

## Central auditory system plasticity and aural rehabilitation of adults

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**Abstract**—Until recently, researchers used behavioral measures of identification and discrimination of speech and non-speech stimuli to assess the effects of auditory deprivation, enhancement, and training. Recent advances in our ability to measure electrical activity in the auditory system in response to sound have made it possible for us to study how changes in auditory input (because of hearing loss, auditory input modification, or training) affect the function of the central auditory system. This article reviews the evidence of changes in the auditory cortex in mature animals and in humans with acquired sensorineural hearing loss as well as changes associated with auditory training in persons with normal hearing. The results of studies that measure psychoacoustic and speech-recognition performance of persons with hearing loss, with and without hearing aids, are interpreted within the framework of our new knowledge about plasticity of the auditory system. Applications of electrophysiologic techniques to hearing aid research and clinical practice are highlighted.

**Key words:** auditory deprivation, auditory-evoked potentials, auditory training, aural rehabilitation, central auditory system, electrophysiologic measurements, monaural amplification, plasticity, sensorineural hearing loss, sudden-onset hearing loss.

### INTRODUCTION

Researchers who study the physiology of the auditory system have provided evidence that functional changes occur in the brain and central auditory pathways of mature animals as a result of auditory deprivation (from damage to the peripheral auditory system) as well as auditory training. This ability of the sensory system to change has been described as “plasticity.” Several review

articles have recognized the potential importance of central auditory system (CAS) plasticity for the provision of hearing aids and aural rehabilitation [1–3], although at the time, no direct evidence of physiologic change in the human CAS and little understanding of how changes in the auditory system of animals effect auditory perception existed.

Willott suggested three ways that CAS plasticity might be relevant to the aural rehabilitation of adults with acquired hearing loss [3]. First, reduced auditory input from peripheral damage (e.g., cochlear pathology) may cause functional changes in the CAS and affect auditory perception. Second, provision of amplification may lead

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**Abbreviations:** AEF = auditory evoked magnetic field, AEP = auditory evoked potential, CAS = central auditory system, DLF = difference limens for frequency, fMRI = functional magnetic resonance imaging, MCL = most comfortable loudness level, MEG = magnetoencephalography, MMF = magnetic mismatch field, MMN = mismatch negativity, PPI = prepulse inhibition, RMS = root-mean-square, SL = sensation level, SPL = sound pressure level, TEN = threshold equalizing noise, VOT = voice onset time.

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to secondary plasticity because of altered input to the auditory system; this might yield secondary changes in auditory perception. Third, learning can cause functional changes in the auditory system and can lead to alterations in auditory perception [3]. Recent advances in our ability to measure electrical activity in the auditory system in response to sound have made the study of auditory system plasticity in humans possible. Information from these studies, combined with information from behavioral studies, allows us to reexamine the issue of how the aural rehabilitation of adults with acquired hearing loss might affect and be affected by CAS plasticity.

In this review, the following questions will be addressed—

- What is the evidence of plastic changes in the auditory system because of acquired hearing loss in humans?
- What is the perceptual significance of such plasticity?
- Does provision of amplification lead to plasticity and to changes in auditory perception?
- Does auditory training in adults lead to plastic changes in the auditory system and to changes in auditory perception?

## **ELECTROPHYSIOLOGIC METHODS FOR ASSESSING AUDITORY SYSTEM PLASTICITY IN HUMANS**

Until recently, the evidence of auditory system plasticity in humans was inferred from animal studies. In animals, invasive techniques can be used to measure functional changes at various points in the auditory pathway and at the cortex. Obviously these techniques are not suitable for studying auditory system plasticity in humans. Noninvasive auditory-evoked electrophysiologic potentials, functional brain imaging techniques such as magnetoencephalography (MEG), and functional magnetic resonance imaging (fMRI) now make studying CAS plasticity in humans possible.

Auditory evoked potentials (AEPs) are used to measure electrical activity in the auditory system in response to acoustic stimuli. Electrodes are placed on the scalp, and information about the time course and strength of the response in the brain stem and brain in response to auditory input is recorded. AEPs are categorized by latency after the onset of a stimulus. The early latency responses are thought to reflect activity in the central auditory pathways. The later responses are thought to reflect cortical activity. The

P1-N1-P2, a complex of positive, negative, and positive waveform deflections that occurs 50 to 200 ms after stimulus presentation, is an obligatory cortical response that can be evoked with the use of simple stimuli, such as clicks and tones, or more complex stimuli, such as speech. The presence of this complex of waves is associated with detection of a stimulus [4–5]. The mismatch negativity (MMN) is another late response (latency 100–200 ms) that researchers have used to obtain information about the brain's detection of a change in the stimulus. The MMN is evoked by presentation of a deviant stimulus among a set of standard stimuli and is seen as an increased negativity in the cortical waveform that occurs because of the change in the auditory stimulus [6].

MEG is a noninvasive technique that is used to measure the magnetic field generated by synchronous activity of neurons in the brain [7]. MEG provides an alternative way of measuring the same cortical activity that can be measured electrically with the use of AEPs. The technique allows good temporal resolution and can be used to infer the site of generation of electrical activity. In the MEG studies discussed in this article, two different measures of activity after onset of auditory stimulus have been used. The N1 response is a peak in the waveform that occurs approximately 100 ms after stimulus onset [8]. The magnetic mismatch field (MMF) or magnetic MMN is a late latency response (180–250 ms) and is a magnetic analog of the electrical MMN. Researchers use the MMF to obtain information about the brain's detection of a change in the stimulus; it is evoked by the presentation of a deviant stimulus among a set of standard stimuli [6].

fMRI is an indirect measure of neural activity. Changes in blood oxygen level from increased neural activity are measured in association with the presentation of auditory stimuli. The fMRI measurement technique can be used to localize brain activity, but has poor temporal resolution [7].

## **AUDITORY SYSTEM PLASTICITY IN HUMANS AND ITS PERCEPTUAL SIGNIFICANCE**

### **Evidence from Animals with High-Frequency Sensorineural Hearing Loss**

Information about auditory system plasticity in animal models serves as a frame of reference for human studies. Results from several animal studies show reorganization of the tonotopic map in the CAS because of

cochlear damage that resulted in high-frequency sensorineural hearing loss; these findings may be relevant for predicting CAS plasticity in humans.

Discrete lesions in the basal turn of the cochlea from mechanical, ototoxic, or noise damage can result in changes to tonotopic frequency mapping in the primary auditory cortex (measured in the hemisphere contralateral to the damaged ear). The area of the cortex that is normally responsive to frequencies from the damaged portion of the cochlea becomes responsive to stimuli from adjacent frequencies. This finding has been replicated in a variety of animals including guinea pigs [9], cats [10], and monkeys [11]. Apparently, significant damage to the cochlea and significant hearing loss are necessary before cortical reorganization will take place in animals [12–13].

