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BRAIN STEM POTENTIALS EVOKED BY ELECTRICAL STIMULATION OF THE COCHLEA IN HUMAN SUBJECTS

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Brain stem potentials were recorded from scalp electrode to biphasic square wave electrical stimulation of implanted electrodes in the cochlea of three patients. Reliable potentials could be recorded that appeared 1.5 to 2.0 msec prior to the customary acoustically-evoked brain stem potentials. The effects of variations in electrical stimulus parameters of rate and intensity were measured. Brain stem potentials can provide objective indices of the effectiveness of electrical stimulation of the cochlea in man.

Recently, permanently implanted devices (cochlear stimulators or implants) have been used to electrically activate the remaining cochlear nerve fibers in deaf patients. The design, site of implantation, and stimulus parameters of these devices are still unresolved. At present the evaluation of these devices is dependent entirely on the subjective experiences of the implanted subjects revealed by various behavioral techniques.

This paper reports the recording of brain stem potentials evoked by electrically stimulating the cochlea in three subjects with intracochlear implants. This is an attempt to define by objective methods the effectiveness of electrical stimuli in activating the central auditory pathways.

The brain stem potentials can be recorded from scalp electrodes using computer averaging techniques. They consist of seven components of submicrovolt amplitudes in the first 10 msec following a brief acoustic stimulus. Wave I is thought to represent the activity of the eighth nerve, waves II and III originate from the pontine portions of the auditory pathway, and waves IV and V arise from the midbrain. The generators of waves VI and VII are unknown. Auditory brain stem potentials have been used to evaluate the auditory capacity of infants and brain stem functions in neurologically impaired patients. In this study the acoustic stimulus usually employed to evoked brain stem potentials was replaced by an electrical pulse applied to electrodes in the cochlea. It is presumed that the electrical stimulus directly activates the fibers of the eighth nerve which in turn transmit the impulses to the central auditory pathway.

METHODS AND MATERIALS

The electrical stimuli applied to the cochlear implants were 0.2 msec biphasic polarity square waves from a constant current stimulator. Current strength was defined by monitoring the voltage drop across a resistor placed in series with one of the electrodes. The stimulus rate varied from 1 to 200/sec.

Stimulation of the cochlear implants was carried out between the most apical electrode located in the scala tympani 16 mm from the round window. The proximal electrode was located just inside the round window. We did not make any systematic attempt to define the effects of stimulating various combinations of the multielectrode array. However, stimulating between one of the intracochlear electrodes and a "ground" electrode located outside of the cochlea resulted in an accentuation of the stimulus artifact that overwhelmed the brain stem potentials. We were therefore unable to make a systematic assessment of the effects of stimulating different electrodes in the cochlea on the brain stem potentials.

Brain stem evoked potentials to the electrical stimuli were recorded between disc electrodes placed on the scalp at the vertex (Cz) and the earlobe contralateral to the stimulus site. An electrode on the ipsilateral earlobe served as the ground. The conventional record-
ing array of vertex to ipsilateral earlobe used to define brain stem potentials from acoustic stimulation was not employed because the artifact of the electrical pulse was particularly large at the earlobe electrode ipsilateral to the implanted cochlear device. Only waves I and III of the brain stem potentials are significantly attenuated if the contralateral earlobe is used as one of the recording sites.

The scalp-derived electrical activity was amplified 100,000 times with a band-pass of 100 Hz to 3 kHz (3 dB down points) and led to a computer for averaging. The computer time base was 10.24 msec and consisted of 256 points with a 40 µsec dwell time at each point. A total of 2,048 stimulus trials comprised each average and a duplicate average was made at each condition to define reproducibility of results. The resulting averages were recorded on an X-Y plotter and the latencies of the various components defined at their peaks. Each patient was tested on at least two separate occasions.

The contribution of facial muscle activity to the evoked potentials was ascertained by recording between electrodes above and below the eye ipsilateral to the implanted cochlea. This control is necessary since the facial nerve passes close to the cochlea and may be activated when the intracochlear electrodes are stimulated. The muscle potentials were amplified $1 \times 10^4$ and averaged in the same manner as when recording the brain stem potentials.

