Auditory Evoked Potentials: Relationship to Stimulus Intensity and Effects of Noise Exposure

Buelke

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AUDITORY EVOKED POTENTIALS: RELATIONSHIP TO STIMULUS INTENSITY AND EFFECTS OF NOISE EXPOSURE

by

Judith L. Buelke

A Thesis
Submitted to the
Faculty of the Graduate College
in Partial Fulfillment
of the
Degree of Master of Arts

Western Michigan University
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INTRODUCTION

Exposure to sounds of high intensities has been shown to result in permanent and/or temporary hearing losses in both man (Davis, Morgan, Hawkins, Galambos and Smith, 1950; Burns, 1968; Cohen, Anticaglia and Jones, 1970) and cats (Miller, Watson and Covell, 1963). Permanent losses are represented in a threshold shift from a pre-exposure baseline level which never fully recovers to the original baseline. Temporary losses are represented by a threshold shift from pre-exposure levels which recovers with time to the original baseline. Such temporary threshold shifts vary in type and degree and are a complex function of the type of sound to which the subject is exposed (pure tone, wide- or octave-band noise), intensity and duration of exposure, etc. (Ward, Glorig and Sklar, 1958; 1959a; 1959b).

Studies concerning measurement of electrophysiological events during pre- and post-exposure conditions deal exclusively with responses at the cochlear level. In the normal ear of cats, minutes of exposure to pure tones of high intensity produce no change in the amplitude of the cochlear microphonic, however, the first neural response ($N_1$) of the ear is enhanced (Galambos and Rupert, 1958). This enhancement is observed in both the anesthetized and unanesthetized preparation. When middle ear muscles are sectioned, both the microphonic and $N_1$ response are severely reduced and show a recovery to the pre-exposure level only after several minutes (Hughes and Rosenblith, 1957; Galambos and Rupert, 1958).
At present, there appears to be a surprising lack of data available concerning effects of noise exposure on responses in central auditory areas. The effects seen at the cochlea would be expected to be reflected in some manner at higher levels, and the value of such a correlate would seem obvious in terms of accessibility and, perhaps less obviously so in terms of neural coding. The gross evoked potential is one electrophysiological event which could be utilized to assess retrocochlear changes following exposure to noise.

The evoked potential is an often used but little understood gross electrical response. The parameters of this potential depend upon the area of the brain from which one records, characteristics of the stimulus and recording techniques utilized. Generally, it is observed as a consistent directional voltage shift time-locked to the presentation of a given stimulus. Attempts to unravel the relations between it and its underlying neural mechanisms have been extensive. It is from the work by such men as Adrian, Bishop and Eccles that the current views of evoked potentials as summated, graded synaptic activity have been developed (Thompson, 1967). The types of synaptic activity and neurons which are responsible for the components of the evoked potential remain uncertain.

Fox (Fox and O'Brian, 1965; Fox, 1971) has suggested that, at least under some circumstances, the form and size of visually evoked potentials are statistically related to the sequential probability of single unit firing. If such were the case, the large initial positive deflection of the surface cortical evoked response might
reflect the maximum frequency of cell discharge. Katsuki (1962) reported that the rate of single unit firing at subcortical auditory centers varied directly with sound intensity. If the relationship proposed by Fox is applicable to the auditory system, one might expect to find systematic variation in form, amplitude and/or latency of evoked potentials with changes in sound intensity.

In a study concerned with the relationship between auditory click intensity and responses at the cochlea and auditory nerve in guinea pigs, Gulick, Herrmann and Mackey (1961) found that the microphonic and $N_1$ response followed a power function of a sigmoid nature. They reported that the slopes of such functions plotted for individual animals were greater than 1.0, and since it is assumed that the magnitude of the $N_1$ response to auditory clicks is determined primarily by the number of neurons firing, it would be reasonable to expect, given a normal distribution of fiber thresholds, that a linear increase in the microphonic would give rise to a positively accelerated $N_1$ response followed at higher intensities by a negative acceleration. Some data are available which support this sigmoidal relationship (Gulick, Herrmann and Mackey, 1961; Rosenblith, 1959). These authors also reported that the latency of $N_1$ varies indirectly with click intensity over a range of 2.0 msec at low intensities to 1.2 msec at higher levels. Similar results from the cochlear nerve (Hughes and Rosenblith, 1957; Teas, Eldredge and Davis, 1962) and the superior olivary complex (Boudreau, 1965) have been reported.
Further evidence for this view has been suggested by Miller, Moody and Stebbins (1969). They reported that the amplitude of auditory cortical evoked potentials in the monkey appeared to be directly related to stimulus intensity, however, little consistent variation in the initial latency of the potential could be seen.

