Topics in Central Auditory Processing

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This About That: *When I was fighting battles with marginal success my son said one person can't get these changes made, you need a group! Well we have a group of 125 people with 125 voices, computers and personal contacts. Wow, how much good could we do if we all acted to let people, schools and institutions know that; Auditory Processing Professionals have been helping people effectively improve their auditory skills for decades despite the chatter.*

In This Issue

Listening Difficulties and Normal Audiograms

Wayne J. Wilson, Ph.D.

We know that difficulties listening in noise in the presence of a normal audiogram point to an auditory processing disorder (APD) presumably in the brain. A recent review of the etiology of difficulties listening in noise by Pienkowski is of interest because it doesn't emphasize the brain, it emphasizes the ear.

Pienkowski reminds us that a "normal" audiogram may not mean normal auditory function. Peripheral auditory dysfunction is possible even with hearing thresholds of 0 dB HL, and is probable once hearing thresholds reach 10 to 20 dB HL. This leads Pienkowski to state: "The current practice of classifying thresholds of 20 dB HL in the 0.1 to 8 kHz range (i.e., 4 to 6 SDs below [poorer than] average) as 'clinically normal' therefore seems dubious at best". Adding to this is the audiogram's poor sensitivity to partial losses of inner hair cells (IHCs) and/or cochlear nerve fibers. Pienkowski provides several examples of this insensitivity such as losses of up to 80% of IHCs having no effect on hearing thresholds in quiet but having significant effects on hearing thresholds in noise in chinchillas, and losses of up to 50% of IHC synapses with little or no loss of outer hair cells (OHCs) following noise exposure in guinea pigs.

Pienkowski reviews several studies that show how insults to the ears could lead to dysfunction in the brain and difficulties listening in noise. He notes that even moderate noise exposure during sensitive periods of development can degrade tonotopic maps and block neural tuning for complex sound features in the auditory cortex.

Pienkowski also notes that unbalanced hearing early in life (e.g., temporary unilateral conductive loss due to otitis media) can lead to imbalances in dichotic listening abilities in the brain (amblyaudia) later in life. These kinds of changes were not limited to children or young animals, with adult cats chronically exposed to narrow-band noise showing suppressed auditory cortical responses to frequencies in that band but enhanced responses to frequencies near to that band. Overall, these studies remind us that altered auditory inputs to the ears can lead to difficulties listening in noise in the brain.

Pienkowski also reminds us of the challenge of separating APD from language and cognitive deficits when considering difficulties listening in noise in the presence of a normal audiogram. He supports continued efforts to improve our ability in this regard, citing the specific example of the Listening in Spatialized Noise – Sentences (LiSN-S) test (developed by the National Acoustics Laboratories in Australia). The

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To EP? or not to EP?

Michael O. Webb, M.S.

Is there a need for auditory evoked potentials (AEPs) in the routine work-up for auditory processing disorders (APD)? More specifically, is there a need for the auditory brainstem response (ABR) in this evaluation process, since consensus has been that typically only the later responses Auditory Middle Late Response (AMLR), ALR & P300 provide relevant diagnostic support? Prevailing opinion would suggest, "No." Financial considerations would likely chime in, "No way!"

Early in my neuroaudiology/auditory processing journey, and influenced by such conventional wisdom, I purchased electrophysiology (EP) equipment, thanks, in large part, to income from hearing aid sales (which subsidized my true professional passions). My motivation was to mine thalamocortical (AMLR) and cortical (ALR & P300) potentials for objective diagnostic—and post-therapeutic—"gold." And to find relevant correlations with my behavioral test battery. I'll share some of my own conclusions (as a clinical, non-research practitioner) about the value (or lack thereof) when it comes to using AEPs.

After 5-plus years of AEP experience supplementing behavioral APD practice, I don't consider myself an expert, but I have distilled down some thoughts and principles about the place of EP in our CAP toolbox.

