Electrocochleography and Experimentally Induced Loudness Recruitment*

James E. Pugh, Jr., David B. Moody, and David J. Anderson

Kresge Hearing Research Institute and the Department of Otorhinolaryngology,
The University of Michigan, Ann Arbor, MI 48109, USA

Summary. The relationship between changes in loudness and the cochlear whole-nerve potential following experimentally produced deafness was studied in an animal model. Reaction time of a subject's response to an auditory stimulus has been shown to be an index of loudness in human experiments and has been adapted to nonhuman primates. In a series of experiments, four macaque monkeys were operantly conditioned to respond to 8-kHz tones over a range of 0–80 dB SPL, and their reaction times to pure tone stimuli were measured. Whole-nerve cochlear action potentials were recorded from chronic inner-ear electrodes. The relationship between behavioral and electrical measures of loudness recruitment were examined in animals with both temporary and permanent noise-induced hearing loss.

Loudness recruitment was demonstrated experimentally after a 1-h exposure to a high-intensity 8-kHz octave band of noise. Excellent agreement was observed between the reaction time function and the action potential input-output function at intervals of 0.5, 12, 24, 48, and 84 h after exposure.

Permanent hearing loss was produced in some of these animals by a much longer duration of exposure to the 8-kHz octave band of noise. Recruitment was observed in both the behavioral and the electrical measures. Histological studies of these damaged cochleas revealed primarily outer hair cell destruction, with a relative sparing of inner hair cells and nerve supply. The findings of this study are interpreted as strong support for the clinical electrocochleogram as an objective indicator of the presence of loudness recruitment.

Key words: Loudness recruitment — Electrocochleography — Acoustic trauma — Action potentials — Monkeys.

Loudness recruitment, the abnormally rapid increase in loudness as sound intensity is raised above threshold, is observed frequently in hearing disorders. Precise observations of the loudness recruitment phenomenon began about 50 years ago, and a physiological model has gradually evolved. The steps in this evolution may be summarized as follows: (1) the first measurement of recruitment by various pychophysical loudness balance techniques (Fowler, 1936, 1937; Luscher and Zwislocki, 1949; Reger, 1936; Steinberg and Gardner, 1937); (2) recognition of its association with cochlear rather than conductive deafness (Fowler, 1937); and (3) the demonstration, from clinical material, that loudness recruitment is found in cases of pathology involving Corti's organ, and is not found in purely neural pathologies (Dix et al., 1948).

In recent years computer-averaging techniques have permitted the clinical study of human cochlear whole-nerve potentials recorded from the external canal or the promontory (Ruben et al., 1976). These electrocochleograms have demonstrated recruitment-like charactristics in the whole-nerve input-output functions of persons with audiometrically-determined loudness recruitment (Portmann and Aran, 1973; Eggermont et al., 1974). These investigators have identified a pattern of threshold shift combined with a high growth rate of the N_1 component of the whole-nerve action potential in the electrocochleograms of patients with audiometrically-determined loudness recruitment. Their findings suggest that the electrocochleogram may be an objective indicator of the presence of recruitment.

The experiments described here are intended to examine the relationship between the electrocochleogram N_1 growth function and loudness growth in nonhuman primates. More specifically, since a recruitment-like phenomenon has been observed in the electrocochleograms of patients with known loudness recruitment, it was desired to produce experimentally a reversible state of loudness recruitment and to study the recovery of baseline conditions. Experimentally induced loudness recruitment, following noise exposure, provides suitable conditions for study of the relationship between loudness and cochlear electrical events in greater detail than has previously been possible.

The development of a chronic implant for recording cochlear potentials in behaviorally trained primates has provided an experimental preparation suited to this study (Pugh et al., 1973). Furthermore, it has been shown (Moody, 1973; Pfingst et al., 1975) that changes in the loudness function of nonhuman primates may be monitored by measurement of response latencies in an auditory detection task. Moody (1970), in experiments with nonhuman primates, demonstrated a close relationship between the growth of response latency and expected growth of loudness (determined from similar experiments with humans). Using this technique, he has demonstrated what appears to the loudness recruitment during recovery from noise exposure. In addition it has been possible to produce equal-loudness curves for humans using reaction time techniques, and these agree quite well with equal-loudness curves obtained by the more traditional loudness-balance method (Pfingst et al., 1975). Such curves are also readily obtainable from nonhuman primates.

This investigation represents a new approach in that behavioral, electrophysiological, and histological data are obtained from the same animal in the course of studying an experimentally produced state of loudness recruitment.

The general approach was as follows: The baseline loudness functions of ma-

caque monkeys were assessed through the reaction time technique. These reaction time data were then compared to the whole-nerve action potential (AP) data recorded from chronic inner ear electrode implants. Loudness recruitment was produced by a controlled exposure to band-limited noise. Immediately following, and at various intervals after the noise exposure, the electrophysiological data and the behavioral index of loudness (reaction time) were recorded and compared as the temporary loudness shift recovered.

Answers were sought to the following questions:

(1) Do loudness recruitment and "recruitment" of the whole-nerve AP after noise exposure follow the same time-course of recovery?

