Design of a portable system for chronic stimulation of the auditory nerve, a hearing aid for the sensory deaf

by
L. J. Leifer, Institut für Biomedizinische Technik der Universtität Zürich und ETH Zürich

N. Dillier, Institut für Biomedizinische Technik der Universität Zürich und ETH Zürich

U. Fisch, Otorhinolaryngologische Klinik Universität und Kantonsspital Zürich

Introduction

Auditory sensations have been known to result from electrical stimulation of the human body since the time of Volta (1800). A wide range of experiments have since revealed three basic types of response to electric stimuli. Simmons (1966) and Flottrop (1953) have reviewed the basic work, these reviews are still valid.

The most commonly observed effect is a *square law* perception in with the perceived loudness is proportional to the square of the applied voltage. Subjects must have an intact cochlea. Fidelity is good. Electrode placement can literally range over the entire body. An intact tympanic membrane and middle ear are not required. The mechanism of transduction is generally unknown, but fits well the model of an electrostatic transducer mechanism outside the cochlea, typically at the electrode, which converts the input electrical signal into an acoustic signal which is perceived by normal mechanisms of the cochlea.

A second type of sensation follows a *linear law* (perceived loadness proportional to input stimulation voltage). A functional cochlea is required. Stimulation is usually delivered directly to the cochlea. While the mechanism is not understood, it is generally assumed to involve an electrostatic transductive process within the cochlea which affords perception via normal haircell mediation of acoustic energy.

The third type of sensation requires delivery of stimuli directly within the cochlea, or directly to the auditory nerve. An intact sensory cochlea is not required. Perceived loudness follows a higher order *power law,* with a sharp perceptual onset threshold and narrow dynamic range before the painthreshold is reached. Perceptions are complex and have not afforded speech comprehension. It is generally agreed that these perceptions result from direct *nerve stimulation.* Recent attempts to develop electric prostheses for the sensory deaf have all elicited type three sensations through stimulation of the auditory nerve. Using simple electrode arrays, and at times even complex electrode arrays, fidelity has been poor with minimal speech intelligibility achieved even after extensive training *(Simmons* et al. 1966, *Michelson* 1971, *House* et al. 1973, *Clark* 1973, *Merzenich* 1973, *Merzenich* et al. 1974). However, numerous patients have attested to the value of even such limited contact with the acoustic world. The advantage of even rough loudness and pitch perception during lip reading is clear. The value of such an aid would be correspondingly greater for the profoundly deaf who must learn to speak.

Problem

As illustrated in *fig. 1,* the signal transmission problem of the sensory deaf lies at the point where acoustic energy is transduced into patterned neural activity. This condition confronts the neural prosthesis designer with the dual problem of introducing a signal into the auditory system, and generating a pattern of neural activity which is meaningful to auditory perception mechanisms which have evolved to process haircell transduced and coded neural signals. The prosthesis must:

1. transduce an acoustic signal into an electric signal;
2. information useful to the patient must be extracted and transformed into a code meaningful to the auditory nervous system;
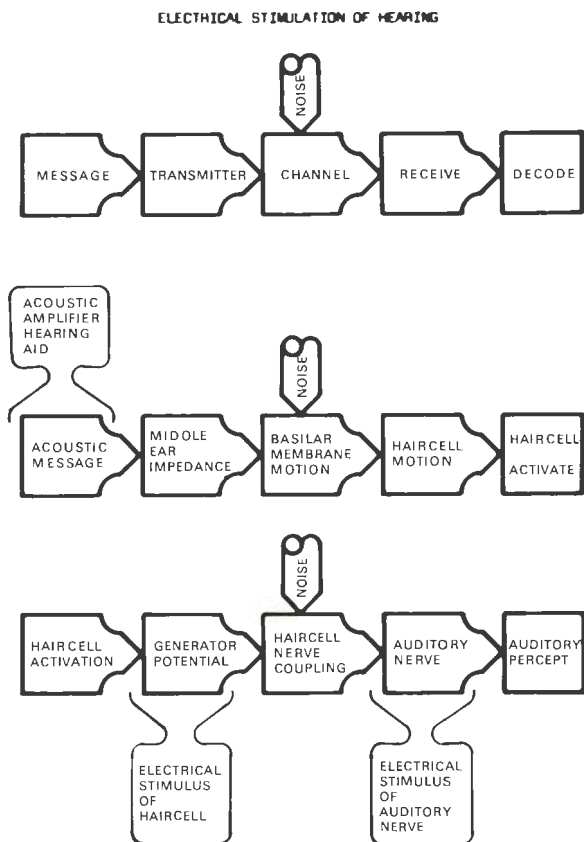3. this code must be transmitted to the auditory system.