1.) First, I do believe that AEPs provide valuable information in CAP evaluations, not the least of which is assisting in **differential diagnosis** (especially the ABR). Auditory Neuropathy/Dissynchrony (ANSD), tumors, demyelinating disease, and some mild TBIs (mTBI), to mention a few presenting conditions, can share the symptomology of CAPD, but with radically different etiologies.

If the audiogram and tympanometry are normal, along with speech recognition in quiet, many audiologists will assume that the way is clear for CAP assessment when symptoms indicate the possible need. Without OAEs. Without middle-ear muscle (acoustic) reflexes (MEMRs). And almost always without ABR. A patient with suspected CAPD with such a scant results profile is not ready for behavioral CAP evaluation alone. And if administered would very likely fail on most measures if those divergent diagnoses were present, if auditory processing was primary or not.

ANSD is a *spectrum* disorder and probably has a wider footprint than we realize. The incidence is fairly low (10-15% among those diagnosed as severely/profoundly hearing-impaired), but those cases may represent only the most involved end of the spectrum.

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If You Haven't Used SIR Yet !!!

Jack Katz

The SSW has been around since biblical times. Actually 56 years. The most important improvement in the past 30 years is the Standard Integration Ratio (SIR).

One of the strengths of the SSW and the Buffalo Model is that we do not rely on just one test finding. Rather we look for support from other aspects of the test or battery.

Integration (INT) is the most severe category of the Buffalo Model. It is associated with interaction between the hemispheres and such disorders as Dyslexia. Yet we have not come up with some strong companions. We have long suspected that we were missing other integration cases because some people have so many errors that mask the Type-A (Ty-A).

We know that some of these cases were initially missed. That's because after the first round of therapy when so many of the DEC and TFM errors were elim-inated; out jumped the Ty-A pattern.

We did find *some <u>support</u>* for the INT category from 2 new measures (Two-By-Three or 2B3 and IX/XX). The latter are Integration Delays or Extreme Delays (see Katz, 2015[1]). They increased our confidence but did not identify additional INT cases.

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[1] *Katz, Integration signs-new addition-further support. SSW Reports, 37:2, May 2015, pp 1-6*

SIR to the Rescue!

A number of years ago Larry Medwetsky told me of a procedure that he found very helpful for identifying some of those missed cases. He compared the L-Competing score with the R-C. If the L-C was more than 1 SD poorer than the R-C it was significant indicating INT. I tried it for several years and found that a number of cases that were significant for SIR were not for Ty-A and also vice versa (but not as many). So this was most promising. Now…

Another Look at Integration

We studied 3 groups with 20 children each. These were incidental samples of children I'm seeing, am refiling and one pile of 10 Type-As that I was looking at. Their ages were 6 to 17 years. Group 1 was children who had Ty-A patterns on their first visit. Gr 2 was for those with no Ty-A, but significant SIR on first visit. Gr 3 was for youngsters who had neither of those indicators on first visit. This is the No Gr/Neither Gr

Figure 1 shows the results for those 3 groups for Ty-A difference score (F - X), SIR SD score (# SDs LC > than RC), 2B3 (<u>≥</u>2 of 9 measure of Buffalo battery being <u>≥</u>3 SDs poorer than the mean), and # IX/XX (number).

You will see the mean Ty-A Gr (F-X) score is almost 6. It's not surprising that it is strongly positive, but the average difference is quite large between

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Dear Ackie on Localization of Sound

Dear Ackie, Au.D.

Hello Everyone,

I know that you have been waiting patiently for another column from Dear Ackie. In fact, one of my biggest cheer-leaders Maggie Montanegro-Galant is also a member of your group, so I decided to answer her via TiCAP. Maggie M-G said that she had not heard anything about localization of sound and wondered if people are still doing that important work. You bet we are. So here goes…

Dear Maggie and all others who want to know the lowdown on localization of sound: I had been playing around with localization of sound for a while because I knew it was an important skill both for practical purposes (e.g., knowing where sounds are coming from) and because it is important for understanding speech-in-noise and in my own case feeling more relaxed. It is one of those skills that requires coordination from the left and right pathways.

