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Research paper

Relationships between otoacoustic emissions and a proxy measure of cochlear length derived from the auditory brainstem response

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ABSTRACT

Brief tones of 1.0 and 8.0 kHz were used to evoke auditory brainstem responses (ABRs), and the differences between the wave-V latencies for those two frequencies were used as a proxy for cochlear length. The tone bursts (8 ms in duration including 2-ms rise/fall times, and 82 dB in level) were, or were not, accompanied by a continuous, moderately intense noise band, highpass filtered immediately above the tone. The proxy values for length were compared with various measures of otoacoustic emissions (OAEs) obtained from the same ears. All the correlations were low, suggesting that cochlear length, as measured by this proxy at least, is not strongly related to the various group and individual differences that exist in OAEs. Female latencies did not differ across the menstrual cycle, and the proxy length measure exhibited no sex difference (either for menses females vs. males or midluteal females vs. males) when the highpass noises were used. However, when the subjects were partitioned into Whites and Non-Whites, a substantial sex difference in cochlear length did emerge for the White group, although the correlations with OAEs remained low. Head size was not highly correlated with any of the ABR measures.

1. Introduction

Otoacoustic emissions (OAEs) exhibit large individual differences even in people having nominally normal hearing (e.g., Bilger et al., 1990; Talmadge et al., 1993; McFadden and Pasanen, 1998, 1999). Even identical twins can differ considerably in their expression of OAEs (McFadden, 1993; McFadden and Loehlin, 1995; McFadden et al., 1996). In addition, group differences are seen in the OAEs of various special populations of humans—differences between the sexes, differences between same-sex and opposite-sex twins, differences between heterosexuals and non-heterosexuals, and differences in boys having and not having attention-deficit hyperactivity disorder (for reviews, see McFadden, 2002, 2008, 2009, 2011). The origins of these group and individual differences are not well understood, although there is strong circumstantial evidence that the degree of exposure to androgens during prenatal development is somehow inversely related to the expression of OAEs (e.g., McFadden, 2011). OAEs, and other aspects of audition, also differ across racial groups (Russell, 1992; Whitehead et al., 1993; McFadden and Loehlin, 1995; McFadden and Wightman, 1983), and these differences may be attributable to melanocyte density in the inner ear (McFadden and Wightman, 1983; Lin et al., 2012). Greater knowledge about the origins of group, individual, and racial differences in OAEs has the potential to provide insight into the cochlear mechanisms that give rise to OAEs.

Over the years, a number of factors have been suggested as possible contributors to the group and individual differences that exist in OAEs. These include differences in the structure of the external ear, the functioning of the middle-ear system, the strength of the efferent supply, the size of the endocochlear potential, and the number and/or arrangement of the prestin molecules in the outer hair cells. Some of these various suggestions have been discussed recently (McFadden, 2009, 2011). Lonsbury-Martin et al.

Abbreviations: ABR, auditory brainstem response; AEP, auditory evoked potential; CEOAE, click-evoked otoacoustic emission; DPOAE, distortion-product otoacoustic emission; OAE, otoacoustic emission; SOAE, spontaneous otoacoustic emission; SPL, sound-pressure level; 2FLD, two-frequency latency difference.

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(1988) explored the possibility that structural anomalies in the organ of Corti could be linked to the pattern of OAEs in a single rhesus monkey.

One possible explanation for individual and group differences in OAEs is individual and group differences in cochlear length. Erixon et al. (2008) measured the anatomy of several dozen human cochleas obtained from cadavers and reported that the anatomical variation was substantial on several dimensions, including length. The individual differences in OAEs also are substantial (e.g., Bilger et al., 1990; Talmadge et al., 1993; McFadden and Pasanen, 1998,1999). Might these facts be related? Several lines of evidence support the possibility that at least one of the known *group* differences in OAEs – sex differences – might be attributable to differences in cochlear length. Measurements of various sorts suggest that the cochleas of human females are about 5–13% shorter than male cochleas (Don et al., 1993; Sato et al., 1991; Kimberley et al., 1993; Moulin and Kemp, 1996; Bowman et al., 2000). (Unfortunately, Erixon et al., 2008, did not report their cadaver data separately by sex.) The cochleas of human females also produce stronger click-evoked OAEs (CEOAEs) and stronger and more numerous spontaneous OAEs (SOAEs) than do the cochleas of human males (e.g., Bilger et al., 1990; Talmadge et al., 1993; McFadden and Pasanen, 1998, 1999). So, perhaps the individual and sex differences in cochlear length contribute to the individual and sex differences in OAEs. One way to think about this is: *if* individual cochleas have approximately equal numbers of those local inhomogeneities (perturbations) in impedance thought to underlie the intra-cochlear reflections that give rise to some forms of OAEs (e.g., Shera and Guinan, 1999), then in shorter cochleas, the perturbations will be more densely packed, which ought to be tantamount to having fewer, but presumably larger perturbations, and thus stronger reflections. The validity of this particular interpretation is not crucial to the basic question, however. If cochlear length does contribute somehow to OAE expression, then measures of the two variables obtained from the same subjects ought to be strongly, and inversely, correlated. (Note that Miller (2007) was pessimistic that the sex difference in cochlear length could be significant for hearing.)

