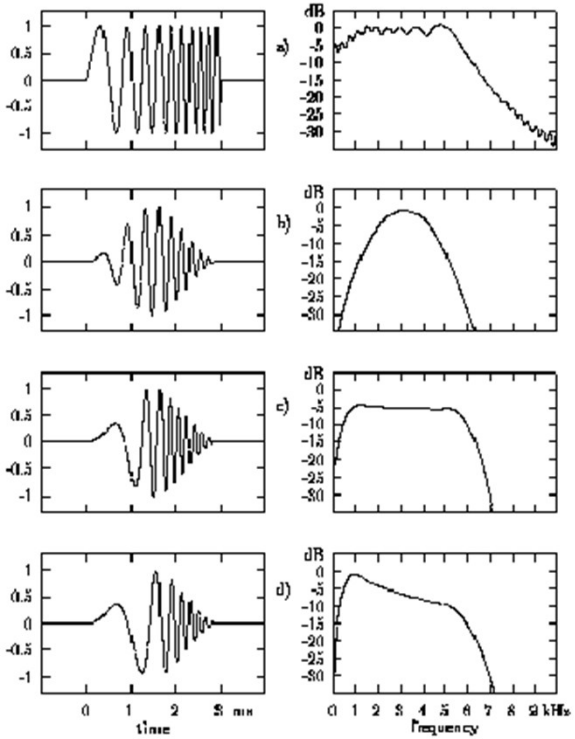
Clicks
(max 2 KHz)



Tone-bursts
(max = Fc)

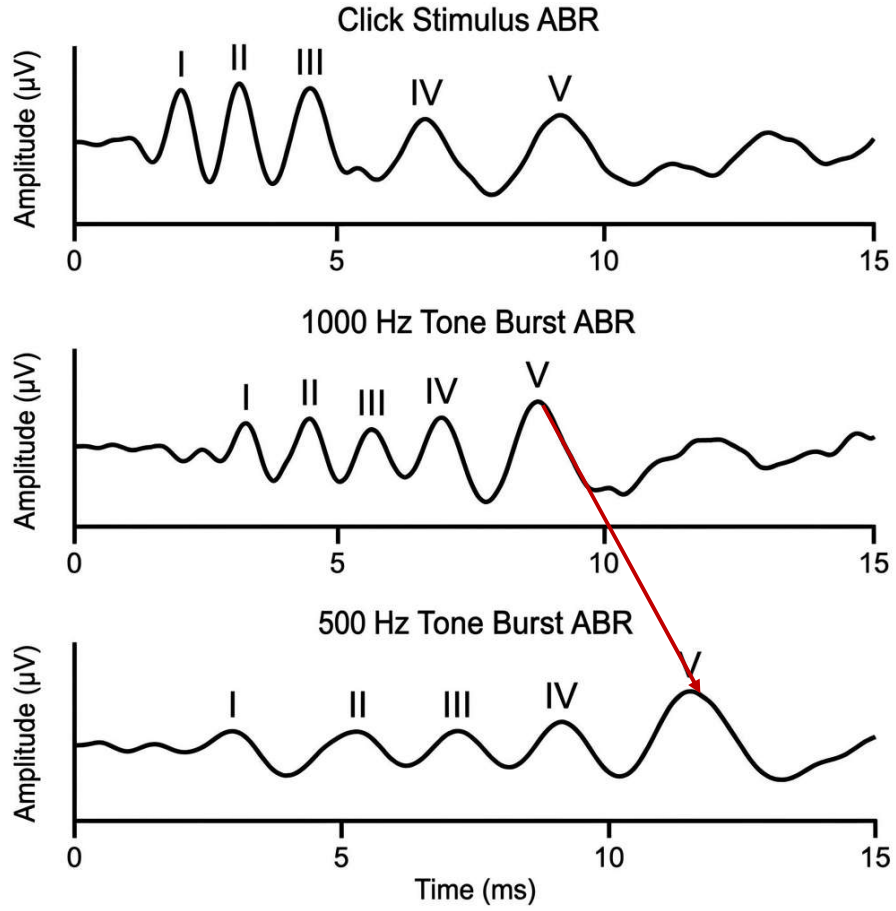


Chirp types



Obviously with different stimuli we record relatively DIFFERENT ABR morphologies !!!!

Section 3 : Common Stimuli (air conduction)

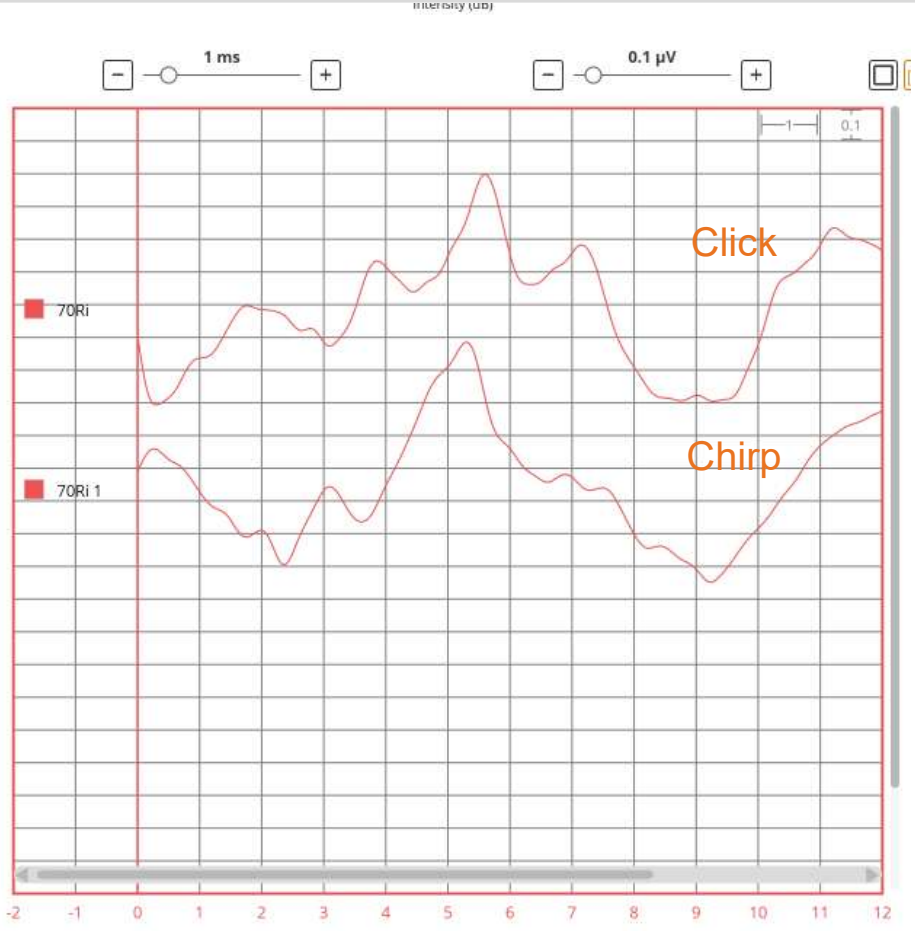


NOTE

With different stimuli

we record relatively DIFFERENT ABR morphologies !!!!

Section 3 : Chirp vs Click stimuli



With chirp stimuli wave V registers as a much larger response. often as a doubling in amplitude. This offers a number of clinical advantages including:

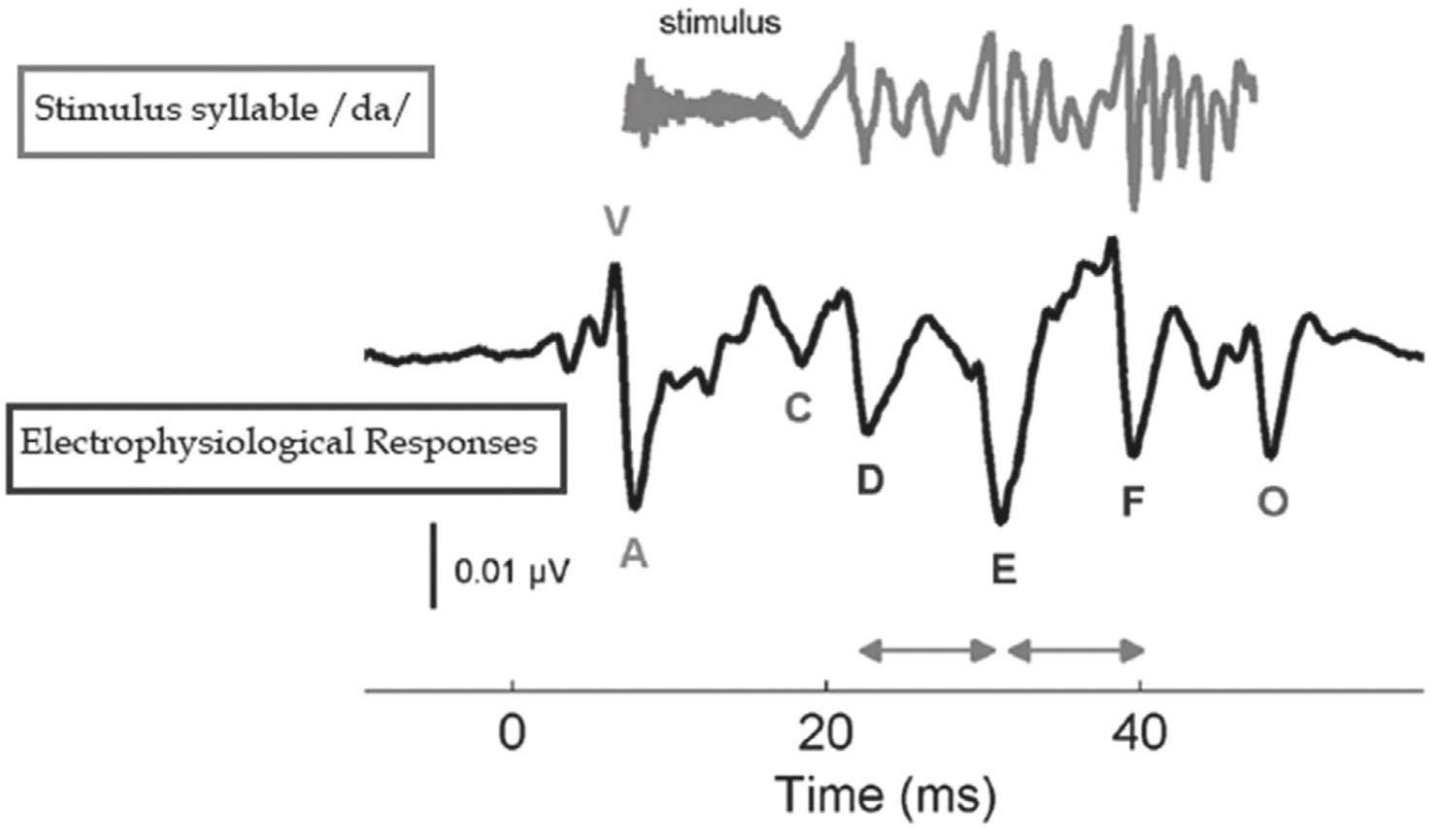
1)a more confident identification of wave V near the minimum response level or threshold;

2) the detection of an ABR at lower intensity levels for presumably more accurate estimations of thresholds; and

3) a decreased ABR acquisition time. The latter is crucial in the Pediatric ABR applications.

Section 3 : A new type of stimulation

Speech Evoked ABR (Frequency Following Response)



This is very similar to the registration of Cortical AEPs BUT at smaller latencies

Sanfins & Hatzopoulos, IJPORL 90,(2016) 12-19

Section 3 : Technical Issues

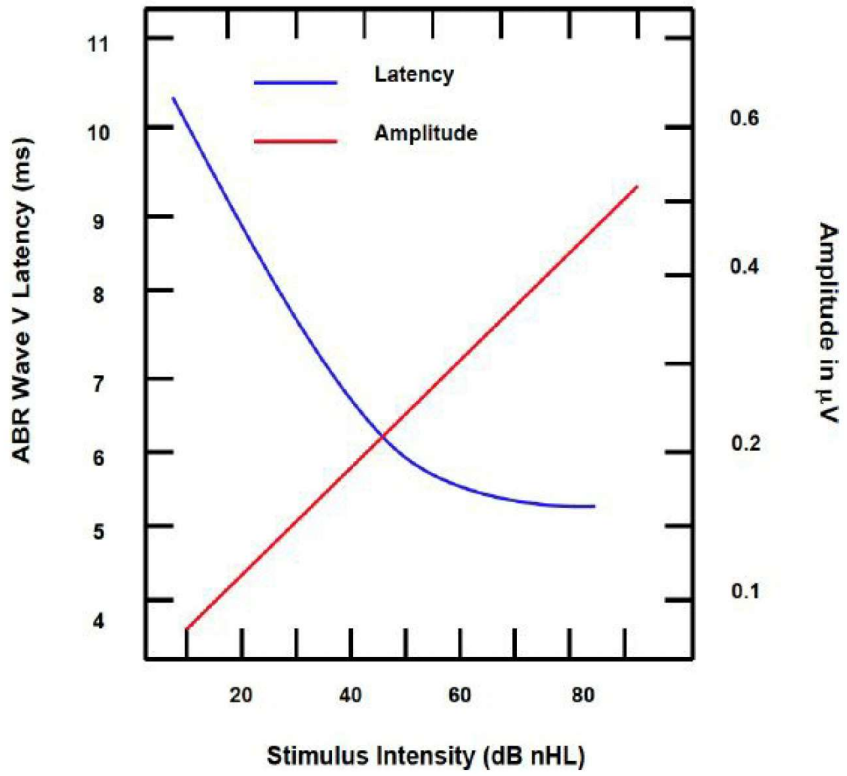
Factors & Parameters affecting the ABR Recording