Mice with bilateral sensorineural hearing loss from presbycusis also exhibit changes in the CAS that are similar to those in mice with experimentally induced cochlear damage. C57 mice, a strain of mice with adult-onset genetic hearing loss, have been studied extensively [14–15]. By comparing measurements of auditory system and cortical function, we can study changes in the CAS of C57 mice versus mice of similar age without genetic hearing loss. CBA mice are typically used as control subjects. C57 mice initially exhibit normal hearing but begin to evince high-frequency hearing loss by 4 to 6 months of age (~40 dB elevation in threshold at 20 kHz). By age 1 year, these mice have moderate hearing loss in the low to middle frequencies and severe high-frequency hearing loss. The changes in the cochlea include outer hair cell damage or loss in the basal turn by age 6 months, but minimal loss of spiral ganglion cells occurs. Total degeneration of the basal region of the organ of Corti and loss of spiral ganglion cells occur by age 2 years. Plastic changes in C57 mice include changes in the tonotopic map in the auditory cortex and the ventral inferior colliculus [14].

Little is known about the effects of tonotopic reorganization from peripheral hearing loss on auditory perception in animals. Willott and colleagues used an experimental technique for measuring the prepulse inhibition (PPI) of startle to examine how the salience of different frequencies changes with developing hearing loss in C57 mice [16–17]. In this protocol, a moderate intensity tone is presented 100 ms before an intense broadband noise stimulus and the amplitude of the startle response (inhibition) is measured. By manipulating the frequency of the prepulse tone, researchers can obtain information about the salience of that test frequency.

Willott et al. [16] and Carlson and Willott [17] provided evidence of the increased salience of midfrequency stimuli in mice with high-frequency sensorineural hearing loss. PPI was measured with prepulse tonal stimuli of 4, 12, and 24 kHz in 1-, 5-, and 12-month-old mice. In 1-month-old mice, inhibition of the startle response was observed after a 24 kHz prepulse stimulus. In older mice, inhibition of the startle response after the 24 kHz stimulus decreased with the onset of high-frequency hearing loss, and the inhibition of startle after the 4 and 12 kHz stimuli increased. By 12 months of age, the 24 kHz stimulus was no longer effective in inhibiting the startle response and the 4 kHz stimulus became most effective. Thus, in the region of hearing loss, the prepulse stimulus was not effective in decreasing startle amplitude. Presentation of a prepulse stimulus in the midfrequency region (frequencies slightly below the region of hearing loss) resulted in the greatest decrease in startle response amplitude.

Carlson and Willott measured neural PPI in the caudal pontine reticular formation of 6-month-old mice with high-frequency (>20 kHz) hearing loss [18]. The neural PPI showed the same response as the behavioral response; i.e., the neural PPI was greater after midfrequency prepulses (4 and 12 kHz). This is evidence of increased salience of the midfrequency region in the neural response. Willott and Turner found that the stronger midfrequency PPI in presbycusic mice was significantly correlated with the percentage of recording sites in the inferior colliculus that responded to the tone [19].

### **Evidence from People with High-Frequency Sensorineural Hearing Loss**

Is there evidence of auditory system plasticity in persons with high-frequency sensorineural hearing loss? Results from animal studies predict that the tonotopic maps of the primary auditory cortex of persons with high-frequency sensorineural hearing loss (and relatively normal hearing in lower frequencies) should show an increased representation of frequencies at the edge of the hearing loss. To test this hypothesis, researchers should compare cortical function in persons with high-frequency sensorineural hearing loss with that in persons with normal hearing.

A study by Dietrich et al. provides evidence of differences between the cortical tonotopic maps of persons with normal hearing and those with steeply sloping high-frequency hearing loss from acoustic trauma or sudden hearing loss [20]. These investigators measured the N1m

component of the auditory evoked magnetic field (AEF), which is a response that occurs approximately 100 ms after stimulus onset. Normal-hearing subjects were tested with the use of randomized tone bursts at 0.5, 1.0, 2.0, and 4.0 kHz. Measurements were obtained over each hemisphere for stimuli that were presented contralaterally. Cortical strength and source location of the N1m wave were estimated at all test frequencies for both hemispheres. In the subjects with hearing loss, AEF was measured over the supratemporal auditory cortex contralateral to the side of hearing loss with the steepest slope. Tests were performed at three frequencies (one at and two below the edge of the lesion frequency) and a presentation level of 60 dB sensation level (SL). The cortical strength (dipole moment) and the source location of the N1m wave were estimated for the lesion frequency and the two frequencies below the lesion frequency.

Six of the subjects had unilateral high-frequency hearing loss and two had bilateral high-frequency hearing loss. All the hearing-impaired subjects had normal hearing at frequencies below the lesion edge (the highest frequency of normal hearing adjacent to the hearing loss). The lesion-edge frequencies were 1.5 (three subjects), 2 (two subjects), 3.5, 4, and 7 kHz. The slope of the high-frequency hearing loss ranged from 30 to 65 dB per octave.

The subjects with normal hearing had similar mean cortical strength at all test frequencies for both the right and left hemispheres. Seven of the eight subjects with high-frequency hearing loss had significantly higher cortical strength for the lesion-edge frequency than for the frequencies below the lesion edge. The cortical strength at the two lower frequencies did not differ significantly. These results were interpreted as evidence of cortical plasticity, i.e., an expanded representation of lesion-edge frequencies in the cortex, and are consistent with findings of plasticity in animals.

This study provides information about how the frequency representation in the auditory cortex in persons with steeply sloping high-frequency sensorineural hearing loss differs from persons with normal hearing. The subjects in this study had abrupt-onset, precipitous high-frequency hearing loss. The pattern of responses in persons with hearing loss was consistent with findings from cortical mapping in animals with experimentally induced cochlear lesions in the high-frequency region. The results of this study cannot necessarily be generalized to gradual-onset hearing loss, other hearing-loss configurations, or

varied degrees of hearing loss. Clearly more research is needed to determine the conditions necessary for plastic changes in the cortex to occur and the time course for such changes.

Whereas researchers can readily assess the type and degree of cochlear damage in animals, they cannot directly assess the exact nature of cochlear damage in humans. The subjects in this experiment all had precipitous high-frequency hearing loss of a degree that suggests inner as well as outer hair cell loss. Otoacoustic emissions measurements might be helpful in assessing outer hair cell function, and results from a test such as the threshold equalizing noise (TEN) test might be useful for detecting the presence of dead regions on the cochlea, i.e., lack of functional inner hair cells or neurons [21]. The study would also have been strengthened if the researchers had obtained information about the auditory perceptual capabilities of the subjects and determined whether purported changes in the cortical map affect auditory perception.

Willott pointed out that auditory system plasticity might either enable the auditory system to compensate for the loss of auditory cues from hearing loss, which would lessen auditory deprivation effects, or make things worse [3]. Many of the differences in psychoacoustic performance between persons with normal hearing and hearing loss can be attributed to cochlear pathology. Is there evidence of changes in auditory performance that might be explained by CAS plasticity?

