Introduction

Descriptions of brain plasticity and perceptual learning were once limited to scientific journals and academic textbooks. Recently, however, these concepts have been expressed in mainstream media and pop culture. Computer games purported to improve cognition and memory, as well as halt biological aging processes, can be found in stores that sell video games. With therapeutic goals in mind, the merging of science and technology has resulted in many types of commercially available training programs. With increased public awareness, individuals are learning that the brain, like the body's muscles, can be altered with exercise.

A common motivational theme underlying training programs is that the brain is “plastic”; meaning it has the capacity to change as a function of experience (Eggermont 1990). With this principle in mind, exercises are designed to activate the brain in ways that help remediate perceptual and/or cognitive functioning. While the technological approaches to exercising the brain might be new, the concept of modifying auditory perception through training is not. The idea that we can alter our perception of sound with training can be found in scientific archives. More than a century ago, Meyer (1899) asked the question: “Is the memory of absolute pitch capable of development by training?” If the answer was yes, the author states they would have to assume that the trained individuals possess a physiological property that distinguishes them from people who cannot acquire this ability (Meyer 1899). However, exactly what these physiological properties might be, the author acknowledges, would be difficult to determine. To a certain degree the author was correct. A century later, we are still searching for the biological mechanisms involved in perceptual learning and how they might differentiate learners from non-learners.

What is Perceptual Learning and why is This Concept Relevant to Audiologists?

Psychologists, neuroscientists, biomedical engineers and school teachers will likely provide various definitions of the term “perceptual learning”; moreover, the definition itself has changed with time (Gibson 1963; for reviews see Goldstone 1998; Fahle 2005). That being said, “perceptual learning” can be defined as a process that invokes relatively long-lasting changes to an organism’s perceptual system in a way that improves its ability to respond to its environment. Within the context of audiology, perceptual learning has been described as the process by which a hearing-impaired adult learns to make use of acoustic information (unfamiliar sounds) when they are first fit with a hearing aid or cochlear implant (Watson 1991; Robinson and Summerfield 1996), or learning to localize sound with only one ear, following a sudden loss of hearing in the other ear (Moore, Peters and Glasberg 1993; King et al. 2001).

While there might be reason to question the purported effectiveness of video games to reverse the aging process, or remediate various perceptual disorders, at the root of the training movement is scientific evidence that sometimes gets lost in translation. Take, for instance, the auditory system. Once sound passes through the peripheral auditory system, it travels along many nerve fibers and through many nuclei before...
reaching the auditory cortex. From the ear to the brain, spectral and temporal acoustic information contained in the signal is represented using place and timing codes. For instance, neural response patterns in the auditory cortex have been shown to reflect perceptually relevant time-varying parameters that correlate with the perception of fundamental frequency, voice-onset-time (VOT), and other important acoustic speech cues (Phillips and Hall 1990; Steinschneider, Schroeder, Arezzo and Vaughan 1994; Eggermont 1995; Steinschneider, Reser, Schroeder and Arezzo 1995; Steinschneider, Schroeder, Arezzo and Vaughan 1995; Schreiner, Mendelson, Raggio, Brosch and Krueger 1997; Steinschneider, Volkov, Noh, Garell and Howard 1999; Ahissar et al. 2001).

It was once believed that these physiological patterns/codes were hard-wired and resistant to change. However, research has since shown that the central auditory system changes as a function of experience, reorganizing throughout the lifespan according to the auditory input that is available to the individual. Sometimes referred to as “deprivation- or injury-related plasticity” in the case of hearing loss – or “use-related plasticity,” when sound is reintroduced to the auditory system (Irvine, Rajan and Brown 2001) – modified sensory maps, synaptic alterations and neurochemical changes can be seen following periods of auditory deprivation and auditory stimulation. This means the typical individual fit with a hearing aid has experienced significant deprivation-related changes in the central auditory system prior to being fit with the device (Willott 1996; Palmer, Nelson and Lindley 1998; Chisolm, Willott and Lister 2003).

