Summating Potentials of the Cochlea

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ABSTRACT

DAVIS, HALLOWELL, BRUCI H. DEATHERAGE, DONALD H. ELDREDGE AND CATHERINE A. SMITH. Summating potentials of the cochlea. Am. J. Physiol. 195(2): 251-261. 1958.—When the ear is stimulated by a steady tone scala media and scala vestibuli become less positive electrically relative to scala tympani. This 'summating potential' (SP) is a d.c. change related to the root-mean-square of the acoustic pressure, integrated over one or two waves. It increases up to injurious sound pressure levels. It is increased by additional positive polarization of scala vestibuli or media. It is modified and may even be reversed in sign by hydrostatic displacement of the cochlear partition. The external hair cells produce CM and may also generate small SPs, usually positive in sign. The SP generated by 7000 cps tone bursts is strong in the basal turn while those by 2000 and 500 cps are very small here but are strong in the second and third turns, respectively. The theory is proposed that the negative SP is the response of the internal hair cells, an amplifier action intermediate between a mechanical detector action of the cochlear partition and the excitation of nerve impulses. Both CM and SP depend on bending of the 'hairs' of the sensory cells in the proper direction.
position of our hypotheses concerning SP than was appropriate in a review of the physiology of the entire inner ear as a whole.

**METHODS**

Nearly all of our observations have been made on guinea pigs anesthetized with Dial in urethane 0.50 cc/kg body weight. The operative procedures of exposing the cochlea of the guinea pig in the auditory bulla and inserting one intracochlear electrode (nicrome steel wire 30 μ in diameter) into scala vestibuli and another exactly opposite in tympani have been described adequately elsewhere (9). Such a pair of intracochlear electrodes may be placed in any of the four turns of the guinea pig cochlea.

Sometimes also a fenestra was made in the bone over the spiral ligament of the basal turn and a pipette electrode 25 μ down to less than 1 μ in diameter was inserted into scala media. The basic arrangement of electrodes and the approximate location and orientation of various potentials are shown in figure 1.

The electric potential differences were amplified by conventional condenser-coupled or d.c. amplifiers (Grass) and displayed on two twin-beam oscilloscopes. Differential (push-pull) amplifiers were employed throughout, and the rejection of unwanted signals was facilitated by an input network, shown in figure 2, that follows the cathode-follower preamplifiers. Here the variable resistances Rs and Rs are adjusted empirically to eliminate unwanted potentials relative to ground that are common to the two intracochlear electrodes. The chief unwanted potentials are the action potentials (AP) of the auditory nerve. For input to the second channel a point is chosen on the potentiometer R7 at which the potential between the two cochlear electrodes due to CM is equal to the corresponding potential at the distant reference electrode. The potential changes between this point on R7 (ideally the average of the potentials at the two cochlear electrodes) and the reference electrode are primarily the nerve potentials, although they are contaminated by potentials from the muscles of the neck. The separation of a mixed record like that in figure 3 into CM and SP in one trace and AP in another, as in figure 4, has been of great assistance in our analysis of SP.

Another important property of our paired intracochlear electrodes is that they are sensitive to differences of potential between scala vestibuli and tympani, but only to those that originate within a millimeter or two of the pair of electrodes. The CM and SP responses of other turns are not recorded due to the attenuation by electrical leakage through the cochlear partition (9, 11, 12). On the other hand the AP record includes the action potentials in nerve fibers from all parts of the cochlea.

Other features of our stimulating and recording devices are described elsewhere (9, 13). Our standard acoustic stimulus is a brief tone burst with rise and fall times of 1 msec. or three.
waves, whichever is longer. The intensity is maintained constant for a few milliseconds. This makes it easy to distinguish a shift in the base line (SP) from a neural response (AP). The use of the tone-burst controlled by an electronic gate instead of the briefer tone pip produced by transient excitation of a resonant circuit has been indispensable in the analysis of the summating potentials. Two important additional features of our gate are: a) a nearly linear opening and closing. This has been standardized at 1 msec. for both rise and fall. This duration gives a good compromise between well synchronized action potentials and a minimum scattering of acoustic energy above and below the nominal frequency. b) Opening always at the same phase of the modulated tone. This phase relation can be varied at will. This feature is essential for proper observation of the action potential responses to tone-bursts at or below 2000 cps in frequency.