Fig. 1. Flow diagram of signal transmission
  a) general system
  b) the auditory peripheral system

Hypotheses

Our project is based upon the hypothesis that a sensori-neural deaf patient with an intact auditory nerve and functional central auditory system will hear normally when the approximately 30,000 fibers of his auditory nerve are stimulated in a manner equivalent to that of the normally hearing person. To apply this hypothesis we ask three questions:

1. What, exactly, is the normal neural activation pattern following acoustic stimulation?
2. How can this activity pattern be reproduced when the normal transfer function of the basilar membrance and haircells is pathologically altered?
3. What constitutes a comprehensive rehabilitation program for patients who receive an electrical stimulation hearing aid?

Motivation

As representative data indicate, there are approximately 300,000 persons in the USA with bilateral hearing loss of 90 db ISO or greater. Of this number, it is estimated that 100,000 persons are real candidates for an electrical stimulation aid *(Carhart* 1974). While the incidence of sensory deafness increases with age, it is the very young who are most drastically affected in their impaired learning of verbal communication skills.

Immediate goal

We attempt to develop and evaluate a practical hearing prosthesis which allows the profoundly deaf patient to reestablish contact with the acoustic environment. Using one or two channels of stimulation, such a prosthesis will provide a crude representation of normal sounds. Through extensive testing and evaluation of auditory performance in a small group of patients we seek to identify the criteria for signal encodement which might make second generation multi-channel prostheses capable of providing speech intelligibility.

Methods theoretical

We believe that the major long range obstacle to the successful development of an electrical stimulation hearing aid lies in the signal transformation, or encodement, phase of the problem. Accordingly, *Dillier, Kobel* and *Leifer* (1975) have constructed a digital computer simulation of the peripheral auditory system. The principle parts of this model are the middle ear mechanics, the basilar membrane mechanics, the haircell transducer function, and the single fiber auditory nerve activity *(fig. 2).* The model is derived from the work of *Flanagan* (1962) with respect to the basilar membrane transfer function and the work of *Weiss* (1966) with respect to the haircell and neuron. Specific parameters for the model are derived from more recent experimental studies by *Helfenstein* (1974), *Johnstone* et al. (1970) and *Rhode* (1971). Our contribution to the modeling effort is directed at identifying the transfer function required to make identical acoustic and electrical input signals elicit the same neuronal activity. This amounts to the signal transformation phase in the problem; given an input, how does one optimally stimulate the auditory nerve to give a sensation equivalent to that perceived by the normally hearing

MIDDLE EAR	HAIR CELL	NEURON
BASILAR MEMBRANE	NONLINEAR	STOCHASTIC
MECHANICAL FILTERS	TRANSDUCER	THRESHOLD

$$y = \frac{k_1 k_2 x}{k_2 + x}$$

SPIKES

time

THRESHOLD POTENTIAL $R - R_r + (R_m - R_r)e^{-t}T_r$
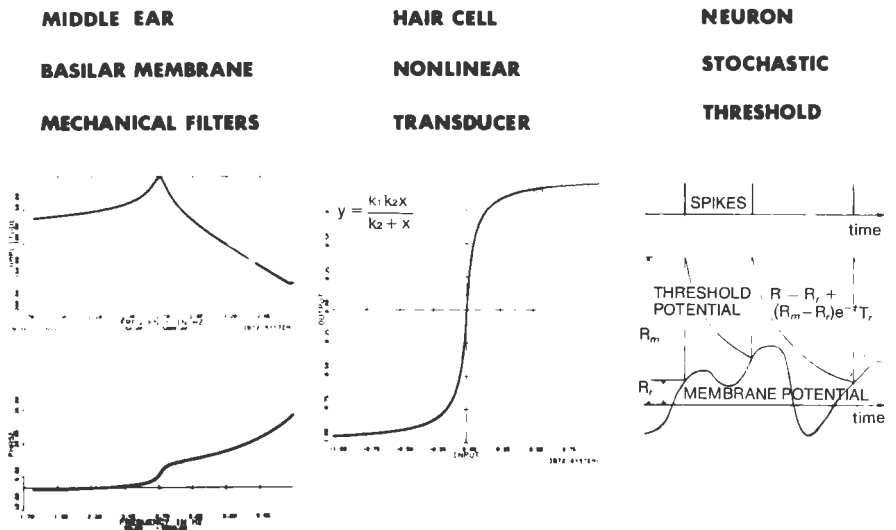
R_m

