

**W**hile electrophysiologic measures often are used for diagnostic purposes, the outcomes of this testing can be extremely useful also in the selection and fitting of hearing aids. For example, some of these measures assist in predicting hearing thresholds—important for determining hearing aid gain and output settings. Aided measures can be used for verification of the fitting; this was discussed by Harvey Dillon on these pages last year. And, electrophysiologic results also can be used to monitor the effects of amplification and rehabilitative audiology over time.

This is all good, assuming that it leads to appropriate decisions regarding the need for and function of amplification. But all is not simple when the various signals used for electrophysiologic testing are processed through modern hearing aids. And clinically things can become even muddier when the electrophysiologic expert is not the hearing aid expert, and vice versa. Perhaps a Page Ten article on this topic would be a good idea?

Our guest author this month is **Kelly Tremblay**, PhD, associate professor at the University of Washington in Seattle. Dr. Tremblay is an internationally known researcher in the area of electrophysiologic measures, and has received numerous awards for her research. She serves on the editorial board for several audiology journals, and publishes extensively herself. In addition to her journal articles, look for her book chapters in the soon-to-be-released book *Auditory Evoked Potentials Basic Principles and Clinical Application*. Dr. Tremblay also provided us with an excellent Page Ten article a few years back. If you happened to miss it, you can find past Page Ten articles at *The Hearing Journal* archives at [www.audiologyonline.com](http://www.audiologyonline.com).

Although many of you think of Kelly as a researcher, she also has been a *clinician* for 20 years, trained by none other than Richard Seewald back at Dalhousie University in Halifax in the 1980s. Her clinical background is apparent in her research, which often involves the search for answers to important clinical questions. Kelly's roots are in Canada, and she is spending this year on sabbatical in Toronto.

At Dr. Tremblay's University of Washington web page, she displays the following quote from Thomas J. Watson of IBM fame: "The ability to ask the right question is more than half the battle of finding the answer." Kelly and her colleagues are definitely asking some good questions about electrophysiologic measures of hearing aid performance, and some of their findings are surprising. You'll enjoy reading about them on the following pages.

**Gus Mueller**  
Page Ten Editor

## Hearing aids and the brain: What's the connection?

By Kelly L. Tremblay



Tremblay

**1 I remember that you wrote a Page Ten article 3 or 4 years ago about "plasticity" of the auditory system. Are you going to provide us with an update?**

I certainly am. But first, how about a quick review? The term "auditory plasticity" is sometimes used to describe the brain's capacity to change. We typically think of hearing as it relates to the ear, but sound travels along many nerve fibers and through many nuclei before reaching the auditory cortex. Along these pathways, the acoustic content of the signal (i.e., frequency, intensity, and timing information) is coded by highly organized neural systems.

Even though we are typically born with the capacity to code this acoustic information, our brain—specifically the central auditory system—"changes" as a function of auditory deprivation and stimulation. It reorganizes itself throughout our lifespan according to the auditory input that it receives.

**2 What do you mean when you talk about "deprivation" and "stimulation"?**

Think of the saying "Use it or lose it." The central auditory system of a person who has been diagnosed with a conductive or sensorineural hearing loss has experienced *deprivation*-related plasticity. For example, physiological maps in the brain used to code frequency information change when they have not been activated for a period of time.

**3 So what happens when you fit the person with hearing aids or a cochlear implant?**

A couple of things are presumed to take place. First, introducing sound to a previously deprived auditory system again alters the way spectral and temporal information is represented in the central auditory system. Irvine and Rajan refer to this process as "stimulation-induced" or "use-related" neural plasticity.<sup>1</sup>

Second, hearing aids and cochlear implants, through signal processing circuitry, modify the content of the incoming sound and deliver a modified signal to the central auditory system. In a sense, these modified signals are new signals because they are unfamiliar to the listener and so are likely to stimulate the auditory system in a new way. It is widely believed that people *learn* how to relate the modified signal, and the altered neural spectral and temporal codes, to an existing memory of sound.<sup>2,3</sup>

**4 Could this influence the success of a hearing aid fitting?**

Most definitely. Hearing healthcare providers are typically trained to approach re/habilitation by improving audibility (by means of hearing aids, cochlear implant, or assistive listening devices). Obviously, this is a critical first step. But successful rehabilitation

depends also on the central auditory system's ability to represent and integrate new spectral and temporal information delivered by a hearing prosthesis.

