the Western Desert. Each calcrete layer formed at the end of an aggradational phase, either by evaporation from a capillary fringe or by pedogenic processes. In either case, the climate was characterized by more effective moisture than it is today.

Eventually Wadi Tushka became fluvially inactive and filled with aeolian sand during Late Pleistocene time. During the Holocene pluvial event, interdunal depressions along the former watercourse held playa lakes around which prehistoric people lived between 9350 ± 400 ^{14}C years B.P. (UCR-831) and 4530 ± 100 years B.P. (SMU-746) (*22*). Wind erosion has removed part of these deposits over the past 4000 or 5000 years, and today the movement of heavy construction equipment has obliterated most if not all surface archeological sites along the canal area.

Ancestral Wadi Tushka is real, but its origin is uncertain. Further testing of the hypotheses presented here will depend upon continued investigations like those in progress, both geological and archeological, to establish a paleoclimate chronology for the Western Desert. The outcrops are limited, the area is vast, and water is scarce, but it is today one of the least explored areas remaining on the earth.

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23. This work was supported by grants from the National Geographic Society, grant EAR-77-10109 from the National Science Foundation, and Smithsonian Foreign Currency Program grants FC 80140100, 90247700, and 90878700; this research is a part of the Combined Prehistoric Expedition of Southern Methodist University, the Institute for the History of Material Culture of the Polish Academy of Sciences, and the Geological Survey of Egypt. Additional assistance was provided by the American Research Center, Cairo; D. L. Johnson, University of Illinois; P. J. Mehringer, Jr., Washington State University; and B. Issawi and El S. Zaghloug, Geological Survey of Egypt. Borehole data and permission to visit the Sadat Canal excavations were provided by engineers A. Hassanein and K. Amin, High Dam Authority. Engineers F. Takla, R. Saad, and G. R. Kishk, Behera Construction Company, provided for our needs at the canal site.

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Changes in Endolymphatic Potential and Crossed Olivocochlear Bundle Stimulation Alter Cochlear Mechanics

Abstract. *Mechanical nonlinearity in the cochlea produces acoustic distortion products that can be measured in the ear canal. These distortion products can be altered by changes in the endolymphatic potential as well as by stimulation of the crossed olivocochlear bundle, which provides efferent innervation to cochlear hair cells.*

The origin of the exquisite sensitivity and frequency selectivity of mammalian auditory nerve fibers has been a topic of great interest in auditory physiology. A number of experimental manipulations (cochlear efferent stimulation, anoxia, drugs, loss of outer hair cells, and changes in perilymph composition) have been shown to alter the sensitivity and selectivity of auditory nerve fibers (*1, 2*). These changes may be the result of changes in cochlear mechanics, since Rhode has shown that the tuning of the basilar membrane is physiologically vulnerable (*3*).

Within the past few years, considerable attention has been drawn to nonlinearities in both auditory nerve responses and cochlear mechanics that seem to be intimately associated with normal cochlear function (*3–5*). Mechanical nonlinearity in the cochlea will distort the incoming sound waveform, and the resulting distortion products will propagate along the length of the cochlea. The finding that the mechanically propagated distortion products measured in the ear canal and in the response of auditory nerve fibers are altered by hair cell damage and fatigue suggests that the hair cells play an important role in cochlear mechanics (*5*). I have shown that the generation of propagating distortion products as measured with the cochlear microphonic depends on the electrical potential (EP) of the endolymph (*6*) bathing the hair cell cilia (*7*). This potential is normally positive 80 to 90 mV with respect to the extracellular fluid at the bottom of the hair cells.

The purposes of the experiments reported here are (i) to confirm that the effect of EP is indeed mechanical and (ii) to test the hypothesis that the effect is mediated by the outer hair cells.

Pigmented guinea pigs (200 to 700 g) were anesthetized with urethane (1.4 mg per gram of body weight) supplemented with ketamine as needed. The bulla was exposed and opened through the use of a ventrolateral approach. For experiments in which the EP was altered, a small hole was made in the cochlea over the stria vascularis of the first turn and a 5- to 8-μm KCl-filled pipette was inserted into the scala media. An optically coupled

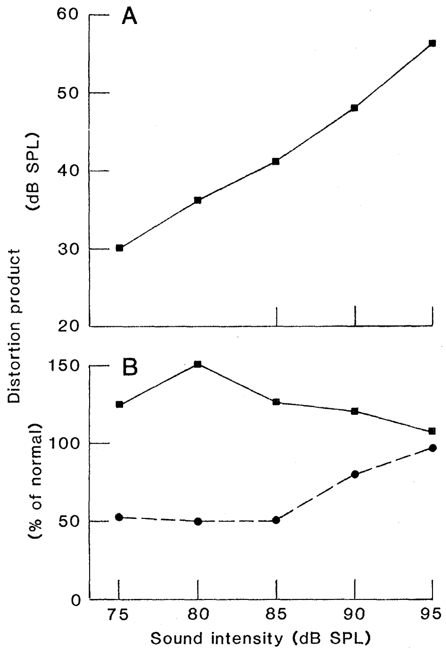

Fig. 1. (A) The distortion product at the difference frequency (700 Hz) measured in the ear canal as a function of the stimulus intensity. The primary frequencies were 10.0 and 9.3 kHz and were of equal intensity. (B) Sensitivity of distortion to changes in EP as a function of sound intensity. These current levels (± 10 μA) would be expected to cause EP changes of 40 to 50 mV (*12*).