Several studies have investigated psychoacoustic performance in the region of hearing loss in an attempt at finding behavioral evidence of cortical reorganization of the tonotopic map. In three studies, difference limens for frequency (DLFs) were measured in persons with steeply sloping high-frequency sensorineural hearing loss [22–24]. The results of all three studies agreed. Enhanced DLFs were found in the region at the edge of the hearing loss. McDermott et al. hypothesized that if an expanded cortical region was associated with frequencies near the lesion edge in persons with steeply sloping high-frequency hearing loss then stimulation of that expanded region might affect the perception of pitch [22]. They measured DLFs in five subjects with steeply sloping high-frequency sensorineural hearing loss and five with normal hearing. The subjects with hearing loss had normal hearing or mild hearing loss at low frequencies and a slope of hearing loss greater than 50 dB per octave. Three of the subjects had congenital hearing loss,

one had hearing loss from ototoxic medication, and one had progressive hearing loss possibly due to cochlear otosclerosis. DLFs were measured with an adaptive, three-interval, forced-choice procedure. For the hearing-impaired subjects, test frequencies ranged from 0.25 kHz to the highest frequency that could be tested. These included frequencies below and above the cutoff frequency ( $f_c$ ), where  $f_c$  is defined as the lowest frequency with a threshold less than 15 dB hearing level and with a slope above that frequency greater than 50 dB per octave. DLF tests were performed at a constant loudness level ( $\pm 3$  dB rove level) so that loudness and pitch cues would not be confused (loudness matching was carried out to a 0.5 kHz tone at 45 dB SL).

For the normal-hearing subjects, DLFs were measured at 0.25, 0.5, 1, 1.5, 2, and 4 kHz at a constant loudness level (70 phon curve). The mean DLFs, (mean frequency difference/reference frequency)  $\times 100$ , of the normal-hearing listeners systematically decreased with increasing frequency from 1.2 percent at 0.25 kHz to 0.6 percent at 4 kHz.

The mean DLFs for the hearing-impaired subjects in the lower frequencies was 3 percent. In this region, subjects either had normal or near-normal hearing. These DLFs were much larger than the DLFs of the normal-hearing subjects. For four of the subjects with hearing loss, the DLF in the region around  $f_c$  was significantly smaller than in regions below or above, and the DLF increased in the regions with the greatest hearing loss. For one subject (this subject had progressive hearing loss possibly from cochlear otosclerosis), no significant differences between the DLF in the region surrounding  $f_c$  and the regions below and above were found. The investigators ruled out learning as the cause of the smaller DLFs in the region of  $f_c$  because analysis of the data revealed no learning effect. They concluded that the smaller DLFs might be explained by an expanded representation of the edge frequencies in the brain. If this was the case, a greater number of cortical neurons would be activated. Different frequencies would cause different patterns of spatial activity in the cortex and lead to better frequency discrimination in the edge region.

Thai-Van et al. were able to replicate the smaller DLF results for persons with bilateral high-frequency hearing loss and also established that the slope of the high-frequency hearing loss is an important factor [23]. Twenty subjects with bilateral high-frequency sensorineural hearing loss were divided into three groups based on the slope of the high-frequency hearing loss (nine with

a slope of 50 dB per octave, six with a slope of 24–50 dB per octave, and five with a slope of  $<24$  dB per octave). Tests were performed at a constant loudness level  $\pm 3$  dB (determined by loudness matching to a 0.5 kHz tone at 30 dB SL). DLFs were measured at 13 frequencies above and below  $f_c$  (from 1/2 an octave below  $f_c$  to an octave above in 1/8th octave intervals).

Analysis revealed that the group with steeply sloping hearing loss differed from the other two groups with regard to the DLF near  $f_c$ . While the groups with more gradual slopes had similar DLFs at frequencies directly above and below  $f_c$ , the group with the steeply sloping hearing loss showed a significantly smaller DLF at 1/8 octave above  $f_c$ . The analysis failed to show a learning effect. The authors concluded that their data supports cortical plasticity. However, they add the caveat that subjects may have used loudness differences in regions of the steeply sloping hearing loss as cues (loudness balancing was only done at 1/2 octave intervals and estimates of loudness levels were made by interpolation at intervening frequencies).

In a later study of frequency discrimination in persons with steeply sloping sensorineural hearing loss, Thai-Van et al. established that loudness cues were not responsible for the finding of enhanced DLFs in persons with steeply sloping high-frequency sensorineural hearing loss [24]. Frequency discrimination was assessed for five subjects with steeply sloping high-frequency sensorineural hearing loss and cochlear dead regions (as established by results from the TEN test). All subjects showed better frequency discrimination within 1/4 of an octave of the edge frequency of hearing loss. The subjects' ability to use loudness cues was minimized by measuring equal loudness contours at smaller frequency intervals than in previous studies (1/8 rather than 1/2 an octave), and stimulus levels were roved over a 12 dB range ( $\pm 6$  dB). They also ruled out the possibility that the presence of spontaneous otoacoustic emissions might be used as cues in the DLF task. None of the subjects had spontaneous otoacoustic emissions. Additionally, the TEN test was used to identify dead cochlear regions. All the subjects had cochlear dead regions and showed better frequency discrimination within 1/4 of an octave of the edge frequency of hearing loss.

In contrast to these findings, Buss et al. failed to find evidence of improved performance at the edge frequency of persons with steeply sloping sensorineural hearing loss on several different psychoacoustic tasks: frequency sweep detection, intensity discrimination, gap detection, or gap discrimination [25]. The performance of seven subjects

with hearing loss was compared with the performance of three persons with normal hearing with simulated hearing loss (masking to simulate high-frequency sensorineural loss). Significant differences in performance were not found between groups. In addition, the 0.04 kHz following response was obtained on the normal-hearing subjects and on three of the hearing-impaired subjects. This measure was included for the researchers to obtain a more objective measure of whether increased neuronal activity was present at the edge frequency. The response of the hearing-impaired subjects did not differ significantly from that of the normal-hearing listeners.

The previous studies have dealt exclusively with persons with steeply sloping high-frequency sensorineural hearing loss. Doherty and Lutfi studied the effect of mild and moderate sensorineural hearing loss on the perception of complex spectral stimuli using conditional-on-a-single-stimulus analysis, which is a method that can determine the weight given to specific frequencies when subjects identify differences between complex spectral stimuli presented at suprathreshold levels [26–27]. In the first study, subjects were presented two multitone-tone complexes (six tones at octave frequencies from 0.25 to 8 kHz) and the level of each frequency in the complex was varied randomly [26]. The listener was asked to identify the stimulus with higher overall intensity. It was anticipated that an ideal observer would give equal weight to all frequency components. The average performance of 11 persons with normal hearing approximated the pattern expected from an ideal observer. In contrast, 14 listeners with bilateral sensorineural hearing loss (10 with gradually sloping and 4 with steeply sloping hearing loss) showed a very different pattern of performance. All listeners showed a greater weight for high frequencies than for low frequencies.