(2) Is there a consistent relationship between loudness growth and growth of the whole-nerve AP during this recovery?

A second purpose of this study was to shed light on the physiological mechanism of loudness recruitment. To this end, a second phase of experimentation followed that dealing with reversible loudness shifts. The same animals were exposed to a very high-intensity band-limited noise of much longer duration, and a permanent hearing loss with loudness recruitment was produced. Electrophysiological and behavioral data were collected and compared at this end-point, and histological studies were then carried out on the animals' inner ears for the purpose of anatomic correlation.

Methods

Subjects

Four male pigtail monkeys (*Macaca nemestrina*), weighing between 4 and 7 kg, were used as subjects in this study. All monkeys had experience in threshold testing and were found to have normal hearing at the outset of the experiment. The animals were individually housed and were given free access to water. A restricted food ration was given at the conclusion of each daily testing session.

Apparatus

Monkeys received all behavioral training while restrained in specially designed portable chairs incorporating a perch with waist and neck restraining plates (Moody et al., 1970). When placed in the soundproof testing booth, a plexiglass head restraint was attached to the chair, and the monkey's muzzle was thus maintained in position between upright metal rods in a constant relationship to the delivery chute of an automated feeder. Calibrated PDR-600 earphones were attached tn the top of the chair, with a reasonably tight fit of the earphone cushions to the subject's external ears. The delivery tube and chute of the feeder were attached to the top of the chair also, with the tip of the chute positioned against the monkey's lips. A yellow stimulus lamp was located at eye level on the plexiglass housing for the chute. A plexiglass tube guide for the monkey's arm, containing the telegraph-type response key, was attached to the front edge of the chair's waist plate. This tube ensured a constant relationship of the animal's arm and wrist to the key, an important factor in obtaining consistent reaction times.

A system of digital logic modules and a PDP-8 computer were employed to automate the behavioral procedure. An 8-kHz pure tone stimulus, ranging in 10-dB steps from well below the monkey's hearing threshold to 80 dB SPL, was preprogrammed into the PDP-8 computer which directly controlled the behavioral testing. The audio signal produced by a Hewlett-Packard 200 CDR oscillator

was fed into a tone switch which could be triggered by a signal from the digital logic. The resultant tone bursts had a rise time of 2 ms. The computer, via a programmable attenuator between tone switch and earphone selector, presented stimuli from the programmed intensity list in a random sequence. In all subjects, only the right ear was routinely tested, since this was the ear selected for electrode implantation.

All electrical recording was carried out under light ketamine and Valium anesthesia to decrease electromyographic activity, thus facilitating identification of cochlear AP thresholds. This recording was done in a double-walled sound-treated room.

For the electrical recordings, the output of a Wavetek model 115 oscillator was fed through a tone switch which produced tone bursts of the desired rise and fall times and duration. Tone bursts of 8-kHz frequency, 2-ms rise and fall time, and 20-ms duration were used throughout the experiments. An attenuator was manually controlled and allowed presentation of various intensities to the animal. The intensities used varied in 10 dB steps over a range from 10 dB to 80 dB SPL, identical to that used in behavioral testing. Tone bursts were delivered to the animal through the same calibrated, closely fitted PDR-600 earphone used in behavioral testing.

An alligator clip connection was made to the exposed end of the electrode implant wire on the top of the monkey's head, and an indifferent needle electrode was placed in the neck muscle. Preamplification within the room was carried out with a Grass P-15 preamplifier set at a gain of 100 with bandpass of 3 Hz to 10 kHz. Its output was fed into a Tektronix 122 preamplifier with identical gain and similar bandwidth settings. Thus, the total gain prior to the computer was 10,000.

Stimulus waveforms and responses recorded from the monkey's electrode could be monitored simultaneously on a Tektronix 565 oscilloscope outside the test room. A PDP-12 computer provided on-line signal averaging of responses, and the program permitted plotting of the computer-averaged responses on a CalComp model 565 digital incremental plotter.

Noise exposures were carried out in a modified IAC double-walled sound-treated room with Masonite panels covering its inside walls. The panels and 1-in by 1-in wooden struts applied at various angles to them served to increase the homogeneity of the sound field. The monkeys were awake and seated in their restraining chairs in the center of the exposure room.

White noise produced by a General Radio model 1382 Random Noise Generator was filtered by an Allison 2BR filter and amplified by a MacIntosh MC2105 stereo amplifier. Two Altec-Lansing "Voice of the Theatre" speaker systems were used as transducers within the room.

Procedure

In the reaction time procedure, a trial was initiated by the monkey's depressing a response key. After a variable hold period of from 1–4 s, which was randomized from trial to trial, a tone was presented through the earphone. In order to receive a reinforcer in the form of a 190-mg banana-flavored food pellet (P. J. Noyes Co.), the monkey was required to release the key within 1 s after tone onset. Digital circuitry incorporating a precision timing device recorded the elapsed time in milliseconds between the onset of the tone and key release. This interval defined the monkey's reaction time. The maximum reaction time which could be recorded was 2,047 ms; consequently, failure to respond during a trial was simply recorded as this value of reaction time. No consequences were programmed for the monkey's failure to respond to a tone, regardless of its intensity. After any key release, there was a 5-s intertrial interval, during which the stimulus lamp was out and the animal was required to withhold key presses. Catch trials could be programmed by disconnecting the earphone from the system and discontinuing the availability of reinforcement for key release.

