most of them being on the vast island of New Guinea, which comprises 330,000 of the 371,000 square miles of land in the conference area.

Financial support for the commission comes from the member governments, contributions being fixed by the agreement which created the commission. Contributions to the annual budget are made as follows: Australia, 30 percent; France, 12½ percent; the Netherlands, 15 percent; New Zealand, 15 percent; the United Kingdom, 15 percent; the United States, 12½ percent. The budget for 1950 is slightly in excess of 144,000 pounds sterling.

Technical assistance to the various territories in its area is of vital concern to the commission. Although the organization is entirely separate from the United Nations, liaison is maintained with the UN and such of its affiliated and subsidiary bodies as will have common concern in the South Pacific. This is particularly true in regard to the World Health Organization, the Food and Agriculture Organization, and the Indo-Pacific Fisheries Council. The S.P.C. is patterned after its successful prototype in the Western Hemisphere, the Caribbean Commission, several of the same member governments being concerned.

Now that the South Pacific Commission has gone through its organizational period and has established a concrete program of activity, it can be expected that there will be an accelerated attack on the problems of the peoples of the South Pacific. Within the present year results should be increasingly evident, although many of the problems of the area are of such a nature that solving them will require cumulative effort over a period of years rather than months. The significant fact is that six nations are working cooperatively to meet the common needs of the island peoples in the territories of the South Pacific in which they hold political responsibilities.

Technical Papers

The Recovery of Auditory Nerve Action Potentials after Masking

Joseph E. Hawkins, Jr. and Michael Kniazuk
Merck Institute for Therapeutic Research, Rahway, New Jersey

The action potentials of the auditory nerve as recorded from the round window of the cochlea in the anesthetized cat or guinea pig are subject to masking by other sounds. This is most easily demonstrated in the response to clicks of low intensity, where the action potentials are well developed and sufficiently separated in time from the aural microphonic for the two components to be readily distinguished. In the presence of a tone or noise of suitable intensity the neural component is immediately reduced in amplitude, whereas the microphonic remains unaffected.

Masking has been explained by Stevens and Davis (*6*) as a necessary consequence of the properties of the auditory nerve: the masking sound excites the nerve fibers and keeps them refractory, so that they are unable to respond synchronously to the clicks. The authors qualify this explanation, however, by stating that such a "line-busy" effect is not necessarily the only mechanism involved in masking.

We have recently observed that when a brief noise or tone is sounded during stimulation of the ear by a train of clicks, the action potentials of the click response, which must represent the sum of the activity in many fibers, recover their full amplitude only after 0.1 sec or more, a period far in excess of the maximum possible duration of the refractory period of the nerve fibers. Here, then, is a phenomenon which must represent a continuation of the masking process, but which cannot be explained on the basis of the refractory state alone. In

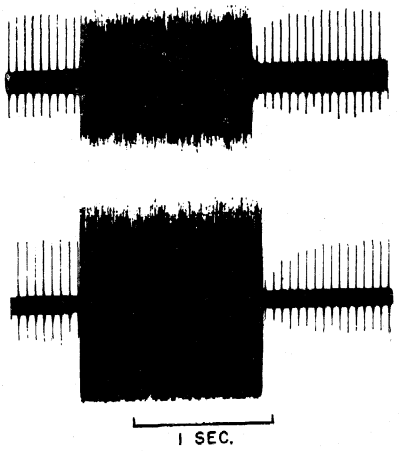

FIG. 1. Photokymograph records of recovery of action potentials of click response from round window after masking by thermal noise for 1.3 sec. Above, noise at 50 db relative to 0.0002 dyne/cm²; below, noise at 70 db. Clicks about 30 db above threshold for action potential. Cat, pentobarbital anesthesia.

Fig. 1 the action potentials return to full size approximately 0.3 sec after a thermal noise[1] of 50 db (as measured at the cat's ear by the General Radio sound level meter) has been turned off, and approximately 1 sec after a noise of 70 db. The rate of recovery after masking at 50 db is independent of the duration of the noise and is unaffected by repetition. When the intensity of the masking sound is increased recovery becomes slower,

[1] That is, noise of wide frequency spectrum, often called "white" noise.

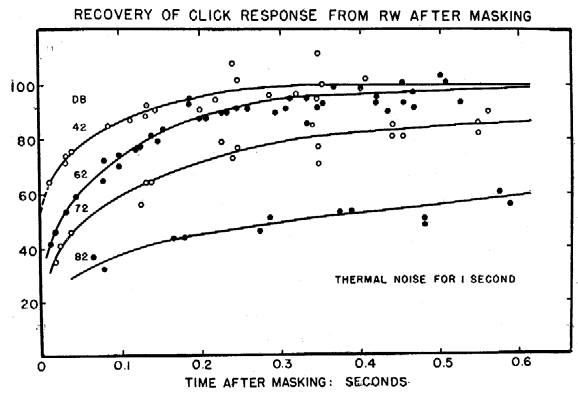

FIG. 2. Recovery of action potentials of click response from round window after masking by thermal noise at levels indicated for 1 sec. The points are plotted from measurements of series of 2 to 5 records similar to those in Fig. 1. Average height of responses immediately before masking taken as controls. Cat, pentobarbital anesthesia.

as illustrated in Fig. 2. Above 70 db there is a cumulative impairment of the response, and an increase in the duration of the masking further delays recovery. After exposure to noise at 95 db for 1 min the neural component is temporarily eliminated and even the microphonic is slightly reduced in amplitude. In 3 to 4 min, however, both components recover completely.

