SENSORY MASKING—A PERIPHERAL OR A CENTRAL PHENOMENON?

DAVIS and Derbyshire[1] have recently reported that "Auditory masking is the diminution of audibility of one sound caused by the presence of a second sound. Study of the electrical responses of the cochlea and of the auditory nerve of the cat shows that this phenomenon depends primarily on the refractory period of the nerve fibers." In other words, nerve impulses initiated by one sound fail to reach the sensorium because blocked by the refractory periods following the passage of impulses generated by another sound; the phenomenon is *peripheral,* depends upon the physiological properties of the nerve fiber and is of the nature of a functional block.

It has been observed in this laboratory[2,3,4] that if, in a conscious person, a cutaneous nerve be stimulated through the skin with a suitable alternating current, a characteristic tingling is felt in the cutaneous area supplied by the nerve, and perception of touch, pressure and pain in that area is dulled. Possibly here, similarly, impulses initiated by the stimuli applied to the skin may be blocked by the refractory periods set up by the electrical stimuli applied to the nerve. But we have come to favor the suggestion offered many years ago by Robert,[5] in explanation of the local benumbing effect of certain electric currents, that the diminished perception results not from anesthesia or blocking, but from diversion of attention or confusion of consciousness; this supposed *central* phenomenon Robert termed "anesthésie de diversion," or "masking." In the spring of 1934 Lorin W. Denny, James C. Luce and I tested this matter upon ourselves; I described the experiments before the Association for Research in Nervous and Mental Disease in New York on December 27, 1934, and a detailed account is almost ready for publication; in view of the interesting work of Davis and Derbyshire, the following brief summary seems timely.

A cutaneous nerve, such as the radial, being completely blocked with procaine,[6] its cutaneous area was quite insensible to all forms of sensation; but the sense of deep pressure remained beneath the anesthetized skin, the impulses ascending from muscles, etc., through other nerves, *e.g.,* the ulnar (Fig. 1). The threshold for this deep pressure was measured

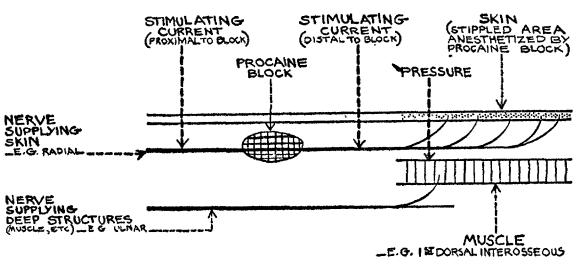

FIG. 1. Diagram illustrating the procaine block experiments described in the text.

(through the unfeeling skin) by the method described by Thompson *et al.*[4] Stimulation of the cutaneous nerve *distal* to the block evoked no sensation, and did not substantially alter the deep pressure threshold. Stimulation *proximal* to the block, however, not only aroused a normal tingling sensation projected into the anesthetized cutaneous area, but significantly elevated the threshold for deep pressure beneath that area, even though the nerves conducting impulses from the deep structures were affected neither by the anesthetic nor by the stimulating current. Moreover, in this type of experiment, stimulation of any nerve in the forearm or hand similarly raised the threshold for deep pressure beneath the anesthetized skin. It seems that perception of deep pressure (free from touch and superficial pressure) may be reduced, without interfering with the end-organs or the nerve involved, by suitably stimulating another nerve, if the impulses flowing from this stimulation succeed in reaching the sensorium and arousing a *sufficiently intense* sensation. We infer that the diminished perception in these experiments depends upon a central effect: to this we have applied Robert's term "masking," using it in his sense.

The important investigation of Davis and Derbyshire indicates that the difficulty in hearing a click simultaneously with a tone depends upon a peripheral effect—a physiological block. To call this "masking" introduces confusion into the literature, "masking" having already been preempted[2,3,5] to designate a *central* phenomenon, postulated by Robert, and demonstrated (we think) by us. It is to be hoped that some other term will be applied to the *peripheral* phenomenon elucidated by Davis and Derbyshire.

I. MACLAREN THOMPSON
UNIVERSITY OF CALIFORNIA
BERKELEY

[1] H. Davis and A. J. Derbyshire, *Proc. Amer. Physiol. Soc.,* April 10–13, 1935, p. 34; also reported in SCIENCE (Suppl.), 81: 6, 1935.
[2] I. M. Thompson and V. T. Inman, SCIENCE, 77: 216, 1933.
[3] I. M. Thompson, *ibid.,* 78: 268, 1933.
[4] I. M. Thompson, G. F. Banks, A. Barron, A. M. Fratis, Jr., and B. F. Mattison, *Univ. Calif. Publ. Anat.,* 1: 167, 1934.
[5] Robert, *L'Union Médicale,* 12: 487, 1858.
[6] Kindly donated by Eli Lilly and Company.