In the second study, Doherty and Lutfi examined subjects' ability to detect a target tone that was embedded in a multitone complex [27]. As in the first study, two multitone complex signals were compared; in the second study, the amplitude of only one of the tones was increased. Target tones were at 0.25, 0.5, or 4.0 kHz. The performance of 15 normal-hearing subjects was compared with that of 15 hearing-impaired subjects who had normal hearing or mild hearing loss in the lower frequencies and moderately sloping loss in the higher frequencies. Again, the hearing-impaired listeners gave greater weight to high- rather than low-frequency stimuli. While the normal-hearing listeners gave greater weight to the 0.25 kHz target tone, the hearing-impaired listeners gave greater weight to the 4 kHz tone.

Doherty and Lutfi speculated that the spectral weighting of the hearing-impaired listeners may indicate a compensatory strategy (i.e., hearing-impaired listeners pay more attention to information in the region of loss) and that greater spectral weighting of the high-frequency region may interfere with the individual's ability to use amplification in that region [27]. However, it is unclear why hearing-impaired listeners would not change their listening strategy if amplification were to change the spectral makeup of the sound they heard regularly. With amplification, the hearing-impaired person may no longer need to "pay more attention" to the spectral region in the area of the hearing loss. If this were the case, then the spectral weighting of the listener would change over time. Longitudinal measures of the perception of complex tonal and speech stimuli that were obtained before and after hearing aid fitting would provide a test of this hypothesis.

These two studies point to differences in the frequency-specific perception of complex stimuli by persons with hearing loss. The weighting pattern found in listeners with high-frequency sensorineural hearing loss is similar to the increased weighting of middle frequencies from the PPI studies in mice that exhibited CAS plasticity. Cortical plasticity might account for the finding of greater weighting of frequencies in the region of hearing loss because frequencies in the region adjacent to the hearing loss would stimulate a larger region of the cortex. If cortical remapping has taken place because of hearing loss, then cortical remapping might be required to restore the original tonotopic map once a hearing aid restores the audibility of frequencies that had been inaudible. Electrophysiologic studies that assess tonotopic maps before and after hearing aid use would be needed to test this hypothesis.

New electrophysiologic evidence of cortical plasticity in humans with high-frequency sensorineural hearing loss exists, but the evidence is limited to studies of individuals with precipitous high-frequency sensorineural hearing loss. As a group, they are likely to have normal or near-normal hearing at lower frequencies and severe to profound hearing loss in the higher audiometric frequencies. Several frequency discrimination studies in persons with this type of hearing loss have revealed enhanced discrimination in the frequency region at the edge of the hearing loss [22–24]. CAS plasticity from either injury or deprivation or from the subject learning to use a different set of cues in the auditory signal might account for the behavioral findings. Direct evidence of altered cortical

activity in listeners who exhibit such behavior would help to prove or disprove the association between the behavior and modified cortical activity.

### **Plasticity Related to Unilateral Hearing Loss**

In the normal auditory system, it is expected that monaural stimulation will lead to bilateral cortical activity. The cortical activity will occur earlier and will be stronger in the hemisphere contralateral to the stimulated ear. In adult animals, unilateral hearing loss has been found to affect the cortical response to signals that were introduced to the opposite (normal-hearing ear). Before receiving an ototoxic drug, normal-hearing adult guinea pigs showed large ipsilateral/contralateral threshold differences in the auditory cortex for frequencies between 4 and 16 kHz (14–23 dB smaller thresholds in the ipsilateral auditory cortex). After injection with sisomicin through the round window membrane of the nontest ear, the asymmetry in cortical activity in response to sounds in the opposite ear was disrupted. Thresholds improved in the ipsilateral auditory cortex, which resulted in only small differences between the response in the contralateral and ipsilateral hemispheres of the cortex in the high frequencies. The amplitude of the contralateral cortical response was much larger than the ipsilateral response, but had similar waveforms. The ipsilateral amplitude increased and latency decreased for low frequencies. Changes in the cortical response occurred over at least a 3-week period. The types of changes that occurred were attributed to changes in inhibitory circuits from unilateral damage [28]. Thus, unilateral hearing loss resulted in bilateral changes in the CAS. Several studies have documented the same sorts of changes in the adult human auditory system after unilateral hearing loss. These studies are relevant to our understanding of how the CAS changes with monaural amplification in persons with bilateral, symmetric sensorineural hearing loss.

Vasama and Makela used whole scalp MEG to study possible cortical plasticity in persons with sudden unilateral sensorineural hearing loss [29]. AEFs were recorded 2 to 5 years after hearing-loss onset from eight adults with sudden unilateral hearing loss (presumably of cochlear origin). All subjects had normal hearing through 4 kHz in the unaffected ear. The degree of hearing loss in the affected ear differed among subjects. Four of the subjects had profound hearing loss, three had severe hearing loss, and one had moderate to severe hearing loss in the affected ear. Eight adults with normal bilateral hearing

were also tested as control subjects. The N1m response was measured with a series of 1 kHz tones. The data were analyzed to determine latencies as well as the number and location of the response sources. In the case of the subjects with unilateral hearing loss, the signals were presented to the ear with normal hearing (four with normal hearing in the right, four with normal hearing in the left) and hemispheric differences in responses were ascertained. Half of the control subjects were tested with signals presented to the right and half with signals presented to the left. Again, hemispheric differences in response were determined.

The distribution of the magnetic fields in space was modeled (equivalent current dipole) to determine the location of the generation of brain activity. In the majority of the subjects, an equivalent current dipole in each temporal auditory area could be used to interpret the pattern of magnetic field activity. As expected, the subjects with normal hearing showed shorter latencies and stronger dipole moments for the hemisphere contralateral to the stimulated ear. Three of the persons with unilateral hearing loss had patterns similar to those of the normal-hearing controls. Five of the persons with unilateral hearing loss showed shorter latencies and/or stronger dipole moments in the cortical hemisphere ipsilateral to the stimulated ear (the better ear). This pattern of performance (shorter latency/stronger activity in the ipsilateral rather than contralateral hemisphere) was interpreted as evidence of reorganization of the auditory system as a result of the hearing loss. Three of the persons with unilateral hearing loss also showed a very different spatial and temporal response in the MEG than did the control subjects, which indicated additional sources of cortical activity. Thus, parts of the brain that are not active in persons with normal hearing appear to be active in those with unilateral hearing loss. The results of this study are mixed in that some subjects with unilateral hearing loss had AEF patterns similar to those with normal hearing while others did not. Subjects with profound and lesser degrees of hearing loss exhibited a pattern of activity that differed from that seen in the normal-hearing subjects. Clearly more studies of this type are necessary. Longitudinal studies on subjects grouped by pathology, configuration, and degree of hearing loss would provide valuable information about the time course and nature of CAS plasticity in persons with unilateral hearing loss. Tests of auditory perception should be included in future studies of this nature.

