Auditory Brainstem Responses Elicited by 1000-Hz Tone Bursts in Patients with Sensorineural Hearing Loss

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Abstract
Auditory brainstem responses were measured in response to 1000-Hz tone bursts from 115 patients with sensorineural hearing loss, presumably of cochlear origin. Mean wave V latencies and variability were comparable to those observed in normal hearing subjects for similar stimuli. The range of interaural differences in wave V latencies for 1000-Hz tone bursts were slightly greater than those observed for clicks, which may not be surprising, given the greater variability in wave V latencies for tonal stimulation, even in normal-hearing subjects. These differences, however, were not affected either by the magnitude or symmetry of hearing loss for frequencies at and above 1000 Hz. These data suggest that tone burst ABRs might be useful in otoneurologic evaluations, especially for patients with asymmetric hearing loss.

Key Words: Asymmetric hearing loss, interaural wave V latency differences, tone-burst ABRs.

C licks are the most commonly used stimulus in otoneurologic applications of auditory brainstem response measurements. Although these broad-spectrum stimuli excite wide regions within the cochlea, factors such as traveling wave velocity and neural density cause the responses elicited by these stimuli to be dominated by high frequency or basal cochlear regions. Figure 1, taken from Kiang (1975), shows single-unit responses to clicks from individual fibers in the cat auditory nerve. Although fibers innervating the entire length of the cochlea are responding, there is greater temporal synchrony among discharges from fibers at the basal end, with greater temporal disparity as one moves towards more apical, lower frequency regions. This greater basal synchrony presumably affects gross potential recordings such that (1) it is easier to record responses to high-frequency stimuli, and (2) responses elicited by broad-spectrum stimuli, such as clicks, are dominated by activity coming from basal or high-frequency cochlear regions (Jerger and Mauldin, 1978; Gorga et al, 1985; van der Drift et al, 1987).

Figure 1 Discharge patterns of auditory neurons. Click neurogram for auditory-nerve fibers. The PST histograms of click responses for 50 units in one animal are plotted in a staggered series to give a three-dimensional visual impression of the response patterns over the array of fibers. Vertical scale, instantaneous discharge rate in spikes per second; horizontal scale, time after the delivery of the click; receding scale, CF of the units. (Reproduced with permission from Kiang NYS. (1975). Stimulus representation in the discharge patterns of auditory neurons. In: Tower DB, ed. The Nervous System. Vol. 3. Human Communication and its Disorders. New York: Raven Press.)
It also is widely recognized that response latency depends upon the cochlear place responsible for generating the response (e.g., Kiang et al., 1965), which also is evident in Figure 1. As a consequence of cochlear tonotopic organization, response latency also depends upon frequency. This pattern is reflected in responses from humans, in which ABRs to high frequency stimuli have shorter latencies than similar responses elicited by lower frequency tone bursts (e.g., Gorga et al., 1988). Since the ABR in response to clicks is dominated by the responses from fibers innervating high-frequency cochlear regions, it is reasonable to assume that hearing loss affecting high frequencies might alter response latency. That is, persons with high-frequency cochlear hearing loss might have longer latency responses than individuals with normal hearing when these responses are elicited by broad-spectrum stimuli such as clicks (e.g., Coats, 1978). This pattern is due to a relative reduction in the contributions to the response from short-latency, high-frequency fibers, with larger relative contributions coming from longer-latency, low-frequency regions. In otoneurologic assessments, these conditions are not problematic if hearing loss is symmetric. Under these conditions, one can compare wave V latency from one side to that observed on the opposite side without concern that any latency asymmetries are the result of differences in peripheral hearing sensitivity.

Patients with asymmetric hearing loss are often seen for otoneurologic evaluations, either because of the asymmetric hearing loss and/or because they present with other symptoms, such as unilateral tinnitus and dizziness. In these patients, as in other patients with symmetric hearing loss, auditory brainstem responses may be measured and wave V latencies examined either in comparison to established norms for absolute latencies or in terms of interaural latency differences.