Stimulus Factors	Recording Factors	Patient Factors
Intensity	Electrode montage	Age
Rate	Filter settings	Gender
Polarity	Time window	Medications
Duration/rise time	Number of channels	Attention
Frequency	Number of sweeps	Body temperature
Monaural/binaural		Muscle artifact

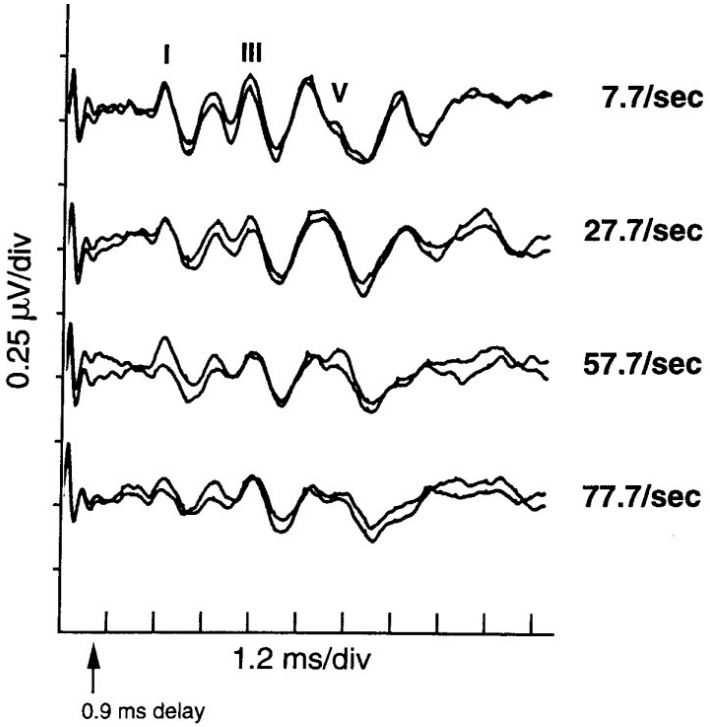
These issues have been covered in detaild in my previous Webinar on ABR. See ALSO the APPENDIX slides

Section 3 : Stimulus Intensity

- As the stimulus intensity ↑
  - Latencies ↓
  - Amplitudes ↑
- The latency changes occur slowly for intensities from 90 to 60 dBnHL and then they increase more rapidly at lower intensity levels.



Section 3 : The stimulation rate



Terence W. Picton: Human Auditory Evoked Potentials

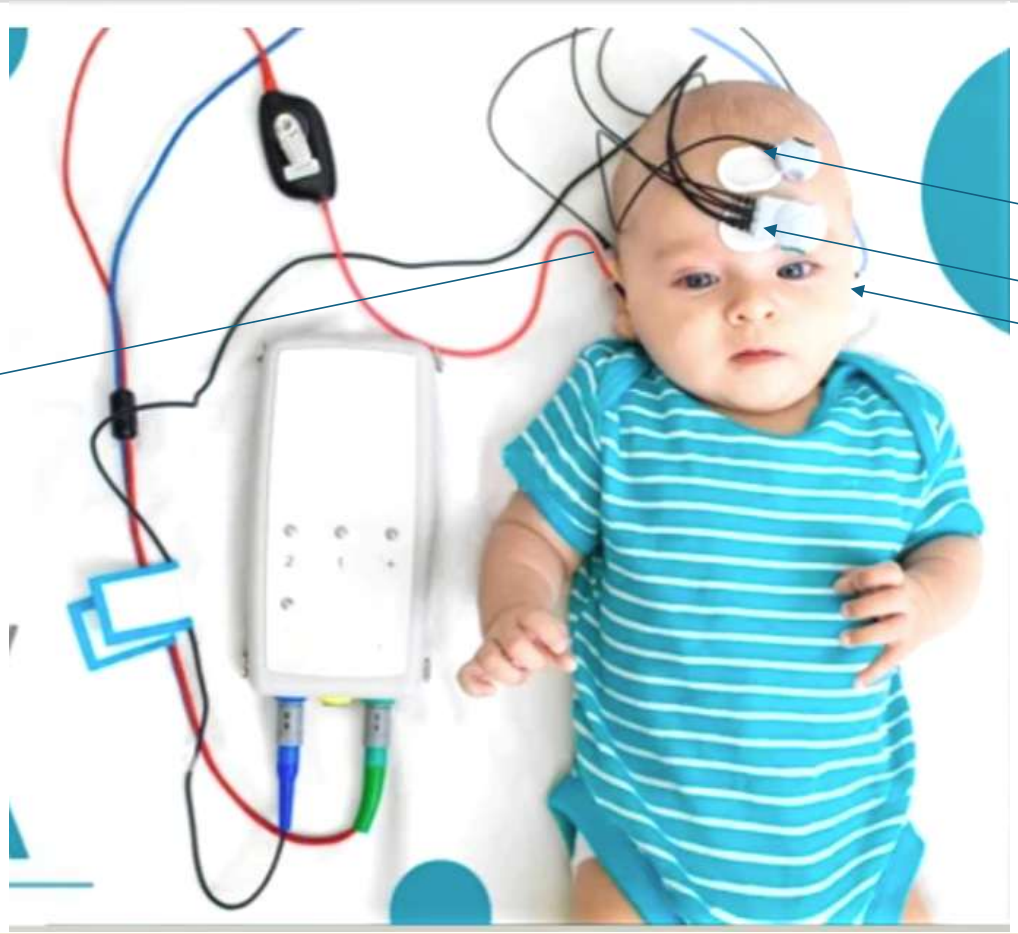
High stimulus rates affect the wave latencies AND the Inter-wave latency differences.

Lets recall that the *stimulus rate has a more pronounced influence on:*

- ABR latency for premature than term neonates.
- For younger children under age 18 months than for older children
- For older children up to age 13 years than adults

Section 3 : Electrode Montage-infants

In the photo you see the two stimulus inserts at the Left & Right ear.



1 channel system (Rx)

Positive (high forehead)

Ground (low forehead)

Negative (Mastoid)

Section 3 : Electrode Montage- AI model creation



Code for from use.AI (subscription)

An image showing the position of 3 small disposable ABR electrodes on a realistic infant as follows: A Red electrode at the center of the forehead , below the hairline. Another Black electrode placed at the lobe of right ear. And another Green electrode placed at the center of the lower forehead, above the bridge of the nose, under the red electrode.
Style: Realistic
Aspect ratio: 1:1 (Square)

Took me 15 min to get the right image

Section 3 : Electrode Montage- AI model creation



Code for use from nanobananaimg.com (subscription)

An image showing the position of 3 small disposable ABR electrodes on a realistic infant as follows: A Red electrode at the center of the forehead , below the hairline. Another Black electrode placed at the lobe of right ear. And another Green electrode placed at the center of the lower forehead, above the bridge of the nose, under the red electrode.
Style: Realistic
Aspect ratio: 1:1 (Square)

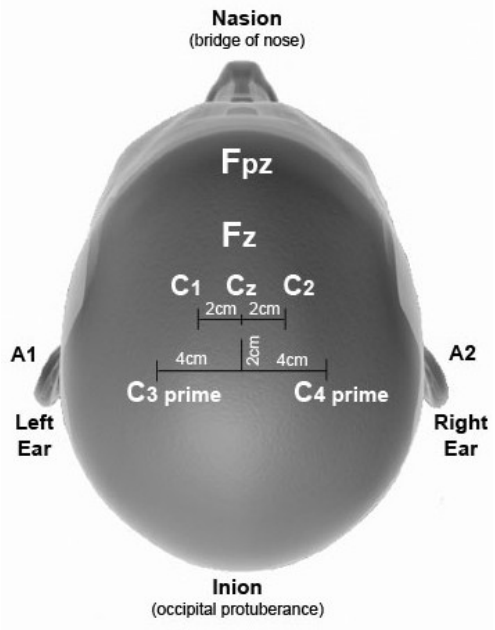
Took me 1 min to get the right image BUT

Can you figure out what is wrong with this picture ??

Section 3 : Electrode Montage 2 channel

- Reccomended electrode placement for most conditions:
  - Cz (vertex); Positive or NON-inverting electrode
  - A1 Lt ear, A2 Rt ear: Negative or INVERTING electrode

Fpz (ground): can be put anywhere on the body, usually on the forehead, above the nose.



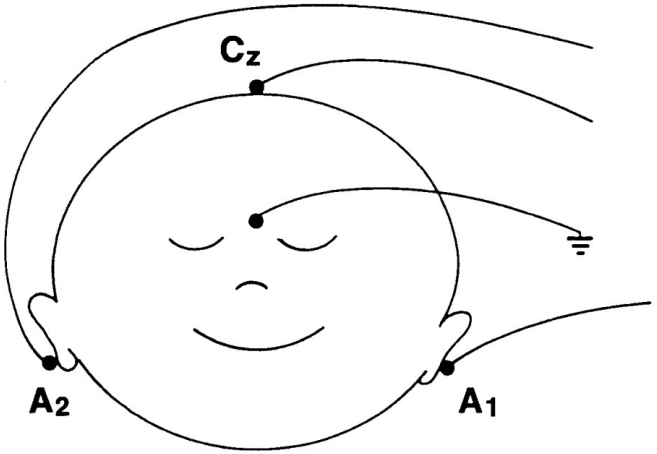
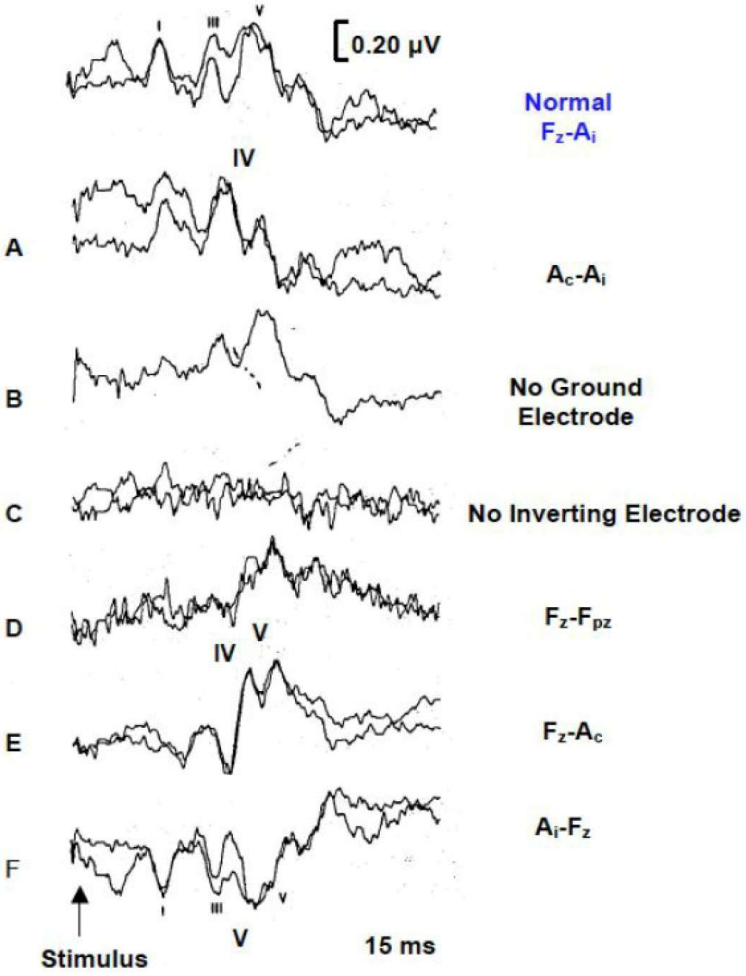


Figure 3-6. Electrode positions used for a two-channel ABR recording.

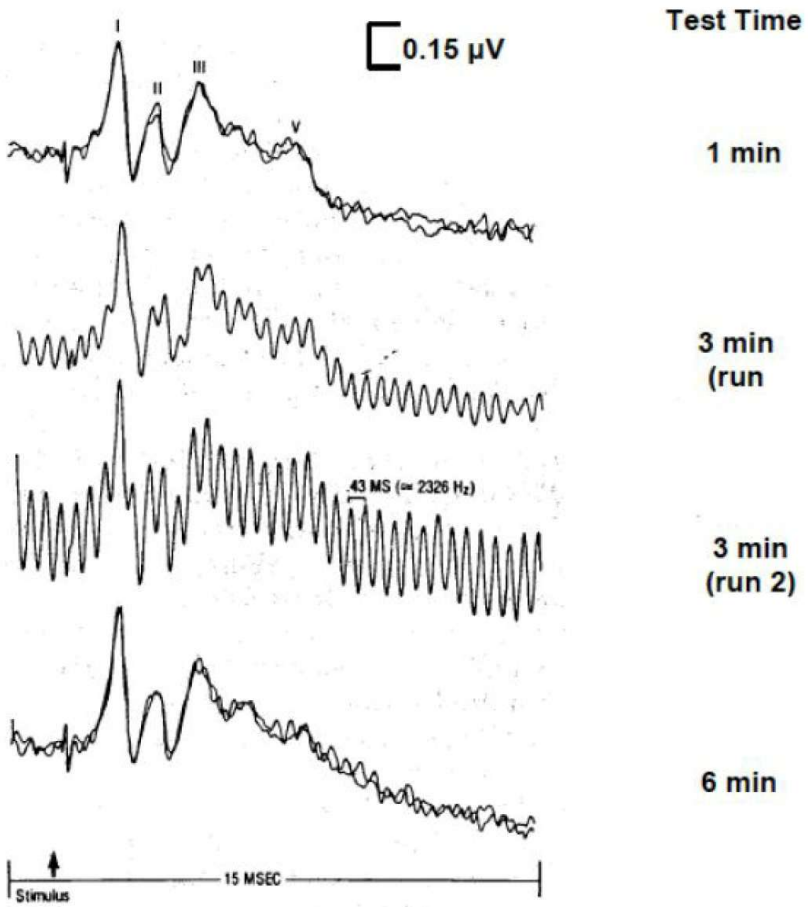
Section 4

Analysis of the ABR response (conventional responses, abnormal ABR patterns, non -pathologic subject factors, the effect of drugs on the ABR morphology)

