Microwave hearing: The response of single auditory neurons in the cat to pulsed microwave radiation

Robert M. Lebovitz and Ronald L. Seaman

Department of Physiology, University of Texas Health Science Center at Dallas, Dallas, Texas 75235

Inferences regarding the mechanism of "microwave hearing" heretofore have been based upon theoretical studies, reports of human observers, animal behavioral experiments, and the recording of auditory field potentials. To gain more insight into the phenomenon we have examined the effect of pulsed microwave radiation (MWR) at 915 MHz on single auditory neurons in the cat. The responses to pulsed acoustic stimuli ("clicks") and to pulsed MWR were compared by means of post-stimulus time histograms. Although the response to MWR was dependent upon parameters of the pulse of incident MWR, it was independent of the averaged rate of energy absorption. Threshold effects were observed at an energy dose as low as 4 μJ/g per pulse. Auditory units with characteristic frequencies (CFs) below 1.2 kHz appeared to be more responsive to pulsed MWR than were units with higher CFs. Many neurons demonstrated a response to pulsed MWR that was similar to the response to acoustic clicks, which may provide a means by which to define the acoustic equivalent of MWR. Our results support the view that microwave hearing is mediated, at least in part, by an electromechanical interaction that is initiated distal to the cochlea.

1. INTRODUCTION

The most frequently studied effects of radio frequency energy, including microwave radiation (MWR), on biologic systems have been those related to its action as a source of volume heating [*Johnson and Guy,* 1972; *McLees and Finch,* 1973; *Michaelson et al.,* 1975]. For the typical laboratory animal, as well as for man, the gross thermal load depends only on the averaged rate of energy absorption, i.e., production of heat. Thus, it has become common practice to delineate environmental effects or allowable personnel exposure levels in terms of the averaged power density of incident energy and of the exposure duration [cf., *Schwan and Li,* 1956; *Michaelson,* 1971]. For mass-normalized rates absorption of MWR, the terms specific absorption rate (SAR) are now utilized [*Johnson,* 1975]. However, it has been suggested that the biologic and behavioral responses to pulsed MWR may differ from those associated with exposure to continuous-wave MWR at an equivalent SAR [see the review by *Frey,* 1971]. Potential implications include the possibility that nonthermal modes of interaction may be neurophysiologically significant and/or that mechanisms may exist to amplify the response to weak or to localized thermal sequelae of radiation [*Czerski,* 1974; *Frey and Feld,* 1975; *Servantie et al.,* 1975]. For example, in neurophysiological studies of the effects of 147-MHz radiation, intrinsic brain rhythms of the cat were found to depend upon the modulation frequency of the radiation and to be independent of gross thermal effects [*Bawin et al.,* 1973]. Related effects of amplitude modulated RF radiation on divalent cation (Ca^{++} and Mg^{++}) mobility in the CNS have also been reported [*Bawin et al.,* 1975].

The observation that pulse modulated (PM) MWR yielded a dose-dependent response in the auditory system of human [*Frey,* 1962; *Frey and Messenger,* 1973] and of animal subjects [*Frey,* 1967; *Taylor and Ashleman,* 1974; *Guy et al.,* 1975] indicates the need to examine more closely the neurophysiological significance of microwave hearing. We report here initial studies of the response of single auditory neurons to PM-MWR. In order to carry out these experiments it was necessary to be able to record from single units for extended periods of time, coincident with MWR exposure and free of significant artifact. Once successful procedures had been developed, it became apparent that the single-unit response is determined by the parameters of the MWR pulse and that it is proper to consider the conditional probability of a unitary response to a single MWR pulse. Thus, the SAR is not particularly relevant to the phenomenon of the RF hearing.

We have observed modulation of single-unit discharges from doses as low as 4 μJ/g per pulse, confirming an acute sensitivity of the auditory system to PM-MWR. A major finding is that the response of single auditory units to acoustic clicks and to PM-MWR stimuli are sufficiently similar to indicate that there is a common intermediate step in the conversion of some peripheral event to a neural signal. These data will be discussed in terms of the competing hypotheses about the origin of microwave hearing.

2. METHODS

2.1. *Surgical.*

Adult domestic cats, all mongrels, were utilized. Surgical procedures were carried out under general anesthesia with sodium pentobarbital (35 mg/kg IP initial dose, with supplemental IV doses as required) or with a mixture of urethane (1 g/kg) and alpha-chloralose (40 mg/kg). The posterolateral aspect of the cerebellum was removed by aspiration so that the recording micropipette could be placed directly into the proximal portion of the eighth nerve. The field was then filled with a 3% agar-saline gel to reduce local pulsation;

subsequent movement of the microelectrode was accomplished via a hydraulically coupled stepping microdrive. The animal was loosely wrapped with a warming pad so that the rectal temperature could be maintained between 36 and 38 °C.

