plitudes of GSR to CS+ and summed amplitudes of GSR to CS− were computed for first- and second-interval responses during acquisition trial blocks, and for first-, second-, and third-interval responses during extinction trial blocks. In all comparisons for the Accurate group, summed amplitudes of GSR to CS+ were significantly greater than amplitudes to CS− (Wilcoxon test, $P < .05$). None of the comparisons were statistically significant for the Inaccurate group.

The data were also analyzed to determine whether conditional GSR differentiation was related to the trial in which an accurate statement of the stimulus contingencies was initially reported. For these analyses, the seven subjects who initially made an accurate intertrial report within trial blocks 6 to 10 were classified as "early verbalizers." The eight subjects who initially reported the stimulus contingencies within trial blocks 11 to 14 were classified as "late verbalizers." For each subject in both groups, amplitudes of GSR to CS− were subtracted from amplitudes to CS+ to yield an algebraic difference score for each trial block. For first- and second-interval GSR's, median difference scores were calculated for acquisition trial blocks 6 to 10 and 11 to 14, and for extinction trial blocks 15 to 18. For third-interval GSR's, a median difference score was calculated only for trial blocks 15 to 18.

For the first-interval responses during extinction trial blocks, the median difference score of 0.247 for the late verbalizers differed significantly from the median difference score of 0.000 for the early verbalizers (Mann-Whitney $U = 11$, $N = 7, 8$; $P < .05$). None of the other comparisons yielded statistically significant evidence for a temporal relationship between intertrial verbal reports and conditional GSR differentiation.

Our findings appear to converge with results of studies in which preparatory instructions and procedural shifts were used. Our data provide additional evidence for congruence between conditional autonomic differentiation and cognitive differentiation of conditional stimulus contingencies. Accurate verbalization of stimulus contingencies during interviews after conditioning was associated with conditional GSR differentiation. When verbalizations concerning the stimulus contingencies were omitted or inaccurate, no demonstrable GSR differentiation appeared. These results are consistent with a theoretical viewpoint that treats human classical conditioning as a problem-solving activity in which verbal processes are of fundamental importance (*7*). This viewpoint requires elaboration by studies of the relationship between autonomic and cognitive changes for different conditioning paradigms and autonomic response modes.

The congruence between autonomic and cognitive changes also could be established by content-analysis of intertrial verbal reports. Intertrial verbal reports were less useful when the trial number of the first accurate report was considered for investigating synchrony between cognitive and autonomic differentiation. The relative lack of positive results may be attributable in part to the limited number of subjects involved. The only reliable finding was that the first-interval responses of a group whose initial accurate report occurred during the later acquisition trials showed greater resistance to extinction than the responses of a group whose initial accurate report occurred during earlier trials. Regardless of whether the initial accurate report occurred promptly during acquisition trials or was delayed, differentiated GSR's appeared in the first few acquisition trial blocks. Some subjects may have delayed reporting until they were confident of the accuracy of their reports. This consideration is amenable to instructional manipulation.

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Temperature Effects on the Peripheral Auditory Apparatus

Abstract. *Cooling with a thermoelectric cold probe, well localized in the region of the cochlea, produces a rapid, reversible decrease in the amplitude and increase in the latency of the action potential induced by clicks. These changes closely resemble those produced by reducing click intensity. Temperature also affects the amplitude of the cochlear microphonic, but the amount of change is considerably less than, and is poorly correlated with, the amplitude change of the action potential. It is speculated that temperature may act on a hypothetical "excitatory process" in the cochlea, which comes after the cochlear microphonic in the sequence leading to production of the action potential of the auditory nerve.*

A study has been made of the effect of temperature change on the cochlear microphonic and on the action potential of the auditory nerve. The temperature of the auditory end organ was varied by placing the tip of a "thermoelectric cold probe" (*1*) firmly against the bony ridge just beneath the round window.

With a previously described technique (*2*), action potentials induced by clicks were recorded from the round window, and, in some experiments, also from the auditory nerve, of anesthetized cats. Temperature was monitored by a thermocouple affixed to the surface of the thermoelectric cold probe near the tip. A heat lamp was used to maintain a normal overall body temperature (measured rectally). Thus, the recorded temperature changes were, in all likelihood, well localized in the region of the cochlea.

The following results are in agreement with the results of other investi-

gations in which the temperature of the entire animal was lowered (*3*): (i) Decreasing temperature decreases the amplitude of the action potential and increases the delay between stimulus and action potential (hereafter referred to as latency). (ii) These changes are reversible within a rather wide range of temperatures. (iii) The amplitude of the cochlear microphonic (but not its latency) is similarly affected by temperature. However, cochlear-microphonic amplitude changes much less than does action-potential amplitude.