DRINKER RESPIRATOR PATENTS HELD INVALID

THE District Court of Massachusetts has recently held invalid three patents numbered 1,834,580, 1,906,453 and 1,906,844, granted to Philip Drinker and Louis A. Shaw in an infringement suit brought

against John H. Emerson by Warren E. Collins, Inc., manufacturers of hospital equipment in Boston, who were sole licensees under the patents. The defendant, John H. Emerson, maintains a machine shop in Cambridge for the manufacture of hospital and research equipment, and began the manufacture of respirators in 1931.

The patents covered the construction of apparatus for producing prolonged artificial respiration, especially for human beings suffering from infantile paralysis as well as in cases of gas poisoning, electric shock, drowning. These respirators worked admirably and were in great demand since 1929 for use in hospitals.

The court found that the defendant began to make his respirators after having seen the Drinker respirator. The defendant, however, excused his infringement on the ground that the patents were invalid because all the alleged novel features covered by the patents were old and disclosed in the prior literature.

After considering all the evidence and prior publications produced by the defendant the court agreed with the defendant that the Drinker patents were invalid. The important features of the patent claims of all three patents were found to be anticipated by prior publications, some as far back as 1876, or by prior issued patents.

The patent statutes permit the defendant in an infringement suit to excuse his acts if he can definitely prove that the patent or patents on which the suit is brought are invalid on account of prior knowledge, use or publication more than two years before the filing date of the patent application on which the patent was granted.

It is the duty of the Patent Office to make an investigation of prior practices and publications before granting patents, but owing to limited facilities, funds and personnel such investigations are not always thorough. Of course it must be borne in mind that defendants in infringement suits often employ experts at a cost of thousands of dollars who spend many months in order to uncover prior practices or obscure publications to anticipate issued patents. It is impossible for the Patent Office to make such thorough investigations at present on account of the larger number of patent applications filed each year. In the fiscal year 1934, for example, there were 60,363 patent applications filed in the Patent Office.

The patent statutes fortunately provide for the invalidation of erroneously issued patents, because it would be an injustice to enjoin the use of those apparatus, processes or compositions which are within the domain of public knowledge and hence free for any one to use for any desired purpose. The courts will therefore sustain and enforce only those patents which cover new and original inventions not known or used before by others within the statutory time limitation previously mentioned.

JOSEPH ROSSMAN

WAUSAU, WISCONSIN

ASCORBIC ACID IN CATARACT WITH SPECIAL REFERENCE TO DINITROPHENOL CATARACTS

THIS is a preliminary report of success in the treatment of the toxic effects of dinitrophenol, especially cataracts, and of cataracts of all types with ascorbic acid, vitamin C.

The presence of vitamin C in the normal lens in fairly high amounts has been confirmed by several workers, including Birch, Harris and Ray, Muller and Buschke, Gurewitch and others. With advancing age and with cataract formation the vitamin C content of the lens diminishes, and in some cataractous lenses may be entirely absent. The drop in the vitamin C content of the cataractous lens has been observed to parallel the drop in its glutathione content, as determined by Goldschmidt, Jess and Shoji, and also parallels the drop in its oxidative activity. Birch and Dann have adduced evidence that cevitamic acid and glutathione form part of the auto-oxidation system of the cell.

These data suggested the use of vitamin C in the form of ascorbic acid in cataract. Slit-lamp microscopic studies revealed that within a few days after administration of from 0.015 gm to 0.30 gm per day to patients with cataracts, marked improvement took place in the pathologic picture. First the swelling of the lens capsule was reduced and it became more transparent; and progressively the cells in the deeper layers improved in the same manner. Within less than a week of treatment mature and hypermature cataracts became sufficiently transparent to permit of examination of the eye-grounds, and vision improved from total or sub-total blindness to counting fingers. Cataracts involving vision less seriously responded at an equal pace, though in no case has the treatment been administered sufficiently long to determine whether the opacity of the lens will undergo total resolution.

The response of the very rapidly progressive dinitrophenol cataracts to ascorbic acid therapy was surprisingly rapid. When the treatment was intermitted, the cataract regressed; and progress was resumed with the resumption of treatment. Treatment was supplemented by food rich in glutathione content, in the hope that this element might also be replenished in the lens tissue, though our knowledge of the metabolism of glutathione is still so inadequate that there is no definite information available on this topic.