2.2. *Stimulus presentation and calibration.*

Acoustic clicks were applied via a pulse-driven condensor earphone that was coupled to one hollow ear bar of the stereotaxic apparatus by a short length of plastic tubing. The acoustic pulse width could be varied over the range of 25 to 200 μsec, although 70 μsec was the usual duration. The acoustic intensity of a train of clicks was determined separately by means of a hollow chamber that fitted over the microphone of a General Radio sound-level meter. The earphone assembly was inserted at the opposite end of the chamber and calibration curves were derived in terms of the sound intensity level relative to 0.002 dyne/cm^2 (dB SPL) versus click-driving voltage (to a maximum of 30 volts) for repetition rates to 10 clicks /sec and pulse widths of 25 to 200 μsec. During each experiment the contralateral ear was stoppled by the solid ear piece, but otherwise the ear remained intact. The broadband background of acoustic noise in the laboratory varied from 40 to 50 dB above SPL.

Pulse-modulated 915-MHz MWR was applied via a MCL Model 15022/6050 source that was connected to a waveguide applicator, which was located 2 to 3 cm from the dorsolateral aspect of a cat's head. The applicator's design was provided in a personal communication from A. W. Guy. The peak output power of the source was 70 watts and pulse durations of 25 to 300 μsec were found to be effective. Pulse repetition rates above 10 were not used. The averaged power density of energy incident on a cat never exceeded 1.0 mW/cm^2, as estimated from measurements utilizing a Narda field-density meter with an omnidirectional probe (Type 8315A). However, the primary measure of applied MWR was the mass-normalized rate of energy absorption, the specific absorption rate (SAR), which was determined as a function of net irradiated power from the applicator. The necessary data for calibration were obtained from separately run experiments by a caloric method similar to that of *McRee* [1974].

At the conclusion of selected experiments, each animal was euthanized by an overdose of pentobarbital. A low-mass thermistor was then inserted into the medulla, at the floor of the fourth ventricle. When the medullary temperature had fallen to about 30 °C a series of CW-MWR exposures at a given net forward power and a range of durations to 80 seconds was carried out. For each trial, the thermistor was removed just before the CW-MWR and was replaced immediately upon termination of radiation (< 0.5 seconds of delay). By projecting back from a series of cooling curves, the rate of incrementing of temperature was used to calculate the medullary SAR. A typical calibration run and a summary of SAR data for nine animals are shown in Figure 1. The energy absorbed per pulse was calculated from the CW (SAR) data and the known pulse parameters

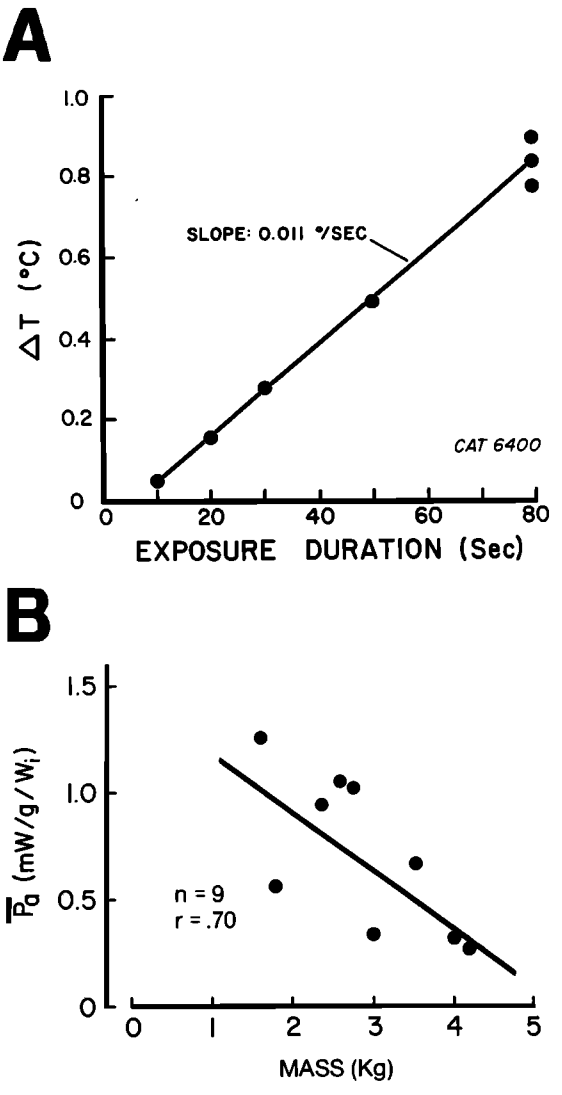

Fig. 1. A. Typical calibration data using CW-MWR. The rise in temperature at the midline of the medulla (see text) is shown as a function of exposure duration. Each data point represents the mean of at least three trials, except for the data on the 80-sec exposure, where each point represents a single trial. At shorter durations, the scatter was too small (less than 5%) to depict on this graph. From the slope of this relation (0.011 °C/sec) and given an effective radiated power (W_i) of 48.6 W, the normalized SAR was determined to be 0.94 mW/g/W_i. B. Midline medullary SAR for nine animals, as a function of body mass. The SAR is normalized with respect to effective irradiated power (W_i). A strong correlation ($P < 0.01$) between normalized SAR and body mass is shown, an expected result in view of the corresponding differences in head size of the experimental animals.