Sometimes the tone bursts are delivered from a loud-speaker in a sound-treated room. The critical observations are made in the few milliseconds before ethos can return to the ear of the animal. Quantitative high-intensity stimulation is achieved by means of closed sound systems (4, 14). The frequency 7000 cps is used routinely in studies of the basal turn because it stimulates a region where it is convenient to insert the intracochlear electrodes and also is within the effective range of our calibrated transducer and coupler.

Properties of the Summating Potential.

Unless otherwise noted the following properties of SP refer to the potential recorded by intracochlear electrodes from the basal turn of the guinea pig in response to a 7000-tone burst with 1 msec. rise and fall time.

**Detector action.** SP is a d.c. response to an a.c. acoustic stimulus. The cochlea thus acts as a ‘detector.’ The name ‘summating potential’ was given to this response (1) because, by virtue of this detector action, it appeared to summate the energy of several high frequency sound waves in a form that would be an effective stimulus to the nerve. The voltage of SP is related to a running time integral of the auditory stimulus. The integration time is short: of the order of two wavelengths for a 7000-cps stimulus. The integration time and the decay time probably vary from one part of the cochlea to another, but have not been precisely determined.

**Location.** SP is best recorded between scala media and either a distant reference electrode or an electrode in scala tympani. With either reference electrode there is a large increase in SP when an exploring electrode enters scala media from the spiral ligament. The increase is greater than the concomitant increase in CM, but this does not prove that SP and CM arise from different structures or are differently oriented. The difference apparently depends on the lower impedance of Reissner’s membrane, etc. for the a.c. cochlear microphonic than for the d.c. summating potential.

**Polarity.** The polarity of SP is defined as the direction of change of potential in scala media (and scala vestibuli) relative to scala tympani. The usual polarity is negative, but under certain circumstances, particularly in fresh preparations and with weak stimuli, the polarity is positive. Also the summating potential recorded in the basal turn in response to a low frequency (e.g. 500 cps) is usually small but positive. Very typically the SP response to a 7000-cps tone burst starts in the positive direction during the rise time of the burst and then
FIG. 4. Cochlear microphonic and summating potential (upper traces) and action potential (lower traces) from basal turn in response to strong 7,000 cps tone bursts. The duration of the plateau of the tone burst for this and most of the subsequent oscillograms was 4 msec. The rise time was always 1 msec. The AP trace shows successive volleys of synchronized nerve impulses following the onset of the tone burst. The stimulus was 10 db stronger for the responses on the right. SP is larger but CM is slightly smaller, although the CM response to the echoes is larger. The first stimulus was maximal for CM, the second was supramaximal. From (8).

Swings negative as the stimulus reaches full strength (fig. 5, right). We shall speak of the negative summating potential (SP−) and the positive summating potential (SP+) as two separate superimposed phenomena, although it is probable that one class of the positive summating potential is generated in the same cells as the usual negative summating potential but in response to a mechanical force acting in the opposite direction.

Latency. The latency relative to CM is short but has not been measured precisely. With a very strong stimulus that is supra-maximal for CM the latency of SP− to a 7,000 cps tone burst is less than half a wavelength, i.e. certainly less than 0.1 msec. (We believe that CM is directly proportional to the amplitude of displacement of the cochlear partition, or, more precisely, to shearing displacement of the tectorial membrane relative to the reticular lamina and to have no latency relative to this movement within present limits of measurement (2).)

Threshold. SP has no true threshold like the action potential but, like CM, it continues to be directly proportional to the sound pressure level as the stimulus is reduced until the response becomes lost in the background noise. If we speak of a ‘threshold of SP’ it is a pseudo-threshold, i.e. the threshold of detection of SP in the oscilloscope trace. The sound pressure level necessary to produce a given small SP− voltage is nearly always at least 20 db above the level needed to evoke a CM of the same voltage (peak-to-peak). An exception is at the upper frequency limit, recording from round window, where the thresholds for CM, AP− and AP converge on the same value. Under certain abnormal conditions (anoxia, NaCN poisoning, streptomycin poisoning, degeneration following chronic venous obstruction) strong SP− responses have been observed below the threshold of detection of CM. The threshold of detection of SP− is actually lowered by a brief period of partial anoxia or mild surgical injury.

