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Evidence for multiple mechanisms of cortical plasticity: A study of humans with late-onset profound unilateral deafness

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HIGHLIGHTS

- Experience-related plasticity is apparent in adult humans with both partial and profound unilateral deafness, reflected by altered cortical evoked potentials.
- Changes occur within 1-month post-onset of profound unilateral deafness, and continue for at least 6-months.
- Differences in the pattern of change between partial and profound deafness, and the time course of changes after onset of profound deafness, suggest the presence of multiple physiological triggers and mechanisms of plasticity in humans.

ABSTRACT

Objective: To investigate 1: plasticity due to partial unilateral deafness of slow onset and 2: the time course of plasticity following abrupt, profound unilateral deafness in adult humans using cortical auditory evoked potentials.
Methods: Baseline data were measured from six participants with partial unilateral deafness due to an acoustic neuroma and compared with data from six controls. Further measurements were made in the unilaterally deaf group at 1-, 3- and 6-months post surgery for acoustic neuroma removal and consequent profound unilateral deafness. Data were recorded from 30 channels in response to pure tones presented to the intact ear.
Results: Baseline data revealed statistically higher amplitudes in unilaterally deaf participants but with normal hemispheric asymmetry. Longitudinal data revealed further increases in P1 amplitudes by 1-month post-surgery, and in N1 and P2 amplitudes by 6-months post-surgery, with statistically different scalp field topographies indicating reduced hemispheric asymmetries.
Conclusion: Different patterns of plasticity occur following partial and profound unilateral deafness. Plasticity occurs both relatively rapidly and more gradually over at least 6-months post-surgery.
Significance: The different patterns of change over time are consistent with multiple physiological mechanisms of plasticity. Unravelling these mechanisms and their time course in humans may be relevant in understanding and, ultimately, influencing plasticity for therapeutic gain.

1. Introduction

One of the main motivations for studying experience-related plasticity is to optimize rehabilitation strategies for individuals with sensory impairments such as deafness (Cramer et al., 2011). Progress in this regard requires knowledge of the physiological mechanisms of plasticity as well as understanding the link between physiological changes and perceptual consequences (Wall et al., 2002). Information on physiological mechanisms of plasticity can be inferred from the time course of events, since different physiological mechanisms are thought to follow a different time course (see May, 2011 for a review). The present study was conducted with the goal of extending our understanding of the time course of plasticity associated with late-onset unilateral deafness in humans. To our knowledge this has not previously been investigated using cortical auditory evoked potentials in humans.

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The time course of plasticity following the onset of profound unilateral deafness has been studied in a variety of animal models. Stimulation of the intact ear in animals with abrupt onset unilateral deafness reveals rapid increases in the responsiveness of neurons within the auditory brainstem and cortex, as measured using various near- or far-field electrophysiological techniques. The increase in neural activity primarily occurs in the hemisphere ipsilateral to the ear of stimulation (McAlpine et al., 1997; Mossop et al., 2000; Popelar et al., 1994). Initial increases may be observed within minutes after the onset of unilateral deafness (Mossop et al., 2000). However, further increases may continue over subsequent weeks, months or years (McAlpine et al., 1997; Moore, 1994; Popelar et al., 1994). Changes over time scales such as these suggest the presence of multiple mechanisms by which plasticity may occur after unilateral deafness. There are at least three candidate mechanisms that have been described previously: unmasking of existing neural connections through removal of the inhibitory effects of the deafened ear, changes in the efficacy of existing connections such as by long term potentiation, and growth of new synapses and/or axons (McAlpine et al., 1997; Salvi et al., 2000; Wall et al., 2002). Functional changes in the existing neural architecture may happen relatively quickly whereas structural changes to the neural architecture may be expected to happen more slowly (Draganksi and May, 2008; May, 2011).

Plotting the time course of events is relatively straightforward in animal studies since the onset of deafness is under the control of the experimenter. However, this is usually not possible in humans because the onset of deafness is not under such control. Therefore, while several retrospective studies have reported evidence of plasticity following late-onset unilateral deafness in adult humans (Langers et al., 2005; Maslin et al., 2013; Ponton et al., 2001; Scheffler et al., 1998), relatively little is known about the time course of plasticity in these individuals.