At each recording session the subjects' subjective experiences to the various stimuli were noted. A quantitative analysis of their perception of electrical stimulation is detailed in several papers.$^{11,12}$

One of the subjects (PH) had intact hearing in the ear contralateral to the implanted electrode. In this subject click-evoked auditory brain stem potentials were also recorded.

**CASE REPORTS**

**Case 1.** PH was a 61-year-old male with left-sided Ménière's disease since 1960 which resulted in a profound hearing impairment and disabling episodic vertigo. A left translabyrinthine vestibular nerve section was performed on April 14, 1975, in which the semicircular canals and all of the vestibular contents were removed. The vestibular nerves were sectioned and Scarpa's ganglion excised. The facial and cochlear nerves were not disturbed. The patient wished to serve as an experimental subject for an electrical implant. He appreciated the risks of the procedure as well as the fact that the prosthesis would not be of any benefit. His informed consent was obtained and the bone work necessary for the cochlear implant (opening of the facial recess) was performed at this same operation.

Six weeks later a five-wire electrode was placed from the mastoid through the facial recess into the scala tympani via the round window under local anesthesia. The most apical electrode was located 16 mm from the round window. The electrodes were separated by 4 mm with the fifth electrode lying just inside the round window. A sixth electrode was placed into the bone of the promi-
TABLE 1. LATENCY OF BRAIN STEM POTENTIALS IN MSEC TO ELECTRICAL STIMULATION OF THE COCHLEA.*

<table>
<thead>
<tr>
<th>Pt.</th>
<th>Pos</th>
<th>Neg</th>
<th>Pos</th>
<th>Neg</th>
<th>Pos</th>
<th>Neg</th>
<th>Pos</th>
<th>Neg</th>
<th>M</th>
</tr>
</thead>
<tbody>
<tr>
<td>PH</td>
<td>ND</td>
<td>ND</td>
<td>1.4</td>
<td>1.6</td>
<td>2.0</td>
<td>2.4</td>
<td>3.0</td>
<td>3.4</td>
<td>5-8</td>
</tr>
<tr>
<td>JB</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>1.9</td>
<td>2.4</td>
<td>3.0</td>
<td>ND</td>
<td>6-8</td>
</tr>
<tr>
<td>CR</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>3.0</td>
<td>ND</td>
<td>5-8</td>
</tr>
</tbody>
</table>

*Polarity designation is vertex referenced to contralateral earlobe.
ND - Not detected; M - Muscle.

ontory outside of its lumen. The seventh electrode was to serve as a ground and was placed beneath the temporalis muscle. All electrodes were connected to an external button of pyrolyzed carbon which protruded through the skin above the mastoid.

Case 2. JB was a 61-year-old engineer with profound hearing loss since birth. The etiology of the loss was unknown but presumed to be due to hypoxia associated with birth. A similar type of cochlear implant as received by the first patient was implanted on April 2, 1975, under general anesthesia.

Case 3. CR was 66 years old at the time of study. He had become deaf bilaterally from syphillis and had been implanted with a silver five-electrode system connected to a Teflon® transcutaneous pedestal six years previously.

RESULTS

The averaged brain stem potentials to electrical stimulation of the cochlea derived from the three subjects are presented in Figure 1. The stimulus artifact occupies the initial segment of all three traces, and is most prominent in the bottom records. Table 1 contains the latencies of the various vertex positive components and following negative troughs in the three subjects to 10/sec stimulus at current strengths that produced a moderately loud acoustic sensation.

A large vertex positive wave occurring at a latency between 3 and 4 msec was designated as the IV-V complex to correspond to the largest brain stem component elicited by acoustic stimulation. Wave III was clearly evident in two of the three subjects (PH and JB), while the negative component that occurs between waves III and the IV-V complex at a latency of 2.5 to 3.0 msec was well-defined in all three subjects. In contrast, wave II was evident in only one of the subjects (PH) and wave I could not be defined in any of the subjects.

