COCHLEAR RESPONSES TO CONDENSATION AND RAREFACTION CLICKS

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ABSTRACT Auditory nerve responses to condensation and rarefaction clicks (CC and RC) have been recorded over a wide intensity range with gross electrodes. At low intensities the RC responses are nearly identical to CC responses. At high intensities RC and CC response waveforms are similar, but the latency of the N_1 peak in the RC response is 0.2 msec. shorter than that for the corresponding CC response. At intermediate intensities the RC and CC response waveforms are quite different. These results can be interpreted in terms of a model in which there are two excitatory mechanisms for the neural response, which are operative in different intensity ranges. The cochlear microphonic potential and a "slow" potential are suggested as possible excitatory mechanisms.

INTRODUCTION

Although it is usually assumed that bending of hair cells in the organ of Corti leads to excitation of the endings of the auditory nerve fibers, the exact mechanism of this process is not known. It is quite well established that the generation of the cochlear microphonic potential, CM, depends on the integrity of the hair cells, and it has been suggested that CM may serve as a generator potential at the nerve endings (1). Davis (2) has hypothesized that the negative summating potential, SP–, is also a generator potential, which enters into play at high stimulus intensities. The present data suggest that two excitatory mechanisms are required to explain certain findings on responses to clicks. The model which we suggest is independent of Davis's hypotheses.

METHODS

The summated response of the auditory nerve to acoustic clicks can be recorded between a wire electrode placed near the round window of the cochlea and a reference electrode. Neural responses to rarefaction clicks (RC) resemble in general responses to condensation clicks (CC). However, several workers have observed that these two classes of stimuli give rise to quite different responses at certain intensities (3–5). Because CM is also prominent in responses that are recorded in this manner, it is difficult to determine precisely the extent to which neural responses change when the stimulus polarity is reversed.

In order to investigate the neural without contamination by the presence of CM, we recorded from a concentric electrode placed in the internal auditory meatus. This electrode configuration and location is known to record relatively little CM(6).

Responses were recorded from cats which were anesthetized with Ciba Dial injected into the peritoneal cavity (75 mg/kg). The skull was opened and a portion of the cerebellum was removed so that the eighth nerve–cochlear nucleus region was exposed. Concentric electrodes (1 mm outside diameter with the center wire extending 2 mm beyond the sleeve) were then placed in the nerve at the most lateral position possible; a wire electrode was also placed near the round window. The animal was in a soundproof room; acoustic stimuli were delivered by a Permoflux PDR-10 earphone through a plastic tube (approximately 6 cm long) which was tied into the external auditory meatus. Clicks were generated by applying a 0.1 msec. rectangular pulse to the terminals of the earphone. Responses were amplified (pass band 8–7000 CPS) and observed on an oscilloscope. Often responses were recorded on magnetic tape; averaged responses were computed either from the tape records or "on line" during experiments by means of an average response computer, ARC-1(7).

At the beginning of an experiment a threshold for neural responses to clicks was determined by inspection of single traces on the oscilloscope screen. This threshold is defined as VDL = visual detection level. The stimulus intensity was then increased in 10 db steps from 10 db below VDL to approximately 100 db above VDL. Responses were recorded for both stimulus polarities at each intensity. The click repetition rate was low enough (5/sec. at low intensities, 1/sec. at high intensities) so that successive responses were for practical purposes independent.

RESULTS

The responses that concentric electrodes record resemble the neural components that are recorded by round window electrodes (Fig. 1). The latency of the first negative peak N_1 is, however, about 0.2 msec. longer for the electrode position in

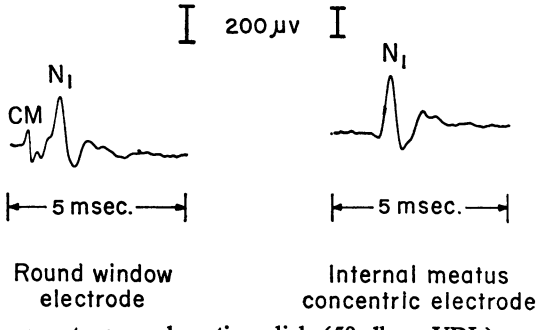

FIGURE 1 Responses to a condensation click (50 db re VDL) recorded simultaneously from two electrode locations. The left trace was recorded between a wire electrode near the round window and a reference lead on the head holder. The right trace was recorded from concentric electrodes in the internal auditory meatus. In all records an upward deflection indicates negativity of the round window electrode with with respect to the reference, or negativity of the core electrode with respect to the sleeve of the concentric electrode. Note that the microphonic potential (CM) is not visible in the response from the concentric electrodes. (C-504).