The present experiment was designed to look at three questions: (1) does amplitude and/or latency of evoked potentials from subcortical auditory areas vary systematically with stimulus intensity, (2) will exposure to intense noise effect one or both of these parameters and (3) will any changes that might occur show recovery with time?
METHODS

Subject Preparation

Two male and four female cats, weighing between 3.0 and 4.0 kg were used as subjects. All were kept on adlibitum diets prior to the experiment and were treated with an ear cleaning preparation a few days before the onset of the experimental session.

Surgery was conducted under aseptic conditions within the experimental chamber. Subjects were anesthetized with sodium pentobarbital (35mg/kg, intraparitoneal) and prepared for stereotaxic placement of the recording electrode. A David Kopf stereotaxic instrument fitted with hollow ear bars was used for electrode placement.

A midline incision was made across the skull and skin and muscles retracted. A burr hole was drilled in the skull appropriate for entrance to the target location. Four subjects received unilateral placement in the pars principalis nucleus of the medial geniculate body, coordinates for which were: anterior - 6.5mm; lateral - 9.0 mm; horizontal - approximately +1.0 mm. The remaining two subjects received unilateral placement within the inferior colliculus, the coordinates for which were: posterior - 2.0 mm; lateral - 6.0 mm; horizontal - approximately +1.0 mm (Snider and Niemer, 1961).

Following surgical preparation of a subject, the recording electrode was lowered to the target area. During this time auditory clicks were presented and brain activity monitored to assure proper
placement. The anterior and lateral coordinates were strictly followed and all final placements were within ±0.5 mm of the horizontal coordinate.

All subjects remained anesthetized throughout the entire experiment and were sacrificed following the session with an additional dose of sodium pentobarbital.

**Apparatus**

The apparatus used for presenting the stimulus and recording evoked potentials is diagrammed in Figure 1. Auditory clicks were produced by a Tektronix type 161 waveform generator coupled to a type 162 pulse generator. The pulse was amplified through a McIntosh MC 2105 power amplifier and fed to two University 808-8A Loudspeakers mounted in the Industrial Acoustics double-walled, sound-deadened experimental chamber. The inside walls were covered with 1/8" masonite strips to make the room reverberant. The intensity of the clicks was monitored in the control room on a Heathkit vacuum tube volt meter. A General Radio 1390-B random noise generator was used to produce sound for exposure. For one session, the random noise generator was connected directly to the amplifier, producing wide-band noise. For the remaining experimental sessions, the output of the random noise generator was fed through Allison 2BR band-pass filters, producing a 2 kHz octave-band noise.

Evoked potentials were recorded with an 0.4 mm platinum-irridium monopolar electrode, insulated to within 0.5 mm of the tip with Form-Var varnish. The reference electrode was attached to the
ear bar of the stereotaxic instrument. The signal was amplified by a Grass P-5 preamplifier and recorded on one channel of an Ampex recorder on magnetic FM tape. The click was recorded on a second channel and the voice channel was utilized for session and trial identification. The evoked potential was simultaneously fed to a Tektronix RM 565 oscilloscope. The oscilloscope was triggered by the pulse generator and records of single and multiple traces were taken with a Grass C-4 camera.

Data recorded on tape for three subjects were later fed into the analog to digital converter and multiplexer of a PDP-8 computer, which averaged 40 individual evoked potentials and displayed the result on the RM 565 oscilloscope. Film records of these displays were taken with the Grass camera.

Experimental Procedure

With the recording electrode positioned, 2 msec squarewave clicks were presented at increasing intensities at a rate of one per second, with a silent interval of approximately 60 seconds between each intensity level. Click intensity was measured in voltage output due to the difficulty of obtaining an accurate absolute sound pressure measure for short duration impulse sounds. However, it is estimated that click intensities utilized in this investigation ranged from approximately 60 dB to 110 dB\(^1\). Table I summarizes the pre-exposure treatment for each subject.