Relations to position and frequency. The magnitude of the summating potential is related to the position of the electrodes along the cochlea and also to the frequency of the stimulating sound. At the round window in the guinea pig, where CM and action potential responses up to 50,000 cps are obtained, strong SP− responses are regularly recorded as part of the normal pattern of response and without any intracochlear surgical trauma. As the upper frequency limit of response is approached SP− becomes more and more prominent relative to CM. In the second quarter of the basal turn there are strong SP− responses to frequencies from 4,000 cps to 16,000 cps, but 2,000 cps (or lower) tone bursts give little or no SP−. In fact at 500 cps the small SP response is usually positive. In the second turn, where the CM response begins to fall off considerably at about 3,000 cps, a clear SP− response to 2,000 cps appears at high intensities, and in the third turn SP− is clear (although never very large) at 500 cps.

Three generalizations emerge from these and
Fig. 5. Left: a pure positive summating potential and CM in response to a weak 8800 cps tone burst. Such responses are exceptional. Right: the response to a stimulus 20 db or stronger. Here the initial positive SP is soon over-balanced by a superimposed negative SP. Lower traces show AP. (This and subsequent oscillograms have been retouched for reproduction.)

Similar observations. First, the SP response is associated more closely with the position of maximum amplitude of displacement (i.e. the peak of the resonance curve of the cochlear partition) than is the cochlear microphonic. 2000 cps and 500 cps evoke strong CM from the first turn but very little if any SP. Apparently the SP— response is associated particularly with the part of the resonance pattern where amplitude is large but the wavelength of the traveling wave is becoming short and the phase differences are considerable.

Second, for electrodes at the best position for each frequency, the SP is more prominent relative to CM the higher the frequency. This relation depends in part on the lower and lower maximum CM voltages at higher frequencies and also on greater absolute values of SP—.

Third, the SP— for frequencies below 3000 cps may be so small, particularly in the first turn, that it is obscured by the peak limiting of CM which occurs for these frequencies. The peak limiting is unsymmetrical, a little more severe on the positive half of the wave than on the negative half, and thus obscures any slight shift of the baseline.

Resistance to anoxia, ionic changes, etc. SP— (for a 7000 cps burst) is very resistant to anoxia. In asphyxia it persists as long as CM and long after AP has disappeared. During the early stages of asphyxia or anoxia the SP— becomes more prominent relative to CM and may actually increase in voltage for a given intensity of tone burst. During death both CM and SP shrink and then very gradually disappear. At one stage in the sequence of mortal changes the polarity of SP in the first turn typically changes from negative to positive and then, at a later stage, it reverts to negative (5). At or near death the SP may be confused with or partly obscured by a partial or complete half wave rectification of CM produced by unsymmetrical peak limiting and, finally, complete suppression of the positive half of the CM response (3, 5).

It is easy to gain the impression that SP— is enhanced by severe anoxia because as AP and CM fail the experimenter is very likely to increase the strength of the stimulus to levels that he previously avoided for fear of injuring the cochlea. As noted above, SP— increases but CM decreases with increase of stimulus above 110 db. Therefore SP— increases rapidly relative to CM and dominates the total response.

SP— also appears to be favored by mild injury of other kinds, such as slight surgical injury to scala media, the mere insertion of a pipette through stria vascularis, or the injection of fluid that differs from endolymph in pH, in its concentration of Na, K, etc., or in containing a small amount of a drug of some sort. In some cases the absolute increase in SP— is perfectly clear at very moderate levels of stimulation. SP— may emerge at levels where previously none could be detected.

