Neuroscience 283 (2014) 64–77

REVIEW

LOUDNESS MODULATION AFTER TRANSIENT AND PERMANENT HEARING LOSS: IMPLICATIONS FOR TINNITUS AND HYPERACUSIS

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Abstract—Loudness is the primary perceptual correlate of sound intensity. The relationship between sound intensity and loudness is not fixed, and can be modified by short-term sound deprivation or stimulation. Deprivation increases sound sensitivity, whereas stimulation decreases it. We review the effects of short-term auditory deprivation and stimulation on the auditory central nervous system of humans and animals, and we extend the discussion to permanent auditory deprivation (hearing loss) and auditory pathologies of loudness perception. Although there is sufficient evidence to conclude that loudness can be modulated in normal hearing listeners by temporary sound deprivation and stimulation, evidence is scanter for the hearing-impaired listeners. In addition, cortical effects of sound deprivation and stimulation in humans, which may correlate with loudness coding, are still largely unknown and should be the target of future research.

This article is part of a Special Issue entitled: Brain compensation. For good?

Key words: loudness, deprivation, stimulation, tinnitus, hyperacusis, auditory physiology.

Contents

INTRODUCTION

Loudness is an attribute of an auditory sensation that can be ordered on a scale from quiet to loud (Moore, 2003). Loudness is determined mainly by physical sound intensity (i.e., pressure), although it is also affected by other stimulus attributes such as duration, frequency, bandwidth, and monaural versus binaural presentation (for a review, see Glasberg and Moore, 2002) as well as psychological factors such as mood and emotional exhaustion (Hébert et al., 2012). Considerable evidence from previous studies suggests that the relationship between sound intensity and loudness is not static in time, but can be modified by sound deprivation or stimulation. Deprivation increases sound sensitivity, whereas stimulation decreases it. The neurological bases of those loudness shifts remain largely unknown. A hypothetical

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Abbreviations: 2-DG, 2-deoxycglucose; AAF, anterior auditory field; ABR, auditory brainstem responses; AEPs, auditory-evoked potentials; AI, primary auditory cortex; ASR, acoustic startle reflex; ASSR, auditory steady-state responses; AVCN, anteroventral cochlear nucleus; CAPs, compound action potentials; DPOAEs, distortion products of otoacoustic emissions; fMRI, functional magnetic resonance imaging; HL, hearing level; IC, inferior colliculus; MG, medial geniculate; MSO, medial superior olive; SPL, sound pressure level.

http://dx.doi.org/10.1016/j.neuroscience.2014.08.007
0306-4522/

central gain process, which regulates supra-threshold sensitivity somewhere in the auditory system, is one of the most commonly proposed mechanisms. We examine the current evidence for such a mechanism and others, by reviewing the neural changes following auditory deprivation and stimulation in normal-hearing humans and animals along the auditory pathway. In addition, we examine whether similar changes exist in hearing-impaired listeners, both humans and animals. Finally, we discuss the clinical implications of the results for loudness pathologies including tinnitus and hyperacusis and potential treatments.

NEURAL CODING OF STIMULUS INTENSITY

Intensity is first coded at the periphery of the auditory system by the basilar membrane: increased sound pressure increases the amplitude of the basilar membrane displacement, which increases the spike rates in primary afferent auditory nerve fibers and the number of responding fibers (for a review, see Philips, 1987). The large dynamic range (~120 dB) of intensity coding in the auditory nerve is achieved by fibers with different spontaneous firing rates and saturation thresholds (Liberman and Kiang, 1978), which project, at least partly, to different target cells in the cochlear nucleus, as well as different thresholds.