\(^1\)All dB values in this paper are in reference to 0.0002 dyne/cm\(^2\).
Following the collection of the baseline data, each subject was exposed to a prolonged period of intense noise. The noise was presented binaurally in a free-field manner. For subject 2m, exposure was to wide-band noise, while all other subjects received exposure to 2 kHz octave-band noise. Table II summarizes type and duration of exposure for each subject.

Immediately following noise termination, a repeated series of clicks at increasing intensities was presented to each subject in the same manner as during baseline conditions. If the preparation remained in good condition and required no further sodium pentobarbital to maintain its anesthetized state, further repetitions of the click series were made at 40 minutes and 80 minutes post-exposure. Table II summarizes post-exposure data collection procedures for each subject.

Data Analysis

Two parameters of the evoked potential, peak-to-peak amplitude and latency, were looked at across intensities and experimental conditions. All measurements were made from filmed records of either multiple traces of five individual evoked potentials, in the cases of subjects 1m, 2m and 1c, or from computer-averaged traces of 40 individual evoked potentials, in the cases of subjects 3m, 4m and 2c. The peak-to-peak amplitude of the evoked response was measured from the outermost limits of the greatest positive to the greatest negative deflection. Latency was measured as the distance from the initiation of the trace to the peak of the first negative deflection.
of the evoked potential for medial geniculate recordings and to the first positive deflection for inferior colliculus recordings.

In all records, upward deflections are negative.
RESULTS AND DISCUSSION

Pre-exposure

Comparing peak-to-peak amplitude of the evoked potentials across stimulus intensities, a clear change in amplitude was found for all subjects (see Figure 2). This increase in amplitude with increased intensity is in general agreement with previously reported data collected from other brain areas (Hughes and Rosenblith, 1957; Gulick, Herrmann and Mackey, 1961; Boudreau, 1965; Miller, Moody and Stebbins, 1969; Guiteras, 1971). This direct relationship with stimulus intensity can be seen clearly in both computer-averaged potentials and multiple traces of five individual evoked potentials (see Figure 4).

In recent years, several attempts have been made to relate the power law which appears to govern psychophysical data with neurophysiological activity of sensory systems (Stevens, 1970). Briefly, this principle states that equal stimulus ratios produce equal sensation ratios. Many of the studies finding a direct relationship between stimulus intensity and amplitude or frequency of electrophysiological events have reported that this interaction takes the form of a power function, at least over relatively moderate ranges of stimulus intensity. The changes in amplitude here, however, more closely approximate a logarithmic relationship, most clearly illustrated by data obtained from subjects 1m, 1c and 2c. Log-log plottings of amplitude data show much less resemblance to a power
function than do semi-log plottings to a general logarithmic trend as is shown in Figure 2.

Latency measures across intensities show a decrease in latency with increased stimulus intensity (see Figure 3). This indirect relationship is again in general agreement with reported findings (Hughes and Rosenblith, 1957; Rosenblith, 1959; Gulick, Herrmann and Mackey, 1961). Except for subjects 3m and 4m, the latency changes seem to rather closely approximate logarithmic functions. The previously mentioned studies make no attempt to relate latency changes to the power law.

Do sensory systems, and the auditory system in particular, generate power-law transformations that can be detected as neurelectric effects? Stevens (1970) has reviewed the evidence supporting such a view and has concluded:

"The same compelling constraints of ratio invariance (in psychophysical data) cannot yet be said to pilot out expectations through the turbulence of electrophysiology. To be sure, the power function has been found to govern the growth of neurelectric effects in numerous experiments but few investigators would feel astonished if their electrodes recorded a different function." (p 1050)

A considerable amount of research will undoubtedly be required to clear up the relationship between psychophysical and neurophysiological events.

The variability among subjects in absolute amplitude for a given stimulus intensity and the magnitude of change across intensities is great. Such results are not surprising and most probably can be accounted for by intersubject variability, the differences between measurements taken from multiple traces of
individual evoked potentials and those taken from computer-averaged single displays, as well as the probability of slight differences in electrode placement across subjects.