Reaction time for each trial was stored in computer memory according to the stimulus intensity value for that trial. Because a typical 1-h session might produce as many as 350 reaction time values, the computer was programmed to perform data compilation and compression. In a typical printout, median reaction time and interquartile range for each intensity value during that session were given. In addition, percent responses to catch trials and histograms of reaction time distributions could be obtained when desired. A discussion of the effect of various experimental parameters on reaction time values and distributions, using this procedure, is given by Moody (1970; see also Moody et al., 1976).

Responses to catch trials were typically less than 10% for all monkeys in this study. After changing over to the reaction time procedure approximately one month of daily sessions was required for stabilization of each monkey's reaction time functions. The criterion for stability was agreement within 10 ms at each of the three highest intensity levels, on a day-to-day basis (at the lower intensities, variability was greater). Upon reaching this stage, where reaction time functions from day to day were essentially the same, the monkey was considered ready to receive an electrode implant.

Chronic recordings of cochlear electrical activity were made using a wire electrode implanted into the bony horizontal semicircular canal of the monkeys. The surgical implantation technique has been described in detail in an earlier paper (Pugh et al., 1973). Anesthesia for the procedure was carried out with i.m. injections of ketamine hydrochloride, 20 mg/kg, and Valium (Roche), 0.75 mg/kg. Atropine, in a total dosage of 0.5 mg, was also given initially to suppress hypersalivation. Through a postauricular incision, the horizontal canal was exposed by drilling through mastoid air cells. A small opening was drilled into the bony canal and into this was inserted the bared tip of a 30-gauge teflon-coated, platinum-iridium wire. The wire was sealed in place with carboxylate dental cement, and its free end was passed subcutaneously to an acrylic plug on the head. Postoperative horizontal nystagmus and some ataxia were transient and all monkeys were able to resume behavioral testing within ten days after surgery.

Baseline AP response data were collected over the identical range of stimulus intensities as were used in the behavioral reaction time testing. In all experiments, electrophysiological data were obtained within 1 h after the behavioral data with which they were compared.

Maximum amplitudes of cochlear whole-nerve action potentials obtained varied from monkey to monkey, although these generally fell within the range of 40 to 70 μV. It was found that averages of 100 responses were sufficient at the higher intensity levels, while averages of 400 responses were required in the vicinity of AP threshold, in order to improve the signal-to-noise ratio and facilitate detection of the averaged waveform. Equal numbers of tone-bursts of opposite polarity were used in signal averaging, in an attempt to minimize the cochlear microphonic contribution to the averaged response. The amplitude of N_1 was measured from the baseline to the peak voltage.

Production of Threshold Shift by Noise Exposure

Following the baseline behavioral reaction time and AP amplitude determinations, each monkey was exposed for 1 h to an octave band of noise centered at 8 kHz at a level of 108 dB SPL.

The 1-h noise exposure at 108 dB SPL has been found to produce a temporary threshold shift in pigtail monkeys which typically recovered completely within 48–72 h. In several of the monkeys, at a later stage of experimentation, a permanent threshold shift was produced. For this purpose, an exposure octave band of noise centered at 8 kHz was again used, but an intensity of 118 dB and a duration of exposure of 160 h (8 h daily for 20 days) were required to produce significant permanent changes in threshold.

Post-Exposure Testing

Immediately following the 1-h noise exposure, each monkey was tested in the behavioral reaction time procedure and then electrophysiological (AP) data were recorded. Recording and comparison of the two measures were also carried out at post-exposure intervals of 12, 24, 48, and 84 h. In some animals, it was found to be advantageous to delete the 12-h testing session, since this allowed more complete recovery from the ketamine anesthetic employed in the previous session.

Permanent Threshold Shifts

Several of the monkeys, following complete recovery from the noise-induced TTS, were subjected to the 20-day noise exposure described above. During the course of this long-term exposure, behavioral reaction time testing was performed daily as an index of the degree of threshold shift and loudness recruitment present. Daily testing was carried out for an additional 2 weeks following the last day of noise exposure to ensure that no further recovery in threshold was to be expected. After these 2 weeks, cochlear AP data were obtained and compared to the behavioral data of the same day.

Each monkey was then killed and the membranous labyrinth was stained in situ by perilymphatic perfusion of 1% osmium tetroxide solution (Zetterqvist). Whole mounts (surface preparations) of the organ of Corti and basilar membrane were examined by phase contrast microscopy, and the percentage of hair cells remaining in each millimeter of the 25—29 mm length of the membrane was determined from a complete count. In counting, a decision was made whether each cell was present or absent, i.e., replaced by a phalangeal scar. No attempt was made to assess lesser degrees of damage which might be thought to signify a decreased capacity to respond to sound stimuli. Details of the histological procedures may be found in Hawkins and Johnsson (1976).