(peak power and duration). Under experimental conditions the averaged medullary SAR did not exceed 0.5 mW/g.

All of the experiments were carried out within an electrically shielded enclosure that was lined on three sides with microwave absorbent material (Eccosorb Type H-4). Further, the stereotaxic head holder was

fabricated from plastic to minimize interactions with the MWR field.

2.3. *Electrophysiological methods and data analysis.*

Extracellular unit activity was obtained via glass micropipettes filled with 1 or 2 M NaCl. The shielded probe assembly of a Grass P-16 microelectrode preamplifier was protected further by a low-pass input filter to reduce the transient overload that frequently resulted from the MWR pulse stimuli. Each amplified extracellularly recorded neural discharge ("spike") was converted to a standard 10-μsec logic pulse via a window discriminator. The logic signal provided all of the information necessary for data analysis in terms of the distribution of interspike intervals. However, the waveform of each detected unit discharge was monitored also on an oscilloscope to ensure reliable discrimination of single units.

Interval histograms and post-stimulus time histograms (PSTH) of the unitary discharge were derived on-line via Ortec Model 4621/4620 histogram and memory units. Given here in terms of counts-per-bin interval, each histogram is equivalent to a distribution of relative firing frequency; *viz.*, firing frequency = (counts per bin) $\times$ (bin width)$^{-1}$ $\times$ (trials per histogram)$^{-1}$.

3. RESULTS

3.1. *General characteristics of the MWR response.*

Stable, extracellularly recorded units were first evaluated for their response to a train of acoustic clicks by means of a PSTH of at least 500 trials at 10/sec. Similarly, a PSTH with respect to a train of MWR pulses (10/sec) was then obtained. If the unit proved to be responsive both to the acoustic stimulus and to MWR, we proceeded to make additional dose-response determinations as long as the unit showed a stable discharge. In this series of experiments, a total of 133 auditory units was studied; 63 units were responsive both to acoustic clicks and to pulsed MWR.

The latencies of the earliest MWR responses that were seen in these studies were 1.5 to 2 msec. The latency increased with decreasing intensity of the acoustic or MWR stimulus and, for, intensities near threshold, latencies as long as 5 msecs were observed. Because of equipment limitations, the maximum available energy dose was 40 μJ/g per pulse. By way of comparison, the minimal single-unit MWR-response threshold we observed was approximately 4 μJ/g. The range of effective MWR intensities was therefore much constricted compared with the 50 to 80 dB dynamic range of acoustic stimulation to which primary and secondary units are responsive [*Grinnell*, 1969]. For this reason it is likely that we often observed only small (less than 1 msec) variations in response latency with respect to intensity of MWR pulses. The failure of more than 50% of the auditory units that were studied to show a response to PM-MWR can be explained similarly by the limitation on the effective range of MWR doses. In other respects, the unitary responses to MWR pulses were not grossly different from those to typical acoustic stimuli.

When the PM-MWR was sufficiently intense to yield an evoked potential, the unitary response could be observed to coincide with it or to lag by several milliseconds, as is the general case with primary sensory evoked potentials. The evoked potentials to PM-MWR and to pulsed acoustic stimuli (clicks) were similar in form. However, the MWR response-latency was frequently as much as 0.75 msec shorter than that for acoustic clicks, corresponding in part to the acoustic transit time from earphone to the tympanic membrane. No change in MWR response latency with change in position of the microwave applicator could be discerned. Because the evoked potentials allowed only limited quantitative evaluation of the nature of the PM-MWR effect, they were not studied in detail. We focused our attention on the unitary discharge patterns and on a comparison of the PSTHs as derived from acoustic clicks with those from PM-MWR.

3.2. *MWR-PSTH.*

Although the MWR-PSTH of a unit was frequently multipeaked, the intensity of MWR was generally insufficient to yield multiple action potentials to a single pulse. Rather, the multipeak response occurred because of a systematic modulation of the post-stimulus firing probability (to be discussed in more detail below). The cumulative probability of a discharge increased as the MWR intensity (energy dose per pulse) increased (Figure 2). For pulse durations in the range of 25 to 300 μsec, it was the quantity of energy per pulse, not simply the pulse duration or peak-pulse intensity, that most strongly influenced the unitary response in most instances (Figure 2A, B). However, as illustrated in Figure 2, equivalent pulse energies did not always yield equivalent PSTHs in a given experiment.

In some units, the relation between the MWR intensity and the unitary response was qualitatively more complex. Figure 3, for example, shows a series of MWR-PSTHs from a unit whose response was a nonmonotonic function of pulse width as well as of pulse energy. In this unit, the maximal response occurred to MWR pulses of 150-μsec duration. No response was seen with MWR pulses greater than 250 μsec or less than 25 μsec in duration. Thus, although MWR pulse energy was a significant factor, the pulse form also influenced unitary response. In units of this type, as with all of the units tested, it was found that for any given MWR pulse width the magnitude of the response always decreased with decreasing energy dose.

