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RESEARCH OBJECTIVES AND SUMMARY OF RESEARCH

The general goal of this group is to find some understanding of nervous action. It is not obvious how such an understanding comes or from what kind of experiment. Accordingly, we use a broad approach and work at several different levels in the hope of revelation.

1. Membrane Processes

National Institutes of Health (Grants 1 RO1 EY01149-01 and 5 PO1 GM14940-07) Bell Telephone Laboratories, Inc. (Grant)

J. Y. Lettvin

Several years ago, in honor of K. S. Cole's birthday, this laboratory developed an operating analogue model of nerve membrane. The model has achieved some unofficial attention in the sense that it is in general use as a teaching aid and has already been handled as a norm in some published work. We, however, have not published it, since we have not been able to justify it. The particular difficulty lay in reducing Hodgkin and Huxley's parameters m, n, and h to a fundamental generating function, W, where

$$W = \frac{1}{1 + \left(\frac{(Ca^{2+})_{R}}{(Ca^{2+})_{R}} \exp\left(\frac{2V}{V_{T}}\right)\right)^{2}}.$$

W expresses the adsorption-desorption characteristic of Ca^{++} to the membrane surface as a function of external Ca^{++} concentration, a reference concentration $(Ca^{++})_R$, and the membrane voltage, V, taken with respect to the outside of the membrane. The difficulty lay in making the account plausible, although the formula predicts quite well that an e-fold change in Ca^{++} ought to shift W, as a curve, by approximately 6 mV, and that h, at least, should have the form

$$h = \frac{1}{1 + \exp[(4V - V_{\rm R})/V_{\rm T}]}.$$

For better or worse, however, the model with its somewhat shaky foundations, must

be published, since various authors of other works have become irritated at a lack of reference outside of "personal communication."

2. Visual Receptor Model

National Institutes of Health (Grants 1 RO1 EY01149-01 and 5 PO1 GM14940-07)

Bell Telephone Laboratories, Inc. (Grant)

J. Y. Lettvin

In almost the same way and at almost the same time as we developed our model of nerve membrane a visual receptor model was imagined — in this case at profound variance with the published accounts on receptors. Here the problem lay in accommodating the psychophysics rather than the microelectrode data, which is, in the end, not quite as trustworthy as the former.

Briefly, a receptor had to be imagined that gave the following properties:

a. A dynamic range of 10^6 .

b. A Weber-Fechner law characteristic, i.e., $\Delta I_{threshold} = KI$, where I is intensity of light, and K ~ 0.01.

Now, these conditions can be met easily by the following sort of device. Imagine a single disc of receptor connected by a neck to the synaptic apparatus. Let all of the visual pigment lie in such a way as to coat the interior of the disc. The total number of pigment molecules is C. An unbleached pigment molecule, of which the number is A, on capturing a photon goes through a transition phase A' for a period τ and then becomes a bleached molecule of which the number is B, so that A + A' + B = C, and if A' is relatively small, $A + B \cong C$. Finally, a bleached molecule returns to the state of being unbleached with a probability/time β . Each pigment molecule governs a patch of membrane. For an unbleached molecule, the conductance of any ion across the patch is 0. For an A' molecule, the apple to μ and the particular to μ and the particular to μ and the particular to μ and μ apple to μ apple to μ and μ apple to μ applies the apple to μ applies the applies to μ applies the applies the applies to μ applies the applies the applies to μ applies to μ applies the applies to μ applies the applies to μ applie

ecule, the conductance is high to, say, Na^+ ions. For a B molecule, the conductance to Na^+ is again 0, but the conductance to K^+ ions is high.