In 2009 I read, Therapy for APD: Simple, Effective Procedures I was pleased to see that Jack included localization of sound. He wrote about testing localization using the Localization Clock (LoC) system and then providing therapy to improve a person's localization ability.

The clock provides a simple way to measure a person's localization errors simply, quickly, and inexpensively. We put 12 little cards (like a clock) on the floor around the patient's chair. The test circle is about 3 to 4 feet around the center of the chair. We also use the half-hour spots between cards so we have 24 measurement points that are 30^{o} apart. Jack considered localization errors of 30^{o} or more to be significant. Ackie uses a blindfolded for the person to prevent a visual contribution and asks the person to point to my mouth. This way they point to a specific spot instead of a vague/unknown place somewhere on the speaker's body.

Of course Dear Ackie has a mind of her own. I had an elderly man who couldn't or wouldn't point to my mouth, so I had him point to the space between my shoes. That was pretty smart of Dear Ackie, huh?

Localization Clock is like a game for the kids so they are getting helpful practice while they are playing a hide-and-seek kind of game. Unfortunately, for some of the kids we do not have time in our 50 minute therapy sessions, so I show the parents how to do it and how to record the therapeutic results. Even Dear Ackie is amazed how quickly the kids improve their skills.

I have the family bring in the summary sheets so I can monitor the program. If you do not do localization testing or therapy let me give you some information.

Localization Test: Find a space roughly 7'x7' and put a chair/swivel chair in the middle with a real or imaginary

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Listening Difficulties & Normal Audiograms

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simple sentences (the stimuli) and running speech (the noise) presented from in front of the listener. In another condition the sentences are presented from the front but the running speech presented from the sides. By calculating the difference score between these two conditions, the effects of language and cognition cancel out (as these are the same in both conditions). This leaves a measure of spatial processing, or how well the listener used the location of sounds to listen better in noise.

Pienkowski gives us food for thought for the next time we have a client in our audiology rooms presenting with difficulties listening in noise in the presence of normal audiogram. We will, of course, look for APD originating in the brain. But we might now take a second look at our client's ear history for insults to the ear that could have started our client on the path to an APD.

Reference

Pienkowski, M. (2017). On the etiology of listening difficulties in noise despite clinically normal audiograms. Ear and Hearing, 38, 135-148.

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Wayne J. Wilson Ph.D.
The University of Queensland 4072, Australia. <w.wilson@uq.edu.au>

To EP? or not to EP?

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Chuck Berlin and Linda Hood[i] described a case being treated for CAPD, with poor outcomes. Because their second-opinion consultation uncovered previously-missing critical diagnostic information (no MEMRs), the boy was investigated further and found to have ANSD, confirmed by a subsequent ABR. His final diagnosis was Charcot-Marie-Tooth Disease, frequently involving ANSD, among other disabilities.

A recent case report (Iliadou and Eleftheriadis, 2017) described a patient with poor speech perception in the right ear with normal audiogram, tympanogram, OAEs and MEMRs. She was therefore referred for CAP evaluation by her physician, which was abnormal in the right ear. At that point only the CAP findings indicated a problem. Fortunately, an ABR was abnormal in the right and later imaging revealed a large right cerebellopontine angle (CPA) tumor, which required surgical intervention. It's not inconceivable then, that a CAP referral may be the gateway to more precise diagnosis, reducing misguided interventions. And ABR may be the key.

Some of the presenting conditions I mentioned may still do well with CAP intervention, like M.S. or mTBI. But the thorough diagnosis and ancillary treatment of these conditions should not be neglected by complacently launching into APD therapy or device fittings until the diagnostic picture is clear.

2) I have generally found that the conventional wisdom which **down-plays the ABR and emphasizes the later potentials (middle-late, late, and event-related) for CAP assessment** may be short-sighted, when it comes to regular clinical practice.

