Reprint from THE LARYNGOSCOPE, Vol. LXXXIV, No. 11, pp. 1887-1893, November, 1974.

SYMPOSIUM ON COCHLEAR IMPLANTS.

II. FEASIBILITY OF MULTICHANNEL SCALA TYMPANI STIMULATION.*†

MICHAEL M. MERZENICH, Ph.D.,
DAVID N. SCHINDLER, M.D.,
and
MARK W. WHITE,
San Francisco, Calif.

We review herewith some of the progress that has been made in studies directed toward development of an electrical stimulation prosthesis for treatment of the sensory deaf.

Before proceeding to discussion of the experiments that are the subject of this paper, it is useful to review what is known about electrical stimulation devices. Without such a review, it is not really very obvious why these particular experiments have been conducted.

As we all recognize, the people that we are trying to help are the 200,000 to 300,000 profoundly deaf individuals in the United States — individuals who cannot gain any sound input from any conventional hearing aid. The acoustic nerve is at least partially intact in the majority of these individuals.

Given the survival of the acoustic nerve, it can be stimulated. It has been known for many years that sound sensation is evoked by its direct stimulation. The basis of any acoustic prosthesis is that one can electrically generate a message to the brain that, by virtue of the central connections of the acoustic nerve, will produce the sensation of sound. The reasoning behind current efforts to develop acoustic prostheses is that one can generate some facsimile of the normal input that the acoustic nerve receives when stimulated with normal sounds, and thereby generate sensation that constitutes a facsimile of normal sound sensation. That is our basic *modus operandi, i.e.,* to attempt to generate an input to the brain that is a facsimile of the input generated by corresponding sound.

Psychophysical experiments conducted with the patients of Simmons[1] and Michelson[2,3] have revealed a great deal about the nature of the sensation evoked by direct excitation of the acoustic nerve with simple bipolar electrodes. We understand that tonal sensation can be evoked by

*Presented at the Meeting of the Western Section of the American Laryngological, Rhinological and Otological Society, Inc., San Diego, Calif., January 25, 1974.

†This work was supported by the John C. and Edw. Coleman Memorial Fund.

low frequency periodic electrical stimulation and that there is some discriminative input across the lowest frequencies; but that with stimulation above 500-600 Hz, there is little or no discrimination of electrical stimulus frequency. This is the most important single result of these psychophysical experiments.

Physiological experiments have revealed (on the first level) the mechanism of generation and encoding of sound sensation evoked by electrical stimulation. The experiments of Moxon and Kiang[4] and Simmons and Glattke,[5,6] as well as those in our own laboratory,[3,7] have given us information necessary to understand how it is that sounds heard by these patients are generated and how, in general terms, they are encoded. Thus, we have an understanding both of the nature of the sound sensation evoked by these simple bipolar electrodes, as well as an understanding of how this sound sensation is encoded in the auditory nervous system.

It is clear from studies of implanted patients that substantial benefit can be gained by the totally deaf from even the very limited sound sensation generated with a single bipolar stimulating electrode. This is beyond dispute.

It is also equally clear that with a single electrode stimulating the acoustic nerve one can never generate the neural input necessary for the direct encoding of speech. First, only periodicity and intensity information can be generated with a single bipolar electrode. No "place" coding of frequency is possible. Only one information stream can be delivered to the brain because there is only one electrode. Imagine the analogy of the cochlea as representing the frequency spectrum as does the keyboard of a piano. The frequency spectrum is represented across a series of discrete elements, the piano keys, just as it is across a series of discrete elements in the cochlea, the acoustic nerve fibers of the spiral nerve fiber array. Given a simple electrical stimulation device, one can effect changes only in the *force* and *time order* at which (in the analogy to the piano) one can depress the keys. Any stimulus constitutes a depression of a series of adjacent keys; the *same* keys are depressed regardless of the stimulus.