Let the measure of interest be the membrane voltage. Then $-dA = \phi A - \beta B$, where ϕ is flux of photons. When dA = 0, $B/A = \phi/\beta$. The current across the disc wall is generated by the A'-governed patches and shunted by the B-governed patches. Thus, when dA = 0,

$$A'(g_{Na}^{+})(V_{Na}^{-}V_{M}) = B(g_{K})(V_{M}^{-}V_{K})$$

whence, given that $A' = (\Delta A)_{\tau}$, and g_{Na^+} is a constant (the Na⁺ conductance of a single A'-governed patch) and g_{K^+} is a constant (the K⁺ conductance of a single B-governed patch) then V and V are also constant. Then

$$V_{M} = \frac{A'(g_{Na})(V_{Na}) + B(g_{K})(V_{K})}{A'g_{Na} + B(g_{K})} = V_{c}, \text{ a constant voltage}$$

since in steady state

$$A'/B = \Delta A \tau/B = 1/\beta$$
.

That is, whatever the steady state of illumination, $V_{\rm M}$ is a constant, as is truly apparent without having to go through the calculation. Similarly, if we set a threshold change of $V_{\rm M}$ as

 $(\Delta V_M)_{thresh}$ is a constant

and suddenly apply a step up or down of ϕ , it is easy to see that

$$(\Delta V_{\rm M})_{\rm thresh} \sim \Delta \phi / \phi$$

since both the numbers of g and g are directly proportional to φ over a wide range.

At the time we imagined this very simple device it did not seem worth presenting because all the physiological evidence was against such elementary notions. Now, however, our own experiments on frog rods have begun to contradict the current view and to suggest that such a model is not entirely foolish. We shall, therefore, be presenting the device in a short technical note.

3. Study of Frog Rods

National Institutes of Health (Grants 1 RO1 EY01149-01 and 5 PO1 GM14940-07)

Bell Telephone Laboratories, Inc. (Grant)

J. Y. Lettvin

As part of his doctorate thesis research, Mark Lurie, of this laboratory, found the following.

1. In a thoroughly dark-adapted eye of a frog, in situ, there seems to be no external "dark current" flowing between the outer and inner segments of the rods.

2. When external current does flow with illumination, it flows from the inner to the outer segment rather than the other way round, as is conventionally taught.

Because these observations are opposed to what Hagins and Tomita and others assert, we were much exercised to check our methods. At the present time, we are confident of these findings but, before issuing them, we must discover what accounts for so great a difference between our results and those of other workers.

4. New Staining Methods

National Institutes of Health (Grants 1 RO1 EY01149-01 and 5 PO1 GM14940-07)

Bell Telephone Laboratories, Inc. (Grant)

E. R. Gruberg, J. Y. Lettvin

Dinitro-blue-tetrazolium substitutes for the cytochromes as a proton acceptor and precipitates out as an insoluble formazan when it is oxidized. It has been used to show the presence and location of various dehydrogenases and/or their associated flavoproteins in mitochondria. Tetranitro-blue-tetrazolium acts in the same way but yields an osmophilic precipitate that permits location under electron microscopy. Both dyes penetrate the cell wall easily, although they have been most used with frozen sections, where the mitochondria are directly exposed.

Jay Enoch of Washington University, St. Louis, Missouri, used the dyes supravitally

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to show that the retina could be made a kind of photographic plate. Staining occurs most rapidly in those photoreceptors that have been hit by light, when the retina is incubated with a succinate dehydrogenase where light adaptation is. This activity appears in the ellipsoids and, if one uses succinic semialdehyde, also in the Landoldt clubs.

We have been extending this work in several directions. First, when the retina is stained supravitally without added substrate, we have observed that the synaptic mitochondria of the rods, at least, seem to stain very well – almost as vividly as the ellipsoids, and, seemingly, along with light adaptation. This finding lends some support, indirectly, to our electrical records that are at variance with current teaching.

Second, if the retina is stained under different substrates, e.g., malate, glutamate, etc., a pattern of dehydrogenase activity emerges that suggests that different types of neurons are characterized by different excesses and deficiencies of the different dehydrogenases. For example, the ganglion cells stain most strongly with glutamate substrate. But it is also the ganglion cells that are killed by glutamate excess. We have begun to think, therefore, that subpatterns of the Krebs cycle characterize the different types of neurons, and this finding may now lead us to follow cell differentiation.