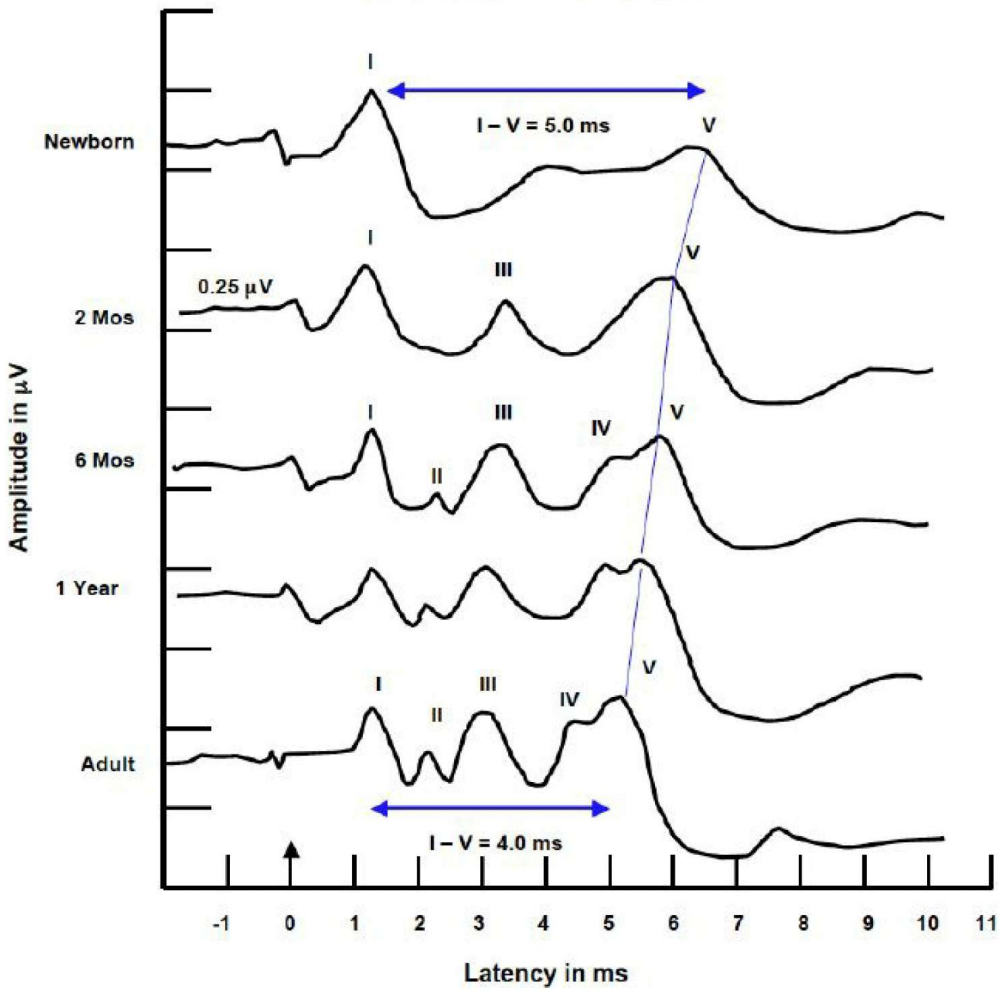
Section 4 : Abnormal ABR patterns. Electrode issues



Electrical Artifact over time



Section 4 : Abnormal ABR patterns. Maturation



Section 4 : Anestesia & Sedatives

- Anesthetic agents, sedatives, potentially ototoxic drugs, and selected other medications must be considered in the analysis of ABR findings for all patients.
- The effect of drugs on ABR latency or amplitude can interact with and add to the effects of other factors.
- Anesthetic agents and sedatives influence the ABR less than cortical auditory evoked responses. None of the medications entirely suppress the ABR.
- Research confirms that some anesthetic agents and sedatives have statistically significant effects on ABR latency or amplitude, which are not clinically significant.

Section 4 : Why sedate (EchoG, ABR only)

- Children >6 months are increasingly unable to remain still.
- Any movement artifact degrades the AEP signal quality.
- Natural sleep is unreliable after ~4–6 months.
- The necessity to apply lengthy diagnostic protocols (60–90 min).
- The necessity to use multiple tests in one session (ABR + OAE + tympanometry + ASSR)

Section 4 : Why sedate (EchoG, ABR only)

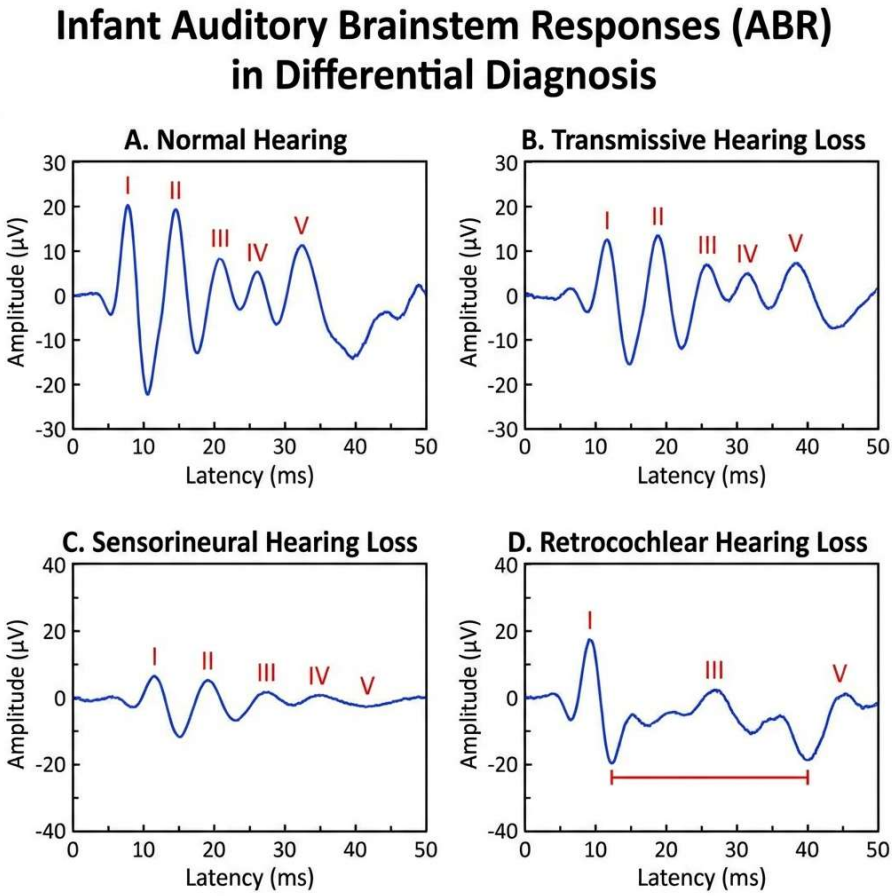
Method	Agent	Route	Age Suitability	Onset	Duration
Natural sleep	—	—	0–4 months	—	Variable
Sleep deprivation	—	—	4–18 months	—	Variable
Chloral hydrate	50–100 mg/kg	Oral/PR	6 mo – 3 yrs	30–60 min	60–120 min
Midazolam	0.3–0.5 mg/kg	Oral/IN	>6 months	15–30 min	45–90 min
Dexmed(etomidine)	1–2 µg/kg	IV/IN	All ages	10–15 min	60–90 min
Propofol/Ketamine	Variable	IV	All ages (supervised)	Rapid	Variable
General anesthesia	Multiple	IV/inhaled	Complex/uncooperative	Rapid	Controlled

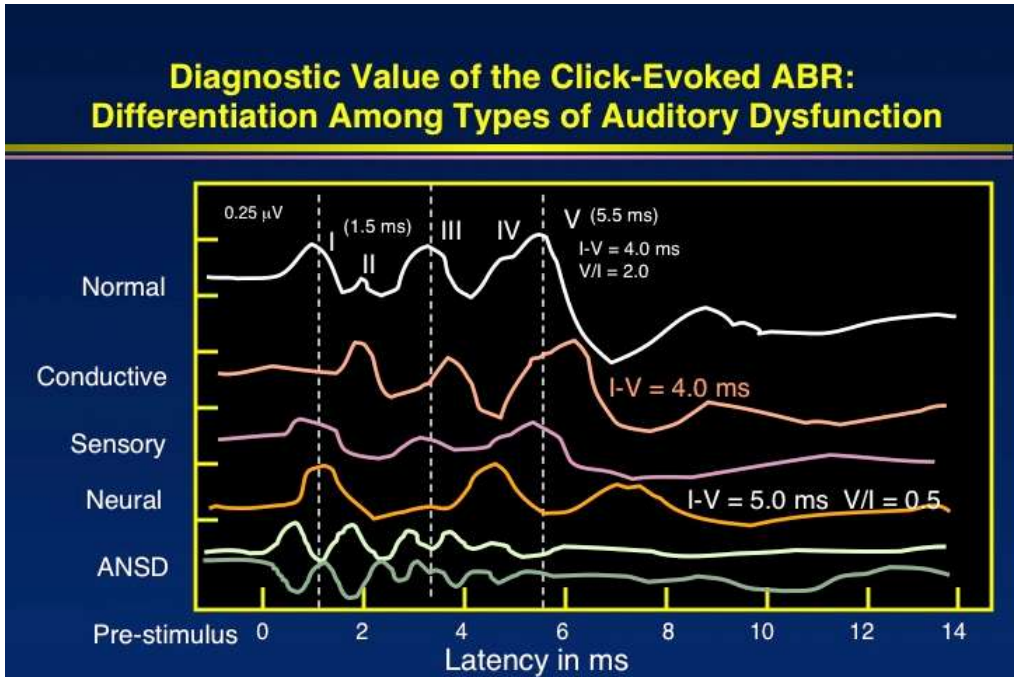
PR = per Rectum (rare), IN = Intra Nasal, IV = Intra Venus

Section 5

Differentiation of HL, Hearing Screening and a short exposure of other clinical applications

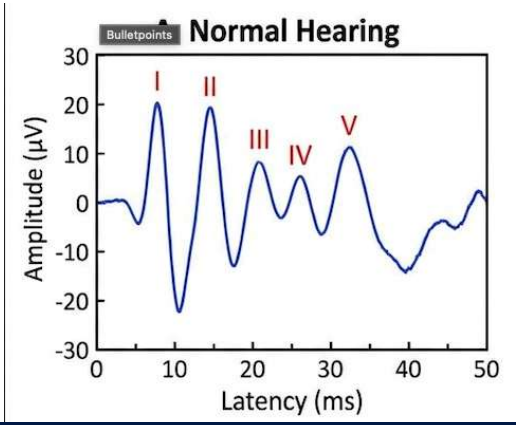
Section 5 : Differentiation of HL

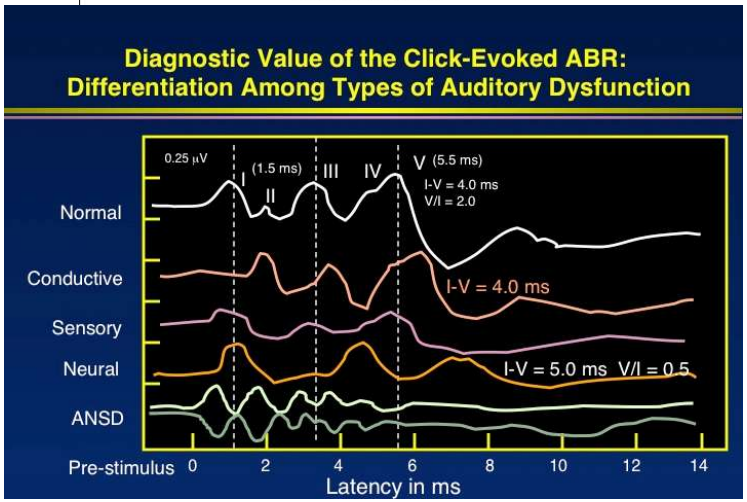




Section 5 : Interpretation of Pediatric ABR, NORMAL children

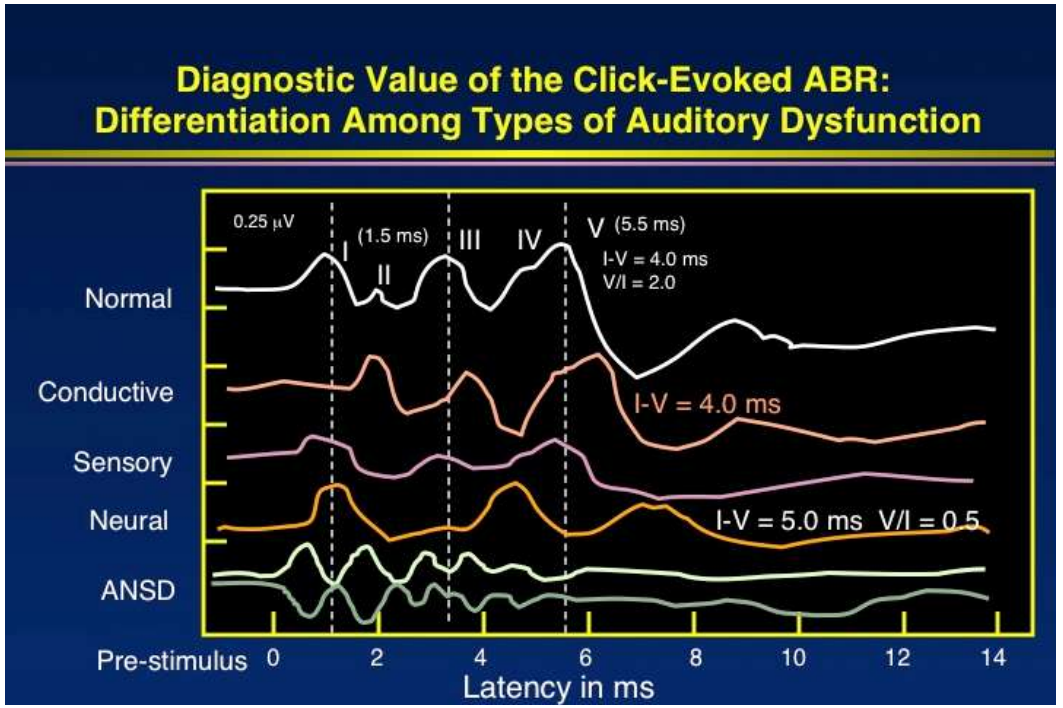
- Wave V clearly identifiable at ≤35 dB nHL (older infants /children)
- Age-appropriate absolute and interpeak latencies
- Wave V / I amplitude ratio ≥0.5 (1.6 -2.2)
- Reproducibility: two waveforms superimposed
- Consistent morphology across replications