Several methods for obtaining indirect estimates of cochlear length have been proposed over the years (e.g., Schubert and Elpern, 1959; Zerlin, 1969; Dallos and Cheatham, 1971; Parker and Thornton, 1978; Kimberley et al., 1993; Moulin and Kemp, 1996; Bowman et al., 2000). The method of greatest interest here was advanced in an elegant report by Don et al. (1993). (Their method was a variant on a procedure originally used by Teas et al. (1962) while studying the whole-nerve action potential in guinea pigs.) The Don et al. procedure was to measure the latency to wave-V of the auditory brainstem response (ABR) evoked by a click stimulus in the presence of noises of various bandwidths. Specifically, the wave-V latency was measured with highpass noises having cutoff frequencies of about 8, 4, 2, 1, and 0.5 kHz. The purpose of the highpass noise was to prevent auditory fibers tuned to frequencies above the cutoff frequency of the noise from contributing to the synchronized neural response evoked by the click stimulus. As the traveling wave produced by the click propagated apically along the basilar membrane, the first location capable of producing a synchronized response (and thus a wave-V in the ABR) lay just apical to the cutoff frequency of the particular highpass noise present. The latency to the peak of wave-V should be systematically different for the various highpass noises, and the difference in the latencies for any two noise bands can be interpreted as an estimate of the time required for the click's traveling wave to propagate the additional cochlear distance from the region of the higher cutoff frequency to the region of the lower cutoff frequency. If the speed of the cochlear traveling wave is similar in short and long cochleas, then the difference in latency between two fixed frequency regions ought to be smaller in short cochleas than in long cochleas. Thus, the magnitude of the difference in wave-V latency across ears could be taken as a proxy for the cochlear distance between those two frequency regions. Here we will refer to the difference in wave-V latency for any two highpass noises as the two-frequency latency difference or 2FLD.

Don et al. (1993) used the data from their version of the 2FLD to conclude that female cochleas are about 13% shorter than those of males. This outcome was particularly interesting because Sato et al. (1991) made direct measurements of cochleas from cadavers and concluded that female cochleas also were about 13% shorter than male cochleas (for a contrary view, see Miller, 2007). These similar outcomes led us to attempt to measure relative travel time using ABRs in a sample of subjects already being tested for individual and sex differences in a collection of psychophysical tasks as well as in OAEs. The goal was to determine if an ABR-based proxy for cochlear length was correlated with the individual differences exhibited in OAEs. In hopes of getting evoked responses from more localized neural populations in the cochlea, we used tone bursts instead of clicks as stimuli. Specifically, we collected ABR data using tonal stimuli of 1.0 and 8.0 kHz in the presence of noise bands highpassed just above the frequencies of those tones. The expectation was that the 2FLD would be larger for males than for females, reflecting the (previously reported) greater cochlear length in males.

Actually, when this study was designed, our hope was that the tone-burst stimuli would produce usable latencies for both wave-I and wave-V of the ABR. Wave-I latencies are inherently more attractive for current purposes. It is intuitive that wave-I latencies should be more "pure" measures of propagation times along the cochlear partition than wave-V latencies because fewer synapses, neurons, and neural delays are involved. Also, we hoped that the menstrual cycle (see below) would affect wave-I latencies less than wave-V latencies. In the end, however, when the highpass noise was present, most subjects produced reliable, substantial responses only for wave-V. (Note that, theoretically, taking the *difference* in latency between the wave-V peaks evoked by the 1.0- and 8.0-kHz tones ought to remove the additional neural time delays and leave primarily the difference in propagation time of the traveling wave to the two locations along the cochlear partition.)

When designing this study, we were aware of the evidence showing that wave-V latency to click stimuli varies with the menstrual cycle (Elkind-Hirsch et al., 1992a, 1992b, 1994). The direction of effect is that wave-V latency is longer (more male-like) during the pre-ovulatory phase (when estrogen levels are high) than during menses; wave-V latency is about the same during menses and the midluteal phase (when the levels of both estrogen and progesterone are their highest). Our expectation was that whatever factors are responsible for this menstrual effect on latency would operate approximately equally on the high- and low-frequency auditory fibers, and, thus, that the 2FLD would be equally good as a proxy for cochlear length during any phase of the cycle. Data were collected from both the midluteal and menses phases of the cycle for all female subjects. At the time of planning this study, there still was considerable disagreement in the literature about using latencies to various OAE responses as measures of travel time from specific points along the cochlea (see Goodman et al., 2004; Siegel et al., 2005), so no OAE latency measures were collected.

Initially the data were analyzed across all subjects tested, partitioned only by sex and menstrual cycle. When some other data obtained on these same subjects were analyzed separately by racial origin, some interesting differences were evident. Accordingly, these cochlear-length data also were reanalyzed separately by race, and several marked differences did emerge; those race differences are presented in Section 4 below.