Second, with only this periodicity information available, there is a significant capacity to distinguish between small frequency differences across the lowest frequency ranges, but no ability to discriminate between differences at higher frequencies, *i.e.*, across the speech frequency range. Just when we get up to the frequency range where it is crucial to provide discriminative input necessary for the hearing of intelligible speech, electrical stimulation with a single channel fails to deliver it. This is a fundamental and everlasting limitation of these devices.

Third, with any single bipolar electrode, there can be no simultaneous representation of different frequency components of sound. Let us imagine our analogy again — imagine music played to the cochlea or on the piano.

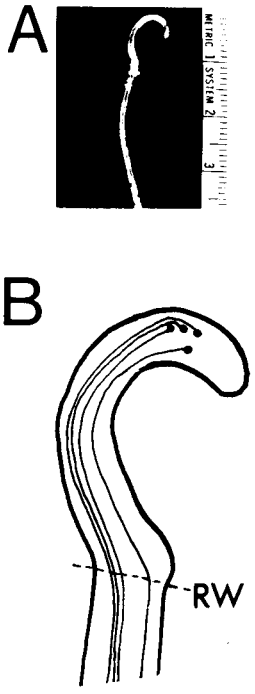

Fig. 1-A. Photograph of a Michelson type electrode,[2] constructed for implantation in the scala tympani in man. The electrodes were embedded in a silastic carrier, molded to fit the most basal 11 millimeters of the scala. B. Drawing of a modified scala tympani electrode used in these studies (see text). Leads were insulated up to the tip. The exposed flat circular tips were approximately 100 microns in diameter; they faced upward toward the basilar partition and bony spiral lamina. Implants had 2-5 exposed electrode tips arrayed across both the transverse and longitudinal dimensions of the scala (there are four in the implant shown). The spread of excitation across the acoustic nerve could be evaluated with stimulation across any electrode pair.

With a simple electrode system, all the keys of the piano are depressed at once regardless of what the input delivered to the system is. There is only one channel of information to the nerve. The individual notes or tones cannot be simultaneously represented in the message delivered to the brain. With any given stimulus, all nerve fibers convey the same input to the brain. We believe that in addition to the periodicity input generated by electrical stimulation, it is necessary to provide *place* input, *i.e.*, to excite discrete, predetermined sectors of the acoustic nerve in an appropriate way. Back to our crude analogy, we must reconstruct our piano keyboard, so that different keys can be individually and independently depressed.

The scala tympani appears to us to be the safest and easiest site at which stimulation of a series of predetermined sectors of the acoustic nerve might be affected. It is safest both surgically and in terms of electrical isolation. The most important advantage of the scala tympani is that it provides access to the spectacular array of the acoustic nerve in the bony spiral lamina.