## **5 Do you think what you just described underlies some of the performance variability seen among hearing aid users?**

It's possible that the degree of benefit a particular patient receives from hearing aids (or a cochlear implant) depends in part on the ability of that person's system to adapt to the modified cues delivered by the device. People who don't experience significant benefits from cochlear implants may have auditory systems that are less plastic, i.e., less capable of representing *new* cues.

Additionally, poor speech perceivers may have more difficulty *learning* how to associate these new neural patterns with an existing memory of sounds. It's this rationale that underlies the use of auditory training exercises—yet another form of auditory stimulation.

## **6 What exactly do you mean by “auditory training”?**

Auditory training is a treatment program that uses repetitive listening exercises to improve a patient's ability to perceive auditory events. These exercises are sometimes conducted with a listening partner at home or during weekly sessions with an audiologist. For people who are computer savvy, interactive computer programs are also becoming popular. In any event, a signal is provided, the person executes a task, and feedback (describing a correct or incorrect response) is given.

Auditory training programs may be categorized as analytic, synthetic, or a combination of the two (for a review, see Sweetow and Palmer, 2005<sup>4</sup>). Let me explain. Analytic (or bottom-up) training focuses on the acoustic content of the signal, and the task involves identifying or discriminating sounds that differ acoustically. One example might involve learning to identify certain consonant-vowel syllables (e.g., “thee”) from a set of sounds that are acoustically similar (e.g., “vee,” “fee”). The idea is that if a person can learn to differentiate acoustic cues, this ability might in turn contribute to improved speech perception. By this I mean that a patient might be less likely to confuse the consonants “th” and “v” when they occur in the context of a word and/or sentence.

## **7 That seems like a reasonable approach. But, how does it differ from the synthetic method?**

Synthetic (top-down) training is designed to improve a person's ability to attend to, integrate, and use contextual information. Many examples can be found in the literature, but I get only 20 questions, so I'll keep this short and describe just one of them.

Think of a task in which a person is asked to repeat a word or a sentence in the presence of competing noise. At first, the signal-to-noise ratio (SNR) might be favorable, so the person may have no problem completing the task. Eventually, the SNR is lowered so the task becomes more difficult.

The purpose of this type of exercise is to stress the system so

that the patient can improve his or her ability to separate signals from noise. Because SNRs are often poor in real-world listening environments (think restaurants, classrooms), the goal of this type of training exercise is to improve a person's ability to “hear” in noisy listening environments.

## **8 I've been hearing about these types of training programs lately. What can you tell me about research in this new area?**

It's interesting that you describe auditory training as a “new” area, because it's been part of audiology for decades. Carhart, for example, described the importance of auditory training as part of the profession of audiology.<sup>5</sup> However, because the effectiveness of auditory training has been questioned and reimbursement for rehabilitative services has become more difficult, many clinics have shifted their focus to simply dispensing devices. For this reason, training exercises are rarely used in clinics and audiologists are often unfamiliar with this approach to rehabilitation.

But to get back to your question, the concept of auditory training has attracted a lot of attention recently. Training programs designed to improve the auditory perception of children with listening, learning, and language problems have become prevalent over the last decade. Many of these training programs would fall under the “analytic” category and are designed to improve the acoustic representation of sound.

## **9 What about people with hearing loss? Are these programs being used with them as well?**

Yes, we're seeing a resurgence of training programs designed to improve the speech perception of people with hearing loss. One reason for this resurgence, I believe, is all the information we have acquired recently about brain plasticity.