We have suggested elsewhere (2) that this paradoxical increase in SP— following anoxia or other mild injury is due to the elimination of a superimposed SP+ that is more vulnerable than SP—. Our various observations are still consistent with this hypothesis. Positive evidence in its favor is found in the shape of the onset of SP. In an ear in good condition the SP usually begins with a slight positive deflection. This suggests a more rapid rise in SP+ than SP—. Following mild injury the SP— begins abruptly and reaches a larger negative value. This change of shape may be reversible (fig. 6). Further evidence for the multiple character of SP is given below.

D.C. response to slow pressure changes. A slow
FIG. 6. SP and CM responses before, during and immediately after injection of quinine dihydrochloride (0.1%) in tyrode solution into scala tympani of the basal turn. Outflow was through a second hole at the apex. The small initial SP+ is abolished by the quinine. SP− apparently increases. CM is ultimately diminished.

(subsonic) or maintained displacement of the cochlear partition causes a change in the potential of the endolymphatic space of scala media relative to surrounding tissues. This d.c. response is obviously related both to SP and to CM. In these cases the cochlear partition all moves in phase or is statically displaced. There is no travelling wave pattern. Examples that we have studied experimentally are the movements produced by contraction of the intra-aural muscles, by increasing the air pressure in the external canal, or by injecting fluid into one or another scala. A general rule states that increase of relative pressure in scala vestibuli causes an increased electrical positivity in scala media and to a less degree in scala vestibuli. Movement in the opposite direction causes the opposite change, i.e. a decrease in positivity. The actual full sequence of events is more complicated. For example, when the intra-aural muscles contract the inward movement of the stapes is followed by equalization of pressure between scala vestibuli and scala tympani as perilymph flows through the helicotrema and then, after about a half a second of maintained contraction, the pressure in scala vestibuli falls during relaxation of the muscles and then equalizes once more. It is during the dynamic phase of relatively increased pressure in scala vestibuli that scala media and scala vestibuli become more positive electrically relative to scala tympani. During the phase of reduced pressure in scala vestibuli they become less positive. This change of potential may be as much as 5 or 10 mv. The direction of change correlates with direction of displacement of the basilar membrane, but not with the direction of movement of Reissner’s membrane. The last point is proved by injecting fluid into scala media (16).

It is no longer satisfactory to explain the slow changes in potential described above by simply stating that the resting positive polarization of scala media is “pressure sensitive.” The endocochlear potential is now known to depend on the stria vascularis, not on the organ of Corti (16, 17, 17a) and it is difficult to imagine how a generator located in the stria vascularis would be directly sensitive to hydrostatic pressure. The slow changes in potential seem to be analogous to the changes in potential that we have observed in the ampullae of the semicircular canals of pigeons in response to angular acceleration, to caloric stimulation and to very strong acoustic stimulation, and also to responses of the lateral line organ of fish (18). Less directly they seem analogous also to the change in polarization of a crustacean sensory neurone associated with a change of mechanical stretch (19). A common feature of the observations on the ampulla and Békésy’s (20) experiments with a vibrating electrode seems to be that bending the hairs of a hair cell in the proper direction causes a change in the electric potential gradients in the immediate surroundings of the hair cell. We believe that CM is caused by a to-and-fro bending of the hairs.

What must be explained in order to account for SP on a similar basis is how a unidirectional bending of hairs can be produced by an alternating sound pressure.

**Electrical polarization.** Following the method of Tasaki and Fernández (21) nonpolarizable Cu-CuSO₄ electrodes were connected by Ringer-soaked wicks and Ringer-filled pipettes to holes in scala tympani and scala vestibuli in the first turn about one-half millimeter from the recording electrodes. The polarizing current applied through these electrodes was measured in microamperes. Responses were recorded from a pair of nichrome steel electrodes in their usual small holes nearby. The SP− evoked by
a 7000-cps tone burst at 120 db SPL was increased when scala vestibuli was made more positive and decreased when it was made more negative by the polarizing current (fig. 7). Similar changes in CM have already been described by Tasaki and Fernández (21). These were confirmed in the present observation. Thus another point of similarity between SP and CM is established.

