Summating Potential with Electrical Stimulation of Crossed Olivocochlear Bundles

Abstract. *Electrical stimulation of crossed olivocochlear bundles resulted in increase of the positive summating potential in response to high-frequency tone bursts when the summating potential was recorded in the basal turn of the cochlea in anesthetized and immobilized guinea pigs. Strychnine locally applied suppressed the changes of the summating potential.*

Electrical stimulation of the crossed olivocochlear bundles (COCB) brings about inhibition of the action potential of the auditory nerve (AP) (*1*) and augmentation of cochlear microphonics (CM) (*2*). The modifications of CM and AP are dependent on the frequency and intensity of the acoustic stimuli (*3*). Fex reported that the endocochlear potential decreased when the COCB was stimulated (*4*). He called this change the crossed olivocochlear potential (COCP) and interpreted it as the postsynaptic activity of the hair cells elicited by COCB stimulation. To date relatively little is know about the effect of stimulation of COCB on the summating potential (SP)—the direct current response (positive or negative) of the hair cells elicited by tonal stimuli (*5*). The positive SP may play an important role in the transition from the broad area of the basilar membrane excursion in response to tonal stimuli to the much more restricted tuning curves exhibited by the first-order neuron (*6*). That is, the positive SP may exert an inhibitory influence on neural units. Indeed, the positive SP may be the agent through which COCB stimulation inhibits AP. If this is so, COCB stimulation should enhance the positive SP. The present experiment was designed to investigate this possibility.

Guinea pigs were anesthetized with pentobarbital sodium; CM, SP, and AP were recorded in the basal turn of the cochlea by means of the differential electrode technique (*7*). The endocochlear potential was measured by a glass capillary electrode inserted into the scala media of the basal turn through the spiral ligament (*8*). A direct-coupled amplifier was used to display changes in the endocochlear potential, while the sound-evoked responses were amplified by an a-c coupled amplifier. Since its overall time constant was 6 msec, the shape of SP was slightly distorted. The magnitude of SP was measured by the shift of the midpoint of CM from the base line 2 msec after the onset of CM. The brainstem was exposed by partial suction of overlying cerebellum, and bipolar stimulating electrodes were placed in the midline of the brainstem at the level of the facial genu (*9*). The electrical stimulus consisted of a train of shocks, and the tone burst was delivered after the end of the COCB stimulation. The middle ear muscles were sectioned; animals were immobilized with gallamine triethiodide and respired artificially during the experiment.

When a tone burst higher than 5 khz at moderate intensity was preceded by the optimum COCB stimulation, we noticed that the inhibition of AP was not accompanied by the augmentation of CM but by changes in SP. Specifically, the modifications of SP and inhibition of AP were related to the parameters of COCB stimuli and the interval between COCB stimuli and tone bursts. Figure 1 illustrates these typical changes under various COCB stimulus conditions; Fig. 1A is a control response without COCB stimulation and Fig. 1B shows the changes in responses when the optimum COCB stimuli were followed by the tone burst 10 msec later. The SP became positive and AP was inhibited. There was no noticeable augmentation of CM. It should be noted that the tone burst was delivered at the moment when the crossed olivocochlear potential was near its maximum. Comparison of B and C of Fig. 1 shows that the longer the interval between COCB stimuli and tone burst, the smaller the change in SP and the less the inhibitions of AP. When the parameters were such that COCB stimulus was less effective in inhibiting AP, the changes in SP became small and COCP was reduced in magnitude (Fig. 1D). As shown in these photographs, it is apparent that inhibition of AP produced by COCB stimulations is associated with modifications of SP.