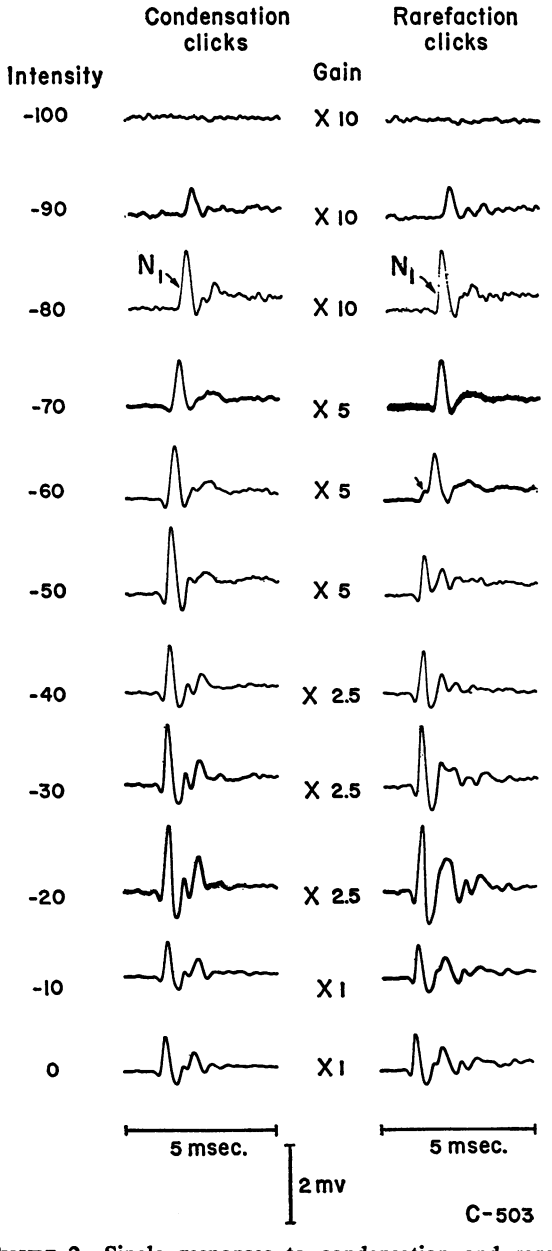

FIGURE 2 Single responses to condensation and rarefaction clicks recorded from concentric electrodes in the internal meatus. Intensity is given in db re 2.5 volts to the earphone. VDL = −95 db. The voltage calibration corresponds to a gain setting of × 1.

the internal meatus. This difference presumably can be attributed to conduction time. Changes in the latency and amplitude of N_1 with stimulus intensity are quite similar for the two electrode positions. At some of the higher intensities CM is as large or larger than N_1 in the round window record so that measurement of N_1 is difficult.

Fig. 2 shows neural responses to rarefaction and condensation clicks over an intensity range of 100 db; the peak-to-peak amplitude of the N_1 components and the latency of the N_1 peak are plotted as functions of intensity in Fig. 3. These re-