When fit with a hearing aid, additional forms of plasticity are presumed to take place (Willott 1996). First, when a hearing aid increases the intensity of a signal, areas of the auditory system that were once deprived of sound are now being stimulated. Experiments that use animal models have demonstrated that electric and acoustic stimulation of a deprived auditory system modify the central auditory system (Javel and Shepherd 2000). Second, signals processed through a hearing aid or cochlear implant are modified; hearing aids alter the stimulus rise time of the incoming signal, and amplitude overshoots can occur from circuitry activation. Thus a “modified” signal is delivered to a previously deprived auditory system. In essence, this modified signal is a “new” signal that is likely stimulating “new” neural response patterns in the central auditory system. Finally, it has been stated that people “learn” how to relate the modified incoming acoustic information, received via the hearing aid – with altered neural spectral and temporal codes – to an existing memory of the sounds of speech (Watson 1991; Robinson and Summerfield 1996).

**Learning? Adaptation? Or Acclimatization?**

It might be intuitive to think that people with hearing loss need time to “learn” how to hear with their new hearing aid or cochlear implant; however, evidence to support this notion is unclear. On the one hand, steady increases in perceptual performance are reported during the first few years following cochlear implantation (Tyler and Summerfield 1996; Tyler, Parkinson, Woodworth, Lowder and Gantz 1997). In fact, perceptual experiments suggest it can take up to 30–40 months following cochlear implantation for some individuals to make maximal use of their hearing prosthesis. Purdy, Kelly and Thorne (2001) report that perceptual and physiologic changes can occur in a shorter period of time. They followed two adult cochlear implant patients for 9 months and measured auditory cortical evoked potentials and speech perception measures at 1, 3, 6, and 9 months after implantation. Perceptual and physiologic changes were observed within a few months following implantation.

On the other hand, Humes, Wilson, Barlow and Garner (2002) report only modest perceptual changes in hearing aid users after one or two years of hearing aid use. They used several speech recognition measures, as well as self-assessment measures, to evaluate hearing aid benefit. Although there were some occurrences of improved perception over time, subjective measures of hearing aid benefit also declined with time. In some instances performance was significantly worse (less benefit) at both the 6-month and 1-year follow-up sessions, than at 1-month after the initial hearing aid fitting. These results are consistent with others who suggest hearing aid benefit increases very little over time (for a review, see Turner and Bentler 1998).

For neuroscientists and clinicians, it is important to understand the effects of hearing aid amplification and cochlear implantation on the mature brain. We therefore ask, does use- or stimulation-related plasticity exist in these populations, and might any effect be different for hearing aid versus cochlear implant users? If physiological changes take place, would we describe physiological changes as correlates of perceptual learning, adaptation, or acclimatization?

The words “learning”, “adaptation” and “acclimatization” are sometimes used interchangeably. There are also instances when clear differentiations in nomenclature...
ure are made (for a review, see Mazess 1975). For example, it is not uncommon to describe adaptation in the context of evolution as a slow process that occurs over many generations due to natural selection. In contrast, acclimatization is sometimes defined as a process that occurs within a single lifetime. A frequently cited example of acclimatization is the ability to acclimate to changes in altitude after spending time at a particular elevation. In the context of hearing aid use, observed changes in perception that occur over time (with hearing aid use) have been described as acclimatization; and thus far evidence for the existence of hearing aid acclimatization is mixed (Saunders and Cienkowski 1997; Turner and Bentler 1998; Humes et al. 2002; Philibert, Collet, Vesson and Veuillet 2002, 2005; Humes and Wilson 2003; Munro and Lutman 2003, 2004).

If a portion of the population does not report being satisfied with their hearing aids, and performance does not improve with time while the individual is using their hearing aid and interacting in various communicative environments (acclimate), then there is motivation to find other ways to intervene and enhance performance. One approach is to revisit the old concept of auditory training.