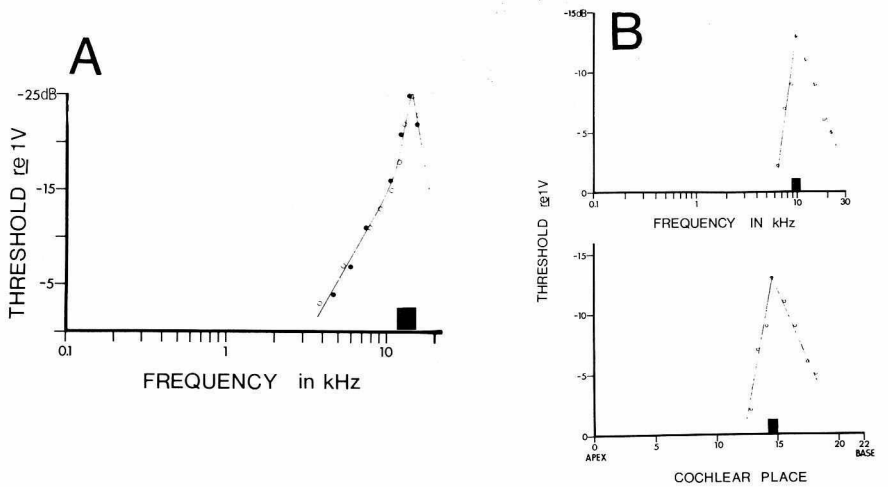

Fig. 2. Excitation of the acoustic nerve as a function of represented best frequency along the cochlear spiral, for two bipolar electrodes within the scala tympani centered at the 13 (A) and 10 kHz (B) positions. The electrode locations and interelectrode distances are indicated by the solid bar along the abscissa. In the lower curve in B, the data in A has been replotted after conversion of frequency position data to corresponding cochlear place. These curves are derived from studies of the threshold to electrical and sound stimulation of binaurally excited neurons within the tonotopically organized central nucleus of the inferior colliculus. They reveal that a discrete sector of the acoustic nerve can be excited from within the scala tympani.

If two conditions can be met, it might be possible to excite predetermined sectors of the acoustic nerve from within the scala tympani, and thereby to generate a much better facsimile of the input generated by the normal cochlea with sound. Those two conditions are: *1.* The nerve must survive long-term intracochlear implantation. There has been great debate over whether it will or not. Some individuals have stated flatly that it will not[8]; the issue is in dispute. *2.* One must be able to excite discrete, predetermined sectors of the acoustic nerve in an appropriate way. Again, there has been great debate as to whether or not this is possible. The resistance to longitudinal current flow in the scala tympani is low, and there is a question as to whether or not one can appropriately restrict stimulation to a discrete sector of the acoustic nerve. The experiments that have been conducted over the past months have been directed toward answering these two questions.

First, does the acoustic nerve survive long term scala tympani electrode implantation and stimulation? Cats implanted for periods up to about two and one half years have been examined histologically. There is some evidence of degeneration in the extreme cochlear base, but never is the nerve severely compromised even by these implants — except in a few cases in which the basilar partition was penetrated by the electrode.[9] Physiological experiments reveal that these surviving ganglion cells are functionally viable.

Can we excite predetermined discrete sectors of surviving nerve? Our

approach has been to use modified Michelson electrodes (Fig. 1) designed for cats. Small electrode contact surfaces face upward toward the basilar membrane. Interelectrode distances are small (200-1,500 microns). Interelectrode distances and electrode impedances are variables in these experiments. The placement of these electrodes *re* the organ of Corti and dendrites of spiral ganglion cells is another important variable.

Results obtained with one such electrode are shown in Figure 2-A. The interelectrode distance was about 1,000 microns. In this graph, we have plotted the threshold of electrical excitation as a function of normally represented frequency along the basilar partition, with the electrical threshold shown on the ordinate and frequency position shown on the abscissa. With this electrode placed at the 13 kHz position, excitation of the acoustic nerve is seen to be restricted to that location. How good is it? How does this discrete, restricted stimulation compare with normal nerve excitation? For the place-frequency representation of a simple tone in the normal cochlea, the slope on the curve relating threshold to place about the resonant point for that tone is probably about 100 decibels per octave in man.[10] On the graph, we see a drop of about 15 decibels per octave for discrete electrical stimulation. It happens that a 15 decibel intensity range for electrical stimulation is equivalent to about 100 decibels for sound stimulation! So that by a stroke of good fortune *we can generate a "place" input that is a close facsimile of the "place" input generated by a simple tone.* This result has now been obtained in several animals.

Results derived with another electrode are shown in Figure 2-B. In this case the interelectrode separation is about 800 microns. The electrodes are centered at the 10 kHz position. Again, if the slopes of the response curves generated by this electrical stimulation are examined, it is evident that these electrodes are generating a response in a cochlear sector with slopes of about 15 decibels per octave. Again, with such an electrode placed within the scala tympani in man, one would generate a place-profile input that is a reasonable facsimile to the place input that is believed to be generated by a simple tone. Back to our analogy, in the experiments shown in Figure 2, we have reconstructed two piano keys. It should, therefore, be possible to reconstruct the piano.