The modification of SP with COCB stimulation was also a function of both frequency and intensity of tonal stimulus. As shown in Fig. 2A, the CM response to 6 khz did not show augmentation in the linear portion of the input-output function, while the inhibition of AP was predominant at low intensity range. In this particular case, when the tonal intensity was increased, the SP in the control changed its polarity from slightly positive to strongly negative. When the tonal stimulus was preceded by COCB stimulation, however, the positive SP persisted until about 120 db sound pressure level. Further increase of intensity of tone bursts resulted in a decrease of the negative SP with COCB stimulation. The differences in magnitude of SP shown in Fig. 2B are plotted as a function of the sound intensity (Fig. 2C). From comparison of A and C, it appears that the inhibition of AP to 6 khz is related to the changes in SP rather than those in CM. With the differential elec-

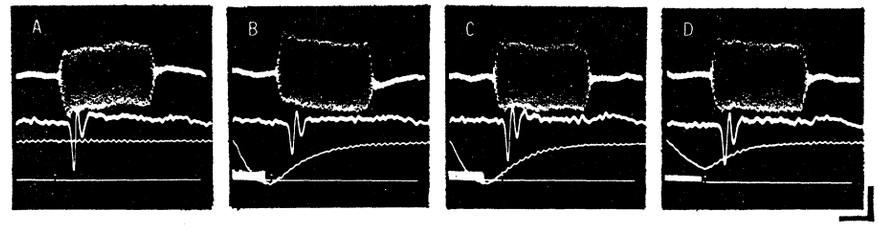

Fig. 1. Changes in sound-evoked responses and the d-c slow negative potential in the scala media [crossed olivocochlear potential (COCP)] produced by electrical stimulation of crossed olivocochlear bundles (COCB). In each record, the uppermost trace shows cochlear microphonics (CM) and summating potential (SP); the second trace, the whole-nerve action potential of the auditory nerve (AP); the third trace, the crossed olivocochlear potential; and the bottom trace shows markers indicating the COCB stimulation and a trigger pulse for the tone burst. The upper two traces are triggered by a single pulse shown in the bottom trace to expand the time scale. The horizontal scale at the right bottom corner is 4 msec for CM and AP and 100 msec for COCP and markers. The vertical scale is 200 μv for CM and AP, 5 mv for COCP. (A) Without COCB stimulation; (B and C) frequency of COCB stimulus is 400 per second and the interval between COCB stimulus and tone burst is 10 and 50 msec, respectively; and (D) frequency of COCB, 200 per second; the interval, 10 msec. Acoustic stimulus, 6-khz tone burst, 40 db above the AP threshold.