constant current source was used to pass current through the pipette. For experiments in which the crossed olivocochlear bundle (COCB) was stimulated, the animals were paralyzed with gallamine triethiodide (Flaxedil) to prevent middle-ear muscle contraction, and the cerebellum was aspirated to expose the floor of the fourth ventricle. The COCB was stimulated where it crosses the midline of the ventricle with a concentric biopolar electrode. Stimulation consisted of 0.1- to 0.2-msec positive pulses with a repetition rate of 100 to 400 per second. The acoustic system consisted of a pair of earphones (Telex) connected by means of a Y-tube to a hollow ear bar. The distortion of the acoustic system, when measured coupled to a small plastic cavity, was below the noise level (equivalent to 20 dB sound pressure level) of the instrumentation system for all acoustic stimulus intensities used. A probe tube connected to a 1/2-inch microphone (Bruel and Kjaer) was inserted into the ear bar with the probe tip approximately 3 mm from the tympanic membrane. The amplified output of the microphone was measured either with a wave analyzer (General Radio) or by sampling the signal with an analog-to-digital converter and using signal averaging followed by Fourier analysis.

When a pair of tones is presented to the ear, a distortion product at a frequency equal to the difference in the frequencies of the primary tones can be measured in the ear canal. In these experiments, primary frequencies were chosen which produced large distortion products. Figure 1A shows the amplitude of such a distortion product as a function of the intensity of the two primary tones. If the EP is altered by passing electrical current through the cochlea in the region of maximum response to the primaries, the amplitude of the distortion product is changed (Fig. 1B). If the change in EP is positive the distortion increases (solid line), and if the EP change is negative it decreases (broken line). The percentage change is greatest at lower intensities of the acoustic stimulus. These results are similar to those reported previously for the intracochlear distortion measured with the cochlear microphonic (*6*).

Stimulation of the COCB alters the distortion much as decrease in EP does (Fig. 2). The change is always to decrease the distortion, and the percentage change is greatest at lower intensity levels of the primary tones. Stimulation of other regions of the fourth ventricle floor has no effect. Stimulation of the COCB decreases the EP by a few millivolts (*8*), but this is much less than the EP change required for the results shown in Fig. 1B. It therefore seems unlikely that the EP is directly responsible for the changes observed in the cochlear distortion. The large change in distortion caused by efferent stimulation indicates that the hair cell membrane potential may be important. A decrease in EP will depolarize the apical membrane of the hair cell and will hyperpolarize the basal and lateral portions of the hair cell membrane. Because the COCB stimulation will hyperpolarize all portions of the hair cell membrane, the basal or lateral portions of the hair cell membrane (or both) seem to be important in influencing cochlear mechanics. Flock has proposed that the hair cell stereocilia may have an important effect on cochlear mechanics since they contain the contractile protein actin (*9*). If the effects on cochlear mechanics mentioned here are mediated by the cilia, a "second messenger" would be necessary for the membrane potential of the basal or lateral hair cell membrane to influence the cilia at the apex of the cell.

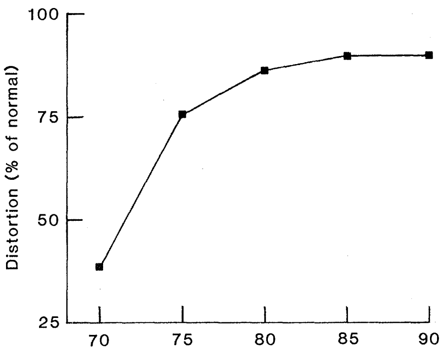

Fig. 2. Sensitivity of the distortion product to efferent stimulation as a function of sound intensity. The primary frequencies were 8.0 and 8.7 kHz and were of equal intensity. Efferent stimulation consisted of 0.2-msec pulses at a rate of 200 per second.

The finding that the stimulation of cochlear efferents brings about mechanical changes in the cochlea may explain the puzzling finding that stimulation of the efferents, which primarily innervate outer hair cells, alter the tuning of auditory nerve fibers that primarily innervate inner hair cells (*1*, *10*). If the hair cell membrane potential influences mechanics, the receptor potential of the outer hair cells would be expected to alter cochlear mechanics; such an alteration may account for some of the nonlinear behavior observed in the cochlea (*11*). In addition, anoxia, as well as changes in perilymph composition, presumably would alter the membrane potential of the outer hair cell, the ionic concentration, or both, which in turn could alter the mechanics of the cochlea.

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