Other findings elsewhere support such an idea, but nobody, it seems, has noted this consequence to the differential responses to such stains. We are pursuing the matter vigorously.

5. Stentor Coeruleus

National Institutes of Health (Grant 5 TOI GM01555-07)

E. Newman

We have been investigating the internal fiber systems of the protozoan, <u>Stentor coer-uleus</u>, with scanning electron microscopy. Pictures of the internal surface of the cortex of Stentor reveal myonemes (contractile fibers) running longitudinally down the entire length of the cell. The posterior region of the myonemes are ribbon-shaped, projecting inward from the surface. The anterior regions of the fibers are flattened in appearance and are interconnected with numerous cross bridges. The internal surface of the frontal field of Stentor is similarly covered with a highly cross-connected myoneme system. A paper is being prepared, entitled "A Scanning Electron Microscopic Study of the Myoneme System of Stentor coeruleus."

6. Optic-Nerve Regeneration

M.I.T. Sloan Fund for Basic Research

Susan B. Udin

Investigations of regeneration in the frog visual system continue. Electrophysiological recording of regenerating optic-nerve fibers reveals a disorganized retinotopic map during the first months after cutting the optic nerve. This map becomes reorganized in subsequent months. In order to investigate the "functionality" of the disorganized map, we have begun to measure the accuracy of prey-catching behavior (orienting and snapping) during the regeneration process.

In a second group of animals, we removed the caudal half of the right tectum. In some of these frogs the left optic nerve was cut too. The aim of this experiment is to determine the fate of the optic-nerve terminals that were originally in the lesioned area.

A third project, in association with Carl Young, is a histological investigation of the fate of the optic nerve following complete bilateral tectal ablation.

7. Activity in Substantia Gelatinosa

National Institutes of Health (Grant 1 ROI EY01149-01)

I. D. Hentall

Experimentation has begun on the spinal cord of cats in order to record activity from single units in the substantia gelatinosa. Many previous investigations have failed in this task, since the small unmyelinated cells of this area are highly susceptible to inhibition by general anesthetics and, we suspect, by mechanical stresses produced by the introduction of a microelectrode into the area. There are several indications of the significance of the substantia gelatinosa in the organization of exteroceptive perception; recent work of Denny-Brown et al. on altered dermatome regions and hyperesthesia in monkeys with chronic lesions of Lissauer's tract, which contains the axonal output of these cells, appears to have considerable significance. But the lack of data from single cells detracts from the value of present hypotheses on the neuronal origin of touch, pain, and so forth. By careful manipulation of the variables of microelectrode technique, we hope to solve the recording problem and obtain more information on the electrical properties of this prominent plexus of small interconnected cells.

8. Color Blindness of Retrobulbar Origin

National Institutes of Health (Grant 5 PO1 GM14940-07)

M. H. Brill

A series of color-blindness tests will be started on people who have recovered from uniocular retrobulbar neuritis symptoms that are typical of the early stages of multiple sclerosis. The tests will be designed to determine how defects that occur proximally to the retinal receptors can be measured. We shall attempt to infer from the pathology some aspects of normal retinal organization. In addition to some conventional tests, we shall measure spectral sensitivity and afterimage latencies of affected as opposed to normal eyes. According to previous published reports, we should expect to find fairly normal visual acuity accompanied by reduction in the apparent saturation of all perceived colors. These tests are being performed in association with Dr. Ronald Burde and Pam Gallin of Washington University Medical School, St. Louis, Missouri.

9. Mechanisms for Color Vision

National Institutes of Health (Grant 5 TOI GM01555-07)

Lynette L. Linden

An evolutionary and mathematical basis for a three-dimensional color space is being formalized. With this background, we shall explore boundary operations for a system that tracks colors under slowly varying broadband illumination.