The reversibility of the changes in action-potential amplitude with changes in temperature is well illustrated in Fig. 1. Besides its reversibility, three other characteristics of the response to rapid temperature change are illustrated by this record: (i) At the beginning of a rapid decrease in temperature, the action-potential amplitude shows a transient increase. However, if temperature was lowered slowly, or if time was allowed for amplitude change to reach equilibrium after a rapid lowering of temperature, the action-potential amplitude always decreased (compare Fig. 2). (ii) The action potential responds to cooling more rapidly than to rewarming. Whether it does so because of physical (heat transfer) or physiological factors is, at present, problematical. (iii) Although there is a significant delay between the temperature change and the response of the action potential, once the action potential begins to respond, it does so relatively rapidly. Much, or even all, of the delay between the temperature change and the response of the action potential can probably be accounted for by the time required for heat conduction from the probe tip to the cochlea. It would there-

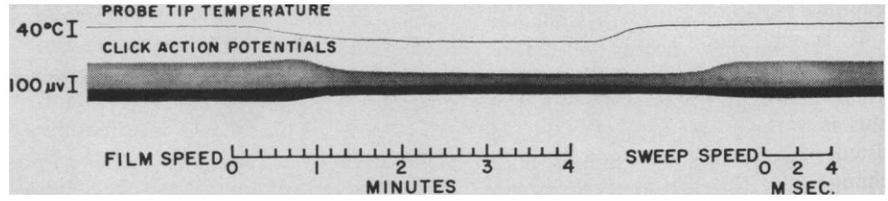

Fig. 1. Response of a train of action potentials to rapid cooling and rewarming in the region of the cochlea. The clicks were obtained from a PDR-600 earspeaker driven by square wave pulses 0.01 msec in duration, 0.1 volt in amplitude, and delivered at a rate of five pulses per second. The action potentials were displayed on the oscilloscope with sweep speed as shown, and were photographed on film moving slowly parallel to the direction of the sweep.