With 7000-cps tone bursts as stimuli the enhancement of CM and SP— is clear both at 80 db SPL (submaximal CM) and at 100 db SPL (maximal CM). With a 500-cps test tone that is strong enough to produce peak limiting in the CM response, other and more complicated effects of polarization appear which we have not yet fully investigated.

Reversal of polarity of SP. During certain phases of pressure change and slow movement of the cochlear partition the polarity of an evoked SP response may shift momentarily from negative to positive. The reversal was first observed during the spontaneous periodic contractions and relaxations of the intra-aural muscles of the guinea pig. We have reproduced the reversal by injecting fluid rapidly into scala tympani of the first turn with only a small outflow opening at the helicotrema. (figs. 8, 9). During the period of static displacement of the cochlear partition toward scala vestibuli the SP response to a 7000-cps tone burst becomes positive. (The positive polarity of the SP response during the displacement must not be confused with the reduction of the positive endocochlear potential that is caused by the displacement itself.) In any case the reversal of polarity of SP closely links the mechanism of SP to mechanical movements of the cochlear partition.

A minor effect that suggests a mechanical readjustment following stress is an exaggeration of SP— for two or three seconds immediately after SP has been reversed by blowing fluid into scala tympani. There seems to be an overshoot of some sort after the pressure is released and then an asymptotic return to the original condition.

Remote masking. Still another phenomenon that links the summating potential closely to mechanical movements is the 'remote response' which is the physiological basis of 'remote masking,' i.e. the masking of a low tone by a modulated high tone or by a high frequency band of noise (22). A 7000-cps tone burst, with a rise time of about 1 msec., at a sound pressure level of 100 db or more generates in the cochlea a slow pressure wave, with a rise time of 1 msec. at the onset and again at the end of the burst. These waves evoke cochlear microphonic responses in the third turn of the cochlea, which of course is not affected directly by the 7000 cps carrier frequency. The CM in the third turn is merely two transients of about one cycle each and is not sustained throughout the burst like the SP in the first turn. The on and the off remote CM responses in the third turn follow the beginning and the end of the SP in the first turn with a delay of about half a millisecond, which is an appropriate delay for a traveling wave of this duration (9) set up by a mechanical movement in the first turn. The slow mechanical waves are associated with changes in SP. They seem to be another expression of the mechanical 'detector' or demodulation action that we have postulated and which evidently takes place in the section of the cochlear partition that is tuned to the frequency of the 7000 cps carrier wave. We believe that SP is the electrical sign of this mechanical detector action.
FIG. 8. Reversal of summating potential by static pressure in scala tympani. The pressure was produced by injecting tyrode solution more or less vigorously into a hole in scala tympani in the basal turn. An outflow hole was at the apex. Lower trace of each pair is AP. The smaller .4P and CM in the bottom pair (strong injection) suggests that in this case sound transmission was impaired by the high intracochlear pressure, probably by stiffening of the oval window. With weak pressure the changes in CM and AP are slight but the composite, dominantly negative SP is converted to a purely positive response. The stimuli throughout are 7000 cps bursts at 100 db re 0.0002 μbar.

SP itself must not be confused with the remote response. SP is a local electrical response: the remote response represents a mechanical wave transmitted toward the apex. The remote response is a transient, related to the beginning and the end of SP. SP itself continues without fatigue or adaptation as long as the stimulus endures.

Generator. The chief generator of the summating potential we now believe to be the internal hair cells. Injury of external hair cells with preservation of internal hair cells was produced in several guinea pigs by injection of streptomycin into the middle ear (8). The streptomycin was apparently absorbed through the round window and caused selective injury of hair cells, most severe in the basal turn. The electrical responses of the cochlea were measured after an interval of 4–7 days. Whenever the external hair cells were absent or obviously injured, as judged by subsequent microscopic examination, CM was absent or was reduced to a few microvolts. SP was absent also when, but only when, the internal hair cells were also severely injured, but was only moderately reduced in several ears in which the internal hair cells remained intact. The number of animals was small, but the correlation with the appearance of the internal hair cells was much better than with any other class of cells in the scala media. These observations do not, however, rule out the possibility that the external cells may also contribute some part of the summating potential.