10. Pattern Display in Flounder

National Institutes of Health (Grant 5 TOI GM01555-07)

W. M. Saidel

Work is in progress on photographing and then analyzing the following phenomenon that has been frequently seen by skin divers, but has not made its way into the literature. When one views a flounder swimming over a well-featured bottom (i.e., rocks or other large objects), it seems that the flounder has a representation of those features

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in the patterns of its upper surface and, furthermore, that these patterns move over the surface more or less as the flounder moves over the bottom, so that it is almost as if one is viewing the bottom through a distorted transparency. Not only is this phenomenon interesting in itself, but it takes on stronger interest if one considers that the flounder has on its back not what it sees (consider the view of a bottom-moving animal) but what a distant observer should see, and it does so without being able to see well the pattern that it has (its own back). These problems call, at least, for a clear statement of what actually occurs.

11. Description and Characterization of Threshold Changes in Frog Peripheral Nerve Axons

Bell Telephone Laboratories, Inc. (Grant)

S. A. Raymond

Two sorts of fluctuation in the threshold of nerve membrane are widely recognized. One includes relatively small amplitude changes in threshold that occur "spontaneously" in a rested nerve. This change is analogous to noise at the input of a perfect trigger circuit, and is measured most simply by evaluating the "relative spread" between the smallest effective stimulus and the largest ineffective stimulus. The second kind of threshold change is a trend that is added to the noisy baseline variations that are always present. These relatively long-term threshold shifts arise whenever the nerve generates an impulse. Similar long-term shifts in threshold can be brought about by other operations such as changing the partial pressure of oxygen or CO_2 in surrounding fluids,

or by adding various drugs. We have developed a method for measuring threshold accurately, and membrane threshold proves to be a very sensitive detector for a wide variety of drugs, including aspirin, alcohol, strychnine, and tranquilizers. We have fixed

our attention primarily on the oscillations in nerve threshold that follow impulses,¹ and our initial characterization is now completed.

For a brief time after an impulse, the membrane will not fire again regardless of stimulus strength. The earliest possible time at which firing is again possible depends strongly on the time course and amplitude of the stimulus, but invariably the absolute refractory period is followed by a period where threshold is much higher than for a rested nerve. Several milliseconds after an impulse, most frog nerves have a period of low threshold where it is actually easier to fire the nerve than it is when the nerve is rested (no prior impulse). This period lasts up to a second or more. Another phase of threshold change becomes dramatic if the nerve has undergone a good many impulses prior to the testing of threshold. This third phase follows the period of low threshold, and is marked by a rapid rise in threshold to a peak that depends monotonically on the frequency and duration of the preceding barrage. At high levels of impulse firing, which are well within the range of firing rates observed in peripheral nerves in vivo, the threshold can rise to two or three times its "resting" value. Even during this "depression" phase, however, when an impulse occurs, it is followed by a brief drop in threshold to a value very near the lowest threshold that can be obtained from a rested nerve. This means that during depression impulses result in threshold oscillations with an amplitude that is two or three times the resting threshold magnitude. Our observations indicate that in vivo nerves normally conduct enough impulses to lead to depression of the threshold. We believe that the normal threshold changes following impulses in animals are large enough to govern the way nerve impulses are distributed throughout the nervous system.

References

1. E. A. Newman and S. A. Raymond, "Activity-Dependent Shifts in Excitability of Frog Peripheral Nerve Axons," Quarterly Progress Report No. 102, Research Laboratory of Electronics, M. I. T., July 15, 1971, pp. 165-186.

12. Development of a Model Frog Nerve Axon Showing Threshold Oscillations

Bell Telephone Laboratories, Inc. (Grant)

S. A. Raymond

Using the computer facility at Project MAC, Paul Pangaro, an undergraduate student in the Department of Humanities, has written a program for a visual display of a nerve axon modeled from the experimental observations made on excised single axons. We have synthesized the behavior of the axon by writing exponential functions for each of the phases in the characteristic threshold oscillation following impulses.