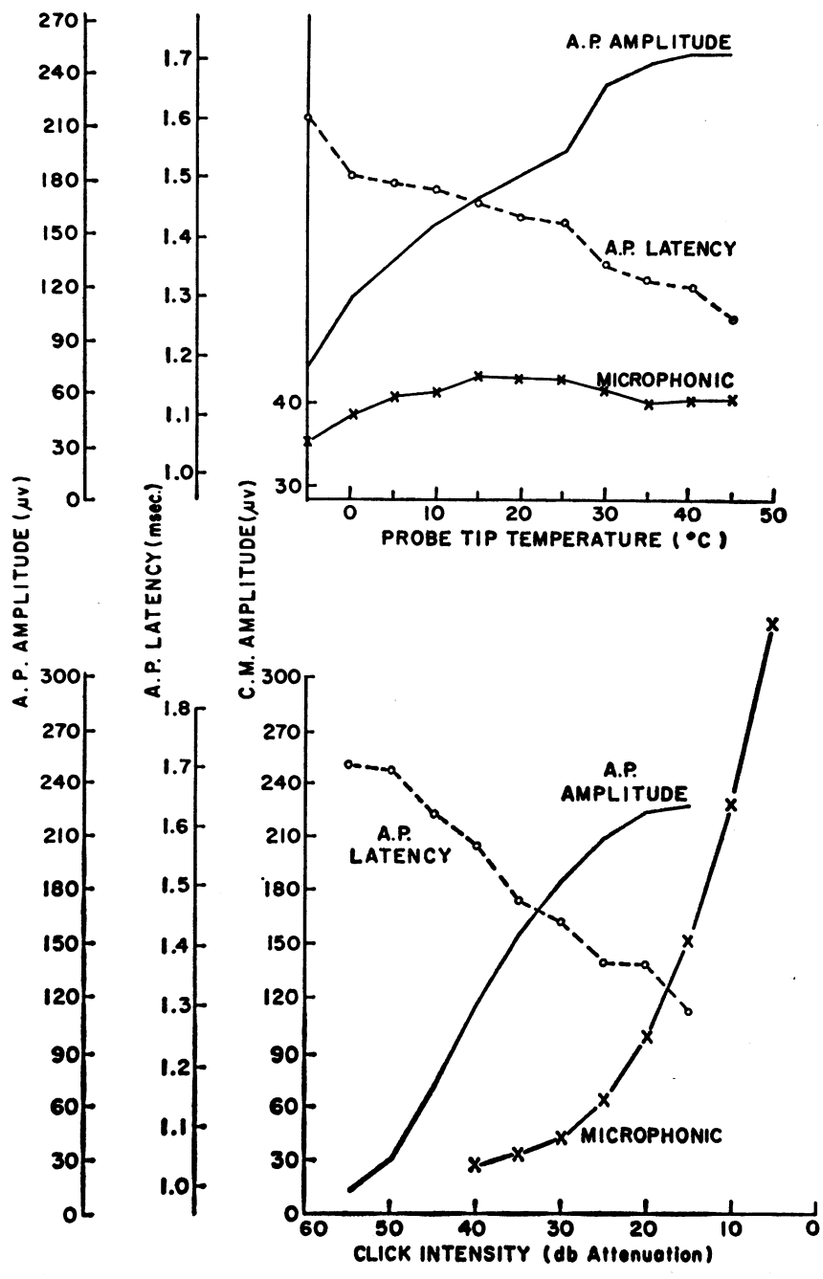

Fig. 2. The effect of varying click intensity and cochlear temperature on action potential (*A.P.*) amplitude and latency and on microphonic (*C.M.*) amplitude. The clicks were produced by driving a PDR-600 earspeaker with an 0.01-msec, 40-volt square wave lead through a decade attenuator. The plot at the top was obtained by varying temperature in the region of the cochlea (probe tip temperature). The decade attenuator was set at 30 db. In order to display the relatively small microphonic amplitude changes better, the scale was expanded to five times the other amplitude scales in this figure. The plot at the bottom was obtaining the varying click intensity, with tip temperature of the cold probe held at 25°C. Action-potential latencies were measured from the beginning of the oscilloscope sweep (which was synchronized with the electrical pulse driving the speaker) to the first action-potential peak. Sound-conduction time from the earspeaker to the animal was about 0.3 msec.

fore seem reasonable to characterize the response as rapid.

It is well known that the latency of the action potential is changed when the intensity of the click stimulus is varied. Part of this study consisted of a comparison of this latency change with the change in latency observed when temperature is varied. An examination of the two sets of curves in Fig. 2 gives the impression that the changes in latency and amplitude produced by varying temperature are at least roughly comparable to the changes produced by varying click intensity.

Figure 3 shows a test of this impression. Here, latency is plotted against amplitude for changes in both temperature and click intensity. All plots appear to be superimposable. Similar plots from three other animals produced identical results.

Included in Fig. 2 are plots of microphonic amplitude. Unlike the action potential, the microphonic responds very differently to changes in intensity and to changes in temperature. As temperature is increased, the microphonic reaches a maximum, while the action potential continues to increase. It is thus apparent that, when temperature is varied, the resulting change in microphonic amplitude is poorly correlated with the change in action-potential amplitude.

In contrast, when click intensity is increased, microphonic amplitude shows a continuous increase. Furthermore, the degree of this amplitude change is an order of magnitude greater than the degree of change produced by varying temperature (*4*).

The mechanism by which temperature acts on the action potential of the auditory nerve remains to be elucidated. However, the poor correlation of the microphonic and the action potential does suggest that the primary effect of temperature occurs sometime after the generation of the microphonic. Furthermore, since the microphonic is probably as vulnerable to changes in blood supply as the action potential is (*5*), this poor correlation makes it unlikely that temperature acts via changes in cochlear blood flow. Further evidence against this possibility has been supplied by Naumann *et al.* (*6*) who directly observed cochlear blood flow during local cooling and reported no change either in diameter of the cochlear blood vessels or in rate of flow. Still further evi-

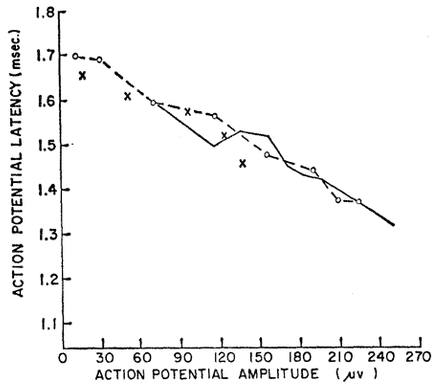

Fig. 3. Action-potential amplitude versus action-potential latency for temperature change (solid line) and click-intensity change (open circles). These plots were obtained from the same data presented in Fig. 2. Also included is a plot (from the same animal) of the effect of varying click intensity at 5°C (probe tip temperature) (crosses).

dence against the possibility that cooling acts via changes in cochlear blood flow is provided by Perlman *et al.* (*7*), who report that the response of the auditory-nerve action potential to generalized cooling is poorly correlated with observed cochlear blood flow changes (which, under generalized cooling, are presumably secondary to changes in the systemic circulation).

The similarity of the relationship between amplitude and latency of the action potential when temperature and click intensity are varied suggests that temperature and intensity act at a common point. Click intensity clearly does not act by changing the excitability of the nerve endings. Therefore, the common point of intensity and temperature action would have to be at the "excitatory process" which delivers the stimulus to the auditory nerve endings. It is tempting to visualize that the response of this excitatory process to temperature and intensity change is analogous to the temperature and intensity responses of the muscle end-plate potential (*8*) and of the generator potential of the pacinian corpuscle (*9*). Thus, the hypothetical cochlear excitatory process would respond to both cooling and reduction of stimulus intensity with a decrease in its amplitude (which would decrease the number of responding fibers and thereby reduce action-potential amplitude) and an increase in rise time (thus reaching firing threshold later and thereby increasing action-potential latency). Since temperature probably acts at a point subsequent to the production of the microphonic, the hypothetical excitatory process would have to occur after generation of the microphonic.

It must be noted that until the mechanism is known by which temperature and click intensity affect action-potential latency, the contention that these factors act at a common point must remain a speculation. However, the present observations have demonstrated that temperature and click intensity affect both action-potential latency and amplitude identically, while producing markedly different effects on the cochlear microphonic. Furthermore, when temperature is varied, a clear dissociation of microphonic amplitude and action-potential amplitude is demonstrated. These observations do not seem compatible with the idea that the cochlear microphonic provides a direct electrical stimulus to the auditory nerve endings.

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