Results

Recruitment During Temporary Threshold Shift (TTS)

In the baseline condition, prior to the high-intensity noise exposure, each subject's median reaction time with corresponding interquartile range was plotted as a function of the stimulus sound pressure. Since cochlear N_1 amplitude increases with increasing stimulus sound pressure, the reciprocal of the averaged N_1 amplitude for each stimulus sound pressure level was plotted, providing a curve that followed very closely the behavior of the latency-intensity (LI) function which decreases with increasing SPL. The corresponding scale, which appears on the right ordinate in each of the figures that follow, was adjusted to the baseline condition such that there was a reasonable fit with the LI function, and the points at the 80 dB SPL approximately coincided. To make this adjustment, the reciprocal N_1 amplitude scale can be moved upward or downward and expanded or contracted. This scaling was established for each animal at the outset of the experiment and was maintained throughout. It was thus possible to examine in a single animal the LI function and the reciprocal N_1 input-output function following the exposure to high-intensity noise.

The baseline data of monkey A-4 are shown in Figure 1. The behavioral threshold is about 15 dB SPL, the sound pressure region where latency is increasing at a rapid rate. It can be seen also that behavioral variability, and hence the interquartile range of the response latencies, increases markedly as threshold is approached. This figure demonstrates that the N_1 amplitude is also rapidly decreasing at about 15 dB SPL. This finding is interpreted as showing good agreement for the two functions. The shape and slope of the functions with increasing sound pressure is also similar.

Following a 1-h exposure to a 108-dB octave band of noise, centered at 8 kHz, median reaction times and corresponding cochlear N_1 amplitudes were compared in
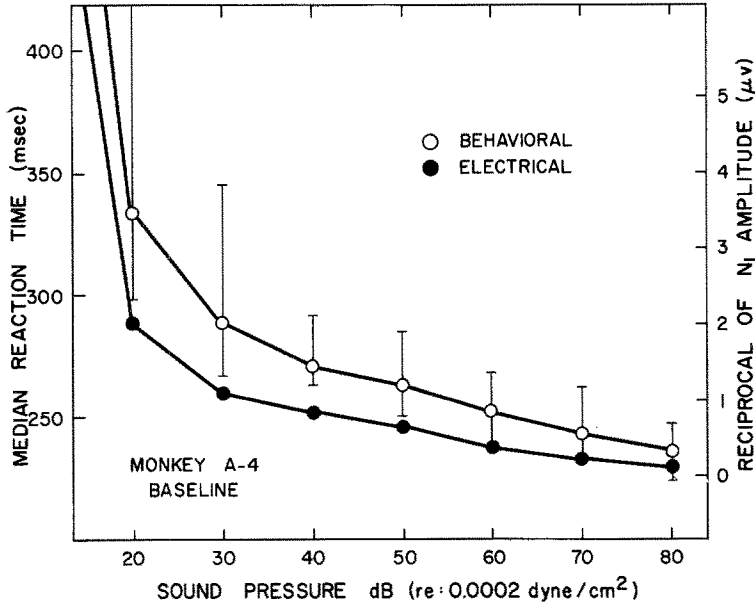
Fig. 1. Baseline data of monkey A-4. Median reaction times are shown as open symbols, with corresponding interquartile ranges indicated by brackets. The reciprocal of the averaged N_1 amplitude for corresponding sound pressure levels is plotted with filled symbols
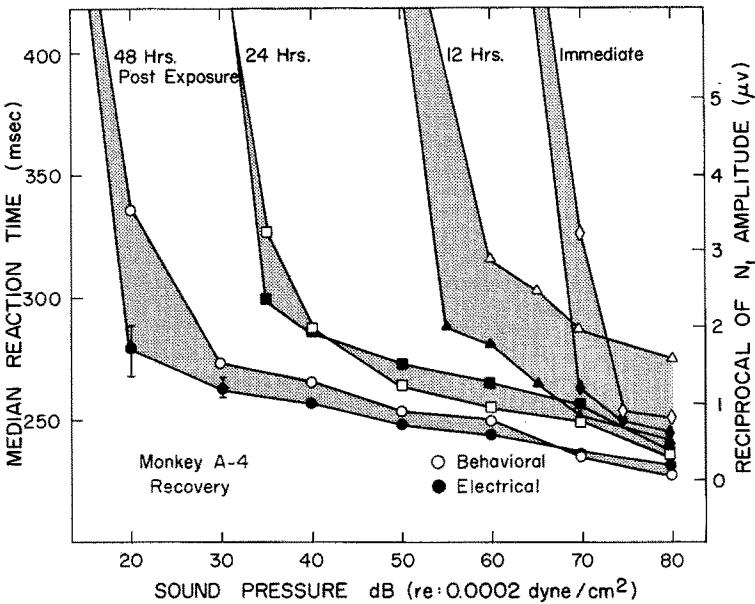

Fig. 2. Post-exposure data for monkey A-4. Elapsed time following noise exposure is indicated next to each curve. The space between corresponding behavioral and electrical curves is stippled

a similar graphic function at various time intervals (see Fig. 2). Stippling is added to facilitate visual comparison of corresponding behavioral and electrical curves. Immediately following the noise exposure, a 50-dB threshold shift can be identified in both functions. In addition, at higher stimulus levels, the post-exposure curves approach the curves obtained during the baseline condition and do so within a significantly smaller intensity range, clearly suggesting the presence of recruitment.