R_r MEMBRANE POTENTIAL

time

Fig. 2. The three major parts of the computer model for the case of acoustical stimulation. The model input waveform (not shown in this diagram) is filtered first, then transformed and converted into a binary pulse sequence by a stochastic threshold mechanism. The output of the model are histograms of this pulse sequence (not shown in this diagram)

person who receives the acoustic signal directly. In the context of this paper we wish only to indicate that this model is used, and that it provides a test vehicle for our ideas concerning signal transformation options in our prosthesis design.

Methods practical

A system for chronic electrical stimulation of the auditory nerve should contain the following five subsystems:

1. *Signal detection*
2. *Signal transformation*
3. *Stimulus isolation*
4. *Transcutaneous communication*
5. *Nerve electrode*

A schematic view of our design is presented in *fig. 3.* Discussion of each of these subsystems will include the specifications of our current design, and our reasons for adopting the specific approach. We begin with the acoustic input signal and follow its transformation and pathway to the auditory nerve.

Signal detection is accomplished with well-known microphone technology. In order to take advantage of existing experience with small, sensitive, speech bandwidth techniques, we have used a standard REXTON

60

model hearing aid for the initial transformation of acoustic signal to electrical signal. This light weight behind the ear device possesses good sensitivity and allows an initial stage of variable signal amplification. This aid also contains a variable non-linear signal compression circuit with which to reduce the dynamic range of the input. Due to the limited electrical stimulation dynamic range possible, this compression feature was particularly sought after. The aid, less the speaker, possesses a bandwidth of about 200 to 2500 Hz. The output of the hearing aid amplifier goes directly to the signal transformation subsystem.

It is in the *signal transformation* systems that we have extended our major electronics effort, and here that we attempt to provide a measure of flexibility in the system which will allow the patient to experience and explore a variety of acoustic stimulation transformations in the context of his everyday-life. A key-factor in the rehabilitation of patients receiving an electrical stimulation prosthesis lies in having the opportunity to use the device daily within the normal living environment in contrast to isolated laboratory testing *(House* et al. 1973). Laboratory testing is also important in that carefully controlled stimuli and quantitative testing is possible.
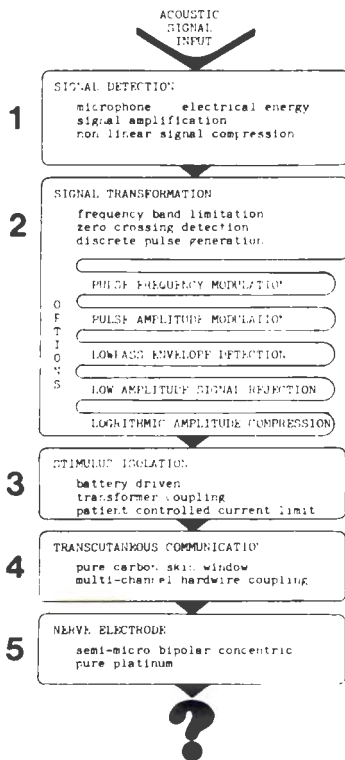

Fig. 3. Schematic view of the transformation and the pathway of an acoustic input signal to the auditory nerve in our stimulation system
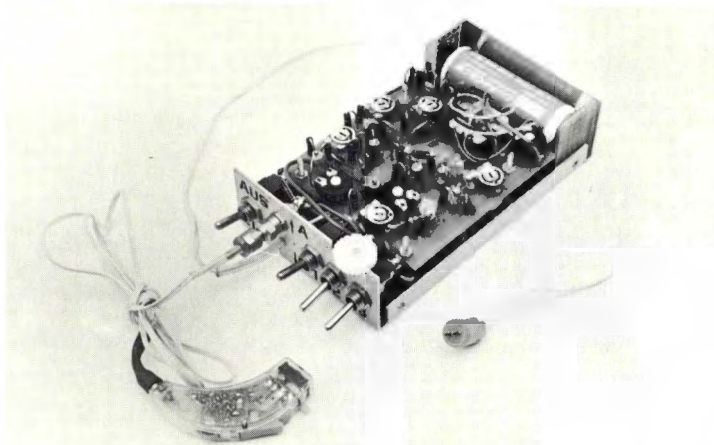
61