The parallel between the brain stem potentials evoked by electrical and acoustic stimulation is shown in Figure 2. The records are derived from the subject whose hearing was intact in the ear opposite the electrode implant. The top traces (electrical) are averaged brain stem potentials to electrical stimulation of the implanted ear at 10/sec. The lower traces (acoustic) are the averaged brain stem potentials to 65 dB HL clicks presented at 10/sec. The two sets of potentials have been arranged so

![Fig. 2. (Case 1) Comparison of brain stem potentials evoked by electrical stimulation (upper traces, electrical) and 65 dB HL clicks (lower traces, acoustic). The traces have been adjusted so that components III and IV-V are aligned. The details of the recording and stimulation are identical to Figure 1.](image-url)
Fig. 3. Brain stem potentials evoked by electrical stimulation of the cochlea recorded from the vertex (top traces, scalp) referenced to the contralateral earlobe and facial muscle activity recorded from electrodes above and below the eye (lower traces, orbicularis oculi). Note that the brain stem components III and V occur prior to the appearance of facial muscle potentials whereas the component designated M coincides with the facial muscle potentials.

that both wave III and the IV-V complexes are aligned.

The designation of a component occurring between 5 and 8 msec as the far-field reflection of facial muscle activity was established by simultaneously recording facial muscle activity from the orbicularis oculi (Fig. 3). In two of the subjects (PH and JB), the threshold of current for evoking brain stem potentials was considerably lower than that for eliciting facial muscle activity. In the third patient facial muscle activity was evident at the threshold for auditory sensation. The initial appearance of a muscle potential occurred at 4.5 msec and achieved peak amplitude at 6.5 msec. Thus any component of the scalp-derived averages occurring after 4.5 msec must be considered as a far-field reflection of muscle activity until proven otherwise.

Parametric studies of both strength and rate of stimulation on the evoked brain stem potentials were detailed in one subject. For this individual, changing the current strength at 10/sec stimulus rate had little effect on either the latency or amplitude of the brain stem potentials once threshold for “hearing” was exceeded (Fig. 4 and Table 2). Thus, 0.2 mamp stimulation was said to be “not much different in loudness” from a 2.0 mamp stimulus.

The effects of signal intensity on brain stem potentials using acoustic stimuli are different. Click-evoked auditory brain stem potentials decrease in latency as signal intensity increases while subjects experience a corresponding growth of loudness. It should be noted that “loudness” functions to electrical stimulation at the 100/sec rate have been reported to grow with current strength.

Changes in stimulus rate of the electrical currents affected both the amplitude and the latency of the brain stem
TABLE 2. LATENCY OF BRAIN STEM POTENTIALS IN MSEC TO ELECTRICAL STIMULATION OF THE COCHLEA AS A (f) STIMULUS STRENGTH*

<table>
<thead>
<tr>
<th>Current Strength in mamp</th>
<th>II Pos</th>
<th>II Neg</th>
<th>III Pos</th>
<th>III Neg</th>
<th>IV Pos</th>
<th>IV Neg</th>
<th>V Pos</th>
<th>V Neg</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.10</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>0.15**</td>
<td>ND</td>
<td>ND</td>
<td>2.0</td>
<td>2.4</td>
<td>ND</td>
<td>3.1</td>
<td>ND</td>
<td>3.6</td>
</tr>
<tr>
<td>0.20</td>
<td>1.4</td>
<td>1.6</td>
<td>2.0</td>
<td>2.4</td>
<td>3.0</td>
<td>ND</td>
<td>3.5</td>
<td>4.4</td>
</tr>
<tr>
<td>0.40</td>
<td>1.3</td>
<td>1.6</td>
<td>2.0</td>
<td>2.4</td>
<td>3.0</td>
<td>ND</td>
<td>3.5</td>
<td>4.4</td>
</tr>
<tr>
<td>0.80</td>
<td>1.2</td>
<td>1.5</td>
<td>1.9</td>
<td>2.4</td>
<td>ND</td>
<td>3.5</td>
<td>4.5</td>
<td></td>
</tr>
<tr>
<td>1.20</td>
<td>1.4</td>
<td>1.6</td>
<td>2.0</td>
<td>2.4</td>
<td>3.0</td>
<td>ND</td>
<td>3.5</td>
<td>4.5</td>
</tr>
</tbody>
</table>

*Polarity designation is vertex referenced to contralateral earlobe.