Input-output relationships. The relations of the output voltage of SP to the sound pressure level of the tone burst are greatly complicated by the dual nature of SP. The observed output voltage is apparently the difference between \( \text{SP}^- \) and \( \text{SP}^+ \). The two responses have different mechanical antecedents and usually originate in different hair cells. \( \text{SP}^- \), \( \text{SP}^+ \) or both may be altered by static displacement of the cochlear partition. They are apparently affected differently by anoxia and chemical agents (10).

The most stable, reproducible and largest \( \text{SP}^- \) responses are produced by using strong stimuli, greater than 90 db SPL, or by causing some mild injury that apparently eliminates or greatly depresses \( \text{SP}^+ \). It seems that the \( \text{SP}^- \) and \( \text{SP}^+ \) responses to weak and moderate stimuli are nearly equal and opposite, but at high sound levels \( \text{SP}^- \) increases more rapidly than \( \text{SP}^+ \) and completely dominates the combined output. In a good fresh preparation \( \text{SP}^+ \) may dominate at low sound levels, as in figure 5.

In figure 10 is shown an input-output curve obtained from an ear that had been subjected to streptomycin injury. It is selected to illustrate the simple, lawful relationships that sometimes appear. We interpret as a 'pure \( \text{SP}^- \)' response, uncomplicated by any significant amount of \( \text{SP}^+ \). We suppose that the

\[ \text{SP}^- \]
generator calls for SP+ have been injured by the streptomycin but that those for SP− and the other structures of the cochlear partition remain intact.

Three general features of the input-output curve are evident here and also appear, more or less overlaid by the SP+ response, in our other input-output curves. First: at low sound pressure levels SP−, like CM, is a linear function of sound pressure level. Second: below 100 db the voltage of SP− is normally less than the peak-to-peak voltage of CM. Third: at high sound pressure levels SP− does not go through a maximum like CM but continues to increase up to at least 130 db SPL according to some power function, usually about the square root or the cube root, of the sound pressure level. Above 110 db the voltage of SP− (in response to a 7000-cps tone burst) is greater than that of CM.

**DISCUSSION**

The chief properties of the summating potentials have been outlined in the foregoing section. The presentation includes the major items of our present interpretation, many of which have already been stated elsewhere (2). Our present views are as follows: a) The cochlear microphonic (CM) is caused by an alternating bending (or shearing displacement) of the hairs of certain hair cells in the appropriate direction. The bending or displacement is produced by shearing forces between the tectorial membrane and the reticular lamina. The energy of the cochlear microphonic is derived from the polarization potential across the interface between the endolymph and the interior of the hair cells. The movements of the hairs somehow modulate the flow of electric current.

b) The summating potentials (SP− and SP+) are produced by exactly the same mechanism except that the bending or displacement of the hairs is a steady one-way (d.c.) bend or shift instead of a more or less symmetrical alternating (a.c.) movement. The d.c. character of SP depends on the series of mechanical events that precedes the bending of the hairs. SP− and SP+ mean that scala media has become more negative or more positive, respectively, relative to surrounding structures.

c) SP− is generated only near the region of maximum amplitude of displacement of the cochlear partition.

d) SP is a normal physiological response of hair cells. Presumably SP− excites the nerve endings, directly or indirectly, just as the scala-media-negative phase of CM is believed to do.

e) SP− continues to increase at sound levels far above the maximum for CM. It thus extends the dynamic range of the ear.

f) SP− is generated primarily by the internal hair cells. If there is a component, SP−, that is generated by external hair cells it has not been identified.

g) SP− is the primary mode of response of the internal hair cells. If these cells generate a cochlear microphonic it has not been identified.
FIG. 10. Input-output curves for cochlear microphonic (CM), summating potential (SP), and initial action potential (AP) in response to 7000 cps tone bursts in an ear slightly injured by streptomycin. The drug had been injected into the middle ear 5 days previously. The maximum CM is about 30% of normal, AP is about 70% of normal, SP is fully normal, d.c. (endocochlear potential) is normal. The shapes of all three curves are normal but the maximum CM output is reached 10 db below the normal sound pressure level, and the linear portion of the CM curve lies 7 db to the right of normal. Reference level for input is 0.0002 μ bar. From (10).