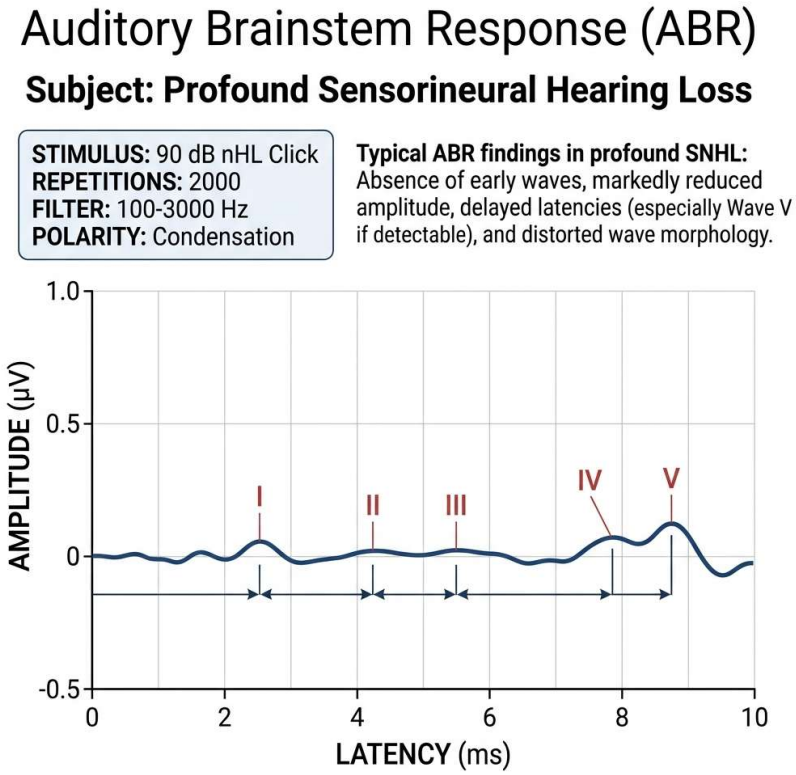
Section 5 : Interpretation of Pediatric ABR, children with a Conductive HL

- Elevated thresholds by air conduction
- Normal bone conduction thresholds
- All waves delayed by equal amount (parallel shift)
- Normal IPLs (I–V within normal limits)
- Common causes: OME, atresia, ossicular anomalies



Section 5 : Interpretation of Pediatric ABR, children with SNHL

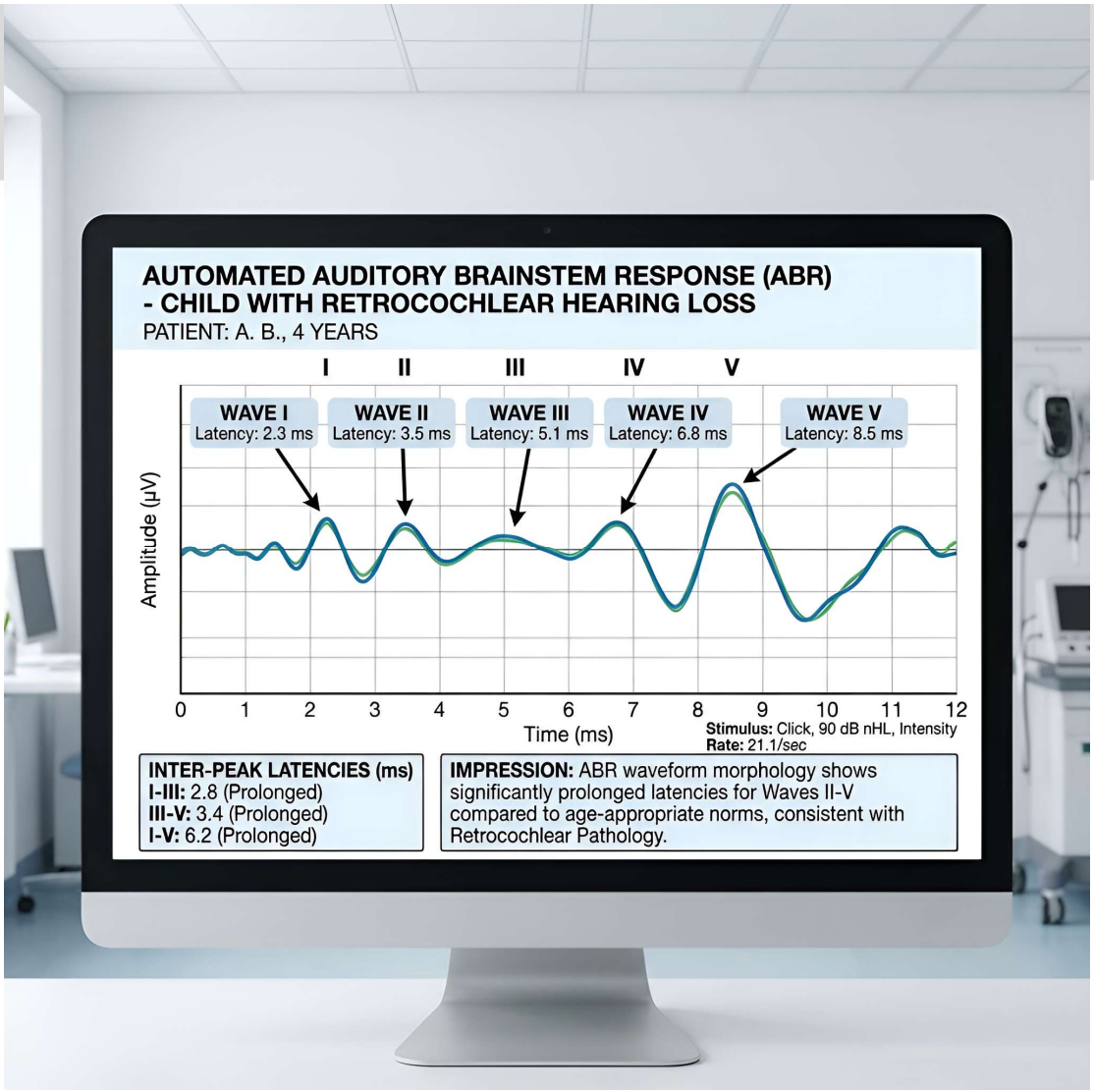
- Absence of early waves or waves present with reduced amplitude.
- Both AC and BC thresholds elevated
- IPLs typically normal (peripheral lesion)
- Wave V morphology may be broad / poorly defined in high-frequency SNHL
- Audiogram estimation using TB-ABR

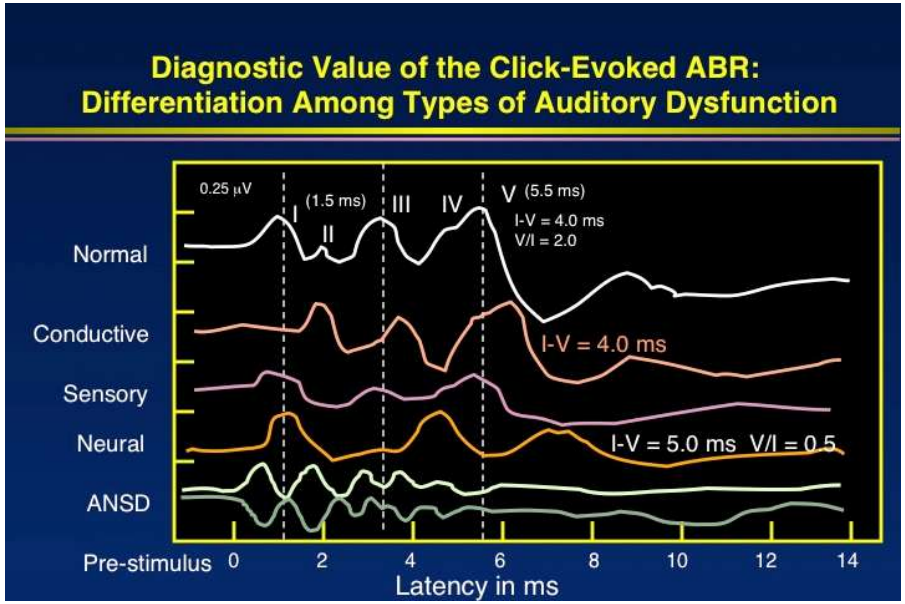


Section 5 : Retrocochlear Patterns (RARE)

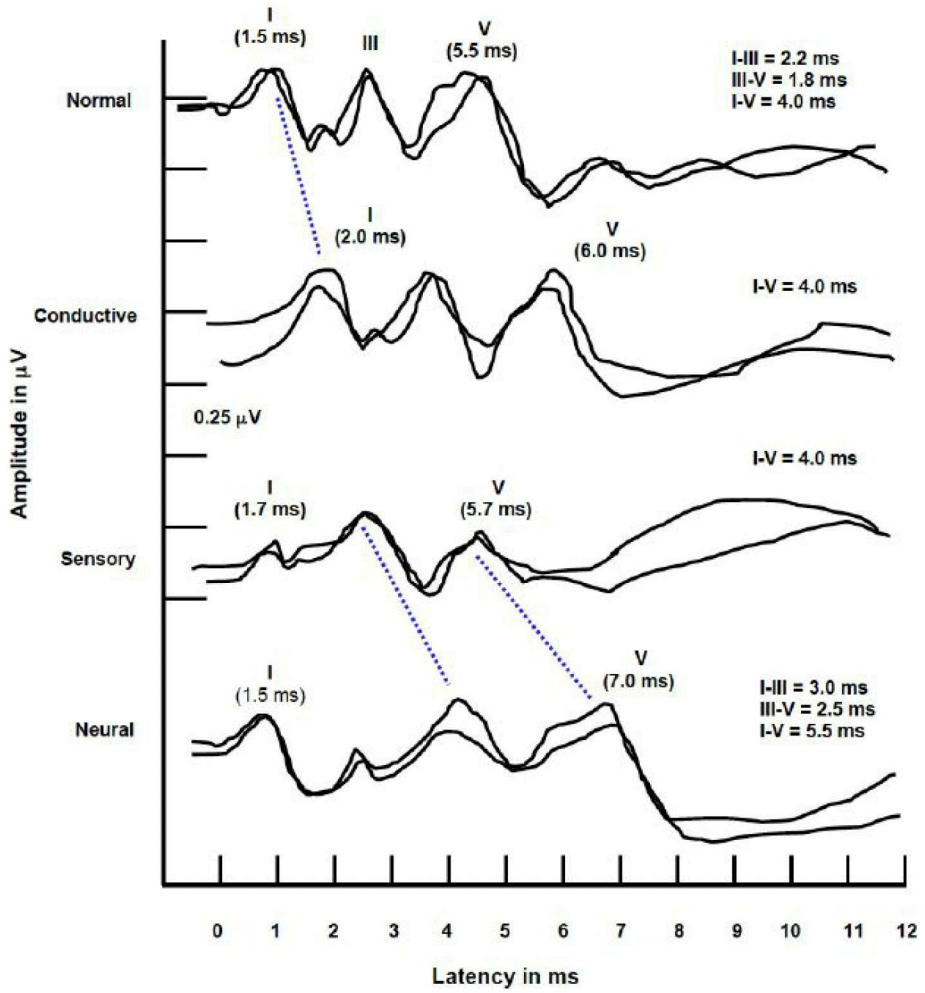
- Prolonged IPLs (inter-Peak Latencies) with normal Wave I
- I–III prolonged: low Brainstem lesion
- III–V prolonged: upper Brainstem lesion
- I–V prolonged: generalized demyelination (Multiple Sclerosis)
- Absent / poorly formed Waves III–V with present Wave I
- Wave V / I amplitude ratio < 0.5
- Conditions: hypoxic-ischemic injury, hydrocephalus, tumors etc

See NEXT Slide


AUTOMATED AUDITORY BRAINSTEM RESPONSE (ABR)
- CHILD WITH RETROCOCHLEAR HEARING LOSS
PATIENT: A. B., 4 YEARS
I
II
III
IV
V
WAVE I
Latency: 2.3 ms
WAVE II
Latency: 3.5 ms
WAVE III
Latency: 5.1 ms
WAVE IV
Latency: 6.8 ms
WAVE V
Latency: 8.5 ms
Amplitude (μV)
0 1 2 3 4 5 6 7 8 9 10 11 12
Time (ms)
Stimulus: Click, 90 dB nHL, Intensity
Rate: 21.1/sec
INTER-PEAK LATENCIES (ms)
I-III: 2.8 (Prolonged)
III-V: 3.4 (Prolonged)
I-V: 6.2 (Prolonged)
IMPRESSION: ABR waveform morphology shows significantly prolonged latencies for Waves II-V compared to age-appropriate norms, consistent with Retrocochlear Pathology.