Scheffler et al. also documented changes in central auditory function after unilateral hearing loss [30]. fMRI was used to map blood oxygen level dependent signal change for monaural or binaural signals (1 kHz pulsed tones, 95 dB sound pressure level [SPL]). The cortical responses of 10 subjects with normal bilateral hearing were compared with those of 5 subjects with profound unilateral hearing loss. Two of the subjects had congenital hearing loss. Three of the subjects had acquired hearing loss from infection, sudden-onset hearing loss, or surgery. The duration of hearing loss was 6 weeks for the subject with sudden-onset hearing loss and several years for the other two subjects. The cortical responses of the normal-hearing subjects to monaural stimulation revealed stronger responses in the hemisphere contralateral to the stimulated ear. The response to bilateral stimulation was similar in both hemispheres. For subjects with profound unilateral hearing loss (either congenital or acquired), similar monaural stimulation responses were found in both the ipsilateral and contralateral hemispheres, i.e., the response pattern to a monaural signal was similar to that of a binaural signal for persons with normal hearing. The lack of hemispheric asymmetry for monaural auditory stimulation provides evidence of differences in responses of persons with unilateral hearing loss versus those with normal hearing. One of the subjects had hearing loss for only 6 weeks and still showed this altered response pattern. It would be important to determine the time course of these changes in response patterns. Longitudinal studies of persons with unilateral hearing loss would provide important information about how and when cortical activity changes with sudden-onset unilateral hearing loss.

Ponton et al. investigated the effects of unilateral hearing loss on representation of the signal at the cortex by evaluating the N1-P2 complex, which is a long-latency AEP that is thought to be generated in the thalamic-cortical portion of the CAS [31]. N1, a negative peak, occurs approximately 100 ms after the onset of the stimulus and P2, a positive peak, occurs approximately 200 ms after stimulus onset. The waveform amplitude is typically larger in the hemisphere contralateral to the stimulated ear.

AEPs were measured in 15 persons with profound, adult-onset unilateral hearing loss (12 from otologic surgery, 3 from sudden hearing loss). The subjects with unilateral hearing loss were divided into two subgroups: eight with hearing loss  $\leq$  2 years, seven with hearing loss  $\geq$  2 years. All subjects had normal hearing at frequencies up to 4 kHz in the better ear. Nine subjects with normal

bilateral hearing served as controls. Subjects were tested monaurally with click trains (10 clicks repeated at a rate of 1.3/s) presented at 65 dB SL. The AEPs were recorded at 30 electrode locations and several analyses were performed for determining whether the monaural stimulation performance pattern differed between the two groups.

In the first analysis, the average root-mean-square (RMS) amplitude of the AEP for the ipsilateral and contralateral hemispheres was compared within and between subject groups. The RMS amplitudes of the AEP for the contralateral hemisphere did not differ significantly between the control group (normal bilateral hearing) and the unilateral hearing loss group. However, the RMS amplitude of the AEP for the ipsilateral hemisphere was significantly larger in the unilateral hearing loss group than the control group. The ipsilateral to contralateral amplitude ratio for the normal-hearing listeners (0.85) indicated hemispheric asymmetry. Subjects with unilateral hearing loss showed almost equal amplitude in the two hemispheres (ratio of  $\sim$ 0.98). This result agrees with Scheffler et al. [30].

In the second analysis, interhemispheric differences in timing of the evoked potential were investigated. A cross-correlation analysis of responses at corresponding locations in the contralateral and ipsilateral hemispheres revealed that both groups with unilateral hearing loss had lower interhemispheric AEP cross-correlations for responses over the frontal cortex than did normal-hearing listeners. The subjects with longer duration unilateral hearing loss had significantly higher interhemispheric AEP cross-correlations for responses over the central cortical regions than those with shorter duration hearing loss. No significant differences were found for the temporal or parietal-occipital regions. Further analysis of the central region responses consisted of linear regression analyses of interhemispheric peak-to-peak amplitudes (P1-N1, N1-P2). The peak-to-peak amplitude correlations increased significantly with increased duration of hearing loss. The authors interpreted the increased symmetry between hemispheres a result of changes in the generators of the N1 peak over time.

One confounding factor identified by the researchers is the age difference between the two groups of subjects with hearing loss. The group with shorter duration hearing loss was older than the group with longer duration hearing loss. Thus, the possibility exists that the difference between the two hearing-loss groups may be attributable to a smaller capacity for plasticity in older persons rather than the duration of hearing loss. An additional issue in

this and many other studies is the use of younger subjects in the control group. The issues of the time course of plasticity and the effect of age at time of hearing-loss onset can be addressed in future studies by collecting longitudinal data for subjects that differ in age at hearing-loss onset. Differences in the N1-P2 peak as a function of age could be addressed by including some older persons with normal hearing as control subjects.

Khosla et al. also measured long-latency AEPs (70–210 ms) in 19 listeners with profound unilateral hearing loss (average duration of hearing loss 2.4 years) and 8 with bilateral normal hearing [32]. Click stimuli were presented monaurally. Data were analyzed with dipole source modeling to determine latency and amplitude differences in the ipsilateral and contralateral hemispheres. The subjects with normal bilateral hearing exhibited the expected interhemispheric differences in latency and amplitude (earlier peaks and larger amplitudes in the hemisphere contralateral to stimulation). No significant differences were found in interhemispheric amplitude as a function of auditory stimulus in the right versus the left ear. In the subjects with unilateral hearing loss, interhemispheric differences depended on which ear was being stimulated and which ear had hearing loss. When the hearing loss was in the left ear, stimulation of the right ear resulted in reduced interhemispheric differences. When the hearing loss was in the right ear and stimulation was to the left ear, the contralateral was greater than the ipsilateral hemisphere activation. Thus, symmetry of response to monaural stimulation seemed to occur only in persons with unilateral loss in the left ear. These studies all provide evidence that unilateral hearing loss can disrupt the normal interhemispheric pattern of cortical response, but do not provide information about the degree of hearing loss that will lead to such changes, the time course over which changes occur, or the perceptual consequences of such changes. The finding of a differential effect of unilateral hearing loss dependent on the affected ear should be followed up in further studies. If such an effect exists in persons with unilateral hearing loss of lesser degree, then use of amplification in the affected ear might counteract such a change.

### **DOES HEARING AID USE LEAD TO AUDITORY SYSTEM PLASTICITY?**

Some behavioral evidence exists that auditory perception of hearing aid users changes over time. This evidence

comes from studies of the phenomena of late-onset auditory deprivation [33] and hearing aid acclimatization [34–35]. Late-onset auditory deprivation refers to the relative auditory deprivation from monaural amplification by persons with bilateral, symmetrical sensorineural hearing loss. It is characterized by a significant degradation of suprathreshold speech-recognition performance of the unaided ear after a period of monaural hearing aid use [33]. Acclimatization is “a systematic change in auditory performance with time that is linked to a change in the acoustic information available to the listener. It involves an improvement in performance that cannot be attributed purely to task, procedural, or training effects” [36].