The response of each unit to PM-MWR showed distributions of the post-stimulus firing intervals that were similar to its response to acoustic clicks (Figures 2 through 4). The PSTHs thus provided a measure by which to estimate the approximate acoustic equivalent of the MWR stimuli. For a typical MWR-sensitive unit, for example, we observed that a 10/sec train of 250 μsec pulses (corresponding to 32.9 μJ/g per pulse) yielded PSTHs comparable to that from a 10/sec

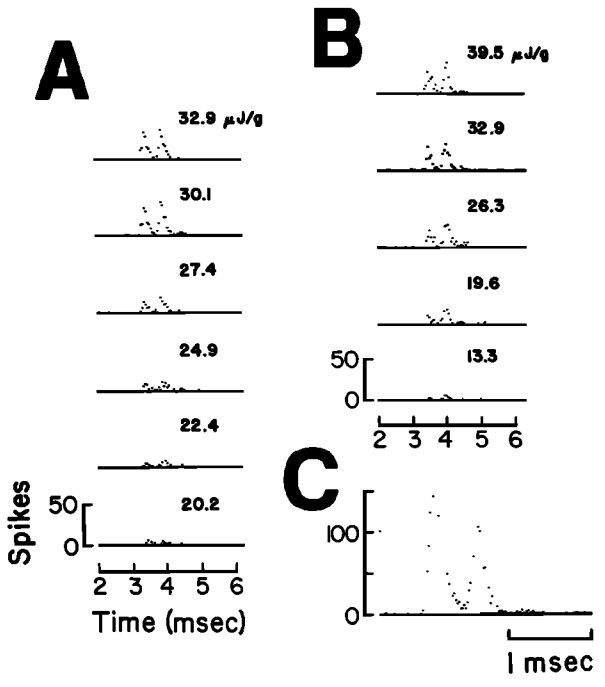

Fig. 2. PSTHs of an acoustic unit for which the MWR response was dependent primarily upon energy dose. A. Responses to MWR pulses of fixed duration (250 μsec) and decreasing intensity, as indicated by the energy dose (per pulse) adjacent to each PSTH. Each PSTH represents 2048 trials, at a bin width of 50 μsec. Time = 0 corresponds to the onset of the MWR pulse. B. Responses of the same unit to MWR pulses of fixed intensity and variable duration. Each PSTH again represents 2048 trials, at a bin width of 50 μsec. From top to bottom, the pulse widths were 300, 250, 200, 150, and 100 μsec, with the energy dose per pulse as indicated. C. The acoustic PSTH of the same unit shown above. Click rate was 10/sec, with a sound intensity level of 58.5 dB SPL. This PSTH represents 3072 trials at a bin width of 20 μsec.

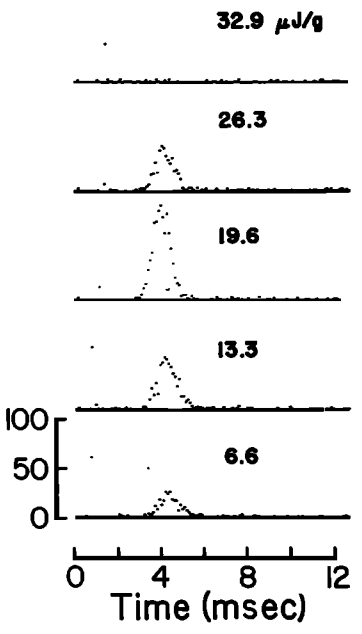

Fig. 3. MWR-PSTHs of a unit whose response was a nonmonotonic function of pulse width and energy dose. The MWR pulse widths were, from top to bottom, 250, 200, 150, 100, and 50 μsec at the indicated energy dose per pulse. Each PSTH represents 1024 trials at 10/sec, with a bin width of 100 μsec.

acoustic click train at 60 to 70 dB above SPL. However, such comparisons on the basis of equal acoustic and MWR-pulse durations did not yield the most satisfactory representation of unitary acoustic responsiveness. It became apparent that an accurate specification of the MWR sensitivity of each auditory unit required consideration of each unit's acoustic frequency response and absolute acoustic threshold. Since these data were not always available, a full analysis was not possible. Experiments currently in progress will provide additional data.

3.3. *Response to MWR relative to acoustic frequency response.*

The characteristic frequency of an auditory unit is defined as the frequency at which its response threshold is lowest. For primary (fibers of the auditory nerve) and most second-order auditory units (cell bodies of the cochlear nuclei or their projecting axons), the characteristic frequency (CF) is determined by the mechanical properties of that portion of the basilar membrane to which that cell is most directly related. Hence, when the PSTH in response to an acoustic click shows multiple peaks, the interval between these peaks will approximate the period of oscillation of the basilar membrane, and thereby provide an estimate of the characteristic frequency of that unit [*Kiang*, 1965]. Similarly, because of the mechanical properties of the auditory periphery, the unitary response latency is inversely related to CF.