Diagnostic Value of the Click-Evoked ABR:
Differentiation Among Types of Auditory Dysfunction
0.25 μV
(1.5 ms)
I
II
III
IV
V (5.5 ms)
I-V = 4.0 ms
V/I = 2.0
Normal
Conductive
I-V = 4.0 ms
Sensory
Neural
I-V = 5.0 ms V/I = 0.5
ANSD
Pre-stimulus
0 2 4 6 8 10 12 14
Latency in ms

Section 3 : Summary
Normal- Abnormal ABR patterns

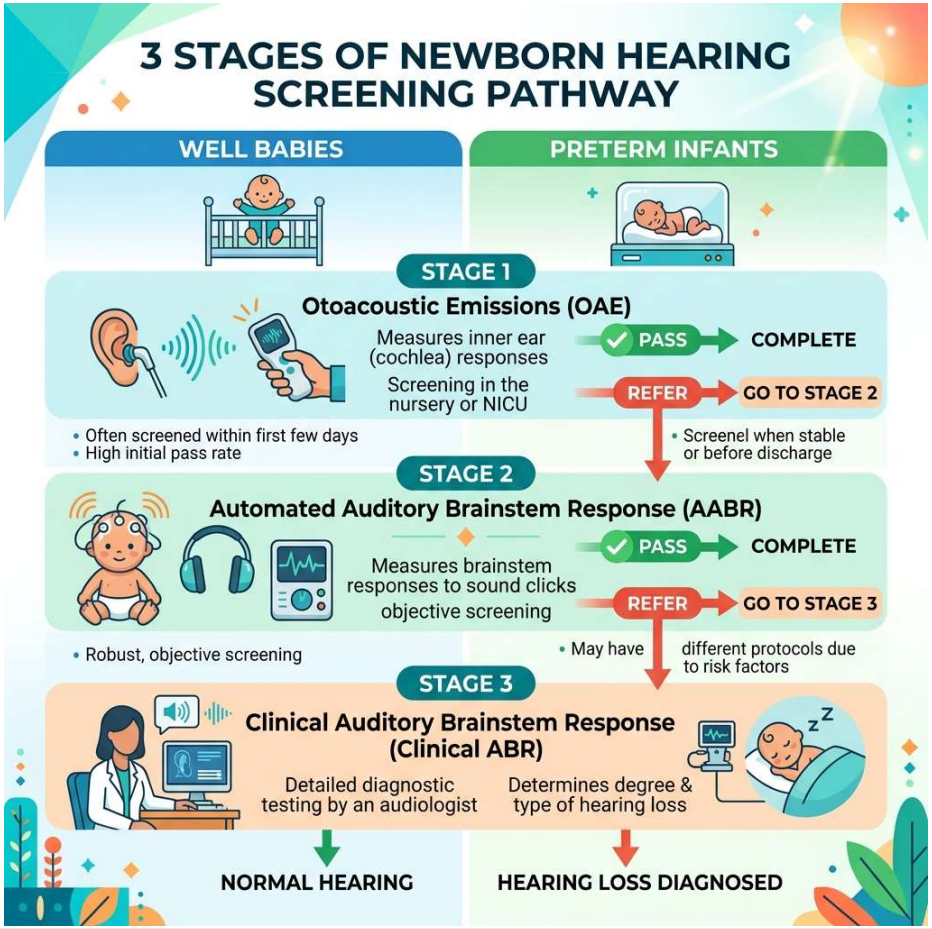


Criteria:

1. Wave Peak Detection
2. Inter-Wave latencies

Wave	Latency
I	< 2 ms.
II	< 3 ms
III	< 4 ms
IV	< 5 ms
V	< 6 ms
VI	< 7 ms

Section 5 : Hearing Screening



- Three-stage screening: OAE, OAE → AABR or AABR alone
- Diagnostic ABR protocol after failed screening (Stage 2):
  - Click ABR (neurodiagnostic)
  - Frequency-specific tone burst ABR (500, 1000, 2000, 4000 Hz)
  - Bone conduction ABR (if air conduction elevated)
- Timing: ideally by 3 months of age
- Importance of natural sleep in neonates (< 3–4 months)

Section 5 : Hearing Screening

- Several clinical trials have shown that the ABR (and the Automated-ABR) testing as an effective screening tool in the evaluation of hearing in newborns, with a sensitivity of 100% and specificity of 96-98%.
- The AABR algorithms usually seek the presence of wave V at a given latency range (at 70-80 dB nHL)

Section 5 : Hearing Screening



- Today ABR and OAE (Otoacoustic Emissions) protocols are widely used for the Identification of the hearing Impairment in the infant population.
- Well-babies are tested twice with OAEs prior to an AABR assessment.
- NICU residents are tested with OAEs upon discharge and then with AABR.

Section 5 : Auditory Neuropathy -ANSD

- The combined use of OAEs & AABR provides the possibility to identify cases with Auditory Neuropathy(AN) and Connexin 26 mutations (non-syndromic sensorineural HL -mild to profound-).

Section 5 : Auditory Neuropathy Spectrum Disorder (ANSD)

- Definition: present OAEs /CMPs + absent / abnormal ABR (asynchronous). The older term is Auditory Dyssynchrony.
- ABR: absent, severely abnormal, or only Cochlear Microphonic present (wave I)
- Cochlear Microphonic: reverses with stimulus polarity change

Section 5 : Auditory Neuropathy Spectrum Disorder (ANSD)

- Prevalence in NICU graduates: ~10% of those failing hearing screening
- Risk factors: hyperbilirubinemia, anoxia, prematurity, various syndromes
- Gender: No consistent sex predilection has been reported
- Bilateral vs. Unilateral: Majority are bilateral

Section 5 : Auditory Neuropathy Spectrum Disorder (ANSD)

Genetic Causes:

- OTOF gene mutations (otoferlin) — most common genetic cause
- PJVK, DIAPH3, OPA1 mutations
- Syndromic: Charcot-Marie-Tooth disease, Friedreich's ataxia, Usher syndrome

Section 5 : Auditory Neuropathy Spectrum Disorder (ANSD). Clinical presentation

Newborns/Infants:

- Fails newborn hearing screening (ABR-based) — but passes OAE-based screening
- Normal startle response to loud sounds but poor speech understanding
- Parents report inconsistent responses to sound

Toddlers/Preschool:

- Delayed speech and language development
- Appears to "hear" selectively — inconsistent
- Difficulty understanding speech in noise
- Better hearing in quiet environments

School-Age:

- Academic difficulties
- Listening fatigue
- Cannot follow multi-step verbal instructions

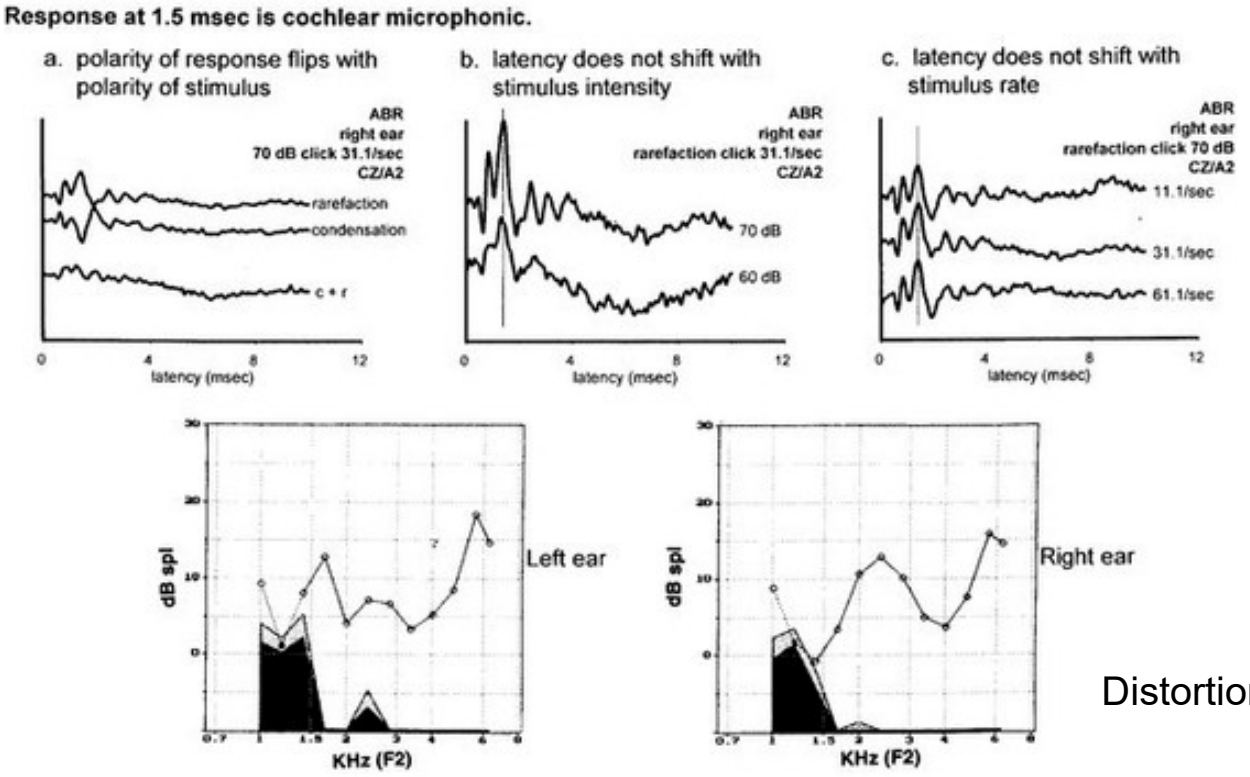
Section 5 : Auditory Neuropathy (ANSD)

- Key ABR Findings:
  - Absent or grossly abnormal neural waves (no reproducible Wave I, III, V)
  - Present cochlear microphonic (CM) — inverts with polarity change
  - Distinguishing CM from an artifact: remove transducer / clamp rigid tube test (rarely done).
- Other Findings:
  - Present OAEs (may disappear over time)
  - Absent acoustic reflexes
  - Behavioral thresholds may range from normal to profound
  - Speech perception disproportionately poor relative to audiogram

Section 5 : Auditory Neuropathy (ANSD)

- **Pediatric** Management considerations:
  - Hearing aids trial → but it needs monitoring, usually benefit children with mild-moderate HL.
  - HAs might not improve speech understanding is neural dyssynchrony is severe.
  - **Cochlear implantation** if there is no HA-benefit (assuming that Imaging shows no cochlear nerve damage)
  - Genetic testing (OTOF mutations → excellent CI outcomes)
  - **Auditory brainstem implants (ABI):** For children with Cochlear Nerve Deficiency (suggested by an MRI) ineligible for a CI.

Section 5 : Auditory Neuropathy (ANSD) Electrophysiologic Profile



The three ABR panels illustrate the absence of ABR waves. The waveforms show only the cochlear microphonic because the polarity of response flips with the polarity of the stimulus (a), latency does not shift with stimulus intensity (b), and latency does not shift with stimulus rate.

Distortion product OAEs

Kraus et al:Consequences of neural asynchrony: A case of auditory neuropathy ARO, 2000:33-45

Section 5 : Summary 1:
OAEs, Tympanometry and ABR Integration of the data

Finding	OAE	Tymp	ABR	Interpretation
Normal hearing	Pass	Type A	Normal threshold / latency	Normal
CHL (OME)	Fail	Type B	Elevated AC, normal BC, normal IPL	Conductive HL
Mild-Mod SNHL	Fail	Type A	Elevated AC & BC, normal IPL	Cochlear SNHL
Severe SNHL	Fail	Type A	Absent / elevated threshold	Severe SNHL
ANSD	Pass	Type A	Absent ABR, present CM	Auditory Neuropathy
Retrocochlear	Fail	Type A	Prolonged IPLs	Neural disorder

Section 5 : Summary 2:
Pediatric scenarios where a Clinical ABR is necessary

- Failed newborn hearing screening (OAE or AABR)
- Family NICU graduates (JCIH high-risk indicators)
- History of childhood hearing loss
- Craniofacial anomalies
- Syndromes associated with hearing loss (Waardenburg, Treacher Collins, Down syndrome, etc.)
- Congenital CMV, bacterial meningitis
- Suspected auditory neuropathy spectrum disorder
- Developmental delay / Speech-language delay
- Neurological concerns (demyelinating diseases, posterior fossa tumors)
- Pre- cochlear implant evaluation
- Monitoring (ototoxicity, post-surgical)

This is the End

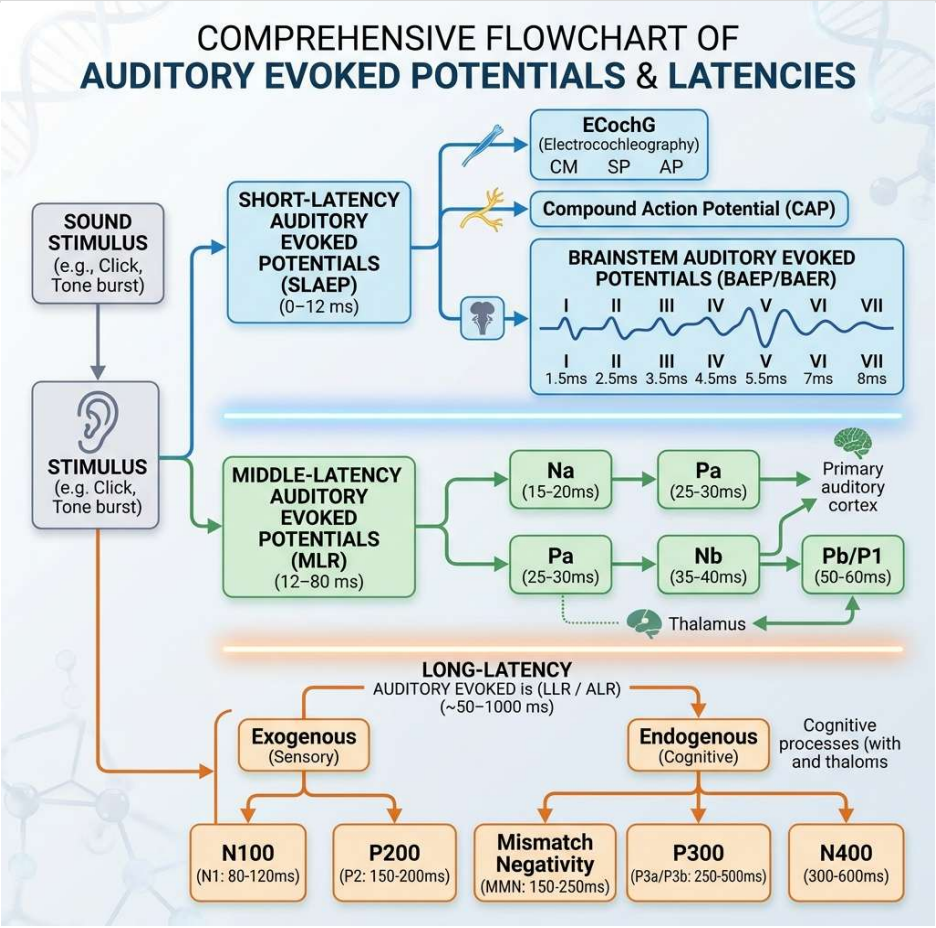


That's all Folks !!