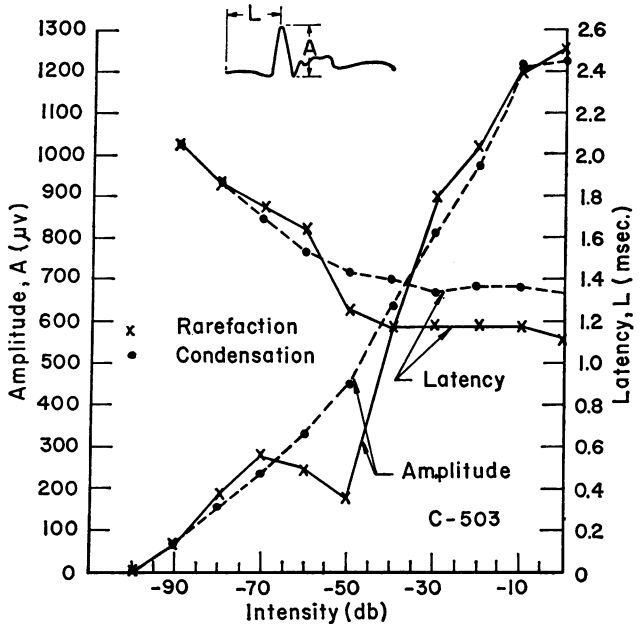

FIGURE 3 Amplitude and latency of N_1 responses to condensation and rarefaction clicks (from data of Fig. 2). Measurements were made as indicated on the inset. When two negative peaks occur in the response (as for rarefaction clicks at −60 and −50 db), the measurements are made to the larger of the two peaks. Latencies are measured from the instant of application of the voltage pulse to the phone; they thus include an interval of 0.3 msec. of sound propagation time.

sults can be described as follows:—(*a*) At low intensities (−90 to −70 db) the responses to RC and CC are nearly equal in amplitude and latency. (*b*) At high intensities (−40 to 0 db) the response amplitudes are nearly equal but the latency of the response to RC is consistently shorter by about 0.2 msec. (*c*) At intermediate intensities (−60 to −50 db) there are striking differences between the responses. The response to CC increases in size monotonically with intensity, but the response to RC changes shape and the earliest component (which by definition is identified as N_1) decreases with intensity in this range. As the intensity is increased an earlier

"bump" appears at the leading edge of the response (see arrow on Fig. 2) to RC at −60 db. At −50 db the early bump has emerged as a component that is larger than the later component. Since the latency is measured to the most negative and earliest peak of the response there occurs a large change in latency between −60 and −50 db.

Fig. 4 shows superimposed averaged responses to CC and RC from another animal. This display shows clearly that the RC and CC response *waveforms* are nearly the same at both low and high intensities but that there is a definite difference