The latencies of the evoked responses from the medial geniculate body for a given stimulus intensity show good agreement across subjects. Agreement between subjects with inferior colliculus placements is not so good, however, both subjects' evoked potentials show a markedly shorter latency at any given click intensity than those potentials recorded from the medial geniculate. This difference averages 4 to 5 msec, a time indicative of an additional synaptic transmission. As placements of electrodes were not verified histologically, the latency measures, in addition to careful adherence to stereotaxic coordinates, provide reasonable assurance as to the reliability of the actual recording sites.

Post-exposure

The peak-to-peak amplitude measure appears to maintain the pre-exposure direct relationship with stimulus intensity, showing a rather uniform decrement at immediate post-exposure trials at low and moderate click intensities in four of the six subjects (see Figure 2). There are at least two possibilities as to why these effects were not seen in subjects 3m and 2c. The first concerns exposure treatment. All other subjects received exposure to noise of either higher intensity (1m, 2m and 1c) or longer duration (4m). Perhaps this combination of intensity and duration was inadequate to produce changes that could be reliably detected as shifts in evoked potential
amplitude. Secondly, studies of temporary threshold shifts following exposure to noise report wide intersubject variability in both humans (Burns, 1969; Cohen, Anticaglia and Jones, 1970; Smitley and Rintelmann, 1971) and animals (Elliot and Fraser, 1970). Exposure to noises of fixed parameters may result in a large temporary threshold shift in one subject but little or no shift in another. The reasons for such variability are unclear, but may account for the present results.

Subject 2m showed a substantial increase in amplitude over the pre-exposure level at the lowest click intensity. This post-exposure trial occurred, through error, at approximately one minute post-exposure and the resulting increase may reflect the sensitization effect reported by Hughes and Rosenblith (1957). They found a slight decrease in the first neural responses (N1) during the first few seconds following noise termination, then a large increase above pre-exposure levels which peaked at one minute post-exposure, followed by an extended period of very great decrease. If, indeed, the evoked potential is a summated response of ongoing single unit activity, and it can be assumed that lower order changes are reflected in higher center activity, this result is not surprising. The estimated time of click presentation corresponds to the sensitization peak time, and this interpretation is further supported by the fact that the measure taken at 40 minutes post-exposure shows a reduction over pre-exposure amplitude.

In those subjects showing an amplitude decrement at moderate click intensities, it should be noted that no decrease occurs at the
highest intensity presented. Such a result resembles the psycho-physical phenomenon of loudness recruitment (Fowler, 1963). An abnormally rapid increase in loudness can often be demonstrated in humans with temporary or permanent hearing impairment due to injury of the organ of Corti and is often seen in humans following exposure to intense noise (Hawkins, 1966). Although the person's threshold of hearing may be higher than normal, he will rapidly increase his perception of loudness as intensity is increased, until at higher intensities, his loudness perception is equal to that of the normal listener.

Since noise exposure produces damage to the organ of Corti (Engstrom, Ades and Andersson, 1963), one might expect the exposures used in this experiment to produce electrophysiological changes which could be correlated with the behavioral phenomenon of loudness recruitment. Figure 2 shows that noise exposure did depress the amplitude of the evoked potentials at low and moderate intensities, while the amplitude at the highest stimulus intensity was not affected. This parallels what is happening in loudness recruitment: responses to low intensity stimuli are depressed while those to high intensity stimuli are essentially unchanged.

It is tempting to accept these results as a neurophysiological correlate of a behavioral phenomenon. However, extreme caution must be exercised in doing so at this point, for at least two reasons. First, it is emphasized that peak-to-peak amplitude of the evoked potential was unaffected only at the highest click intensity presented to each subject, and as can be seen in Table I, this intensity ranged
from 0.8v to 4.2v. This discrepancy in the intensity at which a
decrement is no longer seen is somewhat puzzling, unless we again
call upon intersubject variability to account for it. Additionally,
one would expect to find no decrement in amplitude at the two or
three highest intensities presented, especially for those subjects
receiving clicks of up to 4.2v. That this is not the case casts
further suspicion on the loudness recruitment interpretation.