Section 6

Appendix: EXTRA information from the previous Sections

AEPs classification

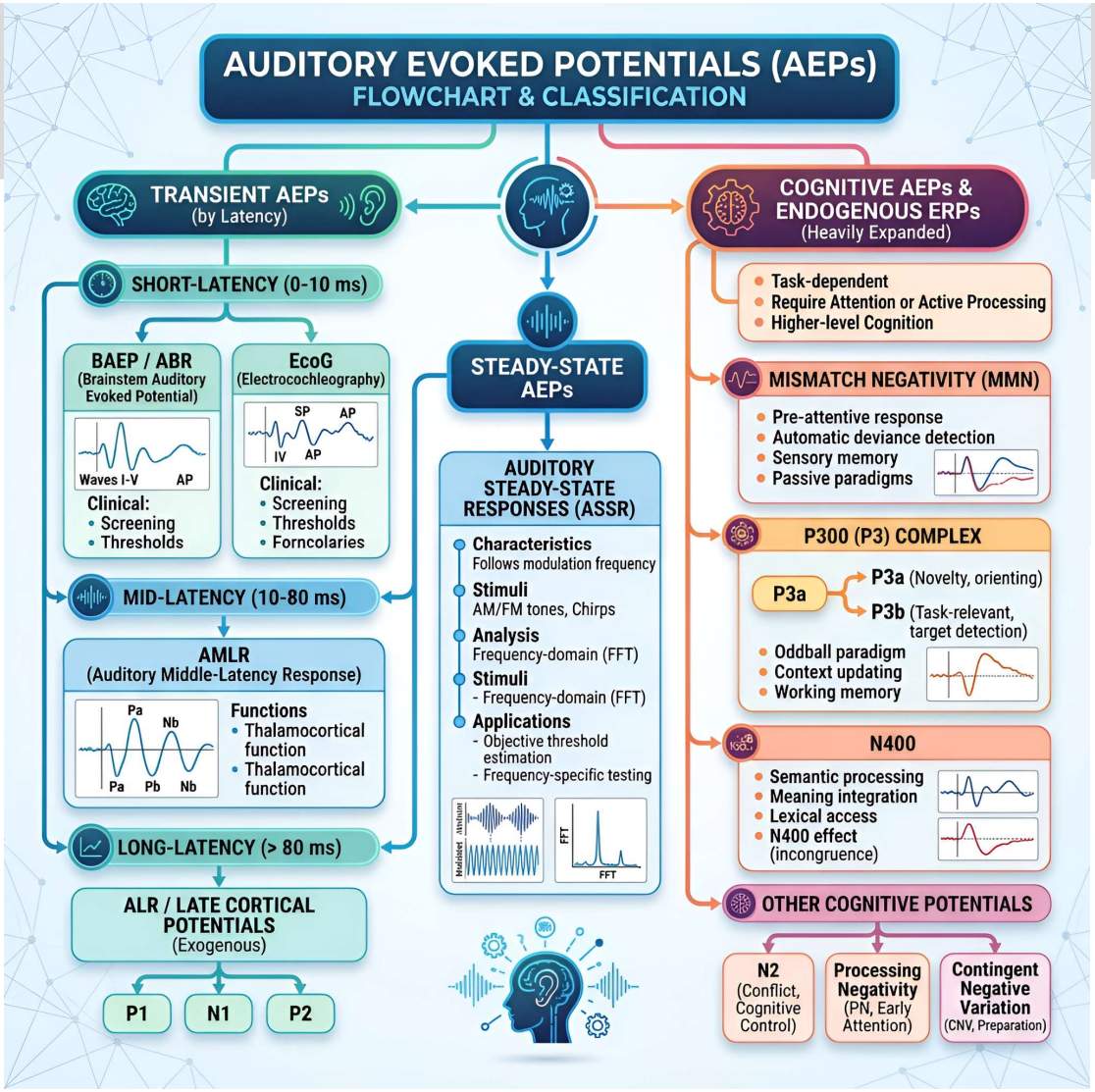


ASSR Webinar 2027

ACA Webinar late 2026

AEPs

Another Classification Infographic



ACA Webinar late 2026

ASSR Webinar 2027

SECTION 1: The Historical Perspective

As early as 1967, Harvey (Haim) Sohmer and Moshe Feinmesser in a paper entitled"Cochlear Action Potentials Recorded from the External Ear in Man" showed waveforms resembling what we now would recognize as an upside down or inverted ABR.

SECTION 1: The Historical Perspective

Although these authors were investigating a non-invasive extra-tympanic technique for recording the ECochG, their electrode array was actually not that different from the electrode locations most audiologists use in ABR measurement with a non-inverting electrode on the earlobe and an inverting electrode on the bony bridge of the nose.

Section 1 : ABR Developments in the 70s & 80s

1970: Jewett described ABR in animal model
1970: Jewett, Romano & Williston first describe ABR in humans
1971: Jewett & Williston systematic study ABR in humans
1971: Moore completed unpublished PhD project revealing ABR
1973: Terkildsen, Osterhammel and colleagues conducted a series of studies on stimulus and acquisition parameters
1974: Hecox & Galambos described ABR in infants and children
1975: Schulman-Galambos & Galambos described ABR in premature infants
1975: Starr described ABR in patients with varied CNS pathologies
1976: Salamy investigated development of ABR in neonates
1976 Robinson & Rudge studied ABR in multiple sclerosis
1976: Davis described ABR slow negative wave (SN 10) in humans
1976: Greenberg applied ABR with other responses in acute head injury
1977: Selters & Brackmann applied ABR in acoustic tumor detection
1977: Clemis applied ABR in acoustic tumor detection
1977: Terkildsen et al applied ABR in acoustic tumor detection
1977: Stockard & Rossiter reported ABR in varied CNS pathologies
1977: Arlinger recorded ABR evoked with bone conduction stimulation
1977: Robinson & Rudge described ABR in multiple sclerosis
1978: Stockard, Stockard & Sharbrough published monograph on measurement
1978: Don & Eggermont investigated high pass masking for frequency specific ABR techniques
1979: Jerger & Mauldin described clinical bone conduction ABR data
1979: Yamada studied the effects of cochlear hearing impairment on ABR
1979: Chiappa described normal ABR variations
1979: Dobie & Berlin investigated binaurally elicited ABR
1979: Don studied frequency specific ABR with masking paradigms
1980: Jerger & Hall studied effect of age and gender on ABR

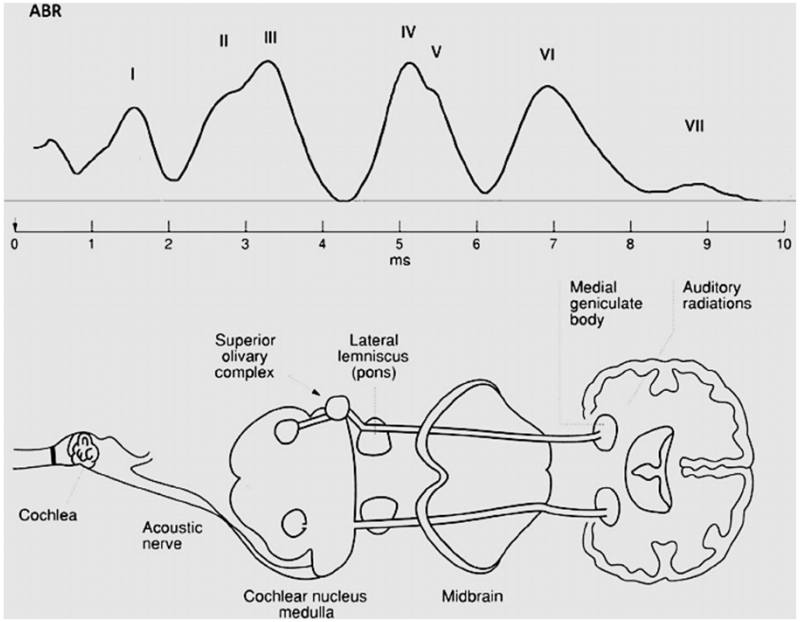
James Hall III : Handbook of Auditory Evoked Responses

Section 1 : Waveform Terminology

Wave I: arises from eighth nerve potentials in man. The same is valid for various animal models. For the human data, multiple studies were conducted in the 80s.

Wave II: arises from the activity of the first order neurons (8th nerve) versus the auditory brainstem.

Wave III: Was traditionally believed that it was generated from neuronal activity in the Superior Olivary Complex (SOC). Data from intra-cranial recordings and EMF dipole models suggest that Wave III is generated beyond the 8th nerve closer to the Cochlear Nucleus (CN), where the negative pattern after wave III, it is believed that it is generated from the Trapezoidal Body (TB).



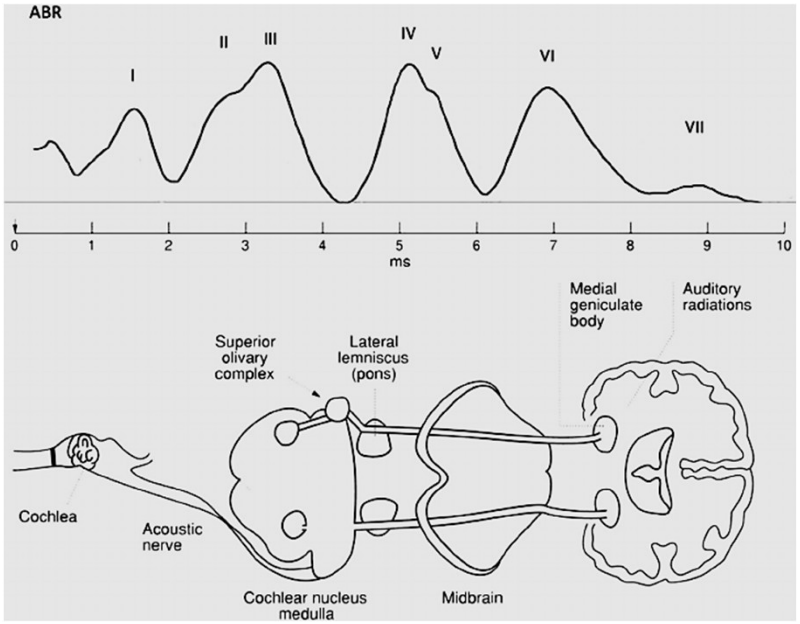
Section 1 : Waveform Terminology

Note: As we move towards the upper Cortex the spatial contribution of the various segments to ABR becomes quite complicated.

Wave IV: Often appears as the leading peak before Wave V. As in the case of Wave III, Intracranial research in the 80s and 90s, suggests that Wave IV arises from neurons in the pons primarily located in the Superior Olivary Complex (SOC).

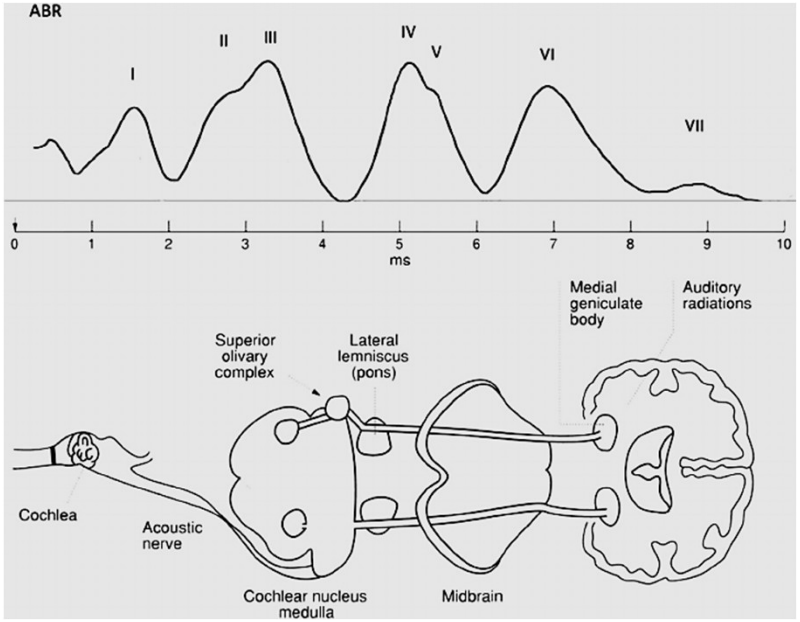
Wave V: Traditionally, Wave V was hypothesized to be generated by neurons in the Inferior Colliculus (IC). But more recent experiments with dipole models suggested that this wave is generated at the end of Lateral Lemniscus (LL) as its fibers enter the Inferior Colliculus.

NOTE: this is in a contralateral scheme to the stimulated ear.