Threshold during the refractory period (R(t)) is modeled as a simple exponential process beginning with the end of the impulse (t=0). We consider it to have an instantaneous value approximately six times the resting threshold and a decay constant τ_r of 3 ms.

$$R(t) = K_{r} \left[\exp(-t/\tau_{r}) \right].$$
(1)

Threshold during the supernormal period (S(t)) is conceived as depending on a simple exponential process acting on a quantity called "supernormality" that has been vaguely named on purpose. "Supernormality" is added by each pulse, although a maximum amount of supernormality is reached asymptotically in accord with our experimental observations. The equation is

$$S(t) = (\Delta S + \Delta S_{last}) [-exp(-t/\tau_s)],$$

where ΔS is the amount of supernormality added by each pulse. It obeys the equation

$$\Delta S = \frac{K_s}{1 + M_s S_{last}},$$

where K_s is the amount of supernormality added to a rested nerve (constant). ΔS is thus K_s attenuated to reflect the observation that less supernormality is added by a pulse if residual supernormality (S_{last}) is high at the time of the impulse. ΔS_{last} is that portion of the residual supernormality remaining at the time of each pulse which is added to the supernormality of the next pulse (ΔS). When supernormality is high, less is conserved after the firing of a new impulse. The maximum amount conservable is stipulated by the value of $L(L \leq 1.0)$. M_L expresses the extent of the dependence of the amount conserved on the value of S_{last} .

$$\Delta S_{last} = \frac{LS_{last}}{1 + M_L S_{last}}.$$

Threshold during the depression phase (D(t)) is modeled as a simple exponential recovery process acting at all times on a quantity called "depression" which is incremented without limit by each pulse. Each pulse adds a constant amount of depression (ΔD) . The lowering of threshold during the supernormal period predominates during the early part of the depression according to a multiplier (M_D) . The equation is

$$D(t) = \frac{1}{1 + S(t) M_D} DL(t),$$

where the depression level, DL, is given by

DL(t) = (DL_{at t=0} +
$$\Delta D$$
) exp $\left(-\frac{t}{\tau_D}\right)$.

Threshold after each pulse is determined as a simple sum T(t) = R(t) + S(t) = D(t). The model reproduces all of the phenomena observed thus far in the nerve. Thus in principle it is possible to conceive of threshold oscillations as reflections of processes requiring varying times and all acting simultaneously on residues of each nerve spike. That is, each pulse can be conceived as instantaneously incrementing the variables that govern at least three processes.

13. Distribution of Nerve Impulses among the Branches of Axonal Trees

Bell Telephone Laboratories, Inc. (Grant)

S. A. Raymond

Sections of nerve membrane characterized by threshold oscillations described in Sections XX-11 and XX-12 are coupled to each other to form a tree. The model tree is displayed visually so that the branches appear to brighten when an impulse invades them. The objective of this arrangement is to study the way the temporal pattern of impulses entering the tree is related to the distribution of impulses within the tree. We assume that threshold changes exert influence on the invadability of branches. Each branch in the model tree has an independent threshold time function that will depend on its own history of invasion. Thus, following an invasion the probability of a second invasion down the same branch becomes a function of time. It is this time dependence that guarantees that the path any particular impulse takes through the tree depends on its timing with respect to impulses preceding it. Since the information being handled by the axon is encoded in the temporal pattern of its pulses, a tree of fallible branches made of model nerve membrane has the following interesting property: Different functional connections will exist between a fiber and postsynaptic elements depending on the information in the fiber.

We believe this notion is the basis for understanding how the nervous system reads information in pulse trains. We are attempting to evaluate the information-handling powers of presynaptic arborizations to determine what sort of operations they can perform. We have recently expanded on the model of the tree to include postsynaptic elements, and we are investigating the throughput of this system for various families of input impulse patterns.