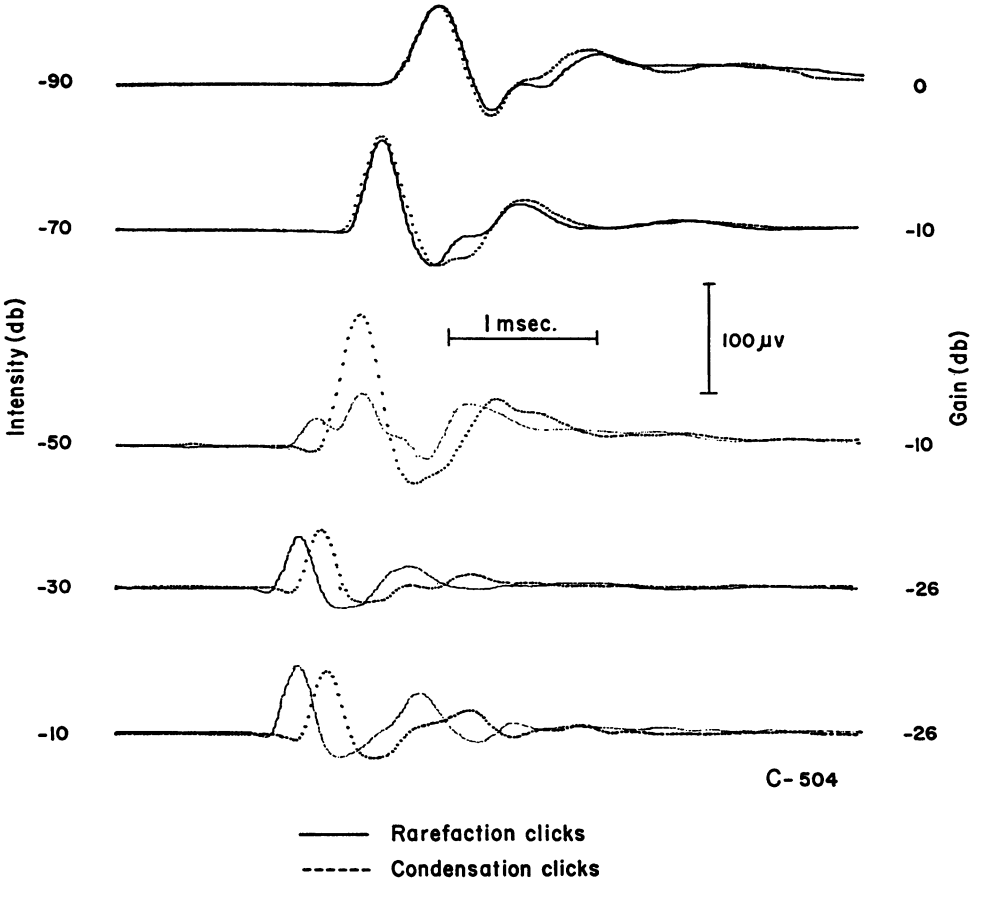

FIGURE 4 Averaged responses to CC and RC. Number of responses averaged by ARC-1 ranges from 128 at −90 db to 16 at −10 db. Voltage calibration corresponds to the 0 db gain setting. VDL = −100 db.

in latency at high intensities. At −50 db there are two early negative peaks in the RC response. Amplitude and latency are plotted in Fig. 5 for this animal as a function of stimulus intensity.

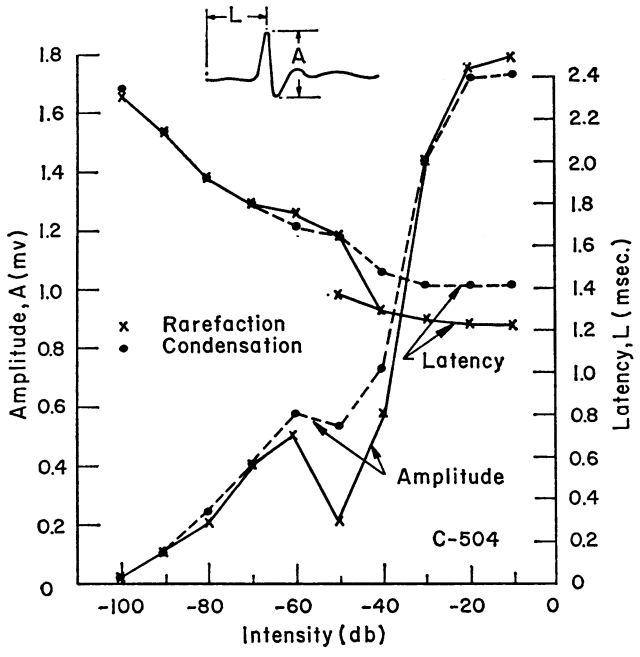

FIGURE 5 Amplitude and latency of N_1 responses for C-504. Measurements were made as in Fig. 3. At —50 db the latencies of both negative peaks have been plotted.

INTERPRETATION

Earlier work has led to the suggestion that motion of the basilar membrane in one direction produces neural excitation but that motion in the other direction does not; the excitatory direction is that corresponding to rarefaction at the eardrum (8). This finding is in agreement with the data of Figs. 2 and 4, which show that at high intensities the latencies of responses to RC are shorter than those to CC. Rosenblith and Rosenzweig (9) found that with stimulation by low frequency tones neural responses occur during the positive half-cycle of CM as recorded near the round window. These data suggest that for round window recording positivity of CM reflects the rarefaction phase of the mechanical displacement. Our data agree with this interpretation, since the initial microphonic deflection in responses to RC is positive.

