

to the left arm was applied, and a week after the patient complained that though her left hand had next day regained its power the right hand had *ipso facto* become weak. The dynamometer showed a power of grasp of 40 k. with the left hand and only 18 with the right. The author's original observations had been made many years before those experiments upon hysterical hemi-anæsthesia in France, in which removal of the anæsthesia was found to be accompanied by its transfer to the other side. He urged that the phenomena pointed to a power of influencing in some way nervous centres by impressions upon the skin, and referred to some trials of a therapeutic character which he was basing upon the observations. His aim was in a case of aphasia to rouse into activity the posterior portion of the third frontal convolution of the *right* hemisphere by directing powerful impressions to adjacent grey matter by means of painful electrical currents applied to the tongue, mouth, and left arm.

J. HUGHLINGS-JACKSON.

Luederitz: Experiments on the Effect of Pressure on Motor and Sensory Nerves. (*Zeitschrift für Klinische Medizin*, Bd. ii., 1.)—In the introduction of his interesting paper, the author observes that a considerable number of cases have been reported in which peripheral mixed nerves were entirely cut in two without producing loss of sensation in the corresponding parts, the paralysis of the motor fibres of the nerve being at the same time complete. *Richet* noticed in a case of neurotomy of the median nerve, that when he happened to touch the peripheral end of the nerve the patient gave signs of violent pain. Evidently in this individual that part of the median nerve contained sensory fibres which were in connection with some other brachial nerves. A similar case of this "sensibilité récurrente" is reported by *Remak*; afterwards *Arloing* and *Tripier* proved by experiments on animals that many nerves contain sensory fibres derived from sensory nerves in the neighbourhood; these fibres run a little way in a centripetal direction, and then enter into the nerve to which they really belong.

These facts explain to a certain extent those well-known clinical cases in which we find complete motor paralysis without loss of sensation; but there are other cases which cannot be explained in this way, and therefore the author made a series of experiments on rabbits in order to ascertain whether the resistance to injury of sensory nerves is greater than that of motor nerves. He applied a

ligature to the sciatic nerve, and then stimulated with a faradic apparatus both the central and the peripheral part of the nerves at a few centimetres distance from the place where the ligature lay. The distance of the two coils, divided in millimètres, expressed exactly the intensity of the current which was wanted for the production of a contraction of corresponding muscles, viz. of a sign of pain in the animal. In order to avoid the influence of "sensibilité récurrente" the crural nerves as well as all other sensory nerves of the leg were cut so that there was no sensory communication between the extremity and the brain but the sciatic nerve.

The result of these experiments was as follows:—The conductivity of motor fibres is invariably impaired earlier than that of the corresponding sensory fibres. It was found several times that 10–15 minutes after the application of the ligature sensibility and motility disappeared simultaneously, but much more frequently the author stated that the strongest faradic current applied above the ligature did not produce the slightest muscular contraction, whereas peripheral stimulation immediately afterwards made the animal display symptoms of pain. Sometimes motor conduction was completely interrupted, sensibility being at the same time absolutely unaltered. On the other hand, loss of sensation without motor paralysis was not observed in a single case. When the ligature was removed in cases of complete motor and sensory paralysis, in some cases both faculties were restored at the same time; in other experiments sensation appeared to have returned, while motor paralysis persisted.

The only way to explain these facts is to assume that sensory nerves have a greater resistance against traumatic influences than the motor nerve-fibres. As a practical consequence, the author points to Duchenne's observation: that the prognosis of traumatic paralysis is much more favourable when, the electro-muscular contractility being extinct, muscular sensibility is unaltered or but slightly diminished.

Brieger: A Case of Paralysis from Fright. (*Zeitschrift für Klinische Medizin*, Bd. ii., 1.)—The popular idea that violent mental shocks are capable of producing severe illness has not gained much ground in scientific researches. Only lately some cases have been reported by Kohts and Leyden, giving indubitable evidence of disease caused by intense fright (during the siege of Strasburg in the late Franco-German war). Similar ob-

servations have been made by Todd, Hine, Pasque, Berger, but all these were deficient with regard to anatomical examination. Leyden only has made autopsies in two cases of spinal paralysis: in the first case he found sclerosis of the middle part of the dorsal region of the cord; in the second case there was diffuse sclerosis of the upper and middle part of the cord in the dorsal region. The following observation, made at Professor Frerich's clinique, will therefore attract the attention of pathologists.