As an example, P300s were touted as a very critical and valuable potential for contributing to the management of CAPD. So when I purchased AEP equipment, which typically doesn't include P300s as a standard feature, the P300 module was a $4000 add-on. But since it came highly recommended and also offered a different event-related potential, Mismatch Negativity (MMN),[ii] I went for it.

For me, P300's have largely been disappointing. When they behave, they are quite valuable. In other words, when I get robust, clear P300s, they are valuable. When I don't, I'm not sure whether to attribute that to attention and its contribution to the P300's endogenous[iii] response, or to some other factor. While initially, the amplitude of the P300 was considered key (and latency less so), recent discussions with Frank Musiek[iv] have revealed that he feels that amplitude is too variable to be reliable because of its state-attention-dependency. Now latency is considered by some to be more indicative as a P300 finding. So the conventional wisdom evolves. And the formerly-disused (in auditory applications) MMN is now getting new attention in some circles. (I'm glad. My MMN results are

more stable than the P300. I'm hoping for better clinical protocols in that direction.)

The middle-late, thalamocortical potential (AMLR) seems to be the hinge-point in this discussion. It is more reliable, stable, and has better clinical utility for this clinician. (And you don't have to buy the P300 module!) It is an exogenous response, so it's not as subject to cognitive effects, like attention. Use of AMLR for post-therapy assessment, may, in my opinion, be more reliable for revealing wave-alterations resulting from therapy than the more volatile P300. The amount of cortical information will, of course, be more limited than that from a robust, clear P300.

3) Lastly, **don't sell the ABR short for CAP diagnosis**. Not only, as discussed earlier, is its differential diagnosis capability stellar, the ABR also provides subtle corroborative data in many cases of CAPD. Absolute wave latency and inter-wave latency prolongation in CAPD is fairly common, especially in patients with disordered temporal resolution and with abnormal masking-level difference (MLD). A number of recent studies have confirmed latency prolongation and some wave morphology effects in cases of TBI, confirming diminished neural synchrony and resultant prolongation of temporal processing speeds. I have seen slight latency prolongations as a frequent feature of non-traumatic CAPD as well. Amplitude effects in binaurally-summated ABRs are also fairly common in my patients. More formal research is certainly needed.

Conclusion: It's my strong feeling that CAPD specialists should pursue one of the following options, all of which involve the more prevalent usage of AEP data in our CAP diagnostic and rehab practices.

1) Purchase (as a practice or as a co-op) AEP equipment (good used unit?).
2) Develop a strong referral relationship with an outside AEP provider. It is important that such professionals be flexible and cooperative to implement some clinical nuances in their testing, which are indigenous to CAP evaluation and which may not be according to their usual protocols. (This particularly applies to the later AEP responses, which are less commonly used than ABRs.)
3) Make sure you have a firm protocol for what outside audiological evaluations must include to be acceptable, prior to CAPD testing. I suggest PT audiogram, tympanometry, MEMRs [ipsi and contra], word recognition in quiet and noise, as well as OAEs. If needed, use other retrocochlear screenings like MEMR decay or Olsen tone decay tests. Also, reserve the right to re-do or supplement anything that is lacking. And consider making ABR a part of that protocol.

References

[i]Berlin and Hood, Handbook of Clinical Audiology, Ed. Katz; *et. al*. 6th Edition. Ch. 22, pp. 529-541.

[ii] Mismatch Negativity (MMN) is a bridge response (part exogenous, part endogenous) that is event-related but occurs pre-consciously, so that the patient doesn't have to perform a conscious task in response to the odd-ball paradigm used to elicit it.[iii]
An endogenous response means that the patient must exercise attention and perform a conscious task (like silently counting the oddball stimuli) to elicit the response. Endogenous = "born from within." An exogenous response only requires the external stimulus to elicit the response, apart from any conscious activity.

[iv] Musiek, Frank, Ph.D., Professor, University of Arizona Department of Speech, Language, and Hearing Sciences. CAPD/Neuroscience Researcher/Author. Editor: Pathways. (Personal Communication)

Iliadou and Elefthiriadis, Journal of the American Academy of Audiology/ Volume 28, Number 1, 2017, pp. 91-101.

