is not as great as might appear, for it may be safely assumed that the severity of the nerve lesion is such as to require operation. In all cases met with, the operation advised was never found to be unnecessary. With regard to injuries to mixed nerves with only partial cutaneous sensory loss, the muscles innervated by the affected nerve seldom fail to react on or below No. 10 stop. If reacting on No. 8 or below, recovery almost invariably ensues without operation. Cases in which the majority of muscles react on No. 10 should be examined every four weeks; if no improvement be apparent after the third examination, exploration should be advised. Some cases appear to remain stationary; in such instances operation usually reveals moderate involvement in scar tissue or a partial division of the nerve, and surgical treatment is followed by rapid improvement.

As regeneration proceeds, there is usually a progressive diminution in the capacity of the condensers required to evoke a contraction in the muscles supplied by the damaged nerve. When the condenser reactions improve progressively month by month, it may be safely assumed that recovery is taking place in spite of the continued absence of voluntary movement. When the reactions approach No. 8 stop from above, voluntary movement may be expected shortly to appear. If, after operation, no change in the condenser reactions takes place month after month, it is probable that the operation has been unsuccessful and that no recovery will occur.

It is not suggested that one should rely solely on quantitative condenser reactions; other important factors often have to be considered, such as the condition of the paralyzed muscles, the absence or otherwise of systematic treatment, sensory changes, etc. The purpose of the communication is to illustrate the value of condenser tests in assisting one to arrive at an opinion as to the extent of a nerve lesion, the nature of the treatment to be recommended, and the ultimate prognosis.

Author's Abstract.

TREATMENT.


The writer is here concerned with that form of vertigo which results from vasomotor or toxic (sympathetic or endocrine) disturbance of the vestibular apparatus, and for which, on examination, no organic cause can be found. Such vertigo is fundamentally a labyrinthine phenomenon, and its cause is irritative in character. The feeling of giddiness is usually as fleeting as the cause which produces it. Vasomotor stasis of the vestibular branch of the internal auditory artery, leading to a congestion of the semicircular canals, produces the pure form of vertigo, i.e., a sensation of giddiness which is unaccompanied by deafness or tinnitus. These latter do occur concurrently, however, if the vasomotor disturbance involves in addition the other (cochlear) branch of the internal auditory artery.
Vasomotor disturbances of the labyrinth may be induced by (1) disorders of the middle and internal ear, (2) abnormal sensory stimuli from any of the cranial nerves or nerves of the splanchnic system, and (3) lesions of the medulla, cerebellum, or cerebrum. The vertigo of the menopause, of chlorosis, of Graves’ disease, of gout, etc., is traceable to vasomotor disturbances which are secondary to a disordered state of the internal secretions.

The author points out that, associated with an attack of vertigo, there are usually nausea, vomiting, sweating, dilatation of the pupils, and various vasomotor symptoms. These occur also in conditions where there is marked intestinal irritation, and are, in fact, well-recognized effects of sympathetic activity (sympathicotonia). Vertigo, he insists, must be regarded in this light, i.e., as one of the several results of a generalized activity of the sympathetic nervous system. The evanescence, variability, abrupt appearance, and disappearance of vertigo, all point to a confirmation of this view.

The general arterial tension in this form of vertigo may be either increased or decreased—it is immaterial which, the important fact being that vertigo occurs as soon as the vasomotor equilibrium in the labyrinthine apparatus is disturbed. As a rule the disturbance is a vasomotor congestion rather than an ischaemia.

**Treatment.**—Quinine, salicylates, and opium are well-established remedies which lead to a general lowering of arterial tension. The drug *par excellence*, however, which is of value in sympathetic disequilibrium is adrenalin. For four years the author has administered it for this type of vertigo. He gives from 5 to 20 min. of the 1–1000 solution twice a day by the mouth. At the end of every ten days the drug is withheld for a short period in order that toxic effects may be avoided. The results have been encouraging.

W. JOHNSON.


The writer classifies individuals into two groups—the vagotonic and the sympathicotonic. The former are hypersensitive to pilocarpine and atropine, and the latter to adrenalin. The former give a normal or exaggerated response when the oculocardiac reflex is tested; and the latter none at all, or perhaps an inverted response. In appearance the vagotonic are pale subjects with cold bluish hands and small pupils. The pulse is sometimes slow, but more frequently the heart-beat is irregular and numerous extrasystoles occur. There is excessive gastric secretion, hyperchlorhydria, constipation, and a condition of arterial hypotension. Sympathicotonic individuals, on the other hand, exhibit marked vasomotor instability (described as waves of heat, etc.), the pupils are dilated, there is tachycardia, dilatation associated with atony of the stomach, hyperæsthesia of the epigastrium, and a definite rise in blood-pressure. There may be attacks of forcible pulsation of the abdominal aorta, attended with considerable discomfort (solar crises).
In such conditions of hypertonia of the sympathetic, the administration of eserine has produced beneficial results. The tachycardia especially is controlled and intestinal activity is promoted. Neutral eserine salicylate is the preparation which the author favours, and, if digitalis is given in combination, the best results are obtained. The dose of the eserine salt has been 10 min. of a 0·1 per cent solution three times a day by the mouth. No adverse effects are produced by this amount. The sulphate and the hydrobromide are not so reliable. In organic visceral disease and conditions of colic, eserine is contra-indicated and atropine should be given.

W. JOHNSON.


**Borico-potassium tartrate or soluble cream of tartar** has long featured in materia medica. The authors have found it to be the most efficacious way of prescribing boric acid for epilepsy. It is not intended to displace potassium bromide, but only for use with patients who show an intolerance towards this drug. Out of a hundred cases of epilepsy, ten patients were selected who were intolerant to potassium bromide. Ordinary borax, sodium tetraborate, and borico-potassium tartrate were the preparations of boric acid experimented with. Both the number and the intensity of major and minor attacks were lessened in every case. A second similar series of ten cases showed the same result. The improvement remained sustained up to eight months. In one or two cases suspension of the drug led to an exacerbation of attacks, which a re-administration was successful in controlling.

The administration of borico-potassium tartrate may lead to digestive disturbances which, however, are slight and fleeting in character. It is usually given in a dose of 20 to 40 gr. Patients have appeared to tolerate it much better than the other salts of boric acid. An important feature is that there is less clouding of the mental faculties than occurs with bromides; also the reflexes are considerably less depressed.

The authors conclude by alluding to the beneficial effects which may follow the administration of potassium bromide, or of luminal, or of both, in conjunction with borico-potassium tartrate. Only experiment in individual cases will show which combination is the most suitable.

W. JOHNSON.
TREATMENT

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