If we assume that positivity of CM gives rise to excitation, and also assume that the neural response is triggered when CM reaches and exceeds a certain voltage level, then we would expect to find a latency difference between neural responses to CC and RC. This difference would amount to about 0.2 msec., since the first positive deflection of CM in response to CC occurs 0.2 msec. later than the first positive deflection in response to RC. (See interval *A* in Fig. 6.) Since the initial

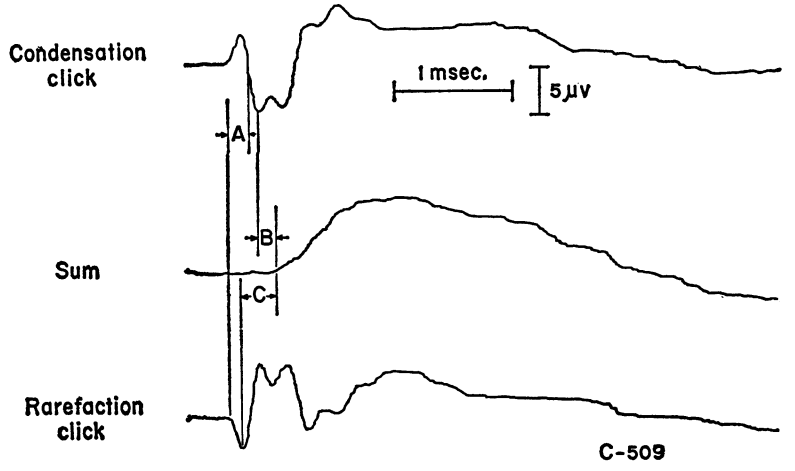

FIGURE 6 Averaged responses to CC (top) and RC (bottom) from a denervated cochlea (10). The middle trace is the sum of an equal number of responses to CC and RC. Note that the early CM components cancel in the middle record, but that a slow component of the response subsists which seems to be common to both CC and RC. Stimulus intensity is —70 db.

The shape of the CM component of the response is determined not by the cochlea only, but by the characteristics of the entire system composed of earphone, acoustic coupling device, middle and inner ears. Since both click polarities produce positive and negative deflections, the terms "condensation" and "rarefaction" refer strictly speaking only to the direction of the initial departure from the equilibrium condition.

rise of CM to its first peak requires only 0.1 msec., the model of a fixed trigger level predicts a comparably small decrease in latency accompanying the increase in CM amplitude produced by increasing stimulus intensity. Also, since the amplitude of CM levels off and even decreases with stimulus intensity above −20 db (Fig. 7) the latency might be expected to remain constant in this region. These expectations agree quite well with the latency data at the high intensities (−40 db to 0 db). Thus the hypothesis that CM is closely related to the excitatory process for the neural response is supported *in this intensity range.*

At low intensities, however, this picture does not seem to fit, since the neural responses for both click polarities are nearly the same in this range and the latencies change rather rapidly with intensity. Perhaps an excitatory process other than CM predominates in the low intensity range. The slow potential which we have described previously (10, 11) (also see Fig. 6) has several properties which make it a suitable candidate for such a process. (*a*) This potential is the same for both stimulus polarities. (*b*) At low intensities it is more prominent than CM (see Fig. 7). (*c*) It has a relatively gradual slope, a property that could lead to latency changes with intensity that are similar to those actually observed.