Rate-level functions are not static, but dynamic. Auditory neurons in the inferior colliculus (IC) of guinea pigs can adapt to the time-average sound level by shifting their thresholds to approach the average (Dean et al., 2005). Within a single neuron, this shift occurs only when the average sound level is higher than the neuron's sound level threshold. This shift happens rapidly, within about 160 ms, and appears to reduce the maximum spike rate and slope of the neuron's rate-level function, particularly at high sound levels (Dean et al., 2008). The underlying mechanism is unknown. This shift has been observed in the auditory nerve of cats (Wen et al., 2009), in the IC of guinea pigs (Dean et al., 2005), and in the auditory cortex of ferrets (Rabinowitz et al., 2011) and marmoset monkeys (Watkins and Barbour, 2008).

Auditory deprivation and stimulation using, for instance, earplugs and noise generators change the average sound level and thus, assuming a similar mechanism in humans, may change intensity coding in the auditory central nervous system. The effect of hearing loss on the adaptation of rate-level functions to average sound level has not been studied.

DISTINGUISHING THE CODING OF INTENSITY AND LOUDNESS IN HUMANS

In this section, we briefly summarize the current knowledge on neural correlates of loudness in humans. We distinguish between neural coding of intensity and neural correlates of loudness, because these are distinct concepts: one refers to a physical attribute of sound, whereas the other refers to its perception. However, because they usually co-vary it is often difficult to dissociate their neural correlates.

Normal-hearing listeners

Neuroimaging studies have shown increased neural activity with increasing sound intensity in cortical and subcortical areas: the cochlear nucleus, superior olive, IC, medial geniculate (MG) body, and auditory cortical areas (Sigalovsky and Melcher, 2006), including Heschl's gyri and the planum temporale (Hart et al., 2002, 2003). To better dissociate intensity from loudness neural coding, Röhl and Uppenkamp (2012) used inter-individual differences of loudness ratings from 45 normal hearing subjects. They correlated subjective loudness ratings for a fixed presentation level of 80-dB sound pressure level (SPL) sound to their associated functional magnetic resonance imaging (fMRI) bold signals. They found a significant correlation between the slope of the fMRI signal change and the subjective loudness ratings in the auditory cortices, but not in the IC or MG body. The authors concluded that loudness coding was completed at the level of the auditory cortex because activity at lower levels was more related to sound intensity. The same group also showed that increasing loudness caused by increasing stimulus bandwidth (loudness spectral summation) correlates with activity in the primary auditory cortices, but not in the auditory brainstem (Röhl et al., 2011).

Sound-level-evoked activity in the human auditory system has also been studied using auditory brainstem responses (ABR; Picton et al., 1981), auditory-evoked potentials (AEPs) such as N1-P2 (O'Neill et al., 2008), evoked gamma-band responses (Schadow et al., 2007), and auditory steady-state responses (ASSR; Picton et al., 2003). Conflicting results have emerged from ABR studies: loudness growth was found to correlate with ABR amplitude measures (Silva and Epstein, 2010) and with wave V latency in some studies (shorter latency equaled higher loudness; Serpanos et al., 1997), whereas others reported no correlation with wave V latency (Howe and Decker, 1984).

There is an extensive literature on the loudness dependence of AEPs (for a review, see O'Neill et al., 2008). The amplitude of the N1-P2 complex increases with increasing sound level (Wutzler et al., 2008; Min et al., 2012; Park and Lee, 2013; Wyss et al., 2013). However, most of these studies did not measure loudness, as mentioned by O'Neill et al. (2008). The relationship between intensity and AEP amplitude appears to be related to the serotonin level in the primary auditory cortex (AI) (Wutzler et al., 2008). A decrease in serotonin concentration increases the intensity dependence of the AEPs, whereas an increase in serotonin decreases it.

Two studies have investigated the relationship between the loudness growth function and ASSR in normal hearing subjects (Ménard et al., 2008; Zenker Castro et al., 2008). In both studies, behavioral loudness growth was measured prior to ASSR recordings at a high-frequency modulation rate, such that neural activity within the brainstem was measured (Picton et al., 2003). Both studies reported a correlation between ASSR amplitude and intensity, and between ASSR and loudness. However, Ménard et al. (2008) found that statistically loudness had better predictive value than intensity for ASSR amplitude, at least for two out of six measured