In cases of asymmetric hearing loss, however, the situation may be more complex because interaural differences in wave V latency may be the result of a neurologic disorder or a consequence of differences in peripheral hearing sensitivity. That is, it may be difficult to determine whether high-frequency, peripheral hearing loss or a mass lesion, such as an acoustic neuroma, is the source of any prolongations in wave V latency. This problem has led a number of investigators to propose the use of latency corrections to account for differences in peripheral hearing sensitivity. For example, Selters and Brackmann (1979) recommend that 0.1 msec be subtracted from wave V latencies for every 10 dB of hearing loss at 4000 Hz exceeding 50 dB. Jerger and Mauldin (1978) recommend subtracting 0.2 msec from wave V latencies for every 30 dB difference in threshold between 1000 and 4000 Hz. Unfortunately, many patients with sufficient hearing loss to meet these criteria provide completely normal and/or symmetric responses before any correction is applied. Thus, it is difficult to know when to use these corrections clinically.

An alternative approach is one in which frequency-specific stimuli are used to elicit the response and these stimuli are selected to fall within regions where hearing sensitivity is more comparable. This approach was recently suggested by Telian and Kileny (1989) and Kileny, Telian, and Kemink (1991) who noted that the latency of wave V for tonal stimuli was quite reliable both within and across normal hearing subjects, a finding that also was reported by Gorga et al. (1988). Kileny and his colleagues showed that in cases of neuropathy affecting auditory brainstem pathways, asymmetries in wave V latency were noted in responses elicited by 1000-Hz tone bursts. The present paper extends this work by examining wave V latencies, evoked by 1000-Hz tone bursts, in patients with sensorineural hearing loss. Such factors as the magnitude and symmetry of the hearing loss at 1000 Hz, as well as the magnitude and symmetry of hearing loss at 2000 and 4000 Hz, are considered in relation to absolute wave V latencies and in relation to the symmetry of response latencies between ears.

**METHOD**

Data are reported from 115 patients for whom sensorineural hearing loss was one of the symptoms that raised concern for possible neuropathy. For this group of patients, other tests, including either click-evoked ABRs or imaging studies, tended to rule out the presence of neuropathy. Thus, the hearing loss in these patients was presumed to be of cochlear origin. Audiograms were divided according to the magnitude of hearing loss at 1000 Hz, enabling us to examine the extent to which hearing loss at this frequency affected the latency of response for a stimulus spectrally centered at this frequency. The audiograms were further divided according to the average hearing loss at 2000 and 4000 Hz. This division enabled us to
consider the extent to which hearing loss at higher frequencies affected the response to lower frequency stimuli.

ABRs were recorded between chlorided silver-silver disc electrodes placed at the vertex and ipsilateral mastoids, with the ground electrode placed at the high forehead. Responses to 2048 stimuli were filtered (100–3000 Hz), amplified (100k), digitized (50 msec per point for 512 points), and averaged (Biologic Navigator). Each averaged response was repeated at least once. Stimuli were delivered by TDH-39 earphones and consisted of digitally generated 1000-Hz tone bursts, gated with Blackman windows, having 2 msec on both the rise and fall, with no plateau. Thus, total stimulus duration from stimulus onset to offset was 4 msec. The time waveform and amplitude spectrum of this stimulus are shown in Figure 2. A stimulus level of 88 dB peak pressure (re: 20 μPa) was used.

**RESULTS AND DISCUSSION**

**Absolute Wave V Latencies**

Figure 3 shows mean absolute wave V latencies elicited by this 1000-Hz tone burst as a function of the average hearing loss at 2000 and 4000 Hz. The parameter in this figure is hearing loss at 1000 Hz. There is a clear trend toward longer latencies as the magnitude of the high-frequency hearing loss increases. That is, in the absence of any hearing loss, the stimuli used to elicit these responses are probably exciting higher frequency cochlear regions. As the hearing loss increases, the contributions to the response from these high-frequency regions are reduced. However, it is important to note that as high-frequency thresholds increase from less than 20 dB HL to over 60 dB HL, the magnitude of this effect is only about 0.5 msec. Furthermore, mean overall wave V latency, collapsed across hearing loss, was 8.05 msec with a standard deviation of 0.3 msec. This value is only slightly longer than what has been observed in normal-hearing subjects for comparable stimuli, where the mean wave V latency was 7.93 msec and the standard deviation was 0.42 msec (Gorga et al., 1988). This agreement, given the differences in hearing sensitivity, suggests that spread of excitation for these stimuli does not differ between normal and impaired ears. It also suggests that stimulus spectrum for tone-burst stimuli is perhaps a more important determinant of response latency than is the relationship between magnitude of the stimulus and hearing loss.