This work was undertaken with Paul Pangaro, an undergraduate student in the Department of Humanities, M.I.T.

14. Conduction of Impulses in Frog Peripheral Nerves

National Institutes of Health (Grant 5 PO1 GM14940-07)

S. A. Raymond

Evidence for impulse conduction block near bifurcations is accumulating. Charles King, an undergraduate student, and I have begun to use intra-axonal electrodes to study the relationship between threshold of the membrane and conduction block at the branch of the unipolar axon in frog dorsal-root fibers. Threshold functions will be compiled by testing threshold at various times after an impulse by current pulses down the microelectrode. In the second stage of this experiment measurements of threshold will be made during periods of natural activity in dorsal-root fibers.

15. Sources of Impulse-Dependent Threshold Changes in Axon Membrane

National Institutes of Health (Grant 5 PO1 GM14940-07)

S. A. Raymond

The projects outlined in Sections XX-11 and XX-12 have stipulated a considerable amount regarding the phenomenology of threshold oscillations. What is the nature of the processes underlying these oscillations? Do such changes in threshold depend on variations in conductance, on ionic pumps, or on "mechanical" relaxation constants in molecular sheets under stress? We are investigating these questions in two classes of experiments.

In the first, we are concerned with the effects of metabolic inhibitors on the depression and recovery phases. In order to determine the contribution of the membrane pump, strophanthidin and ouabain are added to the bath near the stimulating electrodes. Preliminary results indicate that both depression <u>and</u> recovery depend entirely on active transport.

The second class of experiments involves polarization of the membrane. By injecting both hyperpolarizing and depolarizing current into the axon, the conductance of the membrane is altered and the absolute threshold is changed. If changes in membrane conductance are responsible for activity-dependent threshold variations, the time course of the supernormal and depression phases with the addition of polarizing current will result in more than a mere displacement of the control threshold curve. Specifically, one might hope to attenuate or even reverse polarity of the supernormal phase. Results indicate that the magnitude of the supernormal phase is <u>not</u> particularly sensitive to the resting level of the membrane potential.

 The Role of Sodium, Potassium, and Calcium Ions in Activity-Dependent Threshold Shifts

National Institutes of Health (Grant 5 PO1 GM14940-07)

M. Binder, S. A. Raymond

Our working hypothesis is that the supernormal phase stems primarily from the

combined effects of reduction of K^+ conductance and the residual low level of sodium inactivation following the after-hyperpolarization of the impulse. Polarization of the membrane can reveal only that conductance change mechanisms are important during the supernormal phase, but taken alone it cannot indicate how conductance varies for each ionic species during this period. In order to determine which conductances are important, polarization with accompanying changes in ionic concentration is often revealing. Our preliminary observations indicate that when potassium-free Ringer's solution is substituted for normal Ringer's solution, the supernormal phase is accentuated. Depletion of extracellular K^+ generally results in hyperpolarizing of the mem-

brane, reduction of Na⁺ inactivation, and decreased K⁺ conductance. Thus, the initial observations offer mild support to the hypothesis.

Sodium inactivation is extremely sensitive to calcium ion concentration, and we are beginning to study the way altered Ca^{++} concentration affects the threshold oscillations.

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 Nerve Spike Conduction Block in Crayfish Motoneuron Axon Branches

National Institutes of Health (Grant 5 PO1 GM14940-07)

S. A. Raymond

Working with the deep extensor muscles (abdominal medialis, lateralis) in the cray-

fish, I. Parnass¹ demonstrated conduction block for impulses in motor nerves to deep muscles. By recording intracellularly from the medial and lateral superficial extensor muscles of the crayfish, Scott Bammann, Joel Franck (undergraduate students) and I have studied presynaptic impulse block in motor nerve axons innervating these muscles. Presynaptic blockade could be easily distinguished from synaptic "depression" since it occurred suddenly, often during an early period of stimulation when strong synaptic facilitation of the end-plate potential was present. When release of the presynaptic block occurred, the end-plate potentials showed immediate facilitation. These observations make it highly unlikely that the sudden extinction of the end-plate potential during long trains of stimulation of the extensor motor nerve represented synaptic depression. Depression was frequently observed to be characterized by a gradual diminution of the end-plate potential following a period of sustained response. Furthermore, extinction of the end-plate potential during depression was never so complete as during conduction block. Recording the motor nerves in intact crayfish showed that they frequently carry impulse trains in patterns that would be expected to generate conduction block. A manuscript is in preparation.