Section 1 : Waveform Terminology

Wave VI & VII: The exact spatial contributors of these two ABR components are still not well-defined. Some early observations in the 70s suggested that the generators might be located within the Thalamus in the Medial Geniculate Body (MGB). Other research (i.e Møller) suggests that these peaks originate from neurons in the Inferior Colliculus.

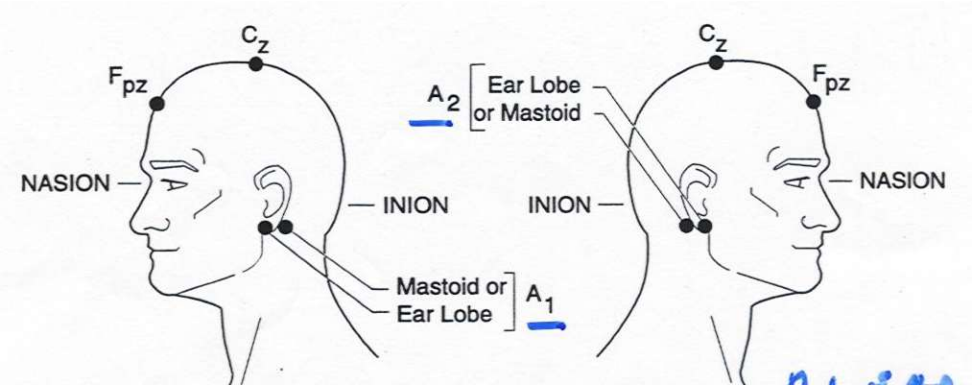


Section 1 : What Happens upon stimulation

- The cochlea, is stimulated by the broad sound spectrum of the transient stimulus (click) activating the hair cells within a wide region of the basilar membrane.
- The responses from the small disposable electrodes are amplified and averaged over time (500, 1000, 1500, or 2000 sweeps).

Section 2 : Electrode Montage

- Wave I-III: more prominent in ipsilateral recordings
- Wave IV, V: often better separated in contralateral recordings
- Earlobes sites: less muscle potential and greater wave I amplitude (than mastoid recording sites)
- Use of non-cephalic site (C7 vertebra): **enhance** wave V, VI

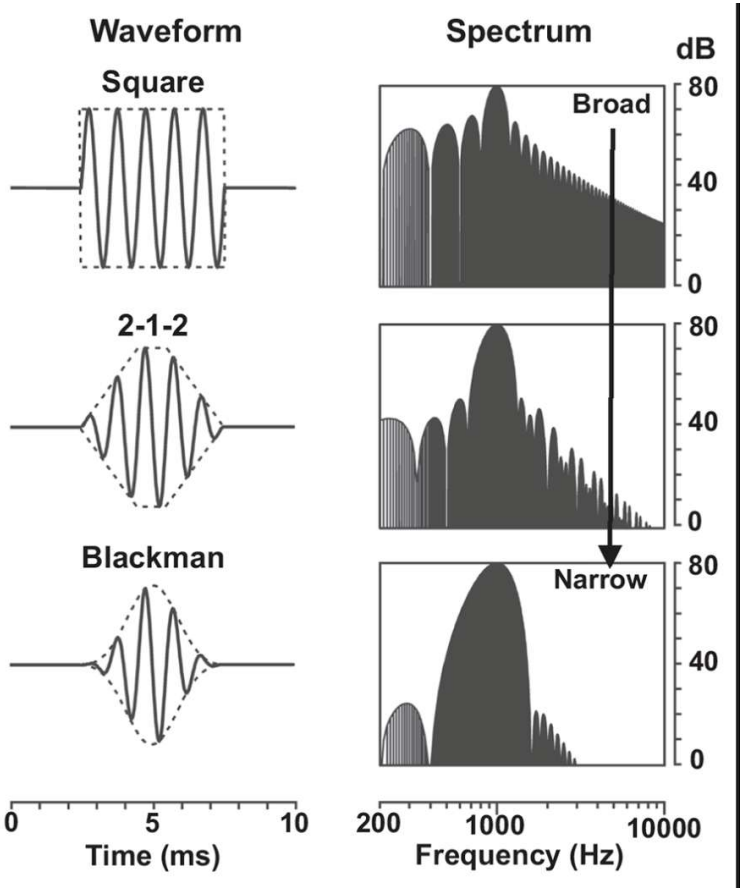


Attention at the Hair !!!!!

They can cause

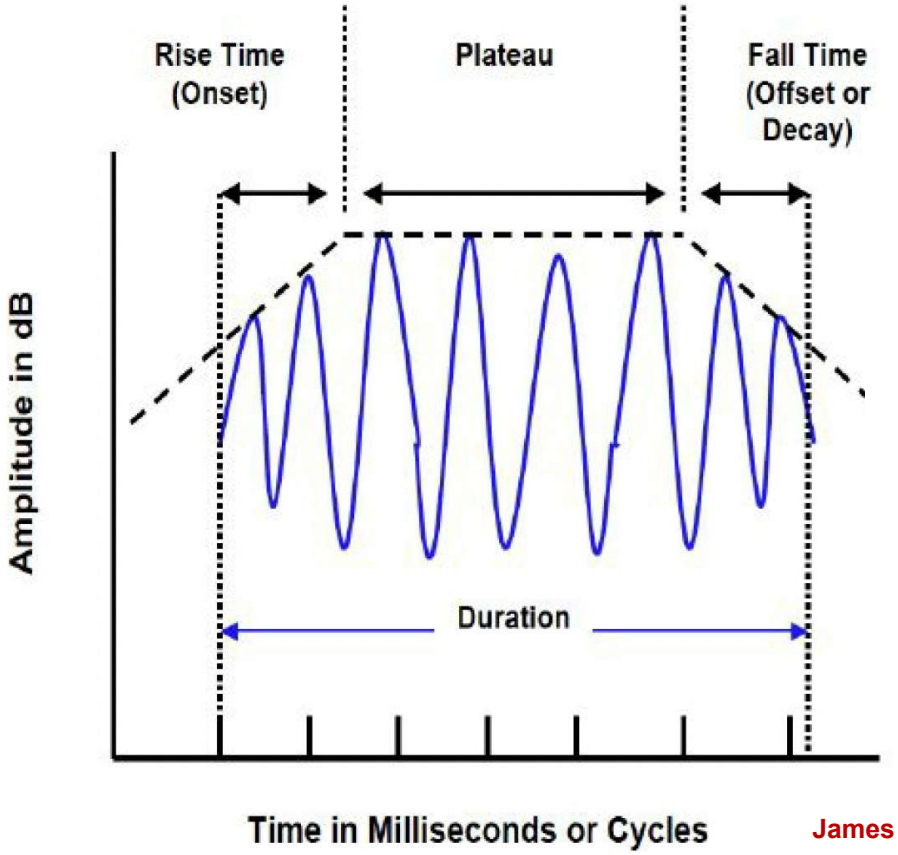
very large electrode impedance

Section 2 : Managing the form of the stimuli



In order to select which cochlear partitions (i.e. tested octaves) are to be stimulated , the ANALOG transient stimuli can be manipulated in such a way that their spectra show peaks in the areas of interest.

Section 2 : Stimulus parameters



Tone Burst Frequency	Duration in Milliseconds			
	Rise	Plateau	Fall	Total
500 Hz	4	0	4	8
1000 Hz	2	0	2	4
2000 Hz	1	0	1	2
4000 Hz	0.5	0.5	0.5	1.5

James Hall III : Handbook of Auditory Evoked Responses

Section 3 : Stimulus Intensity & mean Latency values

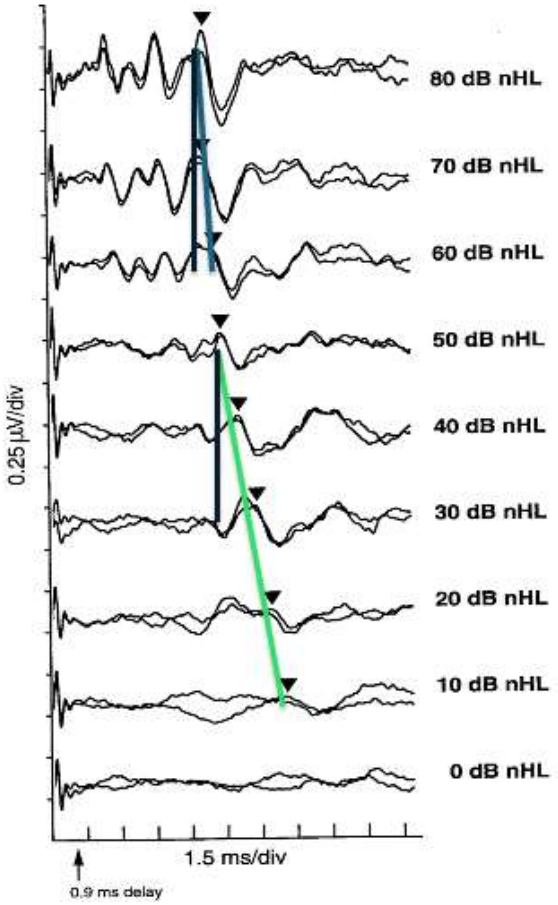
Stimulus Intensity		Absolute Latency (ms) I	II	III	IV	V	VI	Interwave Latency (dB) I–III	III–V	I–V
90 dB nHL 126 dB pSP	Mean	1.53	2.53	3.58	4.56	5.37	7.09	2.05	1.79	3.84
	SD	.11	.09	.09	.17	.12	.39	.14	.14	.16
	n	14	14	14	14	14	14	14	14	14
80 dB nHL 116 dB pSP	Mean	1.62	2.68	3.68	4.68	5.47	7.29	2.06	1.79	3.85
	SD	.12	.11	.08	.22	.12	.17	.11	.09	.14
	n	14	13	14	11	14	13	14	14	14
70 dB nHL 106 dB pSP	Mean	1.82	2.79	3.85	4.92	5.64	7.31	2.03	1.79	3.82
	SD	.17	.12	.13	.24	.16	.19	.11	.12	.11
	n	14	11	14	11	14	9	14	14	14
60 dB nHL 96 dB pSP	Mean	2.04	2.98	4.06	5.11	5.88	7.34	2.02	1.72	3.75
	SD	.20	.15	.21	.31	.25	.31	.12	.10	.11
	n	9	6	10	9	14	9	8	10	9
50 dB nHL 86 dB pSP	Mean	2.43	3.69	4.60	5.43	6.19	8.24	2.02	1.56	3.64
	SD	.17	.10	.23	.25	.32	.34	.19	.18	.19
	n	4	2	13	5	14	2	4	13	4
40 dB nHL 76 dB pSP	Mean	3.01	4.05	4.94	5.65	6.65	—	1.85	1.71	3.60
	SD	.25	.18	.25	.49	.32	—	.14	.14	.11
	n	4	2	7	5	14	—	4	7	4
30 dB nHL 66 dB pSP	Mean	—	—	5.45	—	7.24	—	—	1.74	—
	SD	—	—	.30	—	.42	—	—	.26	—
	n	—	—	7	—	14	—	—	7	—
20 dB nHL 56 dB pSP	Mean	—	—	5.56	—	7.52	—	—	1.88	—
	SD	—	—	.57	—	.63	—	—	.23	—
	n	—	—	2	—	7	—	—	2	—

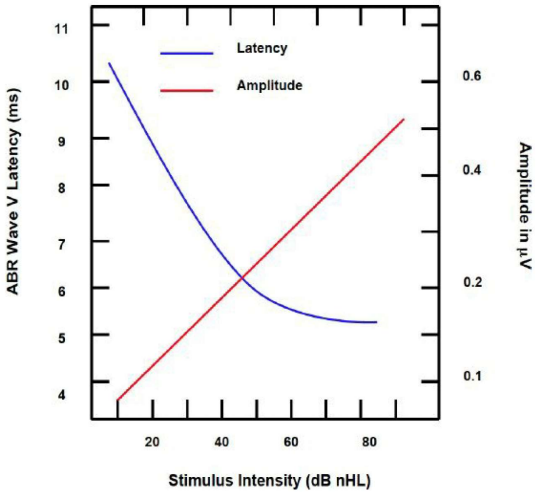
Note: Intensity is expressed in nHL (level relative to a group of normal-hearing listeners) and pSP (peak sound pressure). Latency is in milliseconds and n is the number of subjects.