A consideration of the data shown in Figure 3 also suggests that there is no apparent effect of the magnitude of the hearing loss at 1000 Hz on wave V latency. This conclusion is based on the observation that individual functions in this figure were not displaced upward as hearing loss at 1000 Hz increased. This point may be more clear in Figure 4, where wave V latencies are plotted as a function of hearing loss at 1000 Hz, with high-frequency hearing loss serving as
the parameter. Although there was a slight tendency for latencies to be longer for greater losses at 1000 Hz, this effect was again small and not statistically significant.

The trends in our data are similar to the observations made by Telian and Kileny (1989) and Kileny, Telian, and Kemink (1991). That is, wave V latencies in response to 1000-Hz tone bursts were virtually identical for patients with normal hearing versus those with cochlear hearing loss. Our data differ from theirs only in terms of absolute wave V latencies. In our data, both subjects with normal hearing and patients with cochlear hearing loss had mean wave V latencies that were approximately 8 msec, whereas Kileny and his colleagues report a mean latency of about 6.4 msec for these groups. These differences may be the result of differences in stimulus intensity. We used a stimulus of 88 dB peak SPL. Kileny and his colleagues used stimuli of 75 dB nHL, which was derived from normal behavioral thresholds for their tone-burst stimuli. It is reasonable to expect that behavioral thresholds for these stimuli would be between 15 and 20 dB higher than those observed for longer duration stimuli, due to the effects of temporal integration. Additionally, the transformation from dB HL to dB SPL at 1000 Hz is about 10 dB. (That is, 0 dB HL equals 10 dB SPL at 1000 Hz, which reflects the transfer characteristics of the external and middle ears.) Finally, one would need to correct for the extent to which normal subjects had behavioral thresholds exceeding 0 dB HL (re: ANSI standards) in order to provide an SPL value comparable to the one used by us. In view of these considerations, it is easy to see how a stimulus of 75 dB nHL would actually represent a stimulus amplitude of about 105 to 110 dB SPL. This level is significantly larger than the 88 dB peak SPL used in our study and could help to account for these absolute latency differences. It remains undetermined what stimulus level is most appropriate clinically. While it is the case that a more robust response will be elicited by higher amplitude stimuli, such as those used by Kileny and his colleagues, the lower levels used in our study are less likely to excite distant cochlear regions. These differences in stimulus amplitudes notwithstanding, it is important to remember that our data differ only in terms of absolute wave V latencies. Our findings are in agreement in terms of both the trends in their data and their conclusions.

**Interaural Wave V Latency Differences**

Next we examined response symmetry within subjects because we were interested in knowing the extent to which hearing loss caused asymmetric response latencies. Thus, we examined wave V interaural latency differences for subjects with a variety of hearing losses. Unfortunately, to complete this analysis effectively our database was reduced because the number of patients with bilateral responses to 1000-Hz tone bursts decreased as the differences in audiometric thresholds increased beyond 20 dB at 1000 Hz and beyond 40 dB at 2000 and 4000 Hz. Furthermore, the number of subjects for whom data were available from both ears decreased as the magnitude of hearing loss increased both at 1000 Hz and for the average of 2000 and 4000 Hz. As a consequence, bilateral data were available on only 98 patients whose hearing losses did not differ by more than 20 dB at 1000 Hz and whose losses did not differ by more than 40 dB at 2000 and 4000 Hz. It is important to recognize that in this analysis we are describing the effects of differences in thresholds between ears, not absolute thresholds. Many patients whose average high-frequency auditory sensitivity differed by only 40 dB had significant hearing losses.

Table 1 shows mean differences in wave V latencies when the latency on the nonsuspect side was subtracted from that observed on the suspect side (almost invariably the ear with greater hearing loss). Also listed are the standard deviations of these differences. Small posi-
Table 1  Mean Interaural Wave V Latency Differences and Standard Deviations for Patients Whose Hearing Loss at 1000 Hz Did Not Differ by More Than 20 dB

<table>
<thead>
<tr>
<th>Mean Threshold Difference at 2000 and 4000 Hz</th>
<th>20 dB or less</th>
<th>21 to 40 dB</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean wave V Latency Difference (ms)</td>
<td>0.058</td>
<td>0.185</td>
</tr>
<tr>
<td>Standard Deviation (ms)</td>
<td>0.239</td>
<td>0.229</td>
</tr>
</tbody>
</table>

In all cases, wave V latency on the nonsuspect side was subtracted from that observed on the suspect side. Data are shown for patients whose average hearing losses at 2000 and 4000 Hz differed by 20 dB or less and by 21 to 40 dB.