**Auditory Training**

There is a large body of literature documenting that people with and without hearing loss, can improve their perception of sound through listening training. The effects can be long-lasting, and in some instances generalize to stimuli and environments outside of the training condition. It is for this reason training-related gains in perception are often attributed to and described as perceptual learning.

Sometimes categorized as: “analytic” (bottom up), “synthetic” (top down), or a combination of both, auditory training programs are designed to improve the ability to perceive auditory events through repetitive listening exercises (Sweetow and Palmer 2005). Analytic training emphasizes the acoustic content (spectral, temporal, and intensity cues) of the signal, and the individual is asked to identify or discriminate sounds that differ acoustically. Synthetic training is designed to improve perception by enhancing a person’s ability to attend to, integrate, and use contextual information.

Physiological representations of sound also can be modified through the use of training exercises, and training-related changes in physiology can coincide with improved perception. Sometimes described as “learning-related plasticity,” animal studies have demonstrated that training can alter the physiological representation of sound. Bakin and Weinberger (1990), for example, used a classical conditioning paradigm to investigate changes in cortical receptive fields in guinea pigs. Animals that were exposed to the conditioning paradigm showed an increase in the neural response magnitude to the conditioning stimulus frequency, and a reduction in response magnitude for non-trained frequencies. They also found that the altered receptive fields were retained for as long as eight weeks post-training.

Training-related physiological changes have been attributed to a number of different processes; including, but not limited to: 1) a greater number of neurons responding in the sensory field, 2) improved neural synchrony (or temporal coherence), and 3) neural “decorrelative” processes whereby training presumably decorrelates activity between neurons, making each neuron as different as possible in its functional specificity relative to the other members of the population. In other words, information that is common to two stimuli is disregarded, while responses to unique features of each stimulus are enhanced (Barlow and Foldiak 1989).

Whatever the actual neural mechanisms underlying training-related gains in perception might be, the ability to alter neural response patterns through training is provocative. Because there is a great deal of performance variability among hearing aid and cochlear implant users, the ability to alter the physiological representation of sound (using training exercises), in a way that improves perception, is a new way to view auditory rehabilitation (Tremblay and Kraus 2002; Tremblay 2003; Neuman 2005; Tremblay 2005; Souza and Tremblay 2006; Tremblay, Billings, Friesen and Souza 2006).

That being said, auditory training in its current form (computer exercises, video games, etc.) might appear new, but focused listening training exercises have been a core part of auditory rehabilitation for years (Carhart 1960). However, because the efficacy of auditory training has been questioned, and reimbursement for rehabilitative services has become increasingly difficult, the focus of many centers has shifted to simply dispensing devices, and less about how to listen with the device. For these reasons, training exercises are rarely used in clinics, and audiologists are sometimes unfamiliar about this approach to rehabilitation (for a review, see Ross 1997). For example, in 1980 the number of audiologists who reported providing auditory training services was 31%. By 1990, the number decreased to 16% (Schow, Bal-sara, Smedley and Whitcomb 1993).
What are the Physiological Effects of Amplification and Auditory Training in Hearing Aid Users?

Little is known about the physiological effects of auditory training in people who wear hearing aids and/or cochlear implants. In fact, we know very little about the effects of hearing aid amplification itself, aside from training, on the human central auditory system. For this reason, there is interest in using auditory evoked potentials to study the effects of hearing aid amplification on the brain (for a review, see Souza and Tremblay 2006). Auditory evoked potentials (AEPs) are bioelectrical potentials that are time-locked to an auditory event. The evoked responses are typically defined according to their polarity (positive [P] or negative [N]) as well as their amplitude and latency. Amplitude describes the strength of the response in microvolts (µv). Latency describes the amount of time, in milliseconds (ms), that it takes to generate the bioelectrical response following stimulus onset.