Since the initial report of the late-onset auditory deprivation effect, a substantial number of studies have documented the phenomenon of degraded performance in the unaided ear with both group and individual data [37–44]. After reviewing the literature, Neuman concluded that the evidence to this effect was convincing [45]. The deprivation effect was found primarily in persons with at least a moderate degree of bilateral hearing loss after several years of monaural hearing aid use. At the time of the review, insufficient evidence existed for determination of the time course of the deprivation effect or the effect of subject-related variables on deprivation effects. Retrospective studies since published have continued to compare suprathreshold monaural speech-recognition performance of the aided and unaided ears under headphones [46–47]. The results of these studies confirm those of previous studies but do not contribute substantially to a greater understanding of whether CAS plasticity is involved.

Silverman and Emmer pointed out that the degraded speech-recognition performance in the unaided ear is similar to that seen in persons with asymmetric sensorineural hearing loss [48]. If cortical plasticity is responsible for the performance of persons who exhibit late-onset auditory deprivation, the introduction of asymmetry might lead to a lack of salience for stimuli presented to the unaided ear because of the greater ipsilateral cortical activity from presentation of the stimulus to the aided ear.

The effects of late-onset auditory deprivation appear to be reversible in some hearing aid users with the introduction of a second hearing aid [37,41,43,49–50]. Recovery is seen as a significant increase in speech-recognition performance (under headphones). The increase in performance is not immediate but is seen after use of the second hearing aid for some period of time. Recovery may occur in less than a year or over several years and may be

partial or incomplete. The finding of improvements in speech-recognition performance with restoration of binaural input may be consistent with a secondary plasticity. Measures of cortical activity that show changes in ipsilateral and contralateral brain activity over time would provide evidence of this.

Results from a study by Moore et al. provide further behavioral evidence that auditory performance of persons with unilateral or asymmetric hearing loss differs from that of persons with bilateral, symmetric hearing loss [51]. Speech-recognition performance of persons with unilateral and bilateral hearing loss was compared as part of a study that was designed to assess the accuracy of simulations of hearing loss. This study systematically assessed the effects of simulations of elevated threshold, loudness recruitment, reduced frequency resolution, and dead regions on the cochlea. Subjects with unilateral hearing loss were included in the study to assess the accuracy of the simulation by comparing their performance from the normal ear with that from the ear with hearing loss. Subjects with bilateral hearing loss were included as a control.

Four subjects with unilateral or very asymmetric hearing loss and three subjects with bilateral, symmetric sensorineural hearing loss (similar degree and configuration to the unilateral loss) were tested. In the subjects with unilateral or asymmetric hearing loss, the poorer ear had moderately severe or severe sensorineural loss and the better ear had normal sensitivity through 2 kHz. Recognition of key words in sentences was tested in quiet and in two types of competing noise (a single competing talker and speech-shaped background noise).

Simulations were not particularly successful at capturing the deleterious effects of hearing loss. In quiet and noisy settings, simulations of threshold elevation and loudness recruitment; loudness recruitment and spectral smearing; and loudness recruitment, spectral smearing, and dead regions did not accurately reflect the effects of hearing loss. All four subjects with unilateral hearing loss had poorer speech-recognition performance in the ear with hearing loss than in the normal ear with simulated hearing loss. The subjects reported that the loudness of the simulations seemed accurate but that the speech was clearer in the normal ear. Performance of the impaired ears of the subjects with unilateral hearing loss was much worse than in the subjects with bilateral hearing loss who had similar audiograms. The performance of subjects with bilateral hearing loss was similar to that of the nor-

mal ear (with simulated hearing loss) of subjects with unilateral hearing loss and in some cases was better.

The performance of the subjects with unilateral hearing loss seems consistent with an account of auditory deprivation effects. In daily life, the brain receives normal input from one ear while the input from the hearing-impaired ear is attenuated and distorted. Under these circumstances, cortical reorganization might occur (in this case, possible disruption of normal hemispheric asymmetry from monaural stimulation). The behavioral consequence is poorer than expected speech-recognition performance in the impaired ear. In contrast, in the subjects with bilateral hearing loss no disparity exists between the signals that reach the two ears. Even though the audiometric thresholds are similar to those in the impaired ear of the unilateral subject, the speech-recognition performance is better.

In most studies of relative auditory deprivation, performance has been assessed separately in each ear. Jacobson et al. assessed the effect of a monaural hearing aid fitting on dichotic speech-recognition performance [52]. Twenty experienced monaural hearing aid users (>1 year) with moderate, bilateral, symmetric sensorineural hearing loss; ten with moderate, bilateral, symmetric sensorineural hearing loss who did not use hearing aids; and ten normal-hearing subjects were tested. All subjects were right-handed. Of the hearing aid users, half used the hearing aid in the right ear and half used it in the left. Monaural and dichotic speech-recognition performance was measured with the use of syllables that consisted of a consonant followed by a vowel. Testing was performed at the most comfortable loudness (MCL) level of each subject. MCL was measured in one ear (with the test stimuli), and then a loudness match was obtained for the opposite ear.

Monaural speech-recognition scores did not differ significantly between ears for any of the groups. Dichotic tests revealed the expected pattern of significantly better performance for the right ear than the left ear for the normal-hearing subjects and for subjects with sensorineural hearing loss who did not use hearing aids. The group of subjects who wore the hearing aid on the right ear also showed a right-ear advantage. The right-ear advantage for the normal-hearing group was 11.7 percent, for the unaided group was 9.4 percent, and for the right-ear aided group was 10.7 percent. The group of subjects who wore the hearing aid on the left ear failed to show a right-ear advantage and demonstrated a left-ear advantage of 6.6 percent. These findings suggest that changes in auditory system function because of asymmetric auditory

stimulation may be differentially affected by greater deprivation of speech signals to the right ear than to the left.

The changes reported in late-onset auditory deprivation studies are of suprathreshold speech-recognition scores under headphones that were obtained with standard audiologic assessment tests. These measures do not directly assess performance with amplification. However, in research on hearing aid acclimatization, speech recognition is measured with and without the hearing aid(s) to document changes in performance. Gatehouse has suggested that when a person with hearing loss uses a hearing aid that provides newly audible information, he/she may require time to learn to use these new auditory cues before improvements in auditory performance can occur [34–35]. Thus, benefit from the hearing aid may not be obvious immediately or even within several weeks of the hearing aid fitting. The question of whether acclimatization occurs has been controversial. While some studies show that auditory performance with a hearing aid (usually speech-recognition performance) may improve over a period of weeks or months [34–35,53–59], other studies show stable performance from the time of hearing aid fitting over months and even years [60–65]. The conditions under which an acclimatization effect will be seen in a new hearing aid user or an experienced hearing aid user who has been switched to a new type of processing are unclear (see reviews by Turner et al. [66] and Palmer et al. [2]). Factors that have been cited as interfering with strong demonstrations of an acclimatization effect include speech-recognition tests that lack sensitivity, hearing aid fittings that do not provide sufficiently new information to require acclimatization, and subjects with inadequate hearing loss to demonstrate an acclimatization effect. Additional factors that could mask the demonstration of acclimatization effects are the possibility that acclimatization may only occur at certain aided listening levels and that it may differ in conditions of quiet and noise. Recent studies continue to yield conflicting results and have not added substantially to an understanding of the subject-related, hearing aid-related, and environmental conditions that may lead to changes in performance of a particular listener as a result of extended use of a hearing aid. The use of electrophysiologic measures should be helpful for determining if secondary cortical plasticity occurs in the hearing aid user.