A clinical population who offer the opportunity to study changes in central auditory system (CAS) activity both before and after the onset of unilateral deafness are patients who experience profound deafness as a result of surgery for the removal of a unilateral acoustic neuroma. Acoustic neuromas are space occupying lesions that represent around 10% of all intracranial tumours and arise from Schwann cells on the VIII cranial nerve (Roland et al., 2001). Continued growth of these tumours results in compression of the brainstem and a rise in intra-cranial pressure, hence surgical removal can become necessary (British Association of Otorhinolaryngologists–Head and Neck Surgeons, 2002). There are a range of surgical approaches used to access the site of the acoustic neuroma. The translabyrinthine approach always results in abrupt, profound unilateral deafness. Pre-onset measures of cortical activity in response to sound can be obtained in advance of the surgery and again shortly afterwards. Alternative surgical approaches such as retrosigmoid, middle- or posterior-fossa procedures provide a chance of preserving any residual hearing with suitably located tumours.

To date, there have been relatively few prospective studies that provide any insight into the time course of experience-related plasticity in adult humans following acoustic neuroma surgery (Bilecen et al., 2000; Vasama et al., 1995, 2001). Furthermore, in the studies that have been reported, the results are contradictory and difficult to interpret. At one end of the continuum, some studies report a reduction in cortical activity in the immediate post-operative period following stimulation of the intact ear. Bilecen et al. (2000) used functional magnetic resonance imaging (fMRI) to study one patient and Vasama et al. (1995) used magnetoencephalography (MEG) to study two patients following surgery. Both studies report a reduction in overall cortical activity, within 7–28 days after the onset of deafness. However, over the subsequent period of approximately 1 year, cortical activity returned to normal (Vasama et al., 1995), or exceeded the magnitude seen pre-operatively (Bilecen et al., 2000). The study by Bilecen et al. (2000) suggests that these changes occurred primarily in the ipsilateral hemisphere relative to the intact ear. At the other end of the continuum, Vasama et al. (2001) used MEG to study seven participants undergoing surgery. The results show a short-term (less than 1 month) increase in overall cortical activity with stimulation of the intact ear followed by a decrease to pre-surgery control levels over the ensuing 6 months.

These findings contradict the pattern of changes reported in animal models and retrospective studies in humans, where immediate and permanent increases in neural activity in the ipsilateral hemisphere are observed with stimulation of the intact ear (Maslin et al., 2013; McAlpine et al., 1997; Mossop et al., 2000; Ponton et al., 2001; Popelar et al., 1994).

The reasons for these discrepancies are unclear but one consideration is differences in the degree of baseline hearing loss and the site, duration and size of the tumour prior to surgery, For example, significant plasticity may occur prior to surgery if the acoustic neuroma compresses the auditory nerve or brainstem and results in hearing loss on the affected ear. It is known that such adaptive plasticity can occur following other types of attenuation or alteration of sensory input such as the temporary use of an ear-plug (e.g. Munro and Blount, 2009), following the use of monaural hearing aids (e.g. Munro et al., 2007) or following idiopathic sudden sensori-neural hearing loss in one ear (e.g. Morita et al., 2007) and this has been attributed to changes in central gain mechanisms (i.e. a change in neural firing rate for a given level of stimulation). This homeostatic process is thought to be one means by which the effect of any prolonged asymmetry of sensory input to binaurally sensitive neurons is stabilised in the central auditory system, possibly by functional adjustments in the inhibition of neural activity to the intact ear mediated by the affected ear (Morita et al., 2007). If this is the case then some of the changes observed after surgery may be secondary plasticity in response to the removal of the tumour. Another effect attributed to such adjustments in central gain mechanisms is the phenomenon of a temporary reduction in sensitivity of the intact ear post-surgery, due to the sudden change in both afferent and efferent activity brought about by abrupt deafness on the affected side (Barratt and Prasher, 1988). Such a phenomenon may lead to the impression of a temporary reduction in auditory cortical activity following stimulation of the intact ear for a given stimulus level. In the study by Vasama et al. (2001) it was notable that of the seven participants, three benefitted from preserved hearing post-surgery therefore complete deafferentation could not have occurred, yet the pattern of results was found to be similar to the other participants who had abrupt unilateral deafness. This suggests that the altered responses reported were not due to experience-related plasticity following deafferentation. For example, the authors hypothesise that either the surgical procedures themselves or relief from compression of the brainstem following tumour removal, may lead to changes in cortical responses immediately after surgery. Therefore, in previous studies where results were heterogeneous, the cortical responses from individuals with an acoustic neuroma prior to surgery could already have been different to normal. Furthermore, these differences could have varied across participants. Therefore, the resulting changes post surgery need to be interpreted with caution.

The specific aims of the present study were to: (1) compare cortical activity of individuals prior to surgical removal of an acoustic neuroma with the activity of a control group, and (2) carry out a longitudinal study of the individuals following surgical removal of the acoustic neuroma. The purpose of the first aim was to provide information on the effect of partial, slowly progressing unilateral deafness and the purpose of the second aim was to provide information on the sequence of events up to 6 months following abrupt onset, profound unilateral deafness. It was hypothesised