At 12 h following exposure, there is still good agreement with regard to thresholds for the two functions. Unusually long reaction times were encountered at this testing session, however, and it was felt that the monkey was a bit sluggish as a result of the ketamine sedation during the previous testing session. This sluggishness could account for the divergence of the behavioral and electrical curves above threshold. At 24 and 48 h, excellent agreement in both threshold and overall shape is seen in both curves.

Baseline and post-exposure data for monkey A-9 are shown in Figures 3 and 4. The LI functions and N_1 amplitude reciprocal curves demonstrate good agreement in threshold and shape at all testing intervals. It should be pointed out that immediately following the noise exposure, in this animal as well as the others, N_1 threshold was approximately 5 dB lower than behavioral threshold. This discrepancy was probably due to the fact that, according to the experimental protocol, electrical testing always followed behavioral testing at each time interval. Thus, approximately $\frac{1}{2}$ h of recovery from TTS (during the period of most rapid recovery) occurred between the acquisition of behavioral and electrical data.
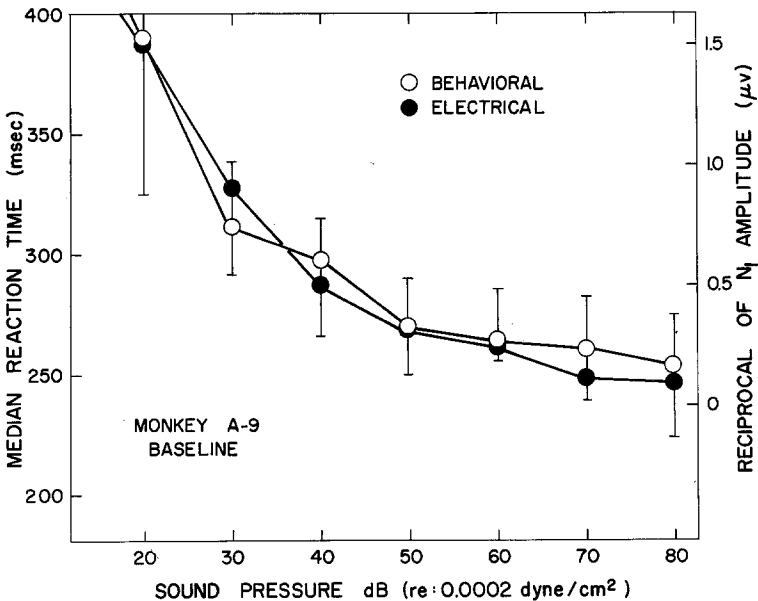

Fig. 3. Baseline data of monkey A-9. Median reaction times are shown as open symbols, with corresponding interquartile ranges indicated by brackets. The reciprocal of the averaged N_1 amplitude for corresponding sound pressure levels is plotted with filled symbols

Figures 5 and 6 present the baseline and recovery data of monkey A-10. Good agreement of behavioral and electrical thresholds is seen, with fair correspondence of curve shape.

Finally, the data of monkey A-12 are shown in Figures 7 and 8. This animal's N_1 threshold was 5 dB lower than his behavioral threshold. This relation holds true, as expected, immediately following noise exposure, but thresholds for the two measures agree quite well at later recovery intervals. As was the case for the other subjects, no drastic changes in the relative shapes of behavioral and electrical curves are observed at any time following recovery from TTS.

In summary, the data of these four animals suggest that, during recovery from a noise-induced TTS, there is excellent agreement between cochlear N_1 threshold and behavioral threshold, and the basic relationship between the N_1 input-output function and the LI function is maintained. In other words, the degree of recruitment determined by one measure is also determined by the other.

Recruitment in Permanent Threshold Shift (PTS)

Following the TTS studies, two of the monkeys were subjected to 160 h of 8-kHz octave band noise at 118 dB SPL, resulting in a permanent shift in their thresholds at the test frequency of 8 kHz.
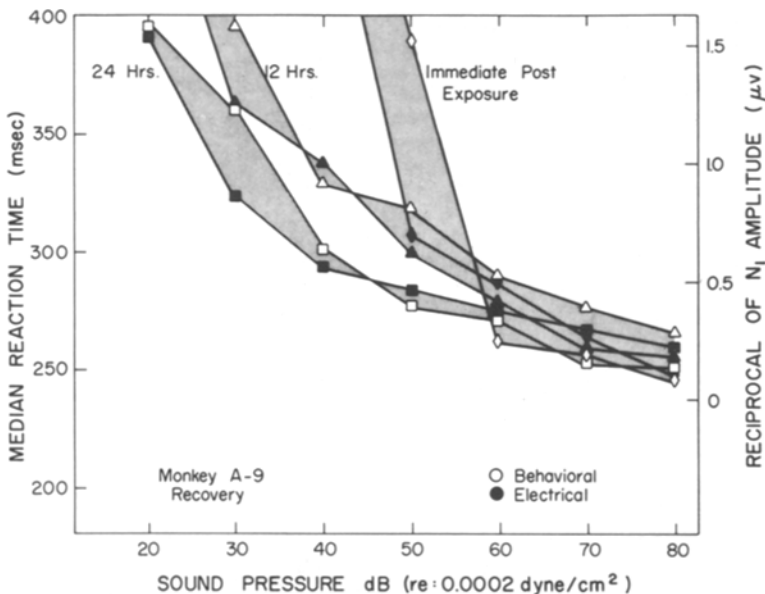