While much more work needs to be done, these preliminary experiments suggest that the necessary conditions for multichannel stimulation of the nerve from within the scala tympani might be realized; that is, the acoustic nerve appears to survive the implantation of an electrode appropriate for discrete multichannel excitation of the acoustic nerve at least over a time course of many months; and physiological evidence indicates that appropriate discrete stimulation of the acoustic nerve from within the scala tympani is really possible. Obviously, both of these sets of results are preliminary. Many more of these kinds of experiments must be conducted.

From these observations, we would draw the following conclusions: First, all of the evidence that has been collected to this time indicates that development of a multichannel nerve stimulation prosthesis is really possible. Second, it is a virtual certainty that multi-channel stimulation devices will provide far more information about sound for profoundly deaf subjects than can any simple electrical stimulation device. Third, given acoustic nerve survival and the capability of generating responses from discrete predetermined sectors of the acoustic nerve, we believe that the scala tympani is the preferred site for this nerve stimulation. Fourth, it is possible that sufficient information about sound can be generated by appropriate excitation within the scala tympani to allow for the direct hearing of speech in a significant population of the profoundly deaf. We believe, in conclusion, that there is a great future in this work — but that the future awaits further experiments of this type, and fabrication of suitable and highly versatile driving systems. The future is *not* now.

ACKNOWLEDGMENT.

The authors acknowledge the technical assistance of Queen Francis, Kent Taylor, Leona Wayrynen and Melvin Bartz is these experiments.

BIBLIOGRAPHY.

1. SIMMONS, F. B.: Electrical Stimulation of the Auditory Nerve in Man. *Arch. Otolaryngol.*, 84:24-76, 1966.

2. MICHELSON, R. P.: Electrical Stimulation of the Human Cochlea. *Arch. Otolaryngol.*, 93:317-323, 1971.

3. MERZENICH, M. M., ET AL.: Neural Encoding of Sound Sensation Evoked by Electrical Stimulation of the Acoustic Nerve. *Ann. Otol.*, 82:486-504, 1973.

4. KIANG, N. Y. S. and MOXON, E. D.: Physiological Considerations in Artificial Stimulation of the Inner Ear. *Ann. Otol.*, 81:714-730, 1970.

5. SIMMONS, F. B. and GLATTKE, T. J.: Comparison of Electrical and Acoustical Stimulation of the Cat Ear. *Ann. Otol.*, 81:731-738, 1970.

6. GLATTKE, T. J.: Electrical Stimulation of the Auditory Nerve in Animals. In: Electrical Stimulation of the Acoustic Nerve in Man. M. M. Merzenich, R. A. Schindler and F. A. Sooy, Editors. Velobind, Inc., San Francisco, Calif., pp. 105-121, 1974.

7. MERZENICH, M. M.: Intracochlear Electrical Stimulation with a Bipolar Electrode in Animals and Man. In: Electrical Stimulation of the Acoustic Nerve in Man. M. M. Merzenich, R. A. Schindler and F. A. Sooy, Editors. Velobind, Inc., San Francisco, Calif., pp. 79-103, 1974.

8. LAWRENCE, M., ET AL.: Discussion: Long-Term Results of Cochlear Implantation: Nerve Survival. In: Electrical Stimulation of the Acoustic Nerve in Man. M. M. Merzenich, R. A. Schindler and F. A. Sooy, Editors. Velobind, Inc., San Francisco, Calif., pp. 161-170, 1974.

9. SCHINDLER, R. A. and MERZENICH, M. M.: Chronic Intracochlear Electrode Implantation: Cochlear Pathology and Acoustic Nerve Survival. *Ann. Otol.*, 83:202-216, 1974.

10. BÉKÉSY, G. V.: The Patterns of Vibrations in the Cochlea. In: Experiments in Hearing. McGraw-Hill, New York, N. Y., pp. 403-484, 1960.