

If, instead of recording the resulting motion of the finger, we record the swellings of the contracting muscle, we gain a deeper insight into the nature of the contraction. We have, in the first place, from such a record, that the activity of the muscles continues after the motion is over and slowly falls back into its quiescent state; and secondly, the innervation and rhythms leave their trace in the curve of contraction. Normal muscular contraction is generally considered to be tetanic, that is, it is not a simple muscular twitch, but a series of maximal contractions. The curve that the swelling of the muscle unites is not a simple up stroke followed by a down stroke, but is composed of several smaller curves of this nature; it shows a rhythm, and a quite constant one, of about ten to twelve per second. It is to be understood that when we move the finger once, however slowly, the muscle gives a series of rapid twitches; what we think is a single innervation is really a manifold series. And in the shortest possible movement there are about four such waves in the curve of contraction, each such wave consumes about one-fifteenth of a second, the curve, of course, continuing after the resultant motion has gone into effect.

Finally, in rhythmical movements, the smaller waves of contraction become variable within wide limits to about forty per second.

These results are confirmed and supplemented by the recent experiments of Horsley and Schäfer. They excited electrically the cortex, the underlying motor fibres, and the motor area of the spinal cord of dogs and other animals, and found the same result from each of these modes of stimulation, viz., a contraction rhythm of about ten per second in the muscle. They varied the frequency of the stimulus from ten to fifty per second, but found that the rhythm of contraction was (generally) independent of the frequency of the stimulation. They found, too, that the same holds for voluntary and reflex motions as for these artificially excited ones.

The most natural explanation of these facts is that the innervations are surmounted in the motor cells of the cord (probably), and are re-issued from those cells at a constant rate of ten per second, no matter at what rate these cells receive these impressions.

Dr. Schäfer has applied the same method of study to voluntary contractions in man with the same result.

We see, then, that there is an innervation rhythm in the nervous system itself, which limits the rate of our quickest motions, and which determines, independently of our will, the activity of our muscles. What to our consciousness is a simple motor act, is really a series of contractions rhythmically exploding themselves at the rate of about ten per second.

J. JASTROW.

PATHOLOGY OF NERVOUS SYSTEM.

"Paralysis of the Isthmus of Panama. Beriberi, Kakke." PIERRE MARIE. (*Progrès Médical*, p. 168, 1887.)

The author, in this article, after briefly reviewing the paralytic

symptoms occurring in beriberi, as described by Scheube in his monograph, goes on to describe a case of paralysis of the lower extremities occurring after a sojourn of several weeks on the Isthmus of Panama, which in the opinion of the writer is certainly a case of beriberi. Cases of this kind have been seen by Charcot, and one also by Hutinel. Marie's case is that of a male, *æt.* 37. Family history unimportant. Patient was in good health until the occurrence of this affection. On the 29th of August he arrived at Colon, where he remained during his entire stay on the Isthmus, with the exception of several visits to Fox River and Monkey Hill (places in which fever is very prevalent). Between the 20th and 25th of September he noticed that he was getting weak, had that his health was growing worse from day to day, although he was unable to assign the cause of his ill-health to any particular organ. The physician who saw him at that time said he was suffering from severe anæmia. During the last few days of his stay at Colon he was exceedingly weak, and fainted several times. During this time his abdomen was very much distended, and seems to have been the seat of an effusion of fluid. The patient also observed the existence of anæsthesia over the abdomen near the umbilical region, the anæsthetic part being of the size of about two hands. On the 11th of November he set sail for home (France). The first day on the ocean he noticed an abundant purpuric eruption on both feet, from tibio-tarsal articulations to the middle of the calves. The second or third day of the trip the legs became œdematous. Until this time no pain, and no special weakness of the legs. On the 22d of November, on attempting to rise, he noticed that both legs were paralyzed. Neither at this time nor subsequently was there any affection of the bladder. Seven or eight days after the occurrence of the paralysis in the legs, the patient noticed a well-marked weakness in both thumbs; the other fingers were not affected. This weakness lasted about two weeks and was not accompanied by any disorder of sensation. The paralysis of the legs began to improve about the 25th of December.

Examination of the patient showed atrophy and flaccidity of the muscles of the thighs. Paresis of certain groups of muscles of the lower extremities, and weakness of the abductors of the thumbs.

Reflexes were all normal, with the exception of the patellar tendon reflex, which was entirely absent. Mechanical excitability of the muscles seems decreased.

Sensation is almost normal, except at the lower part of the legs, where it is slightly diminished. No vaso-motor symptoms. Pupillary reflexes normal.

This case seems to differ from typical cases of beriberi in the rapid development of the paralysis, as well as in the short exposure (about ten weeks) necessary to its production; but both of these abnormalities have been exceptionally noticed by other observers.

The most interesting question is that of the geographical dis-

tribution of this affection. It is apparently new that beriberi is endemic at the Isthmus of Panama, and expressions of opinion by physicians living there would prove very valuable.

G. W. J.

Paralytic Ataxia of the Heart of Bulbar Origin. M. SEMMOLA. (*Gazette des Hôpitaux*, p. 881, 1886.)

In 1876, Semmola published his first observations upon the influence of the nervous system in the production of affections of the heart. In 1881, at the International Medical Congress of London, he clearly formulated his views upon the existence of a cardiac affection, slowly produced as a result of faulty innervation of the oblongata and of the cardiac ganglia. The present communication is the result of further observations upon this subject. All the patients observed were free from any rheumatic, gouty, or syphilitic taint. The results of S.'s observations may be summarized as follows: The prodromal period generally occurs between the forty-fifth and sixtieth years, that is to say, at a time when, in addition to the general impressionability of the nervous system, two other classes of functional disorders are apt to occur: those of the heart and those of the stomach. This period may last for two to three years, and is characterized by a weakness of the systole with an increase in the number of heart beats. This demonstrates simply an insufficient power of recuperation on the part of the nervous system, an exhaustion of the pneumogastric and of the ganglia which preside over the functions of the heart. During this prodromal period the affection is susceptible of a complete cure, if absolute rest and absence of exciting causes are procured. If, on the contrary, the causes persist, the affection enters upon the confirmed stage, curable in some cases, which is characterized by the following symptoms:

- a.* Palpitations. Dysystole. Respiratory difficulties.
- b.* Development of a mottled coloration of the hands, arms, and legs. There is no œdema. This is a purely neuro-paralytic stasis.
- c.* Respiratory uneasiness developed after the slightest exertion. Auscultation reveals the existence of crepitant râles at the base of the lungs; these are also due to a neuro-paralytic stasis.
- d.* Attacks of suffocation which awaken the patient and oblige him to sit up, when they soon disappear. These differ from the attacks of suffocation occurring in organic affections of the heart, by the absence of true dyspnœa, by their short duration, and by the impossibility of detecting any change in the heart itself.

Semmola believes that this respiratory difficulty is caused by the anæmia of sleep, during which condition the bulbar centres of respiration become still more exhausted until paralytic effects ensue.

- e.* Development of œdema in the feet. The dysystole becomes permanent, and all the grave symptoms of organic heart disease appear.

G. W. J.