Fig. 4. Post-exposure data for monkey A-9. Elapsed time following noise exposure is indicated next to each curve. The space between corresponding behavioral and electrical curves is stippled
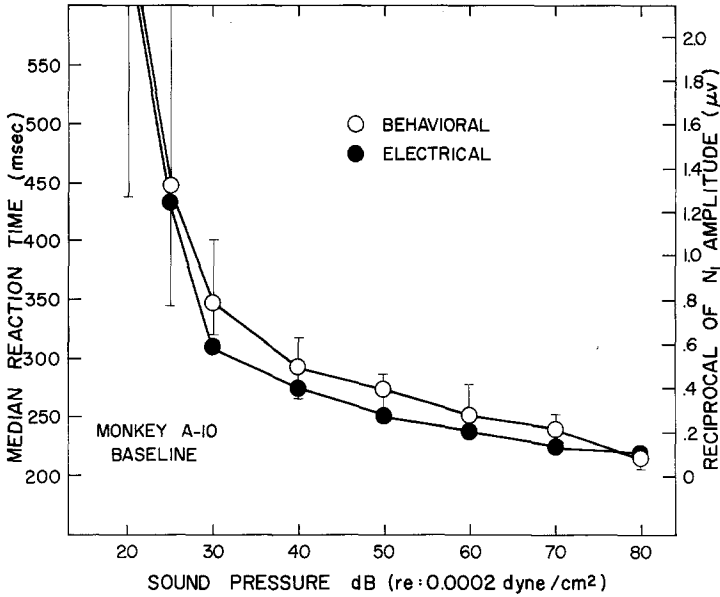
Fig. 5. Baseline data of monkey A-10. Median reaction times are shown as open symbols, with corresponding interquartile ranges indicated by brackets. The reciprocal of the averaged N_1 amplitude for corresponding sound pressure levels is plotted with filled symbols
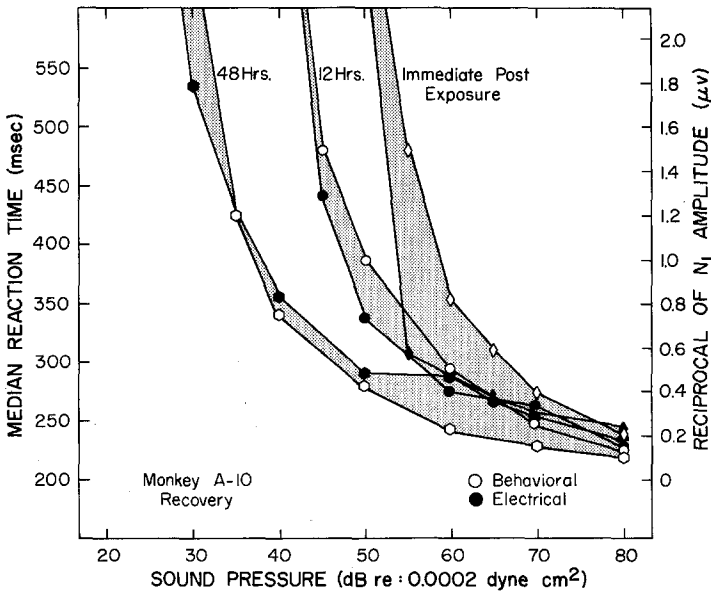