NOTE: For practical Reasons

pSP= SPL

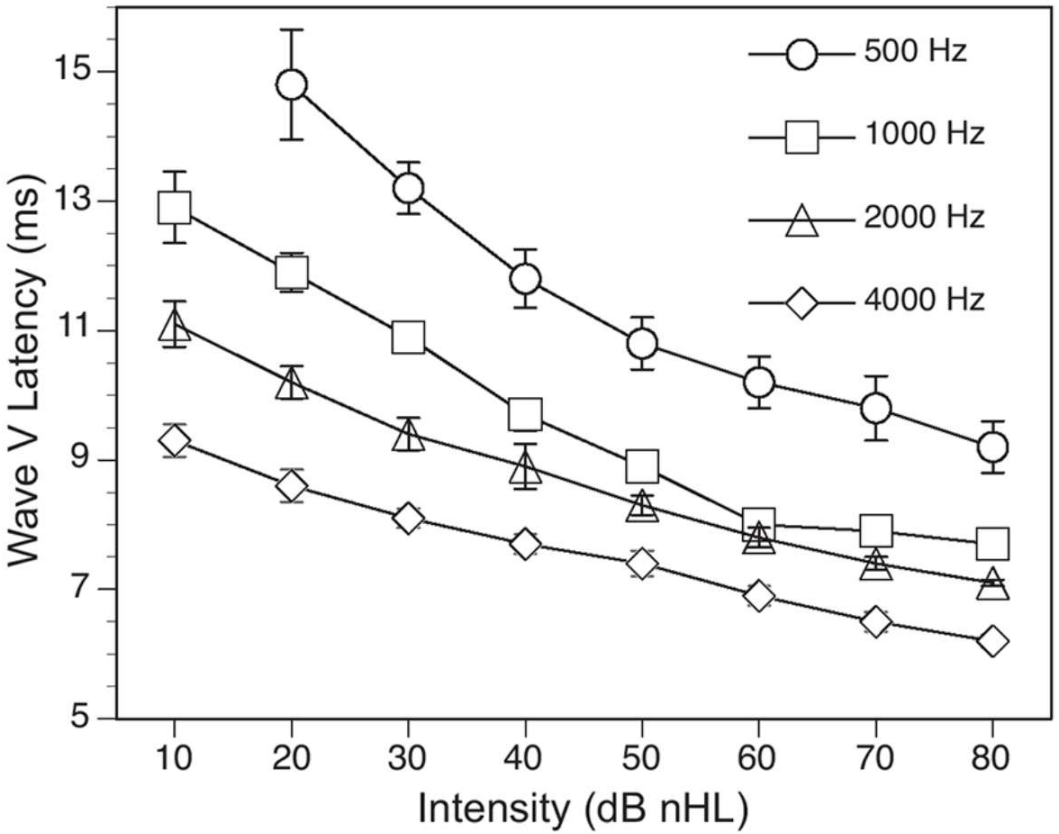
Section 3 : Stimulus Intensity in Wave V



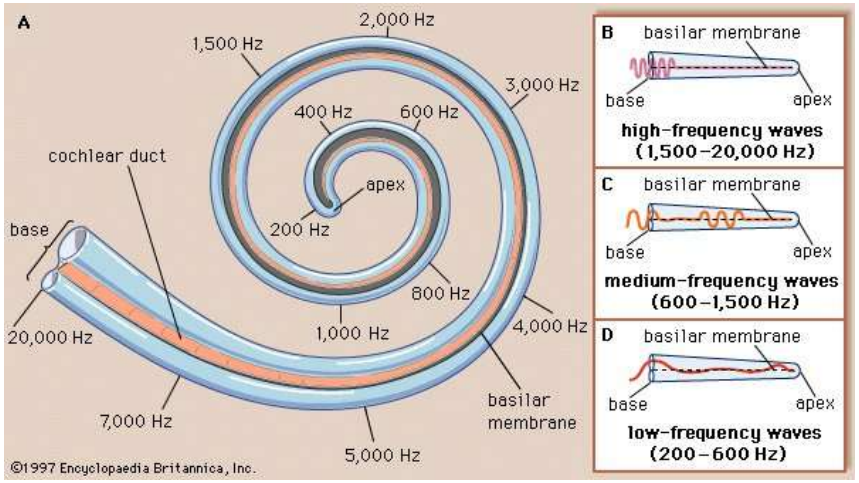


- At lower intensity levels, wave V is mostly visible.
- Earlier components tend to become indistinguishable at 30 dB.
- Near the threshold of the response, wave I occurs at approximately 4.0ms; wave V at 7-9ms.
- Wave I latency shifts more than wave V

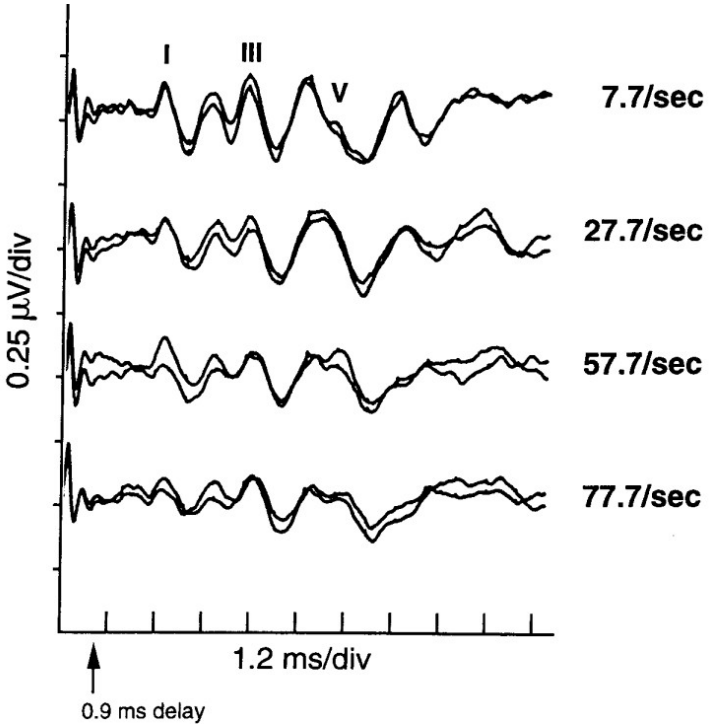
Section 3 : Stimulus Intensity in Wave V



Ananthanarayan Krishnan Auditory Brainstem Evoked Potentials



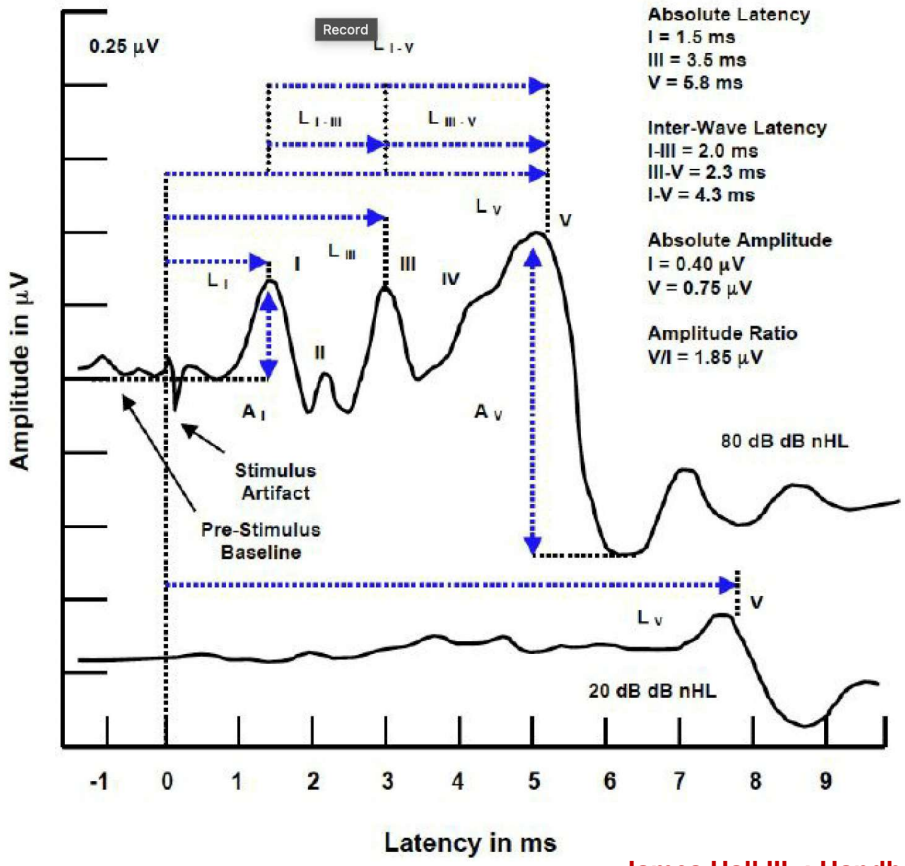
Section 3 : The stimulation rate



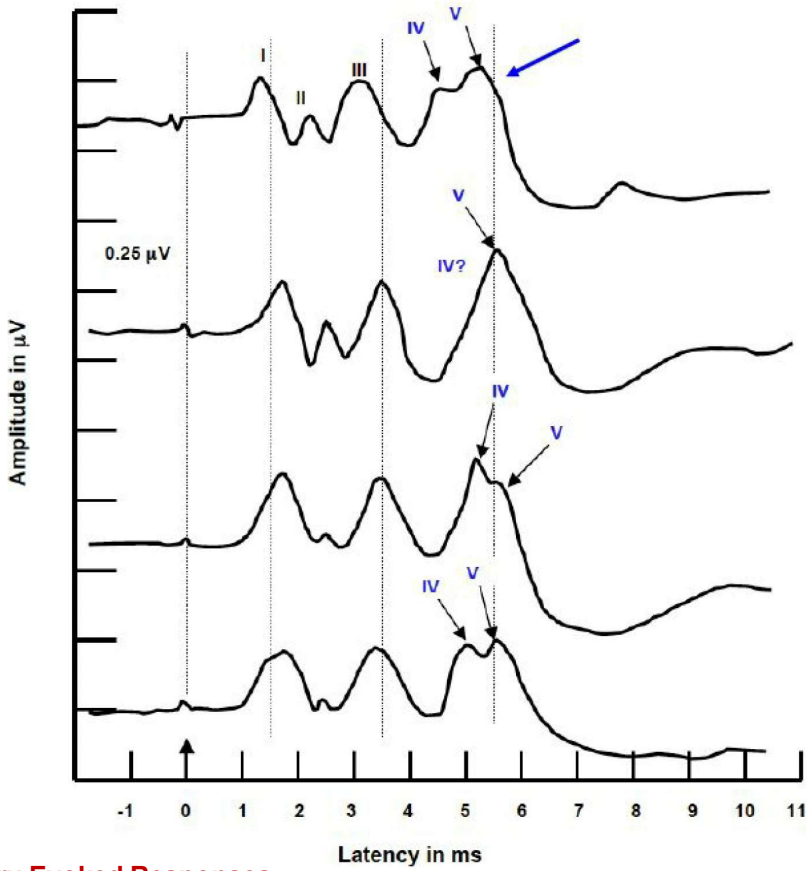
Rate	Wave I	III	V	I-III	III-V	I-V
7.7/s	1.52	3.66	5.89	2.14	2.23	4.37
27.7/s	1.62	3.80	5.89	2.18	2.09	4.27
57.7/s	1.62	3.90	6.11	2.28	2.21	4.49
77.7/s	1.69	3.95	6.30	2.26	2.35	4.61

Terence W. Picton: Human Auditory Evoked Potentials

Section 3 : Response Analysis and Peak selection



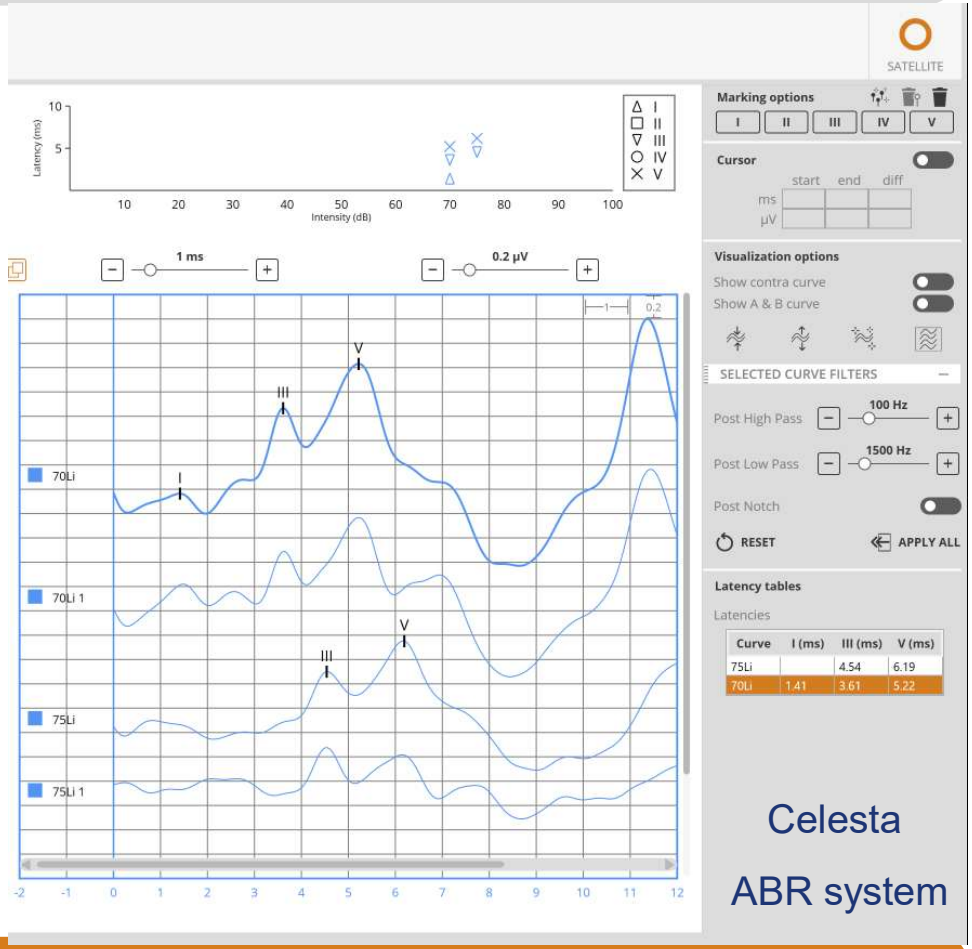
ABRs from normal subjects



James Hall III : Handbook of Auditory Evoked Responses

Section 3 : Filtering

- Allowing more low frequency information into the average:
  - Amplitude increases (particularly later components)
  - The Latency values slightly Increase
- Decreasing the low-pass filter cutting frequency (e.g., from 3000Hz to 1500Hz)
  - Some rounding of the peaks, but less effect on amplitude or latency



Celesta ABR system

Intraoperative monitoring

Auditory brainstem response (ABR), often used intraoperatively, provides early identification of changes in the neurophysiologic status of the peripheral and central nervous systems. This information is useful in the prevention of neurotologic dysfunction and the preservation of postoperative hearing loss.

For many patients with **tumors of CN VIII** or **the cerebellopontine angle**, hearing may be diminished or completely lost postoperatively, even when the auditory nerve has been preserved anatomically.