The interval between the first two peaks of the PSTH will be called $\triangle P$. This interval provided an estimate of a unit's frequency response, given by $CF = (\triangle P)^{-1}$. The limit of resolution was 200 μsec, placing an upper limit of 4 to 5 kHz on the *CF* that could be estimated by this method. If the PM-MWR acted via some mechanical interaction with the basilar membrane or with structures distal to it, we would then expect the mechanical properties that determined each unit's acoustic response to shape its MWR response, *viz.*, the latency and periodicity of the corresponding PSTHs. If hair cells or auditory nerve fibers were activated directly, no such uniform correspondence would need hold.

When the latencies of the acoustic and MWR responses were compared, a distinct though weak correlation ($r = 0.57$) could be seen (Figure 5). Further, for both the acoustic and MWR responses, the estimated CF was a decreasing function of the observed response latency (Figure 6). Each of these results indicates that mechanical properties of the basilar membrane are indeed basic to the form of the MWR -- as well as the acoustic -- unitary responses. However, both acoustic and MWR response latencies would be dependent also

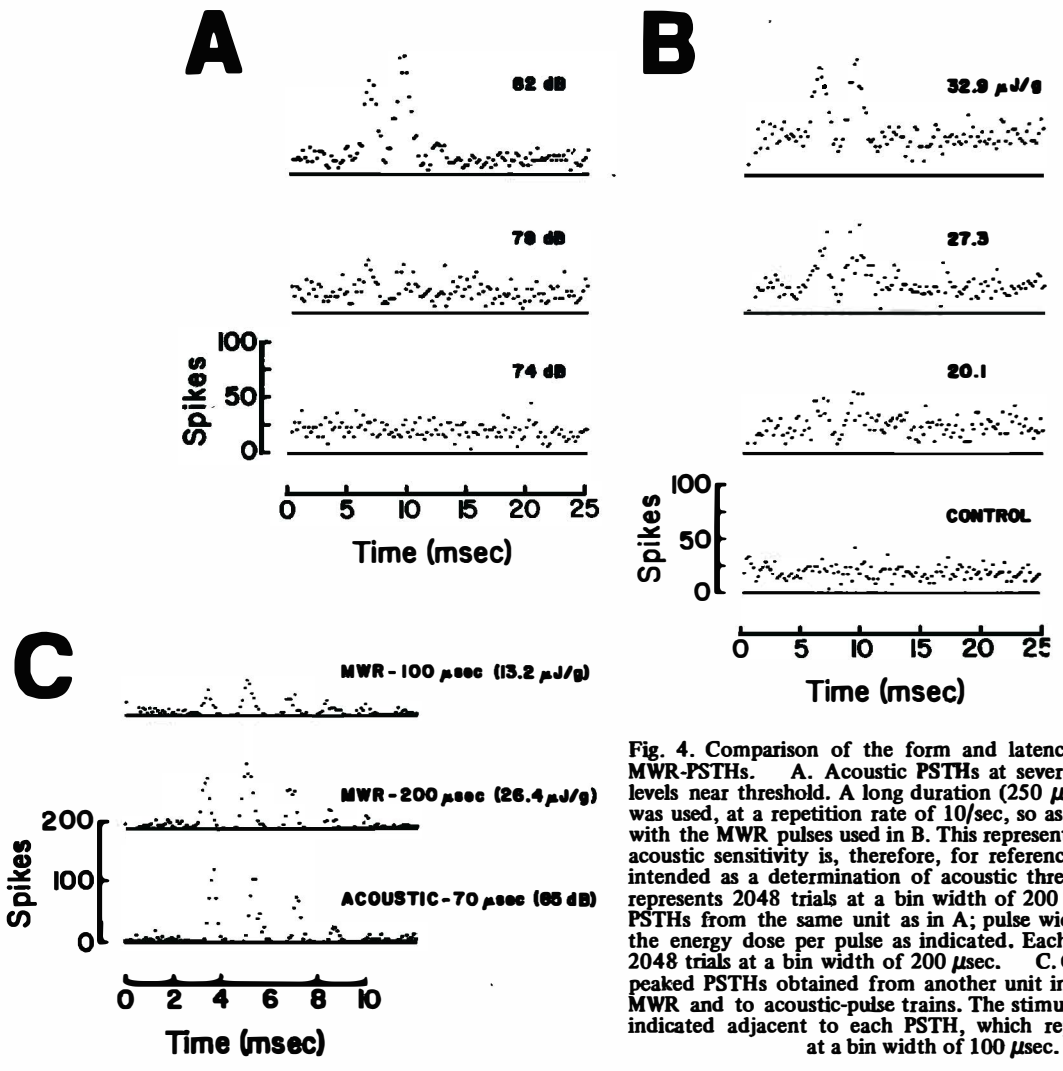

Fig. 4. Comparison of the form and latency of acoustic and MWR-PSTHs. A. Acoustic PSTHs at several sound intensity levels near threshold. A long duration (250 μsec) acoustic click was used, at a repetition rate of 10/sec, so as to be comparable with the MWR pulses used in B. This representation of the unit's acoustic sensitivity is, therefore, for reference only and is not intended as a determination of acoustic threshold. Each PSTH represents 2048 trials at a bin width of 200 μsec. B. MWR-PSTHs from the same unit as in A; pulse width 250 μsec with the energy dose per pulse as indicated. Each PSTH represents 2048 trials at a bin width of 200 μsec. C. Comparable multi-peaked PSTHs obtained from another unit in response both to MWR and to acoustic-pulse trains. The stimulus parameters are indicated adjacent to each PSTH, which represent 512 trails at a bin width of 100 μsec.

upon the stimulus intensity relative to the unit's threshold of response. Thus, it is most significant that when intensity was removed as a parameter by directly comparing the acoustic ΔP with the MWR ΔP, a high degree of correlation ($r = 0.99$) was found (Figure 7). These data clearly indicate that mechanical factors within the cochlea are similarly involved in determining both the acoustic and the MWR responses of a given unit.