Fig. 4. Prototype of a portable signal detection, transformation and stimulation system

---

While nicely packaged *(fig. 4)* the current signal transformation package has not been miniturized. The package is $3 \times 5 \times 8$ cm and weights about 140 gr, this is equivalent to many small pocket radios and is easily portable. External controls include:

1. *On–Off*
2. *Intensity control*
3. *Signal transformation option switches*
   a) *Linear* versus *logarithmic* pulse amplitude
   b) *Speech envelope detection* versus full band signal
   c) *Pulse frequency modulation*
   d) *Pulse amplitude modulation*
   e) *Low amplitude signal rejection*

With the exception of the work of *Simmons* et al. (1965), our use of pulse stimuli is unique in auditory prostheses. The reasoning however is quite simple. It has been firmly established that the minimum charge transfer (μCoul) and minimum pulse energy (μJoul) for equivalent stimulation effects upon peripheral nerve are achieved by rectangularly shaped pulses of durations less than 0.05 ms *(Crago* et al. 1973). The charge per pulse continues to decline as the duration is shortened, and is limited only by the stimulus voltage that can be applied during the pulse. It has also been reported by various workers at the 6th NIH Neural Prosthesis Workshop – Damage Control, 1975 that neural damage is proportional to charge transfer during the stimulus waveform. As a result one may safely conclude that optimum nerve stimulation will be achieved with pulsed stimuli of short duration and sharp rise time characteristics. We currently use bipolar pulses of 0.050 ms. Bipolarity is important to avoid cumulative offset voltages which result when monopolar stimuli are used. These offset voltages may decrease the pain threshold and stimulate remote

62

vestibular structures. Pulse duration can be adjusted over the range 0.020 to 1.0 ms. At a pulse duration of 0.050 ms the device is capable of close to 20,000 pulses per second. Charge per pulse is less than one tenth of the brain tissue damage threshold.

The various signal processing options available in our current circuitry *(Bernasconi* and *Ragaz* 1975) are illustrated in *fig. 5.* The electric output of the hearing aid is first passed through an adjustable logarithmic amplifier to achieve additional amplitude compression. The slope and offset of this circuit are adjustable according to specific patient threshold criteria. The effects of logarithmic compression on the input signal can be observed in the box labeled comparator. The comparator itself tests for positive going zero crossings, the occurrence of which is used to trigger the generation of one unit pulse. The comparator operates with hysteresis such that low amplitude, short duration zero crossings are not detected. The hysteresis may be adjusted, and is used to reduce «noise».
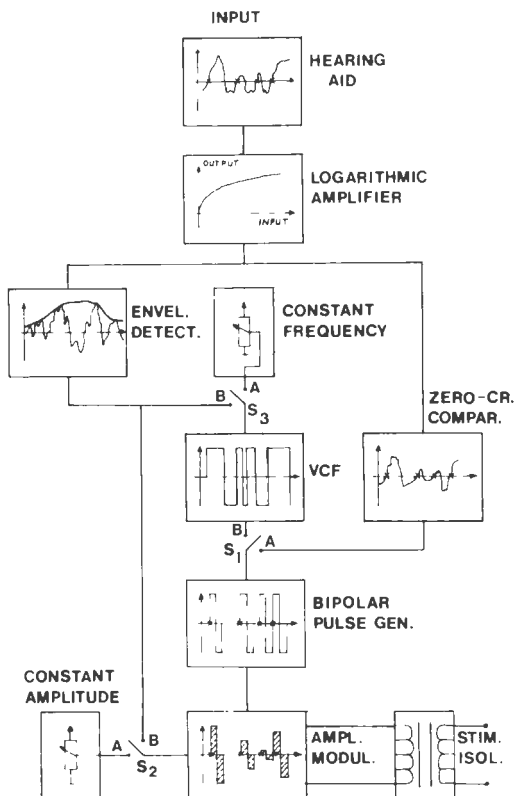