The auditory brainstem response (ABR) is an evoked potential that has been used to examine the physiological representation of amplified sound in the human auditory system; but its purpose was to estimate aided thresholds, rather than acclimatization or perceptual learning (Kiessling 1982; Kileny 1982; Hecox 1983; Mahoney 1985; Beauchaine, Gorga, Reiland and Larson 1986; Davidson, Wall and Goodman 1990). Recording aided ABRs proved to be problematic because click and tone-burst stimuli are brief in duration and they do not activate the hearing aid circuitry in the same way longer duration speech signals do. There also were confounding variables such as stimulus rate and compression characteristics of the hearing aid (Gorga, Beauchaine, and Reiland 1987). In digital hearing aids, for example, the delay characteristics of the digital processor can interfere with the onset response of the ABR. This is further complicated by the fact that the delay in digital hearing aids varies across frequency (Stone and Moore 1999; Kates 2005). Even for normal-hearing unaided listeners, subtle manipulations in the rise time or presentation rate of the stimulus used to evoke the ABR can greatly alter the evoked response pattern (for a review, see Hall 1992). For these reasons, the ABR has not been used to examine the effects of amplification on the central auditory system. One exception is the work by Philibert et al (2002; 2005), who chose to examine the effects of hearing aid amplification and acclimatization on intensity discrimination and loudness scaling by using perceptual and ABR measures. They were able to avoid potential confounds of the hearing aid by recording ABRs in unaided conditions only.

Cortical auditory evoked potentials, such as the P1-N1-P2 complex (figure 1) also have been used to study the effects of hearing aid amplification and training on the central auditory system (for a review, see Tremblay in press). The P1-N1-P2 complex is generated in the auditory cortex; more specifically, from the thalamic-cortical portion of the system (Wolpaw and Penry 1975; Naatanen and Picton 1987; Woods 1995). The N1 component is often described as an onset or change response because its presence indicates the physiological detection of acoustic change (from silence to sound or acoustic changes within a sound). Like the ABR, the P1-N1-P2 complex is sensitive to various stimulus parameters. As stimulus intensity increases, the latency of the response decreases and the amplitude increases. Stimulus frequency also affects the response, with higher amplitude responses to low-frequency stimuli (Naatanen and Picton 1987). Because the response is sensitive to small changes in frequency and intensity (Martin and Boothroyd 2000; Souza, Tremblay and Boike 2003), it has been used extensively to assess the neural detection of sound, as well as estimate auditory thresholds (Naatanen and Picton 1987).

For the purpose of estimating aided thresholds, P1-N1-P2 evoked potentials have been recorded in children and adults with varying degrees of hearing loss (Rapin and Graziani 1967; Gravel, Kurtzberg, Stapells, Vaughan and Wallace 1989; Stapells and Kurtzberg 1991; Korczak, Kurtzberg and Stapells 2005). Without amplification, the evoked neural response pattern is typically delayed or absent. With amplification, an aided neural response pattern can sometimes be seen but results have been inconsistent.

Why the inconsistencies? Well, audiologists are trained to recognize how important their choice of stimulus, rise-time, rate, and presentation level is when recording auditory evoked potentials. This is because the physical characteristics of the stimulus greatly affect
the morphology (latency and amplitude) of the evoked response. Hearing aids and cochlear implants modify the physical characteristics of sound, and as mentioned earlier, 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 hearing aid processing on the physical characteristics of the sound likely affect the evoked neural response pattern.

To date we know very little about the effects of hearing aid signal processing on evoked response patterns. Therefore, to better understand the effects of amplification on the central auditory system, Tremblay et al. (2006) compared unaided and aided responses in young people with normal hearing. Younger listeners were tested because there is evidence that advanced age and hearing loss affect the way sound is processed in the brain, which in turn affects the P1-N1-P2 complex (Tremblay, Piskosz and Souza 2003; Tremblay, Billings and Rohila 2004; Harkrider, Plyler and Hedrick 2005). Normal hearing individuals were tested to isolate the effects of amplification from hearing loss. Two questions were asked: first, can naturally produced speech sounds be used to reliably record P1-N1-P2 responses in individuals wearing hearing aids; and second, does amplification alter neural response patterns? To examine these questions, subjects were tested and then retested within an eight-day period under both aided and unaided conditions. Surprisingly, test/re-test reliability, in both unaided and aided conditions, was remarkably good. These results confirm that sound presented in sound field and processed through a hearing aid can evoke reliable P1-N1-P2 responses in individuals. This point is important because it means that significant changes in waveform morphology, when measured from test to retest (e.g., before and after training or with and without amplification), would likely reflect significant changes in neural activity, and not just poor test/retest reliability (Tremblay, Kraus, McGee, Ponton and Otis 2001; Tremblay and Kraus 2002).