For 40 of the 63 units that were responsive to PM-MWR as well as to acoustic clicks, the acoustic frequency response could be estimated reliably from the acoustic PSTH. The results indicated that the responsivity to PM-MWR decreased with increasing CF (Table 1). For 85 additional units, many of which were nonresponsive to PM-MWR, the PSTH yielded a single peak and no such estimate of the CF was possible. For the majority, however, the response to continuous (tone) stimuli suggested that they were most sensitive to high frequencies (CF above 4 kHz). Thus, it is our impression that the trend shown in Table 1 reflects the possibility that high-frequency units are considerably less responsive than low-frequency units to maximal MWR. We should note, however, that lengthy pulses of MWR were used and that the maximum energy per pulse was achieved by use of a 300-μsec pulse.

3.4. *Acoustic controls.*

The observations (1) that there was a considerable latency for the observed unitary response to the pulsed MWR, (2) that the MWR-PSTH was of the same form as the acoustic PSTH, and (3) that the single-unit response could be a nonmonotonic function of pulse width, confirmed that this component of the MWR-induced response did not in any way derive from electrical artifacts via the microelectrode or its associated electronics. Short latency responses were rarely seen. Such responses, if observed, could have indicated a direct

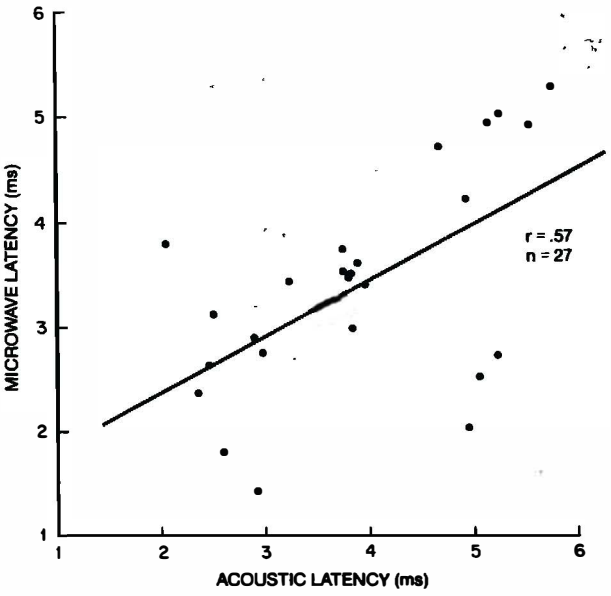

Fig. 5. Latency to first peak of the MWR-PSTH versus latency to first peak of the acoustic PSTH, for 27 units exhibiting multipeaked responses to both stimuli. Acoustic latency was taken as the time from the leading edge of the voltage pulse across the earphone to the first peak in the PSTH; the microwave latency similarly corresponded to the time from the leading edge of the MWR pulse to the first peak of the PSTH. The correlation was significant at the level $P < 0.01$.

neural effect, but the likelihood of intrusion of some electrical or other artifact would have been so compelling as to require detailed control studies. That such responses were not seen at the levels of MWR we used is an additional encouraging sign that we were dealing with a novel but authentic physiological response.

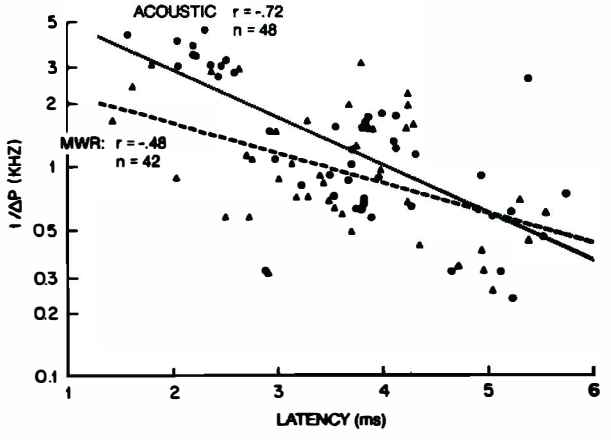

Fig. 6. Comparison of apparent CF with unitary response latency. Shown is the reciprocal of the PSTH interpeak interval, $(\Delta P)^{-1}$, versus the latency to first peak of the multipeaked response for 48 acoustic responses (circles) and 42 MWR unit responses (triangles). Acoustic and MWR response latencies are defined as in Fig. 5. Correlations are significant at $P < 0.01$.

