Physiologic Assessment of Young Infants: Puzzles & Challenges in EHDI

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Focus – how are we doing and in what?

- Initial Dx assessment of AABR referrals in EHDI programs
- **Goals**: coverage, timeliness, validity, accuracy, efficiency, effectiveness
- Dysfunction site, mechanism, cause, functional impact, prognosis, implications for remedy or amelioration, outcome measurement

- From: Gross thresholds, conductive, sensorineural
- To: FS thresholds, suprathreshold functions, afferent OHC, IHC, synaptic, cochlear place, 1st neuron, brainstem pathways, thalamocortical, primary cortical, association cortical, efferent OHC control, cognitive functions, plastic maladaptations, molecular mechanisms, etc

- Some progress ((':', but could try harder.....):
ANSD – a moving target

• Normal OAEs & absent ABR
• Normal OAEs & absent or grossly abnormal ABR
• Normal OAEs and/or CM & absent or abnormal ABR
• Present CM & absent or abnormal click or tonepip ABR...

• Incompatible OAEs & tonepip ABR thresholds....with or without normal high-intensity waveforms

• Broad, late wave V only, no sharpening or I-O change....
ANSD Challenges

• Does ANSD protect against conductive loss?  NO

• Can ANSD & conventional SHL coexist?  YES

• Does ANSD have graded severity?  YES

• Can ANSD have concurrent multiple sites & dynamics?  YES

• Can ANSD occur at specific cochlear frequency-places?  YES
Key points about CM

• May be OHC, IHC or both, from basal to middle cochlear regions
• Presence does NOT guarantee OHC normality – can be seen in severe conventional cochlear hearing loss
• Size at mastoid highly variable over subjects, strongly affected by stimulus level within-subject
• May be abolished by conductive components > 20 dB

• If ‘absent’ OAE, CM/ABR disparity is key predictor of ANSD likelihood

• Given ‘absent’ ABR, does ANY mastoid CM imply ANSD....?
ANSD case? Nov 2013
So what do we do now?

- Likelihood/confidence categories:
  - ‘Definite ANSD component’, Probable…, Possible…, Not suspected, NYD

- OAE < - >ABR, CM <--> ABR
- CM size, duration, oscillation, frequency, I-O, etc

- Extend recording bandwidth (Santarelli R)
- Window 5 or 10 ms, rep rate 91/s (181/s?)
- Wait for ‘gold standard’ outcomes…….VRA? LCPs? Speech perception…
‘Obligatory’ Late Cortical Potentials ‘N1-P2’

• ‘Exogenous’ = determined by stimulus parameters......?
• Yes, strongly but NOT exclusively....
• = Acoustic Change Complex: ANY perceptible change in acoustic env.
• Threshold is just a special case of perceptible change
• Delta I (difference limen, gaps), delta F, delta position, masking, etc

• Eg: FS thresholds (AC, BC) & gap detection at one month in ANSD etc?

• Issues: maturation, practicality, optimal technique & skills
Adult cortical N1-P2 example
Late Cortical Potential mean thresholds

- 0-4 days light
- 0-4 days deep
- > 4 days light
- > 4 days deep

N = 47
Evoked Response Audiometry in newborn infants

N = 250, 88% ‘excellent results’

Taguchi K, Picton T, Orpin J, Goodman W
Acta Oto-Laryngologica 1969, Suppl. 252: 5-17
Adult N1-P2 nonstationarity – attention?
Figure F-2.—Changes in pitch of sound of constant amplitude. Note the absence of responses for triangular frequency modulation.
ABR & ASSR puzzles

• Are they the same thing, measured differently?
• Ear is E integrator, TC ~ 250 ms at threshold
• ABR reflects Δ (intensity) & dI/dt in 0-2 ms.....
• ASSR in profound loss: validity???
• Superior f-specificity: validity???
• Greater efficiency?

• Depends on host of subject, acquisition parameters & detection criteria eg averaging times, ABR rep rates, intensity strategies, FP & FN error rates....

• MUST compare optimised methods in specific populations & in every subject individually, with time & error control; focus not on group means but variation, interaction. Express in terms of ppeSPL...not nHL for ABR
‘Lost in diagnosis’ – ABR test efficiency
Bayesian optimization under time constraint

• Test: Ear, AC or BC? Frequency? Intensity? Etc.
• Which single R+ / R- answer would make the greatest difference to Dx inference, counselling, intervention... if it were the only answer obtained?
• Given an answer, ask it again & again, until you or the baby are done...

• Step info gain greatest if alternatives are equiprobable
• Average ascending threshold variance 3x descending variance
• R-negative proof usually slower than R-positive proof
• Use response characteristics to control down-steps
• Confirmatory averages can be smaller; repeats only on threshold brackets
• Minimum initial level > up 30 dB...down 20 dB given R++, 10 dB given R+, etc
Infant ABR rare-cond differences
Infant ABR rare/cond differences
Infant ABR: rare/cond diff, no CM
‘If you haven’t found something strange during the day
.....then it hasn’t been much of a day’
And now….at last
Main topics - messages
Main topics - messages
Click CM amplitude in Normal & ANSD infants
Young & Cone-Wesson 01, Starr et al 01
• For linear rise-plateau-fall pips, the energy-equivalent duration is 
  \[ \frac{2}{3}R+P : 500 \ 3.33 \ \ 1k \ 1.67 \ \ 2k \ 0.83 \ \ 4k \ 0.42 \ \ \text{click} \ 0.25 \]
• In 250 ms \ 80 \ \ 160 \ \ 320 \ \ 640 \ \ 1,000 \\
• SPL under \ 11 \ \ 8 \ \ 5 \ \ 2 \ \ 0
Click ABR rare-cond differences

- Are more common & dramatic than expected
- Can cause misinterpretation of alt. ABR anomalies
- Are not well-understood –superficial dogma...