Fig. 6. Post-exposure data for monkey A-10. Elapsed time following noise exposure is indicated next to each curve. The space between corresponding behavioral and electrical curves is stippled
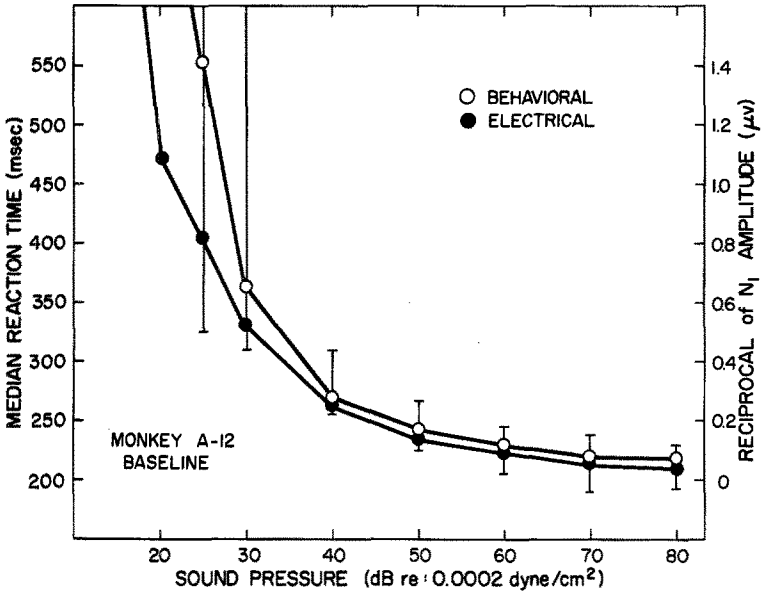
Fig. 7. Baseline data of monkey A-12. Median reaction times are shown as open symbols, with corresponding interquartile ranges indicated by brackets. The reciprocal of the averaged N_1 amplitude for corresponding sound pressure levels is plotted with filled symbols
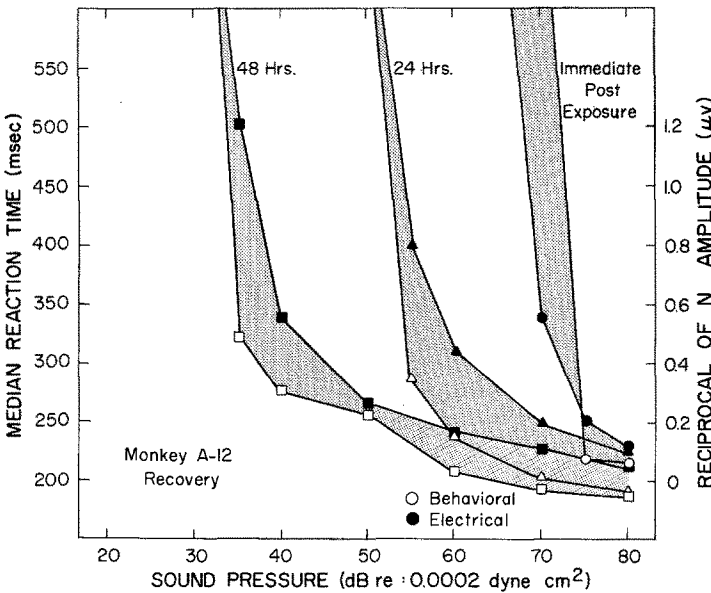

Fig. 8. Post-exposure data for monkey A-12. Elapsed time following noise exposure is indicated next to each curve. The space between corresponding behavioral and electrical curved is stippled

Monkey A-12 sustained a permanent threshold shift with recruitment evident in his 8-kHz LI function, as shown in Figure 9. Electrical and behavioral data from the animal's right ear were compared at the pre-exposure and the 10-day post-exposure intervals, and excellent agreement was observed between them.

Figure 10 illustrates the distribution of inner and outer hair cell loss observed in this cochlea. In the lower part of the figure, the behavioral threshold hearing loss is shown for comparison. A slightly different testing procedure was used to obtain these data (Moody et al., 1976). Outer hair cell loss of uncertain etiology and significance is seen at the very apical end of the cochlea. Of more importance, however, is the outer hair cell loss, unsystematic with respect to row, that begins fairly abruptly at 8 mm from the round window. There is a progressive loss of all three rows of outer hair cells in a basalward direction for approximately 1 mm. At this point, the degree of outer hair cell loss stabilizes and does not progress for several millimeters. However, at 5 mm from the round window, there is a further progression of outer hair cell loss in conjunction with an abrupt and progressive loss of inner hair cells. At a point 3 mm from the round window, there are essentially no hair cells remaining.

The basal distribution of hair cell destruction correlates well with the pattern of hearing loss observed in monkey A-12. The losses are confined to the higher frequencies of 4 kHz (10 dB), 8 kHz (17 dB) and 16 kHz (30 dB).
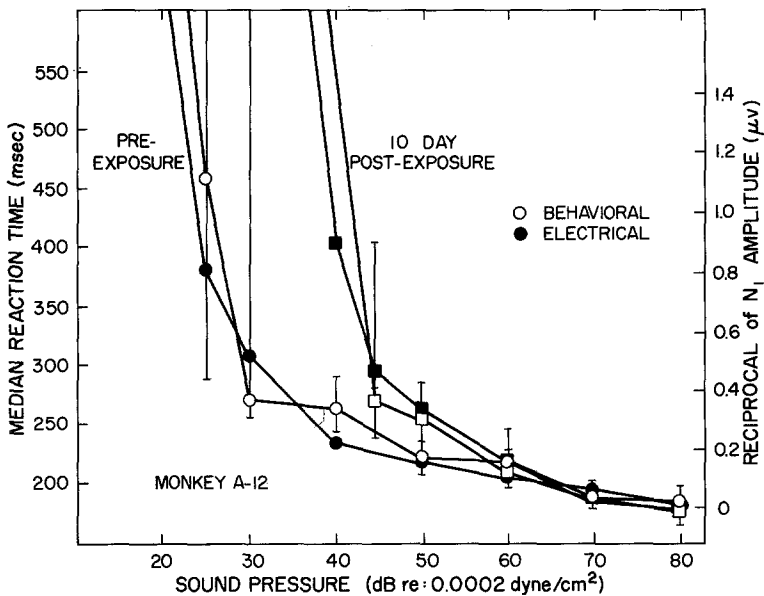