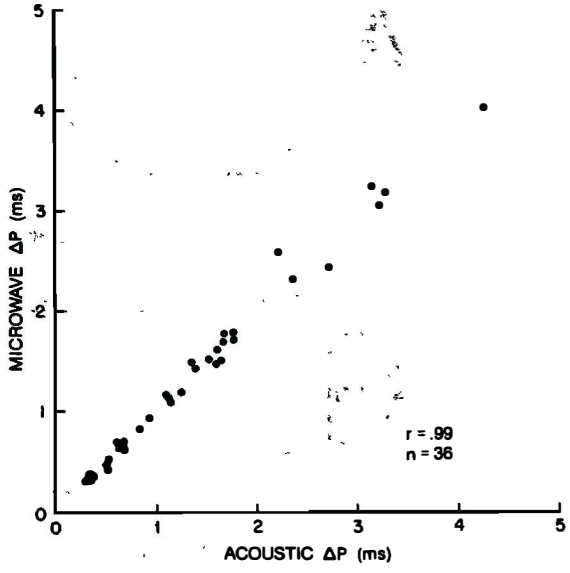

Fig. 7. Interpeak intervals (ΔP) of MWR-PSTH versus interpeak intervals of acoustic PSTH, for 36 units exhibiting multipeaked PSTHs to both pulse MWR and to the equivalent acoustic clicks. Details are presented in text.

Whether the earphone remained in place or was removed prior to MWR, the same PSTHs were obtained. Thus, no earphone-coupling artifact occurred. However, there is the possibility that the MWR pulse impinging on the stereotaxic apparatus or on the surrounding MWR absorbent material could have produced an artifactual acoustic stimulus. This possibility was checked directly by placing a sensitive contact microphone on the stereotaxic head holder. The contact microphone was certainly far less sensitive than the cat's ear, so that this test was primarily to determine whether any PM-MWR-induced acoustic transient, comparable to the acoustic-test stimuli, occurred in the stereotaxic head piece. This was a worse-case test since, with the animal in the stereotaxic frame, the most efficient acoustic coupl-

TABLE 1. Auditory unit response to microwave pulses.

Estimated Characteristic Frequency (Hz)*	Number Tested	Number Microwave Responsive	Percent Microwave Responsive
<400	6	5	83%
400 - 711	11	11	100%
711 - 1265	10	9	90%
1265 - 2249	13	10	77%
2249 - 4000	8	5	63%
No CF Estimate	85	23	27%
Totals	133	63	---

*Based on CF = $(\Delta P)^{-1}$, where ΔP is the interval between the first two peaks of the acoustic-PSTH. Cells for which there was only a single-peaked acoustic-PSTH provided no such estimate.