- Are they diagnostically useful?
- Might they reflect IHC dead zones?
- What would be revealed by ‘stacked ABR’ type masking?
Title

- Text
Threshold: cortical P2-N2 complex in sleeping infants

• Use stimulus unaffected by dys-synchrony
• Use ordinary toneburst eg 10-40-10 ms
• F-spc CHL+SHL components (AC, BC)

• Early data: 1965 – 1986 Barnet AB, Ohlrich ES, Rapin I, Graziani L, Weitzman E, Davis H, Onishi S, Rotteveel J, etc.
• Hall, Reneau & Hnatiow 75 (per mlh)
Obligatory cortical AEPs at 3/12

- $P_{200-250} - N_{350-450}$ prominent (‘P2-N2’)
- Twice the size of N1-P2
- Light sleep acceptable
- Best recorded 3-4/12 corrected age
- Parameters as for adult N1-P2
- Toneburst ISI or frequency jitter may increase amplitude
• Intensity DL: low SL lot of overlap, high SL normal
• Masking: <, >, =, lot of overlap
• ITD lateralization: big difference, difficult?

BUT

• WN Gap, 20 SL: $\alpha$ 2%, $\beta$ 60%, 30 SL: $\alpha$ 2%, $\beta$ 80%

• $\Rightarrow$ Measure P2 gap threshold at P2 threshold + 30dB ? With WN? With low-frequency tones?
Gap thresholds with N1-P2

Michalewski et al 2005 AN/AD = 14

- Psych gap threshold (ms)
- N100 gap threshold (ms)
Gap thresholds vs speech scores

Michalewski et al, Clin Neurophys 2005

psych gap threshold ms vs BKB sentence scores, %
Frequency DL with N1-P2

- Zeng FG et al Clin Neurophysiol 2005
- ‘profound impairment in f-discrim <4kHz’

- Freq DL: 500 Hz N <2 Hz, AN/AD >80 Hz
- AND <30 Hz in SHL (Freyman & Nelson 91)
  - SO

- P2 at 500 Hz, FM 10-40-10ms 50 Hz δF (eg Kohn M, EEG clin Neurophys 1978)
Electrocochleography

- Eg Santarelli R, Arslan E, Hear Res 2002

- CM I-O function (OHC)
- SP I-O function (IHC?)
- AP I-O function??

- ? Feasible in infants, surface electrodes
Priority AEP investigations

- P2 thresholds at 3-6(±12 in all AD, ?AD, PCHI (by ABR) → VRA → difference distributions (ABR-N1, VRA-N1, VRA-ABR)

- P2 Gap & FM: response at specific P2 SL to discriminate, threshold to quantify dysfunction

- CM,SP & CABR I-O fns in AD, ?AD, SHL
- ABR enhancement (low rate, special stimuli)
Efficiency: type, severity in 7 runs or less per f

- In an OAE -> AABR referred ear:
- (not OAE) ABR AC or BC? Hearing WNL? No PHL?
- AC2k 30: R, -> 4k or 500? Other ear?
- AC2k 30: NR, AC or BC? If BC, 30:NR ->60
  - If AC, -> 60:NR, -> 90:R ->70, r ->80....
- If R at minimum or upper bracket level, must replicate
- If NR at lower bracket level, must reach max noise criterion
- Proving NR usually takes longer than proving R
Bone-conduction ABR

How do you determine cochlear origin?

(Which ear does response come from?)
AURAL ATRESIA (RIGHT)

STIM: BC 2000 Hz @ 30 dBnHL
BONE OSCILLATOR: RIGHT

Vertex to Left Mastoid

Vertex to Right Mastoid

Better in ipsi channel (normal) – comes from right (ipsi) ear

Normal cochlea @2kHz
**ASYMMETRIES IN HEARING LOSS**

**BC TONE-ABR IPSI/CONTRA**

Better in contra channel – comes from left (contra) ear

**UNILATERAL SNHL (RIGHT)**

STIM: BC 2000 Hz @ 60 dBnHL

BONE OSCILLATOR: RIGHT

**V_c**

**V**

**V_c**

SNHL @2kHz

STIM: BC 2000 Hz @ 30 dBnHL
Infant Tone-ABR @ “normal” intensities*

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Intensity</th>
<th>Right Response</th>
<th>Left Response</th>
</tr>
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<tbody>
<tr>
<td>0.5 kHz</td>
<td>35 dBnHL</td>
<td>V (14.2 ms)</td>
<td>V (14.4 ms)</td>
</tr>
<tr>
<td>2 kHz</td>
<td>30 dBnHL</td>
<td>V (9.5 ms)</td>
<td>V (9.3 ms)</td>
</tr>
<tr>
<td>4 kHz</td>
<td>25 dBnHL</td>
<td>V (8.3 ms)</td>
<td>V (8.7 ms)</td>
</tr>
</tbody>
</table>

Infant AG (11 mos)

Total recording time: 9.4 minutes

* Infant hearing considered “normal” if responses present at these intensities (BCEHP)