Fig. 5. Diagram of the various switchable and adjustable signal processing options available in out circuitry

63

Alternatively, the output of the logarithmic amplifier stage can be passed through a low-pass filter (20 Hz) to extract the speech amplitude envelope. This function is of specific interest because it has been shown that the speech amplitude modulation function conveys the greatest amount of speech intelligibility information at a minimum signal bandwidth *(Flanagan* 1972). This low frequency signal can then be used to modulate the pulse repetition rate and/or the pulse amplitude. Combined amplitude and frequency modulation provides an improvement in the intensity dynamic range over previously used methods which only modulated signal amplitude. Because we use discrete constant width pulses, changes in pulse repetition rate cause a change in perceived loudness *(Leifer* et al. 1976).

The discrete bipolar pulse is triggered either by the zero crossing comparator circuit or the output of a voltage controlled oscillator. This oscillator is in turn controlled either by the speech envelope detection circuit output, or a manually set constant voltage source. Hence, the timing of the pulses may be determined by any one of three ways: 1.) a pulse is generated each time the acoustic input signal makes one full period of oscillation; 2.) pulses are generated at a frequency proportional to the amplitude envelope of the acoustic input; 3.) pulses are generated at a fixed rate independent of the acoustic input.

The amplitude of the bipolar pulse may in turn be modulated by either of two mechanisms. In the first case it is controlled by a manually set constant voltage source. In the second case it is controlled by the output of the speech envelope detector. There is currently no provision to control amplitude by the momentary amplitude of the acoustic input. To do this would be equivalent to stimulating with the acoustic signal waveform itself. We know from the work of *House* and others that this form of stimulation provides very little speech discrimination.

In the last phase of signal processing, the bipolar pulse is passed through an output limiting circuit and through a transformer coupling to the electrodes. The transformer coupling provides isolation between the circuit and the subject such that any voltage bias in the circuit is not conveyed to the subject. It also assures that the stimulation equipment does not provide an electrical pathway to ground which would allow electrostatic shocks to be conveyed through the implanted electrode.

Stimulation isolation is achieved through use of a battery driven, transformer coupled, output pulse generator. The pulse amplitude is constant current limited. This limit may be adjusted by the user, but is absolute limited at a value well below the intensity known to cause neural damage (0.0125 μCoul/pulse). The pulse repetition rate is also limited to about 10,000 Hz such that the charge per second can not exceed 125.0 μCoul/second.

Transcutaneous communication is accomplished through a hardwire multi-channel coupling. This coupling is itself encapsulated in a pure carbon skin window *(fig. 6)*. The pure carbon device is a very promising solution to the chronic problem of communication across the skin boundary. Devices equivalent to the one we propose to use have been successfully used in humans for periods exceeding 2 years *(Mooney* et al. 1974). Because electrical stimulation of hearing and especially the coding of acoustic information for
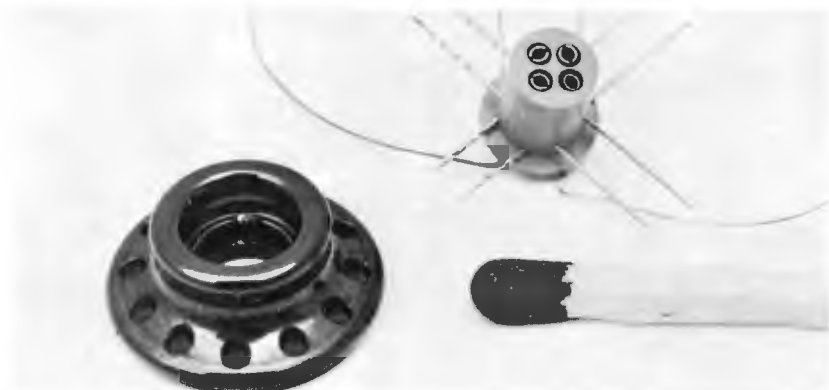
64

Fig. 6. a) Pure carbon skin window with hardwire coupling before final assembling. The four drills indicate how the connectors are mechanically fixed

b) View from bottom showing the mechanical fixation of one electrode. The connections are finally isolated with medical silastic elastomer

direct nerve stimulation is definitely in an experimental stage of development, we have felt strongly that a hardwire coupling is required to carry out the wide range of experiments needed to advance the state of our understanding.