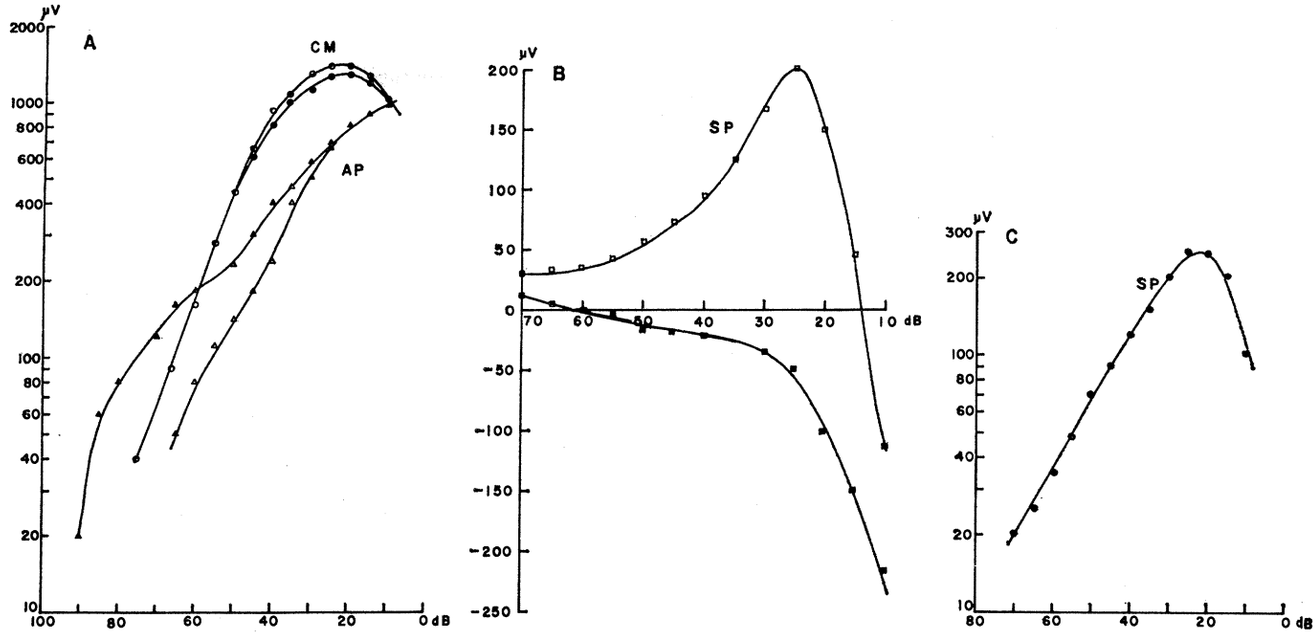

Fig. 2. One example of the input-output function of CM, AP (A), and SP (B) with and without COCB stimulation. Filled circles (CM), filled triangles (AP), and filled squares (SP) are without COCB stimulation and open symbols are with COCB stimulation. The abscissa shows the readings of attenuator (100 db corresponds to 36 db sound pressure level). The ordinate indicates the magnitude of CM or AP in (A) and SP in (B). Acoustic stimuli, 6-khz tone bursts. (C) Differences in magnitude of SP with COCB stimulation as a function of sound intensity. The ordinate indicates vertical deviation between control and COCB stimulation in (B).

trodes in the basal turn, the changes in the SP with COCB stimulation could be detected at frequencies higher than 5 khz.

To ensure that the above-mentioned changes in SP were elicited by COCB stimulation, scala tympani was perfused with artificial perilymph containing 5 μM strychnine. Strychnine has been shown to abolish action of COCB (*10*). This perfusion did not alter CM, SP, AP, or endocochlear potential, but abolished modification of the SP by COCB stimulation within 10 minutes. Washing of strychnine with artificial perilymph was in most cases effective in restoring the inhibitory action of COCB. Little or no modification of SP was produced by COCB stimulation in guinea pigs in which COCB had been previously sectioned surgically 7 to 10 days prior to testing. Thus the modifications of SP could be attributed to the effect of COCB stimulation.

A paradox is immediately apparent when one observes that the inhibitory effect of COCB stimulation on AP becomes progressively less as the positive SP increases. Indeed no inhibition in AP is seen at maximum value of the positive SP (Fig. 2, A and C). To explain this inconsistency, we suggest that the influence of COCB is restricted to the external hair cells. Available anatomical data support this position (*11*). We propose further that the positive SP brought about by COCB stimulation serves to inhibit the firing of afferent fibers associated with the external hair cells. The inhibition of AP is more evident at low acoustic intensity and it is precisely at the low intensities that the external hair cell activity is thought to predominate. As the sound intensity level is increased, the positive SP also becomes larger. Its inhibitory effect, however, is more and more offset by the increment in internal hair cell activity and associated afferent nerve fibers. This appears to be a reasonable assumption, since the internal hair cells are considered to come into play at higher stimulus intensities and their innervation density far exceeds that of the external hair cells (*12*). Moreover, they do not appear to be under control of COCB (*10*). Therefore, at high stimulus intensity level, the AP is almost completely composed of firings from the nerve fibers originating from the internal hair cells, and the inhibitory effect on AP through COCB stimulation is negligible, since the contribution of the afferent fibers associated with the external hair cells is relatively small.

Tasaki (*13*) reported that nerve impulses of the auditory nerve fibers are initiated during the negative phase of CM (the sign of CM refers to the phase of CM in the scala media with respect to the neck). The positive SP with COCB stimulation can be interpreted as a result of suppression of the negative (excitatory) phase of CM and thus the positive SP can be classified as a receptor potential with inhibitory effect on the afferent nerve fibers. The appearance of the positive SP elicited by COCB stimulation plays a major role in inhibition of AP.

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