Secondly, when recruitment is present, the change in the
increment of loudness is greatest near the threshold of hearing and
rapidly decreases with increases in stimulus intensity, and is most
often reasonably complete at approximately 40-50 dB above threshold
(Fowler, 1963). Although reliable threshold data are not available
for the cat, if it is conservatively estimated that threshold for
click stimulation is 20 dB, recruitment would be expected to be
complete at a dB level very near the lowest estimated intensities
presented here.

Anesthesia could also be an important contributing factor in
the results obtained here, but, for obvious reasons, there are no
comparable behavioral data available to suggest its role. Further
examination in chronically implanted subjects is needed to more
accurately assess the implications of these findings.

In those subjects showing a decrease in evoked potential
amplitude, and in one subject that did not (2c), a systematic
increase in latency following exposure can be seen (see Figure 3).
The increases in latency seen for subjects 1m and 1c follow a form
of change similar to that of the amplitude measure, e.g. a rather
uniform change across intensities which maintains the pre-exposure inverse relationship with stimulus intensity.

Subjects 2m, 4m and 2c showed increases in latencies at all click intensities, but with much greater effects seen at the higher intensities. The initial decrement in latency, as compared to pre-exposure measures, seems to represent a failure to respond to increases in stimulus intensity. That is, in pre-exposure measures the latency shows an inverse relationship to stimulus intensity, whereas in post-exposure measures taken five minutes following noise termination, little change is seen in latency with further increases in stimulus intensity. Later measures from these subjects show a partial recovery of this inverse relationship with time.

The reasons for these two different types of latency changes are unclear, and there seem to be no a priori reasons to expect one type of change rather than the other. It is, however, reasonable to expect such a change to be generally consistent. The differences presumably cannot be explained by two central processes, an attractive hypothesis had the two types of changes been found in only one of the two recording sites. This would not seem to be the case, unless the two processes are represented in both the medial geniculate and the inferior colliculus and slight variations in electrode placement within the two regions happened to selectively record one process rather than the other. If one is again inclined to consider the loudness recruitment interpretation of amplitude changes, a more uniform change in latency across intensities might intuitively appear to be more compatible than the greatest increase occurring at the
highest intensities. However, subject 2c, showing no reliable post-exposure amplitude shift, did show a small but consistent latency change, greatest at the higher intensities. Thus, the immediate changes in latency following exposure to noise appear to be very complex. Little more can be said than that noise exposure seems to generally increase latency of evoked potentials over values obtained prior to this experimental treatment.

In those subjects showing post-exposure changes in amplitude, recovery trials were presented to 2m and 4m. Data from both of these subjects suggest a partial recovery of amplitude toward pre-exposure levels with time. Initial recovery trends in the direction of latency baseline values can also be seen for subjects 2c, 2m and 4m. Again, these latency changes for 2m and 2c are most clearly evident at the higher intensities, while recovery for subject 4m seems to be somewhat more uniform across intensities. The partial recovery of measures reported here parallel recovery of temporary threshold shifts in human and animal subjects (Davis, Morgan, Hawkins, Galambos and Smith, 1950; Cohen, Anticaglia and Jones, 1970; Miller, Watson and Covell, 1963). Whether or not the time course of the recovery trends seen in both amplitude and latency measures of the evoked potentials is similar to that of the temporary threshold shift remains to be seen.

More extensive investigations of the phenomena reported here would provide a clearer interpretation of the effects noise exposure has upon these parameters of the evoked potential. The anesthetized preparation utilized in this study limited the available post-exposure
testing period, as greater time intervals would have necessitated further injections of anesthetic and perhaps added to a confounding variable in recovery of the evoked potential measures to baseline values. Neither can results from this experiment be interpreted as wholly temporary. Perhaps permanent effects were induced by the prolonged exposure period. Further research into this area is needed to provide answers to these questions and to determine if, indeed, the changes in auditory evoked potentials seen here can be considered physiological counterparts of documented behavioral and perceptual phenomena.
### TABLE I

Pre-exposure treatment for each subject.