Fig. 9. A comparison of reaction time and N_1 amplitude data of monkey A-12 at a test frequency of 8-kHz, prior to noise exposure and at 10 days following the exposure. Median reaction times are shown as open symbols with corresponding interquartile ranges in brackets. Reciprocal of N_1 amplitudes are plotted as closed symbols
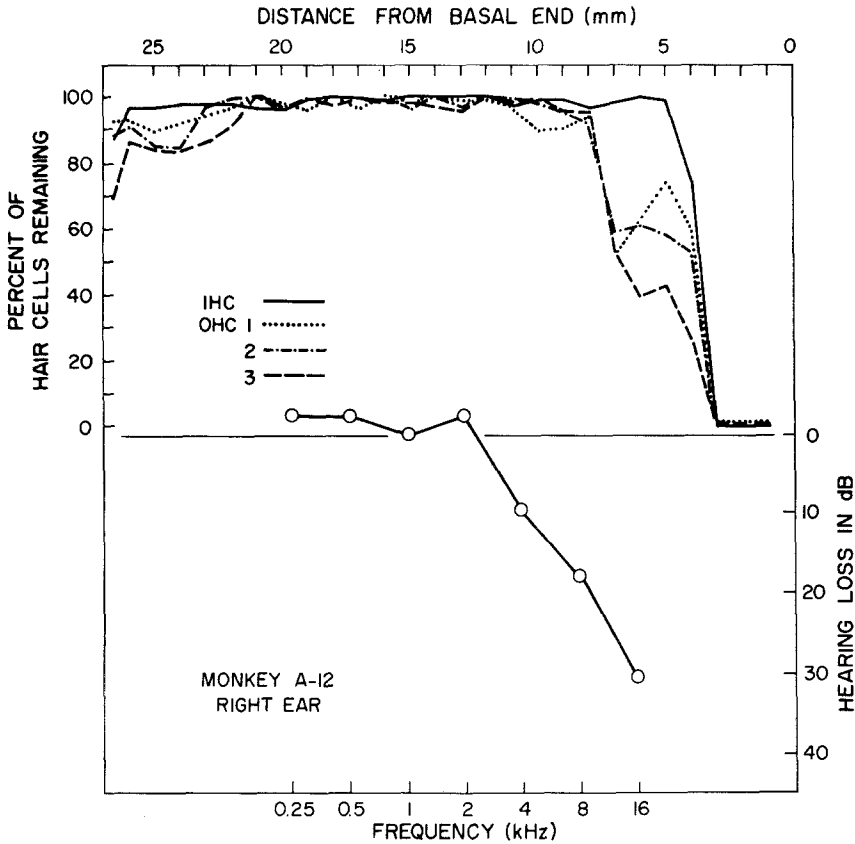
Fig. 10. Distribtution of cochlear inner hair cell (IHC) and outer hair cell (OHC) loss, by row, in monkey A-12. The behavioral hearing loss attributable to the noise exposure is plotted below for comparison

The second monkey exposed to this condition (A-9) suffered a similar, although slightly more severe hearing loss, with complete recruitment. Since he dislodged his implant during the exposure, his electrical data are unavailable.

Discussion

In four monkeys subjected to TTS-producing noise, a close relationship has been observed between the cochlear N_1 input-output function and the animal's reaction time-intensity function. To the extent that reaction time can be used to derive loudness matching data, a relationship has been shown to exist between the N_1 function and the loudness function. As mentioned in the introductory section, Portmann and Aran (1973) observed "recruitment" in the electrocochleograms (N_1 input-output functions) of patients with audiologically-documented loudness recruitment. Furthermore, Eggermont et al. (1974) have demonstrated a close relationship between

patients' results on Alternate Binaural Loudness Balance (ABLB) tests and their N_1 functions at electrocochleography. Finally, Pfingst et al. (1975) have demonstrated excellent agreement between reaction time data and ABLB data from patients with loudness recruitment.

The results of the present study provide a closure between the reaction time data and the electrophysiological data, in effect completing the circle. Eggermont et al. (1974) have suggested that the electrocochleogram may provide an objective index of loudness recruitment, and these findings would support his suggestion.

The results of these experiments indicate an identical time-course of recovery from an experimentally produced "recruitment" condition for both cochlear N_1 growth and response reaction time. Essentially, these results confirm the hypothesis that loudness recruitment is a phenomenon of purely cochlear origin.

An account of the search for an electrophysiological correlate of loudness was given by Stevens (1970) who noted that the growth of cochlear whole-nerve potential amplitude resembles that of the loudness function. He was quick to point out, however, that the growth of N_1 with intensity is not smooth, and different populations of neurons appear to make a contribution to the potential at different intensity levels. While N_1 amplitude growth may be an imperfect correlate of loudness growth, the findings of this study are in accord with the notion that loudness, in the case of a single pure tone stimulus, may be related to the total driven activity in the auditory nerve.

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