The behavioral evidence from studies of late-onset auditory deprivation, on persons with unilateral hearing loss, and of acclimatization might all be explained as manifestations of CAS plasticity effects. Because none of

the studies included measures that could assess CAS plasticity, no direct evidence of changes in the auditory system exists. To establish that plasticity does indeed play a role, researchers should obtain electrophysiologic measures in future (prospective) studies; information about cochlear function would also be important. Otoacoustic emissions measurements at multiple frequencies could serve as a test of outer hair cell function. The TEN test and psychoacoustic tuning curves at multiple frequencies would allow inference of possible dead regions on the cochlea. The electrophysiologic measures should yield information about changes in cortical function with amplification. This information, in combination with information about cochlear status, may help explain differences in hearing aid benefit among listeners with similar audiograms.

It appears that the use of electrophysiologic measures to assess cortical activity in persons who use hearing aids can provide helpful information. Korczak et al. recently established that cortical evoked related potentials can be used to assess how hearing aid use changes the pattern of activity in the brain [67]. Fourteen subjects with either moderate or severe-profound hearing loss listened to speech stimuli syllables (/ba/ and /da/) that were presented in an oddball paradigm. Data collected included measurements of cortical responses while subjects listened with and without their personal hearing aids and while they listened passively or actively. During the passive listening, subjects were asked to ignore the speech and to read a book while the speech was played. During the active listening, subjects were asked to press a button as quickly as possible when they heard the deviant stimulus in the train of stimuli. Measurements were obtained with the use of stimuli at 65 and 80 dB SPL.

Latency and amplitude measures were obtained from the grand mean waveform for each condition. Systematic changes in the evoked potentials and the discrimination task were found as a function of aided condition and stimulus level. Hearing aid use resulted in decreased latency and increased amplitude of the evoked potentials. The change in the waveform was greater at the lower speech level than the higher (either because of lack of audibility at the lower intensity when unaided or because of output limitations in the hearing aid with higher intensity sound). Larger changes in the evoked potentials were seen for subjects with more severe hearing loss. Even with hearing aids, hearing-impaired subjects had greater latencies than subjects with normal hearing. The behavioral measures revealed that the accuracy of deviant syllable identification increased and

reaction times decreased with amplification. Subjects' discrimination was most accurate at the higher intensity level. The paradigm in this study might be useful for obtaining information about changes in benefit from hearing aid use over time once the test-retest reliability is established. The authors point out the large intersubject variability in the evoked potential measurements. This is not necessarily of concern, as long as the within-subject variability is small and changes in latency or amplitude of the evoked potentials with and without the hearing aid could be reliably assessed as a function of time. Future studies should examine acclimatization effects systematically in binaural and monaural hearing aid users with electrophysiologic tests in combination with behavioral tests. Prospective studies should include measures obtained before the hearing aid fitting and at regular intervals thereafter. Hearing aid users should be tested with their hearing aids as worn on a daily basis. Those who wear binaural hearing aids should be tested with them, and those who wear monaural aids should be tested with one hearing aid and the unaided ear unplugged. Hearing aid users who have substantial experience with the type of processing the researchers are testing should be included in the study to control for practice effects [57].

Longitudinal tracking of behavioral measures of auditory performance and electrophysiologic measures of matched groups of persons with bilateral, symmetric hearing loss who are being fit with either monaural or binaural hearing aids might provide direct evidence of a link between CAS plasticity and changes in auditory performance from amplification. Furthermore, this approach might be helpful for determining why speech-recognition performance changes (either improves or declines) in some persons and not in others.

Imaging and evoked potential techniques have been used to investigate plastic changes in the auditory system of persons with cochlear implants. These studies will not be reviewed here for several reasons. First, many of the studies have been performed with children and are more relevant to the issue of plasticity in developing organisms than in adults. Second, the degree of hearing loss and auditory deprivation experienced by cochlear implant users is greater than that experienced by most hearing aid users and, therefore, the auditory system may be more dramatically altered. Third, because input to the auditory system via the cochlear implant differs from that from a hearing aid, the results of these studies cannot necessarily be generalized to the issue of plastic changes because of learned acoustic cues from hearing aids. However, these

studies have proven the value of the approach. The use of these techniques to study the effects of hearing loss and hearing aids should contribute to a better understanding of auditory deprivation and amplification and the possible role of CAS plasticity.

## **ELECTROPHYSIOLOGIC EVIDENCE OF CORTICAL REORGANIZATION AFTER AUDITORY LEARNING**

Cortical reorganization can occur in the auditory cortex after auditory training and learning. In a study by Recanzone et al., owl monkeys were trained to discriminate small changes in frequency over a period of several weeks [68]. After the training period, the monkeys showed an expanded representation of the trained frequencies in the tonotopic map in the primary auditory cortex in comparison with the monkeys who had not received training. The cortical map of monkeys who were exposed to the tonal stimuli but did not receive auditory training (they received training on a tactile discrimination task) were similar to the maps of the control monkeys. This study shows a relationship between learned acoustic cues and cortical plasticity. It is noteworthy that animals exposed to the same stimuli did not exhibit changes in the cortical map. Perceptual learning of acoustic cues was required for cortical plasticity to occur.

A growing number of researchers are using imaging techniques or AEP measurements to study plastic changes in the human auditory system that are associated with auditory learning. The majority of these studies have been carried out on persons with normal hearing, either with nonspeech or speech stimuli. The following summary is by no means comprehensive but rather illustrates the types of information that have been obtained about plastic changes in the auditory system with the use of imaging and electrophysiologic measures.

Pantev et al. compared the N1m response to four piano tones (0.262, 0.523, 1.046, and 2.093 kHz) and four pure tones (0.25, 0.5, 1.0, and 2.0 kHz) from musicians and nonmusicians [69]. It might be expected that musicians would have extensive training in listening to music and that such training might have led to changes in the CAS. The stimuli were presented to the right ear and cortical activity was measured over the left hemisphere. For the musicians, the strength of cortical activation was greater for the piano tones than the pure tones (21%–28% larger

dipole moment). No significant difference was found in the strength of cortical activity for pure tones versus piano tones for the control subjects. The increased cortical activity in the musicians for musical notes may be because of an increase in the number of neurons that process the stimuli or greater synchrony of neural activity.