Nerve electrodes have been chosen from a commercial source which has produced micro and semi-micro electrodes for chronic brain implantation *(fig. 7)*. We plan to use at least two bipolar-concentric electrodes placed within the modiolus. These electrodes are made of pure platinum, have an inner contact diameter of 0.2 mm and outer contact diameter of 0.5 mm. The inner
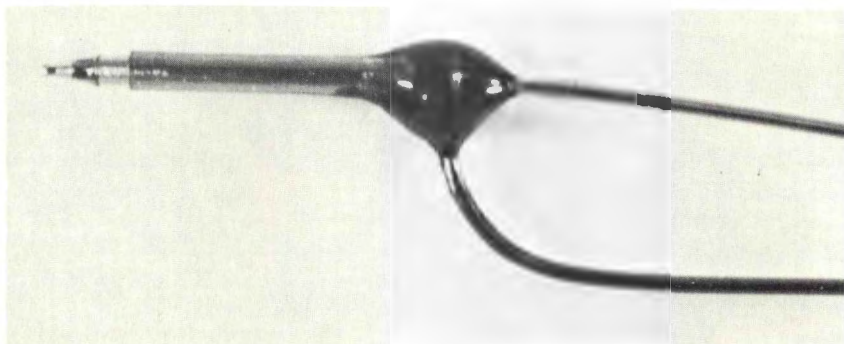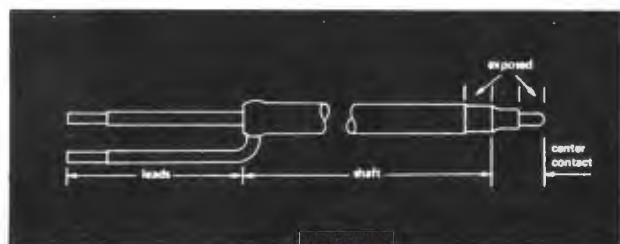
Fig. 7. a) Photograph of the tip of a semi-micro electrode

Rhodes neurological electrode NE-100
Shaft Diameter   0.5 mm
Shaft Length   5.0 mm
Lead Diameter   0.2 mm
Contact Diameter   0.2 mm center
                     0.5 mm outer
Contact Length   0.5 mm center
                     0.5 mm outer

b) Diagram giving the actual dimensions of the electrode

and outer contacts are exposed for 0.5 mm. While our portable stimulation system can only drive one electrode at a time, the reliability factor of placing two electrodes, and the subsequent possibility of studying stimulus interactions within the laboratory strongly motivate us to achieve at least this state of multi-electrode capability.

While the placement of the electrode has not been finalized, we are planning to place two bipolar-concentric electrodes within the modiolus at a separation equivalent to one full turn of the cochlea. We hope thereby to

66

achieve separation of the stimulus effects of the two electrodes and to take some advantage of the place/frequency separation of nerve fibers prior to their relative mixture in the main body of the auditory nerve. The general surgical approach is shown in *fig. 8a.* The extra dural, subcranial, approach affords the

Fig. 8. Surgical Approach
a) General view

b) Cochlear view of transcochlear electrode tip placed within the modiolus

best exposure of the cochlear axis (modiolus axis) along which we seek to place the electrodes. The bony wall of the cochlea must be pierced once for each electrode. A hole must also be drilled in the softer bone separating the cochlear duct from the modiolus. The electrode is then passed through the cochlea such that the active stimulation surfaces are within the modiolus *(fig. 8b)*. We expect damage to the basilar membrane and residual haircell population only along the path of the electrode (0.5 mm). A somewhat similar approach was used by *Simmons* (1966) and in several cat cochlea studies by *Moxon* (1973).

Summary

Three questions were posed together with our hypothesis that a reconstruction of hearing for the sensori-neural deaf is feasible.
1. What is the normal pattern of auditory nerve activation? We have addressed ourselves to the problem through use of a digital computer simulation of the peripheral auditory system *(Dillier* et al. 1975). This simulation is designed to study the type of signal transformation required to convert acoustic into electric stimuli for equivalent neural activation.
2. How may normal nerve activation be accomplished through electrical stimulation? Our experimental work is directed at this question. Our approach is at the psychophysical level and will require studies with chronically implanted electrodes in humans. Our prosthesis is designed to facilitate these studies.
3. What means are required for patient rehabilitation? Our prosthesis is designed to enhance the opportunity for an effective self-help program. Clinical studies are planned for rehabilitation and prosthesis evaluation. Objective means of hearing assessment are being implemented in our laboratory *(Spillmann* et al. 1976) to quantify patient/prosthesis performance in terms of electrode/perception channel capacity *(Corliss* 1971).