Case Studies

In the cases to follow, we will present data from a few patients, who have either cochlear or retrocochlear pathology. Although our overall neuro-otologic caseload is not very large, these cases may serve to illustrate some of the ways tone-burst ABRs might be used as part of an evoked potential evaluation.

Case 1  Cochlear Hearing Loss

Figure 5 shows data from a 78-year-old female with a principal complaint of hearing loss in her left ear. She had no other auditory/aural complaints. Her audiogram is presented in the top of this figure. Notably, the hearing loss is similar at 1000 Hz, with some asymmetry for higher frequencies. Acoustic-reflex decay was observed for left-ear stimulation. Responses elicited by 80-dB nHL clicks (116 dB peak SPL re: 20 μPa) are shown in the lower panels while responses for 1000-Hz tone burst at 88 dB peak SPL are shown at the upper right. For clicks, both wave V latencies and interpeak latency differences were within normal limits and symmetric between ears, although there was a slight tendency for wave V latencies to be longer on the left side. These findings would be consistent with hearing loss of cochlear origin in the left ear. This conclusion is supported by the observation of symmetric wave V latencies in response to 1000-Hz tone bursts. Wave V latency was 7.95 msec in the left ear and 7.75 on the right side.
Case 2 Cochlear Hearing Loss

Figure 6 shows data from a 60-year-old male with a principal complaint of sudden hearing loss in his right ear. He denies any dizziness or tinnitus. For this patient, hearing sensitivity was asymmetric, both at 1000 Hz and at 2000 Hz, but not at 4000 Hz. Click-evoked ABRs, however, would be more consistent with peripheral hearing loss bilaterally. Once again, these conclusions are supported by the observation of symmetric wave V latencies for 1000-Hz tone bursts stimuli.

Case 3 Acoustic Neuroma

Figure 7 represents data from a 62-year-old female with principal complaints of tinnitus and aural fullness in her right ear. Hearing sensitivity is essentially symmetric through 4000 Hz, although some asymmetries are evident at higher frequencies. Notably, a CT-scan with contrast was interpreted as normal, although an MRI scan with gadolinium contrast revealed a 2-cm intracanalicular acoustic neuroma extending into the cerebellar pontine angle on the right side. Click-evoked responses clearly indicated an abnormality on the right side, and these observations were supported by the results that were obtained with 1000-Hz tone bursts. For tonal stimulation, wave V latency was 8.15 msec on the left side and tentatively taken at 10.4 msec on the right.

SUMMARY

It appears that ABRs, evoked by 1000-Hz tone bursts, result in reliable wave V latencies in patients with cochlear hearing losses. These latencies are reasonably close to values observed in normal-hearing subjects and interaural wave V latency differences are only slightly greater than those observed for clicks. These observations were made regardless of whether the patient had symmetric or asymmetric hearing losses. In cases of confirmed neuropathy, tone-burst ABR latencies showed effects consistent with the presence of a lesion.

Perhaps a better test of the usefulness of tone-burst ABRs in otoneurologic assessments would be provided by examining data from patients for whom the click-evoked ABR was either inconclusive or unable to rule neuropathy in auditory brainstem pathways. This application has been reported by Telian and Kileny (1989)
and Kileny, Telian, and Kemink (1991). Unfortunately, our otoneurologic practice is limited, and we do not have sufficient data to provide such a test. However, the observation that, within limits, absolute wave V latencies in response to 1000-Hz tone bursts are relatively insensitive to hearing loss either at or above 1000 Hz suggests that such stimuli may be usefully applied clinically with patients having asymmetric hearing loss. In such cases, the use of tone-burst stimuli provides supplemental information relative to click-evoked responses and may obviate the need to apply corrections to observed latencies, especially in cases when click-evoked responses are ambiguous.

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