Over the past decade, animal and human experiments have taught us a lot about physiological and perceptual plasticity. The ability to alter neural response patterns, through training, is provocative and the translation of this basic science to auditory rehabilitation is obvious for those of us interested in the rehabilitation of hearing-impaired patients. Now the challenge is to extend these neuroscience principles successfully from the laboratory to the clinic.

## **10 Is there any research evidence showing that these training programs work?**

A literature search will uncover many training experiments that report varying degrees of training-related improvements in performance scores. However, the important question is, what constitutes “significant improvement.”

It is not uncommon to observe statistically significant gains of 5%-10% (or more) on a specific test following training. However, does this numeric change translate into significant changes in the ability to communicate in the real world? Ultimately, the answer to your question, “Do these training programs work?” will come from hearing aid and cochlear implant users.

## **11 Here we're talking about auditory training, but I know you're an electrophysiologist. So, I have to ask, can you use electrophysiological measures to determine if hearing aids alter the neural representation of sound? Or to measure the effects of auditory training?**

Okay, now we're getting to the good stuff! As you might have guessed, this is a question we're working on, so I'll share with you what we know so far. Tempting as it is to start recording electrophysiological measures after fitting a hearing aid, or before and after training exercises, things are not that simple.

## **12 What do you mean?**

Well, audiologists are trained to recognize how important their choice of stimulus, rise-time, rate, and presentation level is when they are recording auditory evoked potentials (e.g., the ABR). This is because the physical characteristics of the stimulus greatly affect the morphology (latency and amplitude) of the evoked response.

Now, think about hearing aids and cochlear implants. These devices modify the physical characteristics of sound. Hearing aids introduce noise, compress signals, and alter the frequency content of the signal. So, when evoked potentials are recorded using sound delivered through a hearing aid, the effects of the hearing aid processing on the physical characteristics of the sound likely affect the evoked neural response pattern. This interaction might seem obvious, but you'd be surprised to discover how rarely these issues are taken into consideration when you read publications.

Here's the take-home message: We know very little about how signal processing (through a hearing aid or cochlear implant) affects neural activity. Therefore, we need to be careful not to assume that our interpretation of latency and amplitude patterns recorded in aided and unaided conditions reflect similar neural properties.

## **13 I need more specifics. Let's start with the aided ABR. There is research on this topic, right?**

Oh yes, research using aided ABR recordings can be found in the literature.<sup>6-12</sup> Unfortunately, however, recording aided ABRs proved problematic for several reasons. First, because clicks or tone-bursts are very brief, they do not activate the hearing aid circuitry in a way that longer-duration signals, such as naturally produced running speech, do. Also, brief stimuli are often distorted by the hearing aid and introduce artifacts. Other confounding variables include stimulus presentation rate and how compression characteristics of the hearing aid interact with the stimulus onset.<sup>13</sup>

Another important point to keep in mind is that the delay characteristics of the digital processor in digital hearing aids, often referred to as "group delay," will interfere with the onset response of the ABR. This is further complicated by the fact that the delay in digital hearing aids varies significantly across frequency and among hearing instruments.<sup>14-16</sup>

## **14 I know you've done a lot of work with cortical auditory evoked potentials. Do these concerns apply to cortical potentials as well?**

Absolutely. In fact, they likely apply to all physiological measures that are sensitive to the acoustic content of the signal. When sound is processed through a hearing aid (or cochlear implant), the content of the signal is modified. To date, though, we know very little about the effect of these acoustic modifications on the physiological encoding of sound.

This point might also explain the inconsistency of the findings in the limited number of studies that have recorded cortical evoked potentials in hearing aid wearers.<sup>17-22</sup> In most cases, the functional status of the hearing aid, and circuitry information, was not reported.

## **15 Does this mean we shouldn't measure the physiological responses of hearing aid wearers?**

No. It just means we need to learn how hearing aid processing interacts with the neural representation of sound. Whether you're estimating aided thresholds or assessing stimulation-related changes in the brain following amplification or auditory training, when sound is delivered through a hearing aid, the hearing aid itself becomes a source of variables.