The inner-hair-cell SP−i can be reduced, and even reversed to SP+i, by static displacement of the basilar membrane toward Scala vestibuli.

h) The external hair cells generate CM. They also generate a positive summating potential, SP+i. At low levels SP+i partly or even completely obscures SP−i, but at high levels SP+i is always dominant.

i) The external hair cells are in general more sensitive to anoxia, to ionic changes and to drugs, than the internal hair cells. This explains the relative vulnerability of CM and SP+ and the apparent enhancement of SP− by mild injury.

j) The mechanical detector action that is responsible for SP− is tentatively attributed (2) to a one-way apical movement of the tectorial membrane relative to the organ of Corti produced by the same forces that generate the eddies (11, 15, 20) in the perilymph. However, this is not the only possible mechanism for a detector action.

In one respect the interpretation given above differs from the theory recently published by one of us (2). The earlier version attributed SP− to the external hair cells. Since that article went to press the evidence from acute venous congestion and from streptomycin poisoning has shown clearly the correlation between CM and the external hair cells and (somewhat less clearly) between SP− and the internal hair cells.

Our present assignment of SP− to the internal hair cells is in agreement with the observations by Békésy (20) on the optimum direction of movement for eliciting an electric response from the organ of Corti. Near the limbus the optimum direction is perpendicular to the fibers of the tectorial membrane and nearly longitudinal along the basilar membrane. At the outer edge it is parallel to the fibers of the tectorial membrane and nearly transverse to the basilar membrane. The chief discrepancy that now appears is between our conclusion that the external hair cells are more sensitive to anoxia and Békésy’s (20) clear statement that the internal hair cells are the more vulnerable. For the present this contradiction stands unresolved.

The new insights into the origin and significance of the summating potential do not assist in deciding whether CM and SP− stimulate nerve endings and/or the nerve fibers directly (2) or whether there is an intervening step of chemical mediation. They merely leave us with a rather long and unexplained latency between CM and SP− as electrical stimuli and the appearance of the action potential spike in the auditory nerve. When we imagine a strong SP− acting on the radial fibers to the internal hair cells it is rather difficult to attribute the latency entirely to ‘slow conduction in the nonmedullated terminal twigs.’ SP is generated by the hair cells but it must be enhanced by the positive endocochlear potential of the stria vascularis just as it is by artificial hyperpolarization. The experiments with streptomycin and with chronic obstruction of the inferior cochlear vein show clearly that the resting endocochlear potential (about +80 mv) is generated by the stria vascularis and not by the organ of Corti. Both cochlear microphonic...
and summing potential were sometimes present when the endocochlear potential was much reduced, provided the hair cells were still intact, but they were reduced to about half their normal magnitude when stria vascularis was injured and the endocochlear potential was much reduced. In utricle, saccule and semicircular canals the positive resting potential is normally not more than 5 mv (23). On the other hand, we have recorded both microphonics and summing potentials from these structures. The positive endocochlear potential generated by the stria vascularis, may enhance both the cochlear microphonic and summing potential responses like artificial electrical polarization, but it is not a necessary condition for these responses or for the excitation of nerve impulses by hair cells.

SP− now appears to be an electrical link, like CM, in the sequence of normal response. As yet we have no evidence as to the possible function of SP+. By analogy we should expect it to represent an inhibitory or moderating influence.

The present interpretation of cochlear action makes a clear separation between the internal hair cell mechanism and the external hair cell mechanism. Studies with the electron microscope have shown important differences in ultrastructure between the two types of sensory cell (24). The former is less sensitive, more rugged, and depends on a detector action that seems to be related somehow to longitudinal waves and longitudinal movements of the cochlear membranes. Its localization with respect to frequency is sharper. The external hair cell mechanism is more sensitive to weak stimuli, but is restricted in its output at high levels. It seems to be associated directly with transverse bending of the basilar membrane according to the classical concepts.

REFERENCES