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, 1\textsuperscript{st} 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
Amatuzzi M et al., JARO 2011;12(5):595-604

Pre-Term Cases

Case 24

Case 11

Case 5

Case 14
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....?
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
Ishida, Stapells & Small (in prep)

INFANT ONSET

Adults (Cz)

Infants (C3)

onset

P1

N1
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 $\Delta$ (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
Group A: ≥ 30 dB/octave

- S1
- S2
- S3
- S4
- S5

Stimulus intensity (dB nHL)

Multiple ASSR

0

10
‘Lost in diagnosis’ – ABR test efficiency
Bayesian optimization under time constraint

- 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

<table>
<thead>
<tr>
<th>Group</th>
<th>CM amplitude uV</th>
</tr>
</thead>
<tbody>
<tr>
<td>YCW 90dB</td>
<td></td>
</tr>
<tr>
<td>YCW '80dB'</td>
<td></td>
</tr>
<tr>
<td>S et al ANSD 80dB</td>
<td></td>
</tr>
</tbody>
</table>
For linear rise-plateau-fall pips, the energy-equivalent duration is

<table>
<thead>
<tr>
<th>Frequency (kHz)</th>
<th>Duration (ms)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.67</td>
<td>2/3</td>
</tr>
<tr>
<td>1.00</td>
<td>1.67</td>
</tr>
<tr>
<td>2.00</td>
<td>0.83</td>
</tr>
<tr>
<td>4.00</td>
<td>0.42</td>
</tr>
<tr>
<td>Click</td>
<td>0.25</td>
</tr>
</tbody>
</table>

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?
Threshold: cortical P2-N2 complex in sleeping infants

- Use stimulus unaffected by dys-synchrony
- Use ordinary toneburst eg 10-40-10 ms
- F-spec 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
Zeng FG et al JN 2005
AD vs Normal Functionality

• 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%

• => 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

N100 gap threshold (ms) vs psych 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?)
BC TONE-ABR IPSI/CONTRA
ASYMMETRIES IN HEARING
LOSS

AURAL ATRESIA (RIGHT)

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

Vertex to Left Mastoid

$V_c$

Vertex to Right Mastoid

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

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

Normal cochlea @2kHz
BC TONE-ABR IPSI/CONTRA ASYMMETRIES IN HEARING LOSS

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

UNILATERAL SNHL (RIGHT)

STIM: BC 2000 Hz @ 60 dBnHL
BONE OSCILLATOR: RIGHT

SNHL @2kHz
Infant Tone-ABR @ “normal” intensities*

<table>
<thead>
<tr>
<th>Frequency</th>
<th>Intensity</th>
<th>Right Response Time</th>
<th>Left Response Time</th>
</tr>
</thead>
<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)