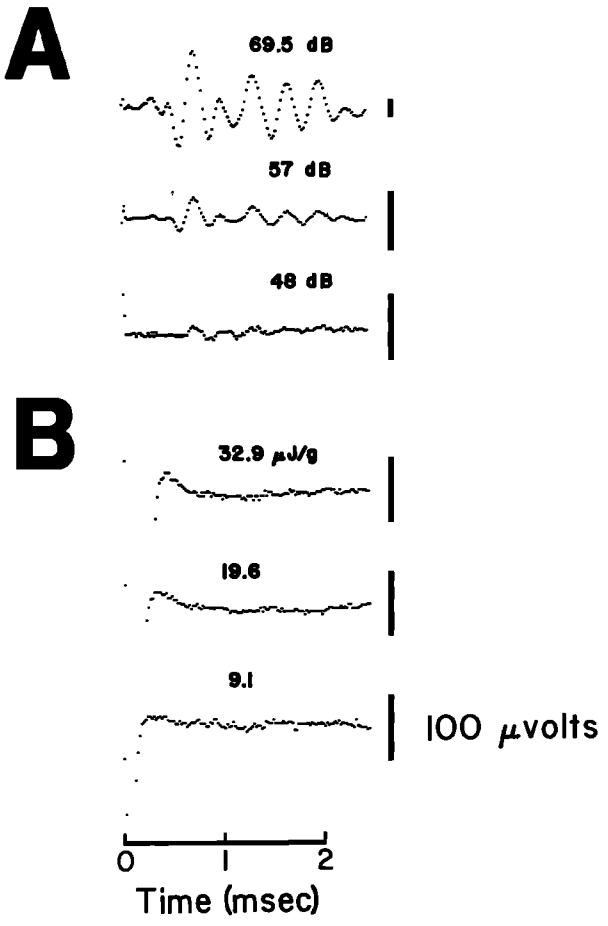

Fig. 8. A. Mechanical transients induced in the stereotaxic apparatus by a click train in condenser microphone that was mounted in ear bar. Each response represents the output voltage of a sensitive contact microphone mounted to the stereotaxic head frame (average of 256 trials). Acoustic clicks were 70 μsec in duration, at 10/sec, with sound levels as noted. B. Microphone output voltage as above, in response to a train of MWR pulses at the maximum peak of power density. Each response was the average of 256 trials at a rate of 10/sec, but with the condenser earphone in place and inactive. The MWR pulse widths were, from top to bottom, 250, 150, and 70 μsec, with the equivalent energy dose per pulse as indicated.

ing would indeed be via the head support and ear bars. No significant acoustic artifact from the pulsed MWR was found (Figure 8). In addition, no sound could be detected by the investigators when the animal was being irradiated. The effective PM-MWR acoustic stimulus must have originated, therefore, within the animal.

4. DISCUSSION

We were able to demonstrate directly an acute effect of single pulses of MWR on cells of the auditory system. The precise nature of the interaction is still open to question, although the range of possibilities is now somewhat reduced by these data. For reasons that are outlined elsewhere [*Lebovitz*, 1975] it is clear that the interaction underlying microwave hearing is within the head. The results reported here -- specifically, the form of the MWR-PSTHs -- support our contention that there is little likelihood that microwave hearing arises from an interaction of the MWR pulses directly with the fibers of the cochlear nerve or with cell bodies at higher stations in the auditory system. On the basis of poor coupling efficiencies, electromechanical interactions either with the tympanic membrane or with the ossicles of the middle ear are unlikely to explain the threshold levels we observed [*Sommer and von Gierke*, 1964; *Guy et al*, 1975]. The demonstrations of subjective auditory perception [*Frey*, 1962], of auditory evoked potentials [*Taylor and Ashleman*, 1974], and of cochlear microphonics [*Chou et al.*, 1975] imply collectively that auditory unit responses to pulsed MWR should occur. Thus, the primary utility of our data lies not in the demonstration of single unit responses as such, but rather in providing data by which to specify the transduction process, that is, the locus and mode of interaction of the MWR with the test animal.

The most satisfactory hypothesis to date for explaining microwave hearing is that the incident MWR pulse elicits a thermoelastic wave of pressure in the head [*Foster and Finch*, 1974]. The mechanical wave may be initiated in or coupled to the cranium and would thereby stimulate the inner ear via bone conduction. An implication is that the response of neurons in the auditory pathway to pulsed MWR would necessarily be similar to their response to transient mechanical stimuli, such as acoustic clicks. In our studies, the MWR-PSTH and acoustic-PSTH of each MWR-responsive unit were indeed sufficiently similar to suggest some level of equivalence between the two stimuli with respect to the auditory periphery. The nonmonotonic dependence of the response on MWR pulse width in some units (Figure 3) was similar to the effect of acoustic pulse width on responses of primary fibers [*Kiang*, 1965]. The latter effect is known to derive from the relation between the frequency content of the click and the tuning curve of the unit. Thus, we have additional evidence that the frequency content of the envelope of the MWR, relative to the characteristic frequency of a given unit, determines the magnitude of the MWR response of a neuron.

The periodic MWR-PSTH suggests also that the excitation of the peripheral receptors (hair cells of the cochlea) was similarly periodic after each MWR pulse. Magnitude of the response aside, the close correspondence between acoustic and MWR $\triangle Ps$ (Figure 7) is strong evidence that this periodicity was determined at the acoustic periphery rather than by the MWR stimulus as such. As a counter example, one could conceive of the PSTHs having grossly different forms in response to acoustic and to MWR stimuli. To the extent that such differences could be demonstrated, one could support the concept of a direct interaction of the PM-MWR with the hair cells of the cochlea, or perhaps with discrete portions of the organ of Corti. However, such unique responses were not reliably demonstrated and the

data are most consistent with physiological activation of the auditory periphery by mechanical means.

Some difficulties with the current thermoacoustic hypothesis arise because of the observed decrease in MWR sensitivity of high-frequency auditory units. The thermoacoustic hypothesis itself implies a pronounced high-frequency mechanical component to the MWR response [*Foster and Finch,* 1974]. *Chou et al.* [1975] have likewise reported a 50-kHz component in the MWR-induced cochlear microphonic. These results would suggest that high-frequency auditory units would be most responsive to PM-MWR. The contrary result was obtained, although our data in this regard are admittedly incomplete. However, it is worth considering that the cochlear microphonic, although present, might be secondary to some other primary acoustic transient or that the envelope of this microphonic better represents the acoustic equivalent of the MWR pulse. Clearly, microwave hearing is more complex than at first presumed and an interaction of the MWR directly with some intracochlear component cannot yet be totally ruled out.

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