A prostitute girl, 23 years of age, without hereditary disposition to nervous diseases, going home late at night from a dance, ran against a man who was lying drunk on the stairs. She managed, however, not to fall, by clinging to the handle of a door, and there was certainly no definite injury to the spine. Trembling with terror she mounted with difficulty to her room in the third story and tumbled on her bed, almost fainting. After a little while she recovered from her fright, undressed and went quietly to bed, without feeling anything abnormal. At 4 o'clock in the morning she awoke, in consequence of intense pressure of urine, but found herself incapable of passing any water. At the same time she felt violent shivering. Hot bottles and tea applied by the landlady did her no good; she therefore went into the warm bed of the latter, in doing which the patient observed a considerable weakness in her legs. She then went to sleep; when she woke at 7 A.M. she found her legs entirely paralysed, and at the same time had a sensation of numbness and tingling. A few days afterwards she was brought to the hospital. There was now complete paraplegia, pain in the lumbar region when she tried to sit up, sensation of cold and heaviness in both legs. Mental functions normal, great depression of spirits. No fever. Residues of syphilis, for which she had been treated at the same hospital two years before this accident. Absolute anæsthesia of the lower part of the body up to the height of the second lumbar vertebra. Retentio urine. The treatment consisted in application of 4 grammes of mercury ointment pro die, and 2 grammes of iodide of potassium internally. No improvement in the following days. On the 12th day sloughing began in the sacral region, extending rapidly. A few days later, symptoms of spinal meningitis came on. Patient died on the 27th day after her reception at the hospital.

The *autopsy* revealed diffuse myelitis, extending from the upper lumbar region to the level of the 8th dorsal vertebra, chiefly localised in the posterior columns and the parts of the white

substance adjoining the anterior roots. Upwards there was found secondary degeneration of the postero-median columns (Goll's columns) reaching as far as the fourth ventricle of the brain.

The author thinks that in this case there can be no doubt as to the cause of the disease; he does not believe that the syphilitic history of the patient has anything to do with it, because there were no symptoms of visceral syphilis, and the other residues of the infection had been quickly improved by the anti-syphilitic treatment. He only admits that the patient's resistance against disease had been diminished by the venereal infection. Concerning the way in which mental shocks act on the cord, the author suggests that the fright brought on a sudden intense contraction of the blood-vessels, lasting for a considerable time and leading to necrotic destruction.

R. H. PIERSON (Dresden).

Onimus on Modifications in the Excitability of Nerves and Muscles after Death. (*Journal de l'Anatomie et de la Physiologie*, 1880, No. 6.)—We know that after death muscles and nerves gradually lose their excitability. The vital functions are rapidly, almost instantaneously, lost as regards the brain, while in the spinal cord they persist for a short period, the grey matter losing its experimental excitability sooner than the white matter. With regard to the nerves, the loss of excitability does not, strictly speaking, occur in a regular order, commencing from central parts and gradually invading peripheral parts; the loss of excitability pervades the nerves of a limb as follows: first, the large cords; secondly, the filaments supplying extensor muscles; thirdly, the filaments supplying flexor muscles—a sequence that calls to mind common pathological events, e.g. dropped wrist. The author states that in the arm the radial nerve, in the leg the peroneal nerves are the first to lose their excitability.

The nerves of the ganglionic system preserve their excitability longest of all; the intestine has been seen to contract five or six hours after decapitation, and M. Robin elicited rhythmic pulsation of the right auricle many hours after death by distension of its walls. With regard to the muscles, their excitability during the first few minutes after death is enhanced; they are at their maximum excitability when the large trunks are inexcitable, their excitability is already on the decline when the smaller filaments are inexcitable. After a gradual death, which is an exhausting process, muscular farado-contractility is more rapidly lost than