If You Haven't Used SIR Yet !!!

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columns X and F. At the same time the other 2 groups scored .5 or less. Yes, they were chosen because they did not have Ty-A.

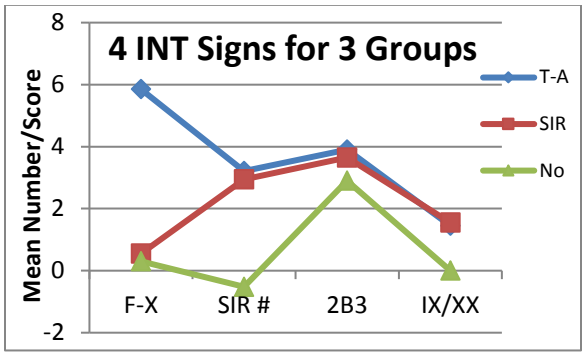

Figure 1. Mean difference (F-X) for Type-A and the SIR score as well as the mean number of significant 2B3s and integration/extreme delays for the 3 groups.

Of course, the SIR group, had a pretty big SIR score (≥1 SD difference is significant) but interestingly the Ty-A group was equal to it. That was despite 2 Ty-A Gr cases who didn't have significant SIR scores). While both INT groups had significantly positive differences the No-Gr had a slightly negative mean score.

The supportive measure 2B3 was similar for both INT groups. But, the No-Gr had almost as poor a score. In part, 4 of the 20 No-Grs were likely false-negative-INT subjects that had high positive 2B3 scores. Nevertheless, that score was greater than expected.

The 4th column shows the number of Integration and Extreme Delays. As in the past there were not as many of these as 2B3s and ever so slightly more likely to be found in the 2 INT groups.

So far, SIR looks like a pretty good measure of INT.

Assessing for Integration Issues

The Type-A Group

These children were chosen only because they had the Ty-A. Of the 20, 16 had SIR as well. So that shows an impressive relationship between these 2 strong indicators of INT. Actually, all 20 Ty-As were significant for INT. This was because each of the other 4 cases had significant 2B3 in addition to 16 that had both strong signs. And 3 of the 4 also had IX/XX. For the Ty-A Gr one child had all 4 INT signs, 15 had 3 and 4 had 2 signs. So, all 20 were positive for INT.

In the Ty-A Gr, 11 had retests; of these 4 had Ty-A and 6 had SIR. So even without specific training for dichotic listening about half of the retests showed normal Ty-A and/SIR on retest.

So for the 20 Ty-A Gr 100% met the criterion for INT. Seventy-five percent had 3 significant INT findings, 5% had 4, and 20% had just the required 2 indicators.

The SIR Group

For this SIR-Gr (by definition) they did not have Ty-As; so their maximum INT signs were 3. Six of the 20 had 3 and 12 had 2 indictors. The remaining 2 had only SIR to indicate INT. Fortunately, they both had retests. One had both Ty-A and SIR on retest while the other had neither. So of the 2 that were positive for just SIR initially; one was clearly supported as INT on retest. Ninety-five percent of those who had SIR showed it was a powerful sign of INT (even with no Ty-A).

The 18 with significant 2B3 gave support to the SIR for 90% and the remaining 2 did not have significant IX/XX either. But IX/XX gave further support to 6 cases for the INT category.

So for the SIR Gr 90% appear to be correctly identified as INT with 5% likely false negative (i.e., actually had INT) and 5% false positive (not likely INT).

Neither INT (Sign) Group

The No-Gr is important to study to see there may be additional INT cases with no strong signs. Because this group had no strong signs the criteria say we cannot demonstrate INT, but can we be sure that they don't have it?

For the No-Gr; 11 of the 20 had significant 2B3s (as suggested in Figure 1) and just 1 had IX/XX. For the No-Gr 11 had 1 INT supportive sign, 1 had 2 signs and 8 had neither of them. So these results are distinctly different than the 2 INT groups. Only 1 child had as much as 2 *support* signs.