<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>PLACEMENT</th>
<th>CLICK INTENSITY IN VOLTS</th>
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<tbody>
<tr>
<td></td>
<td></td>
<td>.015</td>
</tr>
<tr>
<td>1m</td>
<td>medial geniculate</td>
<td>x</td>
</tr>
<tr>
<td>2m</td>
<td>medial geniculate</td>
<td>x</td>
</tr>
<tr>
<td>3m</td>
<td>medial geniculate</td>
<td>x</td>
</tr>
<tr>
<td>4m</td>
<td>medial geniculate</td>
<td>x</td>
</tr>
<tr>
<td>1c</td>
<td>inferior colliculus</td>
<td>x</td>
</tr>
<tr>
<td>2c</td>
<td>inferior colliculus</td>
<td>x</td>
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## TABLE II

Exposure treatment and post-exposure trials for each subject.

<table>
<thead>
<tr>
<th>SUBJECT</th>
<th>Type</th>
<th>Duration</th>
<th>Intensity</th>
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<th>40'</th>
<th>80'</th>
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<tr>
<td>1m</td>
<td>2K octave-band</td>
<td>75 min</td>
<td>106 dB</td>
<td>x</td>
<td></td>
<td></td>
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<tr>
<td>2m</td>
<td>wide-band</td>
<td>60 min</td>
<td>110 dB</td>
<td>x</td>
<td>x</td>
<td></td>
</tr>
<tr>
<td>3m</td>
<td>2K octave-band</td>
<td>90 min</td>
<td>100 dB</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
<tr>
<td>4m</td>
<td>2K octave-band</td>
<td>105 min</td>
<td>100 dB</td>
<td>x</td>
<td>x</td>
<td>x</td>
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<tr>
<td>1c</td>
<td>2K octave-band</td>
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<td>108 dB</td>
<td>x</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2c</td>
<td>2K octave-band</td>
<td>90 min</td>
<td>100 dB</td>
<td>x</td>
<td>x</td>
<td>x</td>
</tr>
</tbody>
</table>
FIGURE LEGENDS

Figure 1 Diagram of apparatus used for stimulation and recording.

Figure 2 Changes in peak-to-peak amplitude of evoked potentials from the inferior colliculus and medial geniculate with increased stimulus intensity under all experimental conditions presented to each subject.

Figure 3 Changes in latency of evoked potentials from the inferior colliculus and medial geniculate with increased stimulus intensity under all experimental conditions presented to each subject.

Figure 4 Pre-exposure evoked potentials collected across click intensities from the medial geniculate (subject 3m). The left column shows five superimposed evoked potentials at each stimulus intensity presented. The column on the right gives the computer-average of 40 potentials from the same click series.

Figure 5 Pre- and post-exposure evoked potentials from the inferior colliculus (subject lc), for two click intensities (0.8v and 2.5v). The top four traces contain five superimposed potentials.
The bottom four traces show individual potentials recorded during the same series of clicks at each intensity.

Computer-averaged evoked potentials from the medial geniuculate body (subject 4m). Traces indicate changes across the three highest intensities presented to this subject, as well as changes at a given intensity across experimental conditions.
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Figure 3

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Subject 3m

Five Superimposed Evoked Potentials

0.8 v

0.42 v

0.25 v

0.15 v

0.08 v

0.042 v

0.025 v

0.015 v

0.0 v

100 μv — 25 msec

Computer-averaged Evoked Potentials

FIGURE 4

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Subject 1c

Five Superimposed Evoked Potentials

<table>
<thead>
<tr>
<th>0.8 v</th>
<th>2.5 v</th>
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<tbody>
<tr>
<td>Pre-exposure</td>
<td>Pre-exposure</td>
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<tr>
<td>5' Post</td>
<td>5' Post</td>
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</tbody>
</table>

Individual Evoked Potential

<table>
<thead>
<tr>
<th>0.8 v</th>
<th>2.5 v</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-exposure</td>
<td>Pre-exposure</td>
</tr>
<tr>
<td>5' Post</td>
<td>5' Post</td>
</tr>
</tbody>
</table>

100 µV — 25 msec

FIGURE 5
Subject 4m

Computer-Averaged Evoked Potentials

0.8 v

0.42 v

0.25 v

Pre-exposure

5' Post

40' Post

25 msec

100 μv

FIGURE 6
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Fowler, E.P. Loudness recruitment. Archives of Otolaryngology, 1963, 78, 748.


