

tain persons, but it is none the less true that this drug, in doses of eight or ten drachms, is sometimes found to produce a very marked depression of the circulation, bringing on a notable lowering of the cardiac pulsations.—*London Med. Record*, April 15, 1881.

Neurotic Atrophy.

Professor VIRCHOW (*Berl. Klin. Woch.*, No. 29, 1880) brought before the Berlin Medical Society two cases of neurotic atrophy, of the type to which he would apply the term "circumscribed," to distinguish from those cases of crossed and hemi-atrophy due to lesions of the nervous centres. One of the cases shown, a man named Schwahn, was the original case upon which Romberg founded his doctrine of tropho-neurosis, and of which Professor Virchow has notes taken by himself in 1859. The other case was a woman. In both the face was the principal part affected, the atrophy affecting one half. In the man's case, the bones were affected, because the disease began during development. In the woman, the bones were unaffected, the condition having commenced when she was 25 years old. Professor Virchow pointed out that the facts do not support the view that the atrophy depends on vaso-motor spasm; the vessels are easily seen, and the skin reddens very readily when rubbed; there is no definite alteration of sensation, though here and there slight numbness is complained of; there is no true paralysis of the muscles; they are only weakened by their bad nutrition. The affection does not involve the whole area supplied by particular nerves or branches of nerves, but only portions of those areas. The distribution of the lesions in both cases was described by Dr. Virchow. In the man's case, the origin of the condition was when he was nine years old; he had swelling of the entire neck and region at the angle of the lower jaw, which got well in a week; the skin at this part became pigmented, but in other places the atrophied patches are notably white. Dr. Virchow believes that the atrophy had made no progress since his former notes were taken in 1859. The woman's case began after a confinement, but the relation to the confinement is not clear, as it was six months or a year afterwards that she first noticed anything. As in Schwahn, the first symptom was inflammatory swelling, in her case near the left eye. A little after, she slipped down and struck the back of her head. There is no abnormality now to be found at the seat of the blow. Dr. Virchow refers to some published cases, in which a blow has been assigned as the exciting cause. In conclusion, he says that complete anatomical examination has never been made on one of these cases, and that there are few phenomena in the range of neuropathology which so much need explanation, or in which nature apparently offers so neat an experiment for distinguishing the different kinds of fibres in the peripheral nerves.—*London Med. Record*, April 15, 1881.

Atrophic Infantile Paralysis.

M. ONIMUS has just written a paper, entitled "Considerations on the Etiology and Diagnosis of the Atrophic Paralysis of Childhood," in which he asserts (*L'Union Médicale*, April 3d) that the examination of the very much weakened electro-muscular contractility by induced currents, and less frequently by continuous currents, is the only criterion by which to distinguish infantile hemiplegia, or the peripheric paresis of the muscular groups, from the atrophic paralysis of childhood, most generally due to changes in the cells of the anterior cornua of the spinal cord. M. Onimus proves, by cases observed, that chill is the most frequent cause of this disease, by directly bringing on congestion of the gray substance of the atrophic nerve-cells. In certain cases, however, the rheumatismal influence

primarily affects the muscles and the peripheric nerves, in which inflammation sets in at once. It is especially in the spring, summer, and autumn, more than in winter, that this disease originates, by transition from heat to cold, and from imprudences committed at this period of the year. M. Onimus is convinced that modifications of circulation in the divers organs of locomotion are more logically the cause of the atrophic paralysis of childhood, than dentition, heredity, breast-milk, or the so-called internal convulsions.—*British Med. Journal*, April 23, 1881.

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Paradoxical Muscular Contraction.

Professor WESTPHAL has given the above name to the phenomenon which he describes as follows (*Archiv für Psych.*, Band x.). If, in certain diseases of the central nervous system, we quickly and strongly flex the ankle of the patient while lying in bed (occasionally slow flexion will also succeed), we find that the foot left to itself remains in the position given to it, not sinking by the force of gravity into the natural position. On observing the tendon of the tibialis anticus, we find that a moment after the flexion it suddenly starts out and remains in this condition. Corresponding to the action of the tibialis anticus, the foot is adducted. The foot remains in this position for some time (on one occasion observed by Professor Westphal for twenty-seven minutes), and then sinks gradually into its natural position. Dr. Westphal named this phenomenon "paradoxical muscular contraction," because here the passive shortening of the muscle acts as a stimulus, and this method of producing a contraction is in direct opposition to the method already described by Erb and himself, viz., by sudden extension or concussion. Dr. ERLÉNMEYER adds (*Centralb. für Nervenheilk.*, 1880, No. 17) that he finds patients are unable by voluntary contraction of the antagonists to overcome this contraction of the tibialis anticus. He has seen the contraction last for three-quarters of an hour. As to the cause, Dr. Erlenmeyer believes that it lies, not in the relaxation of the tibialis anticus, but in the lengthening or extension of its antagonist, the gastrocnemius. To prove this, he instituted two experiments, in the first of which he considers that he changes the lengthening of the gastrocnemius into a shortening; in the second, that he prevents altogether the extension or lengthening of the muscle. To accomplish the first object, he flexes the knee, and pushes with the hand the muscle of the calf towards the heel, a proceeding he found possible for a distance of nearly an inch. The contraction of the tibialis anticus fails in every such experiment, he says, the foot falling directly into its natural position. To accomplish the second object, he uses either the same method as the last, or with the knee flexed as before, he pushes the calf-muscle so far towards the knee that the ankle can still be fully flexed without resistance. In both cases the contraction of the tibialis anticus fails. He considers that here we have a tonic muscular contraction produced by extension of an antagonist, and differing in character from the foot-clonus or "foot phenomenon" of Westphal, which results from passive restraint of clonic spasms in a muscle stimulated by its sudden extension. He proposes for it the name "muscle phenomenon." Dr. Westphal (*Centralb. für Nervenheilk.*, 1880, No. 20) replies to the above. He says it is not the pushing upwards or downwards of the calf-muscles that prevents the occurrence of the phenomenon, but the pressure on the muscles, which acts as a mechanical stimulus, producing extension of the ankle-joint. He had already weighed and rejected the propositions made by Dr. Erlenmeyer.—*Lond. Med. Record*, April 15, 1881.