During the high frequency stimulation the number of ganglion cells discharging gradually gets less. At the end of the period of stimulation there is not a sudden, further decrease in activity, but only gradually and over a period of 0.2 to 0.5 seconds do the cells go out of action. Then for the short space of 0.2 to 1.0 second there is no discharge, following which a certain amount of activity again develops and continues for 15 or more seconds. The intervening period of inactivity is apparently related to the phase of decreased irritability revealed by the positive after potential following the period of intense activity.

It is not possible to say what proportion of the ganglion cells participate in this after-discharge or at what frequency, until we have many records of the activity of small groups of cells. For only thus can individual impulses be observed and quantitative measurements made. At the present time we can estimate that in the initial phase the number of cells in action may well be 25% of those responding at the beginning of the period of stimulation. Several seconds after the end of the stimulus the activity falls to 5 or 10%.

How many units continue in action is probably of less significance than the fact that certain of them do continue to discharge for 15 or more seconds after the end of the trains of impulses which developed the excitatory state at the synapse. We are, therefore, forced to conclude that the altered state of the nerve cells or the altered state of their environment is capable of persisting for many seconds at a level which causes the rhythmic discharge of impulses from the cells.

10066 P

Relation of Potassium to Family Periodic Paralysis.

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The older literature on family periodic paralysis contains observations which might suggest a relationship of disturbed K metabolism to the attacks. K citrate was thought by Mitchell[1] to have a

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1 Mitchell, J. K., Flexner, S., and Edsall, D. L., *Brain*, 1902, **25**, 109.

slightly beneficial effect on the seizures, and McCann[2] found descendants of Mitchell's patient still relying on the drug. Herrington[3] has recently stated that K citrate will prevent an oncoming seizure but will not cure an attack once developed. Furthermore, earlier Japanese investigators[4,5] showed that certain substances (adrenalin, sugar, insulin) which are now known to lower serum K would induce a seizure.

We were thus led to examine the level of serum K in a patient with this disease and have found a definite lowering (25 to 30%) during severe attacks. Aitken and Allott[6] independently observed a similar fall during severe seizures, but in milder attacks they found that the decrease was less, and at times not even detectable.

The experiments which are here reported have been conducted on a 16-year-old boy over a period of 15 months. Our purpose has been to gain information concerning the mechanism of the changes in serum K and their relation to the periodic attacks of the disease.

We have found no excessive elimination of K by the kidney preceding the seizures and have, therefore, sought some other cause of the lowering of serum K during the attack. The seizures generally developed at 3 to 4 A. M., and urinary excretion studies showed that at this time the absorption of K from the gut was quite low. Furthermore, the K taken in with the day's diet had been largely eliminated before midnight. We have, therefore, concluded that such an individual has an unusual need for K salts to maintain contractility of certain of his muscles.

Making up this need for K by administration of K salts early in the morning (4 g KCl at 2:30 nightly) has prevented the development of seizures or made them very much less frequent and severe. No such effect, however, has resulted from the administration of similar amounts of K during the day because it was eliminated before being required.

An attack which has already developed can be cured by the administration of K (5 g of KCl orally), an observation which is also reported by Aitken and Allott. In our case this procedure was effective even when the serum level was not low. On the other hand, several other bases involved in muscle metabolism, such as NaCl, $NaHCO_3$, creatine hydrate (15 g orally), and also prostigmin (1.5 mg hypodermically) failed to diminish the weakness.

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2 McCann, W. S., personal communication.

3 Herrington, M. S., *J. A. M. A.*, 1937, **108**, 1339.

4 Shinosaki, T., *Z. Ges. Neurol. u. Psychiatr.*, 1926, **100**, 564.

5 Yoshimura, K., *Z. f. d. Ges. Exp. Med.*, 1930, **70**, 251.

6 Aitken, R. S., Allott, E. N., Castleden, L. I. M., and Walker, M., *Clinical Sci.*, 1937, **3**, 47.

The manifestations of this disease are almost surely peripheral in origin for certain muscles are electrically inexcitable during an attack. Attention is thus directed to an abnormality of the muscle itself as a cause of the weakness. The facts so far known could be explained on the assumption that from time to time some alteration in the muscle arises which leads to inexcitability and which then requires additional K for its correction. This need is partially met by diffusion of K from serum into muscle as might be concluded from the fact that serum K falls in a severe attack. The increased requirement is not adequately met, however, unless K can be absorbed from the gut. McEachern's observation[7] that the intravenous administration of KCl in small amounts can partially relieve the seizure without raising the lowered serum K could be explained on the basis of direct diffusion of the administered K into the muscle with resulting relief of the weakness.

In this patient the need for extra K is more or less constant for he develops weakness whenever there is no source of available K in the gut. Indeed the urgency of this need is shown by the observation that the washing out of large amounts of K during a simple water diuresis results in a severe attack. Apparently he is unable to compensate for the reduced concentration of muscle salts which follows ingestion of large quantities of water. In other individuals who have infrequent attacks the metabolic change in muscle postulated above must occur only periodically.

The K taken into the muscle during the attack should, on this hypothesis, be eliminated after the abnormality has been corrected. Experiments to check this point are in progress.

10067 P

Immunological Properties of a Sonic Extract of Pneumococci.

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Formalin-killed, young cultures of pneumococci have been found by one of us (Weil)[1] to evoke infiltration when injected intra-

[7] McEachern, D., personal communication.

[1] Weil, A. J., and Phillips, S. W., *J. Immunol.*, 1937, **33**, 149.