## **16 Do you have an example of what you mean by that?**

Sure do. Let me tell you about a recent experiment we did. We asked a very simple question: Does hearing aid amplification (providing approximately 20 dB of gain) affect the latencies and amplitudes of P1-N1-P2 responses?

Just as with ABR responses, we would expect P1-N1-P2 peak latencies to become shorter in latency and larger in amplitude as the intensity of the evoking stimulus increases. In this study, however, instead of increasing the intensity of the stimulus using the evoked potential equipment, we used the hearing aid to provide 20 dB of gain.

We presented a speech sound in soundfield at 64 dB SPL (a level similar to conversational speech) and tested participants in unaided and aided conditions. Remember, in theory, when sound is amplified by a hearing aid (e.g., 20 dB), we would expect P1-N1-P2 peak responses to be larger in amplitude (strength) and shorter in latency (neural conduction travel-time) when compared with unaided neural responses. This assumption is based on decades of research on the P1-N1-P2 response demonstrating that increases in stimulus intensity, even as small as 2 to 3 dB, result in shorter peak latencies and increased peak amplitudes.<sup>23,24</sup>

So, are you ready for the results? Despite 20 dB of gain provided by a hearing aid, we saw no significant differences in peak latencies or amplitudes when comparing unaided and aided recordings.<sup>25,26</sup>

## **17 That's very strange! How do you explain it?**

I don't know. It's bizarre. Why wouldn't 20 dB of gain affect peak latencies and amplitudes? What I can tell you is that these results show that the physiological encoding of sound deviates from our textbook rules (which are based on unaided recordings) when sound is delivered through a hearing aid. Presumably, the hearing aid is introducing some type of effect.

## 18 What type of effect?

A number of possibilities come to mind. We're currently examining the effects of signal-to-noise ratios and compression on evoked neural activity. As an example, because the stimulus input level was 64 dB SPL and compression was activated at output levels approximating 70 dB SPL, compression activation might have altered the signal in a way that interfered with the physiological detection of the 20-dB gain in intensity.

While some of these possibilities might be obvious to hearing aid experts, they might not be to electrophysiologists, who are often unfamiliar with hearing aid processing. So, the point I want to emphasize here is this: When you're recording evoked potentials in response to sound that's been processed by a hearing aid (or cochlear implant), two specialty areas are being combined (hearing aids and electrophysiology).

It's important to understand variables specific to each of these fields, as well as potential interactions, if you want to understand the aided evoked recordings. Without a detailed understanding of amplification effects on neural encoding, conclusions about brain plasticity related to hearing aid amplification might not be valid. For example, individual evoked response patterns might reflect differences among hearing aid processors rather than differences in brain activity. Or, inadequate gains following auditory training could be erroneously attributed to reduced plasticity of the person's auditory system when, in fact, the inadequate gains were related to reduced audibility or distortion introduced by the hearing aid.

## 19 So what's your advice to people interested in recording aided evoked potential measures?

I'm going to sound like a broken record, but I'd tell them to resist the temptation to ignore the hearing aid and spend some time figuring out what the device is doing to the signal. Whether you're interested in estimating aided thresholds or assessing stimulation-related changes in the brain associated with amplification, when sound is delivered through a hearing aid, the hearing aid itself becomes a source of variables. We recently published separate articles that provide tips for quantifying the output of the hearing aid<sup>27</sup> and discuss how these measures can assist in interpreting evoked cortical responses.<sup>28</sup>

## 20 So now you have most of the answers, right?

Ha, you're funny. We're just getting started!

So here's my plea to readers: Consider returning to school and completing a research degree (a PhD)! We need more scientists

with clinical experience to take on issues like these and to re-ignite the excitement of auditory rehabilitation. Whether or not evoked potentials play a role in our quest to improve the communication abilities of hearing-impaired people, combining the expertise of clinicians and scientists will undoubtedly translate into new, and hopefully more effective, approaches to auditory rehabilitation.

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