By making certain assumptions about triggering levels associated with the slow

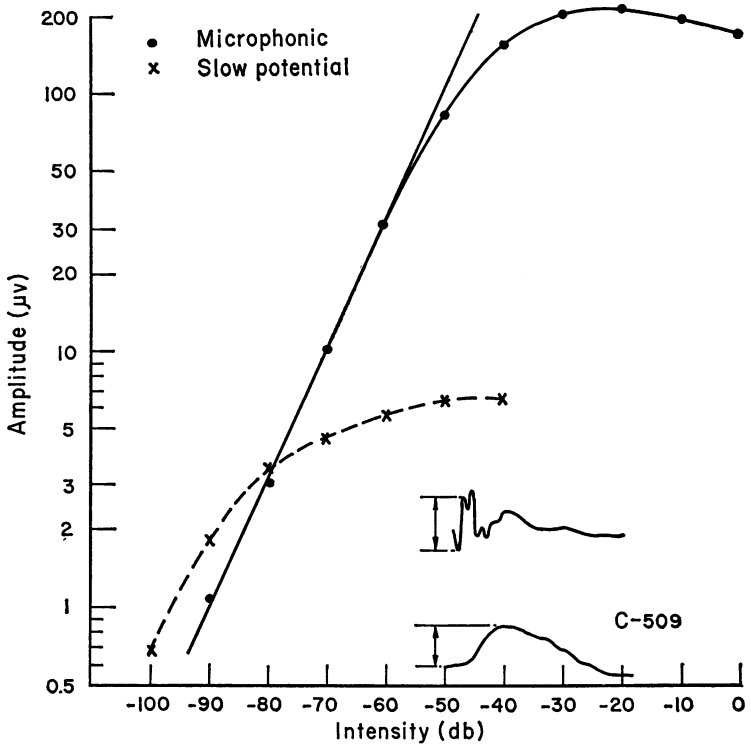

FIGURE 7 Amplitudes of microphonic and slow potentials *versus* click intensity. The amplitudes were measured as indicated in the sketch. The slow potential cannot be measured at high stimulus intensities, since a faster component overrides it in the records (10). The straight line indicates the linear growth (amplitude increasing by a factor of 10 for a 20 db intensity increase) of the microphonic in the low intensity range. 0 db = 3.8 volts.

potential and CM we can plot theoretical latency *versus* intensity curves (Fig. 8). The triggering level for the slow potential is assumed to be the maximum (negative) value that it reaches at an intensity near VDL. For CM the maximum positive value at −60 db is chosen as the trigger level, since the CM process is assumed to come into play at about that level. The predicted curves parallel experimental data that were obtained from another animal. Since the slow potential and CM response were recorded from denervated cochleas, there was no opportunity to make this comparison on data from a single animal. The relatively constant difference between the two sets of curves can be ascribed to the delay that occurs between the initiation of the response at the nerve ending and the arrival of this response at the recording electrodes (*i.e.* conduction time).

It might be argued that the slow potential does not have the right polarity to be an excitatory process, since it is negative at the round window whereas the rarefac-

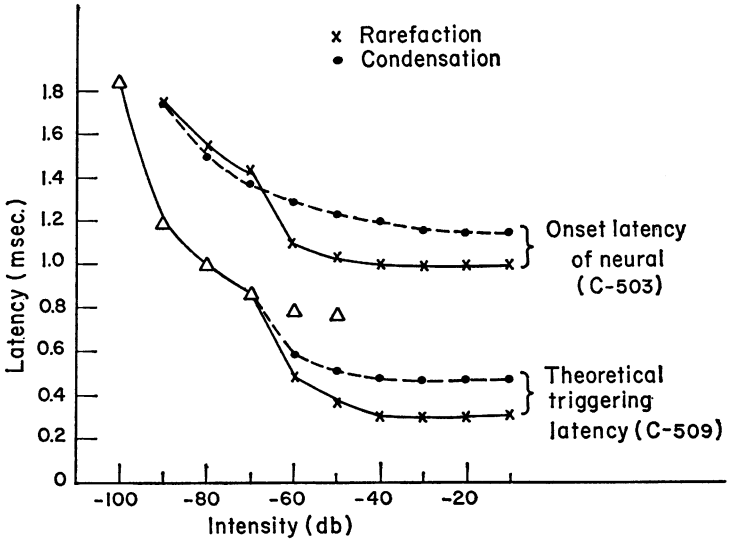

FIGURE 8 Latency of onset of N_1 *versus* intensity. The top curves show onset latencies for responses to CC and RC (from data of Fig. 2). The lower curves show the latency at which the "trigger level" is first reached by the assumed excitatory process. The triangles indicate the time at which the slow potential recorded from a denervated cat (Fig. 6) reaches its trigger level. This level was chosen as the peak value of the slow potential at −100 db which corresponds approximately to the VDL for the neural response. The dots and X's on the lower curves indicate the time at which the trigger level is reached by CM in the same denervated preparation. This level was taken as the value of the first positive peak of the rarefaction click response at −60 db, since that is the lowest intensity at which the early bump is observed.

tion phase of CM and the so called negative summating potential, SP_, are positive at this location. However, since the mechanisms through which these potentials excite the nerve are not known, there is no *a priori* reason that the same polarity particularly at the round window should be excitatory for different processes. Even if the same kind of process is involved, the polarity difference might result from a difference in the spatial orientation of the sources of these potentials.