Menning et al. used MEG to measure both the MMF and the N1m responses in subjects who had received frequency discrimination training [70]. Data were obtained in the 3 weeks before frequency discrimination training, during the 3 weeks of training (15 sessions), and 3 weeks after completion of training. This made direct assessment of how cortical activity changed with training possible. An adaptive training procedure was used to train frequency discrimination at 1 kHz. Discrimination was tested before and after each training session. During the MEG testing, data were collected with the use of three deviant frequencies (1.005, 1.010, and 1.050 kHz) in separate blocks.

Discrimination improved quickly in the first week for all subjects. Subjects learned to discriminate a 0.002 kHz difference from the standard 1 kHz tone by the third week. The MMF responses increased in amplitude during training. The largest increase was for the 1.050 kHz stimulus; the smallest for the 1.005 kHz stimulus. The amplitude of the MMF decreased by the 3-week posttraining measurement but was still significantly greater than before training. The MMF latency was shortest for the 1.050 kHz deviant frequency and approximately equal for the other two frequencies. N1m amplitude also increased during training and decreased 3 weeks posttraining. The amplitude for the 1.050 kHz deviant was largest but did not differ significantly from the other deviant frequencies. Both measures revealed that neural response strength increased with auditory training.

Learning of speech cues has also been shown to lead to plastic changes in cortical activity. Kraus et al. used the MMN to determine whether speech discrimination training led to changes in cortical activity [71]. Thirteen normal-hearing adults were trained to discriminate between two variants of the synthesized syllable /da/ (/da<sub>1</sub> and /da<sub>2</sub>) that differed only in the onset frequencies of the second and third formant transitions. A same-different, two-alternative, forced-choice discrimination task with feedback was used to train discrimination in six daily sessions over the course of a week. MMN measurements were obtained before and after training. For the tests, /da<sub>1</sub> was the standard stimulus and /da<sub>2</sub> was the deviant stimulus. Subjects showed a significant increase in discrimination of the speech stimuli

after training that remained stable 1 month after training. The MMN was detected in a majority of the subjects before training. The MMN increased in amplitude and response duration (earlier onset and later offset) after training. Thus, training led to a change in the MMN response.

The study of Kraus et al. established that the MMN could be used to investigate neurophysiologic changes that occurred because of training [71]. In that study, researchers used the same stimuli for training and testing. Tremblay et al. investigated the generalization of the learning of a voice onset time (VOT) distinction that is not used in English [72]. Eighteen normal-hearing subjects participated in the study; half of the subjects received training and the remainder served as control subjects. Behavioral and electrophysiologic measures (MMN) were obtained for discrimination and identification of labial and alveolar prevoicing VOT cues (−20 ms and −10 ms VOT) from all subjects at the beginning of the study. The experimental group underwent 5 days of identification training on the labial prevoiced stimuli. After the training period, behavioral and electrophysiologic measures were repeated on all subjects for both labial and alveolar prevoiced stimuli.

Behavioral tests revealed significant improvements in discrimination and identification of the trained labial stimuli and the untrained alveolar stimuli by the group of subjects who had received training. These results indicate generalization of learning of the prevoiced stop cue from the trained labial stimulus to the untrained alveolar stop cue. Performance did not change significantly in the control group. Similar results were seen in the MMN measures. After the training period, a significant increase occurred in the MMN area on both the trained labial prevoiced stimulus and the untrained alveolar stimulus. Changes in the MMN were largest over the left hemisphere. The control group did not show changes in the MMN between the initial and retest measurements.

Tremblay and Kraus investigated whether learning from auditory training would be reflected in measures of the N1-P2 complex [73]. The N1-P2 complex is thought to reflect stimulus detection. Seven normal-hearing subjects (right handed) were trained to discriminate the prevoiced labial distinction between −20 and −10 ms VOT. Behavioral measures of performance and measures of the N1-P2 complex (at multiple electrode locations) were obtained before and after training. Training resulted in significant increases in behavioral identification performance. The N1-P2 complex showed changes after training; the sub-components of the N1-P2 complex were differentially

affected by training. The P1 amplitude was found to decrease after training at frontal electrode sites. N1 amplitude increased after training at all electrode sites. P2 amplitude increased at all midline electrode sites. P1 and N1 amplitude changes were only seen in the right hemisphere, while the P2 changes were seen over both right and left hemispheres. Because the P1 and N1 response changes were seen only in the right hemisphere, the authors inferred that the learned cue is processed acoustically rather than linguistically. The authors interpreted the changes in N1 and P2 amplitudes as results of increases in neural synchrony after training to an acoustic cue that has increased salience.

The new as well as the experienced hearing aid user who is switched to a new type of hearing aid may be required to learn new auditory cues. There is a paucity of research on the efficacy of auditory training as part of an aural rehabilitation program. Walden et al. demonstrated increases in consonant recognition performance for new hearing aid users with high-frequency sensorineural hearing loss after completion of a training program on consonant recognition [74]. Rubinstein and Boothroyd [75] and Kricos and Holmes [76] failed to show that subjects improved on consonant recognition tests after analytic training; they did demonstrate that subjects improved on sentence recognition tasks after synthetic and analytic training. AEPs might be useful for identifying specific auditory contrasts that are and are not "recognized" by the brain, deciding upon specific identification or discrimination tasks that might be beneficial for the individual, and tracking changes in the detection of the trained stimulus at the cortex.

## CONCLUSIONS

The plasticity of the CAS has been well established in animal models. Results from studies of cortical activity in persons with acquired high-frequency sensorineural hearing loss and unilateral hearing loss provide evidence of cortical activity that parallels that found in animals. However, more information is needed about the types of hearing loss that will result in cortical plasticity and about the perceptual consequences of plasticity. Does plasticity occur in persons with mild, moderate, gently sloping hearing loss? Does plasticity occur when onset of hearing loss is gradual? What is the time course of any change that might occur? AEPs can help provide answers to these questions.

Systematic cross-sectional studies of the relationships between patterns of cortical activity in response to sound as a function of degree and configuration of hearing loss are needed. Longitudinal measures would be required to monitor changes in cortical patterns as a function of duration of hearing loss and to determine the time course of auditory system changes. Studies are needed in which both behavioral measures of auditory perception and electrophysiologic measures of CAS function are measured for researchers to determine how perception changes with changes in the pattern of activity at the cortex.

In the past, researchers have suggested that results from behavioral studies of late-onset auditory deprivation and acclimatization may be from plastic changes in the auditory system. The data that show a direct link between changes in auditory performance and CAS changes do not yet exist, although the electrophysiologic measurement techniques available currently may soon make it possible to confirm or disprove this hypothesis.

The paradigm researchers use to study the relationship between auditory training and cortical plasticity has not yet been used to study performance with hearing aids or aural rehabilitation programs. This paradigm is likely to become an important tool in hearing aid research and research relevant to aural rehabilitation training programs. This approach provides a powerful tool for answering questions about the status of the auditory system after hearing loss. It should also be useful in tracking changes in the auditory system from amplification and auditory training. Ultimately, it may provide the information necessary for evidence-based clinical provision of aural rehabilitation services.

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