References

1. I. Parness, J. Neurophys. 35, 903-914 (1972).

 Development of an External Reed Larynx for Rehabilitation of Laryngectomized Patients

Bell Telephone Laboratories, Inc. (Grant)

D. W. Schoendorfer, S. A. Raymond

In the United States, approximately 6,000 people undergo surgical removal of the larynx each year, generally for treatment of cancer. The trachea is terminated at a stoma at the bottom of the throat, and all normal vocalization is lost. Several forms of therapy are available; none is wholly satisfactory. Esophageal speech is understandable but, because of its tonal quality, it carries social stigma. It is also difficult to learn. External vibrators held against the throat also permit understandable vocalization, but the sound produced colors the voice with a monotone mechanistic quality. Dynamic range is small and pitch change is virtually impossible. A third main form of therapy makes use of an external air column introduced into the pharynx (air tunnel method). If the appropriate vibrating air column plays into the pharynx, astonishingly normal speech can be produced. Although this therapy results in superior speech, it has not been much employed clinically.

We have built a mechanical testing station for evaluation of various silicon polymers for use as artificial "vocal cords." We believe that considerable clinical benefits will follow the perfection of an external "larynx" capable of modest pitch change and a wide dynamic range. Dr. Shedd has nine patients with external reed appliances, and the results obtained with these units are extremely impressive. We have designed an entire prosthetic unit, but the critical feature is the scheme used to originate the vibrating air column. We have nearly completed an engineering effort to optimize the vibrating element. Materials of varying elastic modulus and hardness were molded into several main classes of shapes and subjected to vibration analysis in a carefully regulated air stream. Several of the best models have been evaluated clinically. They show great dynamic range and reliability.

This work was undertaken in collaboration with Dr. Donald Shedd, Chief of Head and Neck Surgery, Roswell Park Memorial Hospital, Buffalo, New York.

19. Design and Construction of a Portable Adjustable Hearing Aid

National Institutes of Health (Grant 5 PO1 GM14940-07)

S. A. Raymond

It is nearly impossible to obtain a hearing aid that does the job. Most aids attempt flat amplification with frequency, and succeed in amplifying rather well the frequencies between 500 Hz and 5,000 Hz. This often results, however, in amplification of external noise more than desired sounds. Deaf children often show residual hearing, and with the use of hearing aids can learn to speak; but since much of the voice quality is in the lower frequencies, the speech of children using aids that do not amplify these frequencies is sometimes unintelligible. Despite their poor performance at low frequencies, aids now in use are affected by low frequencies; intermodulation distortion is commonly great enough to degrade seriously speech intelligibility even for people with normal hearing who try such aids.

As an M. I. T. Undergraduate Research Opportunities Program project, three students, J. Ringo, D. Zimmerman, and J. Liebschutz, constructed a portable hearing aid using earphones with a nearly flat amplification from 50 Hz to 15 kHz. Since most people have hearing losses that are greater at some frequencies than at others, we planned our aid to incorporate a filter to allow output power of the aid to be tailored to the user's hearing deficiencies (or to his preferences). We have designed a ten-band filter with high adjacent band rejection using parallel Butterworth filters and a mixer. Separate attenuation and amplification is available for each band. Since each ear often has a different hearing-loss contour, we have independent contouring for each ear with two independent amplifying systems. The system will be ready for clinical evaluation in a few more months. .