The striking difference between responses to CC and RC in the intermediate intensity range can now be considered in terms of these two excitatory processes. In this middle range both processes may be assumed to be effective and to overlap to some extent. Excitation by CM becomes predominant as the intensity increases from −60 to −40 db. The positive swing of CM in response to CC reaches its peak a little ahead (0.15 msec.) of the onset of the slow potential (interval *B* in Fig. 6). Hence, as one process takes over from the other there will be a smooth change in neural response latency. However, the first positive deflection of CM in response to RC peaks 0.3 msec. earlier than the onset of the slow potential (interval *C* in Fig. 6). Hence, the two processes might be assumed to excite separate neural re-

sponses. This changeover in effective excitatory mechanism could account for the appearance of the small early bump at −60 db (Fig. 2) and the jump in latency of the largest negative peak (Fig. 3). The decrease in peak-to-peak amplitude of N_1 with increasing intensity (for the RC intensity change from −60 to −50 db) might be the consequence of refractoriness of the neural units at the onset of the slow excitatory process because of previous excitation by the CM-related process. Such an explanatory scheme implies that the two processes act, at least in part, on the same population of neural units.

This model is, of course, speculative and incomplete. The assumption of a fixed trigger level associated with excitatory potentials is undoubtedly a gross oversimplification. Certain features of the data (for example the N_1 peak latency to CC being somewhat shorter than to RC near −60 db) are not accounted for by the model. No attempt has been made to relate the size of the neural response to behavior of the slow potential or CM. Perhaps the most important criticism of this model is that it ignores the possibility of changes in latency of N_1 resulting from excitation of nerve endings at different positions along the basilar membrane. If the waveform of the membrance deflection in response to our clicks were known as a function of position along the cochlear partition, and if the relative contribution to N_1 by nerve elements arising from different positions were known, perhaps a different picture of the mechanisms of excitation would emerge. In the absence of such data we have assumed that CM as recorded near the round window reflects the displacement in phase of a large part of the basal turn of the cochlea, and that fibers that innervate the basal turn make a major contribution to N_1 as recorded from near the round window.

The existence of a low intensity mechanism which is not CM helps to explain the occurrence of relatively large neural responses under conditions for which CM is so small that it is not easily detectable. Conditions for which this observation has been made include low intensity acoustic stimulation, acute damage to the cochlea (8), and cochlear poisoning by antibiotics (12, 13).

Possible relations of this model of excitation of responses from the auditory nerve to that proposed by Davis (2) are not clear. It may be, however, that the two models fit together. Davis has suggested that the negative summating potential, SP_-, is the dominant excitatory process at high intensities, and that CM is dominant at lower intensities. We have suggested that our slow potential is the dominant process at low intensities. Perhaps all three mechanisms are involved, each in a different intensity range. Perhaps also there are other excitatory mechanisms which are as yet undemonstrated. Part of the difficulty in comparing results lies in the fact that Davis and his colleagues have generally used tone bursts as stimuli whereas we have generally used clicks. It is possible that the relative importance of different excitatory mechanisms depends not only upon intensity but also upon the type of stimulation, so that a process which is important for tone bursts may not be important for clicks.

For example, since SP_{-} is observed most easily in response to bursts of tone above 7 kc, this potential (SP_{-}) may be very small in response to our clicks, which produce a rapidly damped CM oscillation having a high frequency component at about 3.5 kc. Hence, SP_{-} may not be a significant process for click stimulation. On the other hand we find that responses to high frequency tone bursts do not show detectable changes in latency for changes in polarity at *any* intensity. Possibly CM is not so important a process for high frequency tone bursts as for clicks. In summary, the existing experimental data are difficult to account for by theories involving only a single excitatory mechanism for generating nervous activity. At the moment it is easier to incorporate the data into a theory that assumes multiple mechanisms for excitation.

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