Does amplification modify evoked neural response patterns? In other words, when 30 dB of sound enters the hearing aid and the hearing aid provides 20 dB of gain, does the evoked response resemble that of a response evoked by a 50 dB signal? As shown in figures 2 and 3, the answer is no. Surprisingly, when 20 dB of gain was provided by a hearing aid, there were no significant differences between unaided and aided electrophysiological response patterns (Billings, Tremblay, Souza, and Binns 2007; Tremblay et al. 2006).

When sound is amplified by a hearing aid, neural response patterns should 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 documenting the effects of incremental in-

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**Figure 2.** Latency and amplitude functions for N1 and P2 cortical evoked potentials recorded in aided and unaided conditions. As stimulus intensity increases, N1 and P2 latencies decrease and amplitudes increase; however there are no significant differences between unaided and aided results as a function of stimulus intensity level (modified from Billings et al., 2007).
creases in stimulus intensity (as small as 2 dB) on electrophysiological recordings (Adler and Adler 1989; Rapin, Schimmel, Tourk, Krasnegor and Pollak 1966; Beagley and Knight 1967; Picton, Goodman and Bryce 1970). Increasing the intensity of a signal by 20 dB (using hearing aid amplification) should therefore result in significant changes in the magnitude and timing of synchronous neural activity – but this was not the case.

These results demonstrate that long-standing principles underlying electrophysiological recordings, which are based on unaided recordings, do not necessarily apply when sound is processed by a hearing aid and then delivered to the auditory system. Presumably, some sort of interaction between the way sound is processed through the hearing aid and encoded in the auditory system is taking place. These concerns likely also apply to cochlear implants when sound is presented in sound field and then modified by the speech processor. Therefore, before conclusions about the effects of auditory training on the brain can be made in people who wear hearing aids or cochlear implants, it is necessary to understand the basic effects of signal processing on the central auditory representation of sound. Without a detailed understanding of amplification effects on neural encoding, conclusions about brain plasticity related to hearing aid amplification might not be valid.

Conclusion

Various types of training exercises and/or video games designed to capitalize on the brain’s capacity to change can be found. Their effectiveness will be determined through the scientific peer review process. In time, methods that are determined to be effective might be incorporated into mainstream medical care. For instance, patients who have experienced a stroke or traumatic brain injury participate in exercises designed to retrain their brain to learn to use a damaged limb. Similar motivations underlie auditory training programs. Through focused listening exercises, the goal is to improve perception in a way that significantly enhances a person’s ability to interact with their communication environment. While there is ample evidence to show that training programs can alter brain activity in normal hearing animals and humans, we do not yet fully understand what these physiological changes reflect. Do they reflect changes in sensory representation, or top-down functions associated with learning?

If we are still improving our understanding of training-related physiological changes in the normal hearing system, we are certainly still naïve about how training affects brain activity in people who wear hearing aids and/or cochlear implants. Nonetheless, as our methods for quantifying physiological activity improve, we will be able to learn more about how sound is encoded in the brain and how these codes are modified with training/perceptual learning. Progress can come from at least two directions: first, we can develop behavioral methods that significantly improve perception and find out if and how these exercises are modifying the brain; second, we can learn more about how the brain works and let this information motivate new training strategies. Eventually, converging evidence from both approaches will provide information that can potentially redefine how we approach auditory rehabilitation.

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