Bibliography

Bernasconi, F., Ragaz, R., Entwicklung eines tragbaren Hörgerätes für Patienten mit sensorineuraler Gehörlosigkeit zur direkten elektrischen Stimulation des akustischen Nervs, Semesterarbeit SS 75, ETHZ-III b (1975).

Carhart, R., Sensorineural Hearing Loss: An Overview, in Electrical stimulation of the acoustic nerve in man, editors.

Clark, G. M., Kranz, H. G., Minas, H., Behavior thresholds in the cat to frequency modulated sound and electrical stimulation of the auditory nerve, Exp. Neurobiol. *41,* 190–200 (1973).

Corliss, E. L. R., Estimate of the inherent channel capacity of the ear, J. Acous. Soc. of America *50,* Nr. 2 (Part 2), 671–677 (1971).

Crago, P. E., Peckham, P. H., Mortimer, J. T., and *Van Der Meulen, J. P.,* The choice of pulse duration for chronic electrical stimulation via surface, nerve, and intramuscular electrodes, Annuals of Biomedical Engineering *2,* 252–264 (1974).

Dillier, N., Kobel, W., and *Leifer, L.,* Towards an acoustic/electric stimulation model for cochlear nerve activation, 1st European Neurosciences Meeting, Munich, Sept. 1975.

Flanagan, J. L., Speech Analysis Synthesis and Perception (Springer-Verlag 1972).

Flottorp, G., Effect of different types of electrodes in electrophonic hearing, J. Acoust. Soc. Amer. *25*, 236–245 (1953).

Helfenstein, W. U., Beitrag zur Messung der akustisch bedingten Bewegungen und Identifikation des mechanischen Teils des Innenohrs der Katze, Diss. ETH, Nr. 5309 (1974).

House, W. F., Long term results of electrode implantation and electrical stimulation of the cochlea in man, Ann. Otol. *82*, 504–517 (1973).

Johnstone, B. M., Taylor, K. J., Boyle, A. J., Mechanics of the Guinea Pig Cochlea, JASA *47*, 2, 504–509 (1970).

Leifer, L., Dillier, N., and *Spillmann, Th.*, Experiences with electrical stimulation of human hearing leading to the design of a prosthesis system, Sonderforschungsbereich 88, Münster, BRD (1976).

Merzenich, M. M. et al., Neural encoding of sound sensation evoked by electrical stimulation of the acoustic nerve, Ann. Otol., Rhinol. and Laryngol. *82*, 504–518 (1973).

Merzenich, M. M., Schindler, R. A., and *Sooy, F. A.*, editors, Electrical stimulation of the acoustic nerve in man, Velo-Bind, Incorporated (1974).

Michelson, R. P., Eletrical stimulation of the human cochlea, Arch. of Otolaryngology *93*, 317–323 (1971).

Mooney, V., Hartmann, D. B., McNeal, D., and *Benson, J.*, The use of pure carbon for permanent percutaneous electrical connector systems, Arch. Surg. *108*, Feb. 1974.

Moxon, E. C., Neural and mechanical responses to electric stimulation of the cat's inner ear, Ph. D. Thesis, MIT, Boston, Mass. (1971).

Rhode, W. S., Observation of the vibration of the basilar membrane in squirrel monkeys using the Mössbauer technique, JASA *49*, 4, 1218–1231 (1971).

Simmons, F. B., Electrical stimulation of the auditory nerve in man, Arch. Otolaryngol. *84*, 2–54 (1966).

Spillmann, Th., Dillier, N., and *Leifer, L.*, Objectivication of clinical ERA and ECOG data assessment, Sonderforschungsbereich 88, Symposium 1976, Münster, BRD,.March 1976.

Weiss, T. F., A model of the peripheral auditory system, Kybernetik, 3. Bd., 4. Heft, 153–175 (1966).

Correspondence:
Prof. L. Leifer, Institut für Biomedizinische Technik, Moussonstrasse 18, CH-8044 Zürich, Switzerland