Tonal stimulation with frequencies in the vicinity of 4000 cycles per sec is particularly effective, both in masking the click response and in causing delayed recovery after masking. This may mean only that the click activates predominantly those nerve fibers which respond to such high frequencies; on the other hand, it is in accord with the known sensitivity of the human ear to the injurious effects of this portion of the audible spectrum. After exposure to 4000 c at 112.5 db for 1 min, the microphonic is reduced by approximately 30% and the action potentials are abolished for at least ½ hr.

Since the refractory period of the auditory nerve fibers, which is estimated by Davis (*6*) as approximately 1 msec, is far too short to account for the slow recovery of the action potentials after masking, we were at first inclined to attribute this delay to the time required for relaxation of the intra-aural muscles, which we supposed to be reflexly contracted in response to the masking sound. This explanation is incorrect, however, for two reasons: 1) At moderate levels of masking only the action potentials recover slowly, whereas the microphonic remains at full size throughout; both components should be affected in the same way by a change in tension of the intra-aural muscles. 2) When the guinea pig gives a brief contraction of these muscles during stimulation with a low tone and a train of clicks, the microphonic response to the tone is temporarily suppressed but the responses to clicks show little if any change in amplitude. This is illustrated in Fig. 3, where the effect of a spontaneous contraction is compared with the masking action of noise at 70 db. From this one may conclude that the intra-aural reflex causes little or no loss in the transmission of the click stimulus across the middle

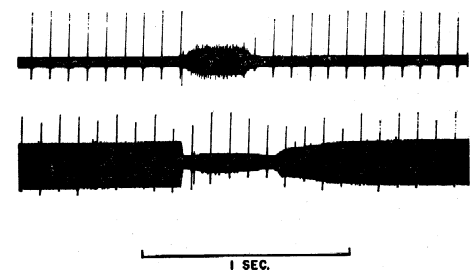

FIG. 3. Above, recovery of action potentials of click response from 3rd turn of cochlea after brief burst of noise at 70 db. Below, effect of spontaneous contraction of intra-aural muscles on responses to 500-cycle-per-sec tone and train of clicks, both continuous throughout record. Guinea pig, pentobarbital anesthesia.

ear and therefore cannot be responsible for the slow recovery of the action potentials after masking.

Our observations, which indicate that masking is not a matter of maintained refractory state alone, unfortunately do not reveal the nature of the other factors concerned in masking. Intracochlear inhibition as described by Galambos (*3*) may be involved, but can scarcely account for the prolonged effect of more intense sounds. The change need not reside in the nerve fibers, but may represent a temporary depletion or "equilibration" (*2*) of the mechanism which transmits excitation from the hair cells to the fiber terminals, so that excitation becomes inadequate for most of the fibers previously responding to the clicks. This mechanism appears from other evidence, such as the remarkable sensitivity of the auditory nerve to anoxia (*6*), to be the vulnerable link in an otherwise rugged system.

The more lasting reduction of the neural response after intense stimulation is probably the electrophysiological correlate of auditory fatigue or temporary deafness in man after exposure to loud sounds.[2] Acoustic injury has previously been studied in animals only after exposures to intensities sufficient to affect the microphonic response or to cause structural damage to the organ of Corti (see references *1*, *4*, *5*, *7*, among others). In its reversible form it now appears to be an extension in time and in degree of the process underlying auditory masking.

References

1. DAVIS, H. *et al.* *J. gen. Psychol.*, 1935, **12**, 251.
2. DERBYSHIRE, A. J. and DAVIS, H. *Amer. J. Physiol.*, 1935, **113**, 476.
3. GALAMBOS, R. *J. Neurophysiol.*, 1944, **7**, 287.
4. LURIE, M. H., DAVIS, H., and HAWKINS, J. E., JR. *Laryngoscope*, 1944, **54**, 375.
5. RÜEDI, L. and FURRER, W. *Das akustische Trauma.* Basle: S. Karger, 1947.
6. STEVENS, S. S. and DAVIS, H. *Hearing.* New York: John Wiley, 1938.
7. WEVER, E. G. and SMITH, K. R. *J. exp. Psychol.*, 1944, **34**, 239.

[2] A more intensive study of this aspect of the phenomenon has been made by Rosenblith and Galambos following their independent discovery of it at the Harvard Psycho-Acoustic Laboratory. Their findings and ours were reported at the November, 1949 (St. Louis) meeting of the Acoustical Society of America.