In addition, several cases appeared questionable, I wondered if they might have INT. One of them had the most significant 2B3s of all 60 subjects with 9. Another had 6 scores $\geq$ 3 SDs poorer than the mean. That score put her in the top 13th percent of all the subjects. Another child in the No-Gr had 4 2B3 signs. I checked all 3 of these questionable cases and on retest all of them had Tp-A, SIR or both! That convinced me that they were false negative for INT.

These results are similar to the Katz & Medwetsky findings). Based on the correction for the 3 false negative cases Figure 2 shows the more likely result.

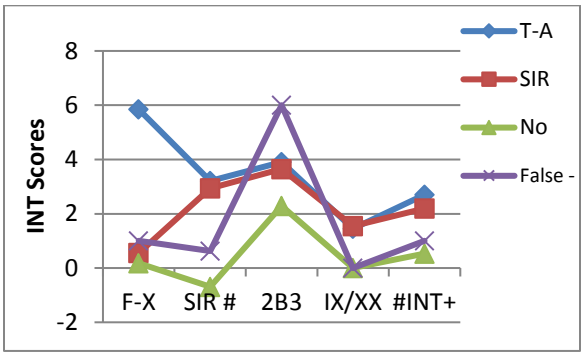

Figure 2 shows the INT mean results for the 3 groups: Ty-A, SIR and 16 No Gr as well as medians for the 3 subjects who likely have some INT issues but not Ty-A/SIR on initial test.

Does this improve clinical practice??

In a word, "Wow".

a. INT is likely the most severe APD
b. We had 1 strong sign since 1966

c. We now have 2 strong INT signs and 2 supportive signs
d. Suggestion: the higher 2B3 the more likely INT, probably same for is true for Ty-A, SIR, and IX/XX, but it's not an INT diagnosis
e. The addition of SIR with the help of the 2 support signs could double the number of INT diagnoses
f. But we need more research

Summary

The 4 INT indicators form a powerful array of finding that produce a high hit-rate and a low false-positive rate. We have no 'gold standards' in APD, but these data look very good and make logical sense. Not only are the 2 INT signs (Type-A and SIR) powerful but they relate to the supportive signs (2B3 and IX/XX) that are very different tasks but tend to support one another.

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Jack Katz <jackkatz@buffalo.edu>

Dear Ackie on Localization of Sound

[Continued from page 5]

circle around it about 7' in diameter. Put the '#12' card, on the circle directly in front of the chair. Then '6' directly behind the chair etc. for each position.

Test Procedures

For the test the person is a blindfold (but not for therapy). We demonstrate the task standing at 12 o'clock with the person without the blindfold. "Each time I say 'test' I want you to point to where you think my mouth is. Make believe you can't see me. When I say 'test' please point to my mouth." Often the person will point to your body, so this is a good opportunity to reinforce pointing to your mouth. Do it again so the person does it correctly. Then put on the blindfold and begin the test.

If you have the therapy book you can copy out the response sheet. If not make up a sheet with the 24 times sort of randomly listed and a place for entering the response (time) or a dot if correct and a column the +/- difference.

When giving the word, 'test' face the person's closest ear and project the word clearly. When the test is over calculate the differences and plot them on a chart to see the person's error pattern. I addition to the weak regions we also look at 3 factors (# Off Target, Off By Hours, and Maximum Hours Off) and can compare the findings to other children with CAPD who have taken the LoC test. This enables a rating of normal to severe which may suggest whether to do therapy or not and whether to ask the family to carry out the procedures.

Figure 1 demonstrates a child's pretest performance. You will note that when standing half way between 1:00 and 2:00 o'clock (i.e., 1:30) that the response was +2. Therefore, it sounded to the person as though it was spoken from 3:30. So that is simple for most + or – responses. However, at 1:00 the response was -2:00. So we count back 2 hours and that is 11:00. This is an exception in understanding where the person pointed.