**Threshold for sound sensation.

ND - Not detected.

potentials (Fig. 5 and Table 3). As the stimulus rate increased, the potentials decreased in amplitude and shifted slightly in latency. The amplitude decrement eventually resulted in the loss of evoked potential waveforms at 200/sec stimulus rates.

The latency shift with changes in stimulus rate was slight. The positive component of wave V shifted from 3.6 msec at 1/sec to 4.0 msec at 10/sec, but did not shift further when the rate was increased to 100/sec. This is in contrast to the prolongation of wave V latency by up to 1.0 msec by click signals when the click rate is increased from 10 to 70-100/sec.15-27

DISCUSSION

These results show that brain stem potentials can be reliably evoked by brief electrical pulses applied to the cochlea in man. The potentials correspond in form and amplitude to those customarily derived by transient auditory signals (clicks) with two principal exceptions. First, wave I could not be detected using the electrical stimulus because of the occurrence of a large stimulus artifact at the time that wave I would have appeared, and the electrode configuration employed (vertex-contralateral earlobe) is not optimal for this component. Secondly, the components evoked by electrical stimulation occur 1.5 to 2.0 msec before those evoked by acoustic stimulation since electrical stimulation bypasses the temporal requirements of an acoustic signal to travel through the ear canal and middle ear and be transduced into nerve impulses in the cochlea.

While the exact location of the stimulating electrode in relation to the surviving nerve fibers is unknown, several phenomena were observed as to the effects of stimulus intensity and rate on the brain stem potentials. First the amplitude and latency of brain stem potentials evoked by electrical stimulation of the cochlea changed little once the threshold of “hearing” was achieved. This is to be contrasted with the brain stem potentials evoked by acoustic signals in which latency shortens and amplitudes increase as stimulus strength is raised. These differences indicate that the dynamic range for electrical stimulation is considerably restricted (approximately 10 dB) compared to acoustic stimulation (approximately 80 dB). The
observation that central conduction times, i.e., the latency differences between wave I and the other components of the auditory brain stem potentials, are unaffected by signal strength\(^ {18}\),\(^ {19}\) also reflects the primary role of the cochlea in transforming signal intensity into appropriate neural codes. The graded activation of the eighth nerve fibers by electrical stimulation is unlikely because the fibers have the same general size and degree of myelination. Thus, the electrical stimuli probably engage all of the remaining eighth nerve fibers at about the same current strengths.

Changes in stimulus rate from 10/sec to 100/sec had little effect on the latency of brain stem potentials evoked by electrical stimulation whereas these potentials can shift up to 1.0 msec when acoustic stimulation rates are correspondingly changed.\(^ {13}\)\(^ {-17}\) This result suggests that the shift in latency with increasing rates of auditory stimulation also derives from cochlear processes.

Changes in electrical stimulus rate did affect the amplitude of potentials. The components decreased in amplitude above 50/sec and could not be detected at 200/sec. Correspondingly the amplitude of acoustically-evoked brain stem potentials also decreases with stimulus rate. Even though the amplitude of both the electrically and acoustically evoked brain stem potentials diminish to the point of absence, auditory sensations persist. Thus, it is likely that these components of the brain stem potentials have little direct relation to the neural mechanisms underlying loudness discrimination.\(^ {20}\)

The results of these studies on three patients provide objective evidence that electrical stimulation of the cochlea in man can activate some of the same neural pathways that are also involved by natural acoustic stimulation. The ability to detect brain stem potentials from electrical stimulation of the cochlea could provide a means for objectively defining the function and optimum placement of a stimulating prosthetic device. Certainly these measures can never substitute for behavioral tests, but they provide another level of analysis which can provide useful parametric data as to the functioning of the cochlear nerve fibers and the prosthesis.

### REFERENCES


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