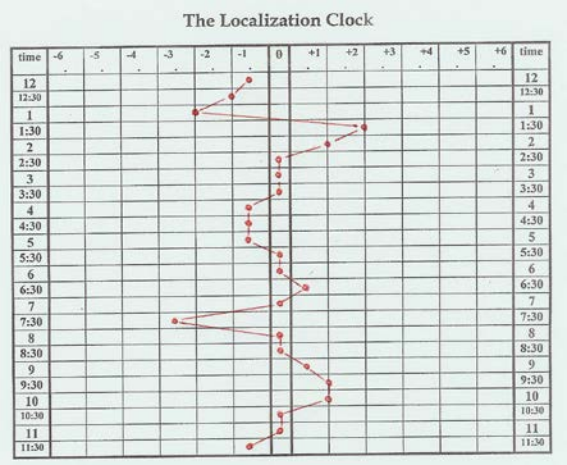

Figure 1 shows the results of a LoC test for a child who had Moderate performance initially. The top axis '0 column' shows correct localizations. Minus & plus hours/half-hours are shown to each side for each of the 24 presentations (from Katz, 2009).

Therapy Procedures

Once we know there are significant problems in localizing sound we begin as we do with B-M therapies, with an easy challenge. We begin with the per-

son's eyes closed and facing forward (toward #12). A noise source is used to conceal the tester's movements (as we do in the test). The word we use is "test" because it is appropriate to the situation and because it is brief and weighted more with high frequency sounds. I have not tried a low frequency word (e.g., moon) that would exercise intensity differences between the 2 sides as opposed to the temporal measures used for the high frequencies.

When there is a teachable moment the person is asked to open their eyes and see where the speaker is actually standing. Then the word is repeated with the person facing forward and eyes closed.

For therapy we follow a protocol (can be copied from the therapy book) that is not known by the person. In the first session the person is told that you will be in one of 4 positions, right in front (#12), back (#6), left (#9) and right (#3). The person points with their forefinger.

Each session adds positions unless the person needs some work on the previous one. See Figure 2 for the results of an initial test and retest. The person was Off Target = 20 (sev), Off By Hours = 18 (sev) and Maximum Hours Off = 3 (mod), so localization appears to be severe. Following therapy the 3 criteria were reduced to 13 (nor), 6.5 (nor), and 1 (nor). So it appears that the therapy was highly successful going from severe to normal after a brief therapy period.

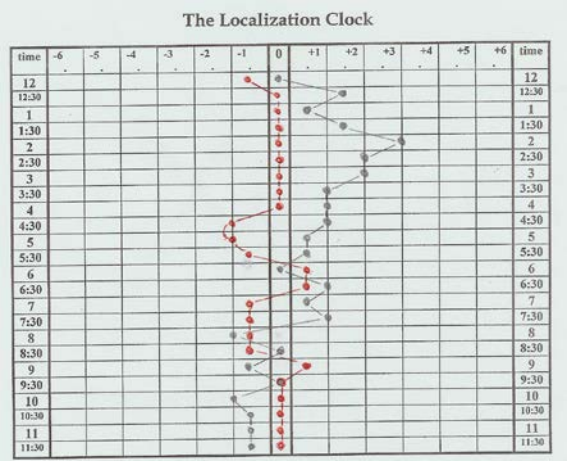

Figure 2. Retest following several sessions of LoC training. The black dots show the initial test and the red show the retest. The important improvement was from about 1:30 to about 3:30, but it looks like there was some over-compensation from 4:30 and 5:30 (from Katz, 2009).

The Benefits of Localization Clock

1. LoC is easy to give
2. Requires no equipment (I cover my tracks using a remote to play a noisy CD when changing positions)
3. Needs little space
4. Shows quick positive results
5. Can be given by parents at home
6. Can indicate where and whether therapy is needed
7. Kids enjoy LoC especially when you sometimes let them see that they were correct.

Reference

Katz, J. (2009). Therapy for Auditory Processing Disorders: Simple, Effective Procedures. Educational Audiology Association: Westminster, CO.