

(6) *Endemic Neuritis; Beri-beri*.—This is a widely spread disease in parts of India, and in China and Japan. To Sheube and Baelz are due the credit of determining its true nature. It is probably due to a micro-organism. Food appears also to have a large share in its causation and it has been attributed to a fish diet. Some have thought it might be due to the presence of parasites in the intestines, but there are no grounds for this belief. There are several types of cases. In the acute pernicious form the nervous phenomena are not so marked. There are fever, anæmia, and general anasarca. In another group of cases there are numbness, loss of tendon reflexes, areas of anæsthesia, and muscular atrophy and anasarca. In other cases the paralysis and atrophy are the most prominent symptoms and the clinical picture is that of a rapidly progressing multiple neuritis with sensory and motor disturbances. The mortality varies from three or four to fifty per cent. Great difference of opinion still prevails concerning the cause of the disease. Special interest has been aroused in the subject in this country, owing to the fact that J. J. Putnam has described a similar disorder among the New England fishermen who frequent the Grand Banks. It occurs in epidemic form, and has, as prominent symptoms, general cedema, shortness of breath, and sensory disturbances with paralysis. In other instances, the paralysis is more extensive and proves fatal. In 1881 and 1889 there were epidemics among the crews of vessels fishing in this region. Birge describes eleven cases which occurred on one vessel in a crew of thirteen, two of whom died. One patient of this crew I saw with F. C. Shattuck, in the Massachusetts General Hospital, with the well-marked symptoms of multiple neuritis. The disease also exists in the West Indies, whence cases have come to this country (Seguin).

**Diagnosis.**—The electrical condition in multiple neuritis is thus described by Allen Starr: "The excitability is very rapidly and markedly changed; but the conditions which have been observed are quite various. Sometimes there is a simple diminution of excitability, and then a very strong faradic or galvanic current is needed to produce contractions. Frequently all faradic excitability is lost and then the muscles contract to a galvanic current only. In this condition it may require a very strong galvanic current to produce contraction, and thus far it is quite pathognomonic of neuritis. For in anterior polio-myelitis, where the muscles respond to galvanism only, it does not require a strong current to cause a motion until some months after the invasion."

"The action of the different poles is not uniform. In many cases the contraction of the muscle when stimulated with the positive pole is greater than when stimulated with the negative pole, and the contractions may be sluggish. Then the reaction of degeneration is present. But in some cases the normal condition is found and the negative pole produces stronger contractions than the positive pole. A loss of faradic irritability and a marked decrease in the galvanic irritability of

the muscle and nerve are therefore important symptoms of multiple neuritis." \*

There is rarely any difficulty in distinguishing the alcohol cases. The combination of wrist and foot drop with congestion of the hands and feet, and the peculiar delirium already referred to, is quite characteristic. The rapidly advancing cases with paralysis of all extremities, often reaching to the face and involving the sphincters, are more commonly regarded as of spinal origin, but the general opinion seems to point strongly to the fact that all such cases are peripheral. The less acute cases, in which the paralysis gradually involves the legs and arms with rapid wasting, simulate closely and are usually confounded with the subacute atrophic spinal paralysis of Duchenne. The diagnosis from locomotor ataxia is rarely difficult. The *steppage* gait is entirely different from that of tabes. There is rarely positive incoördination. The patient can usually stand well with the eyes closed. Foot-drop is not common in locomotor ataxia. The lightning pains are absent and there are no pupillary symptoms. The etiology, too, is of moment. The patient is recovering from a paralysis which has been more extensive, or from arsenical poisoning or has diabetes.

**Treatment.**—Rest in bed is essential. In the acute cases with fever, the salicylates and antipyrin are recommended. To allay the intense pain morphia or the hot applications of lead water and laudanum are often required. Great care must be exercised in treating the alcoholic form, and the attendant must not allow himself to be deceived by the statements of the relatives. It is sometimes exceedingly difficult to get a history of spirit-drinking. In the alcoholic form it is well to reduce the stimulants gradually. If there is any tendency to bed-sore an air-bed should be used or the patient placed in a continuous bath. Gentle friction of the muscles may be applied from the outset, and in the later stages, when the atrophy is marked and the pains have lessened, massage is probably the most reliable means at our command. Contractures may be gradually overcome by passive movements and extension. Often, with the most extreme deformity from contracture, recovery is, in time, still possible. The interrupted current is useful when the acute stage is passed.

Of internal remedies, strychnia is of value and may be given in increasing doses. Arsenic also may be employed, and if there is a history of syphilis the iodide of potassium and mercury may be given.

## II. NEUROMATA.

Tumors situated on nerve fibres may consist of nerve substance proper, the true neuromata, or of fibrous tissue, the false neuromata. The true

\* Lectures on Neuritis, Medical Record, New York, 1887.



neuroma usually contains nerve fibres only, or in rare instances ganglion cells. Cases of ganglionic or medullary neuroma are extremely rare; some of them, as Lancereaux suggests, are undoubtedly instances of malformation of the brain substance. In other instances, as in the case which I reported,\* the tumor is, in all probability, a glioma with cells closely resembling those of the central nervous system. The true fascicular neuroma occurs in the form of the small subcutaneous painful tumor—*tubercula dolorosa*—which is situated on the nerves of the skin about the joints, sometimes on the face or on the breast. It is not always made up of nerve fibres, but may be, as shown by Hoggan, an adenomatous growth of the sweat glands.

The true neuromata, as a rule, are not painful, and occasionally are found associated with the nerve fibres in various regions. Those which develop at the ends and along the course of the nerves of the stump after amputation consist of connective tissue and of medullated and non-medullated nerve fibres. The most remarkable form is the *plexiform neuroma*, in which the various nerve cords are occupied by many hundreds of tumors. The cases are usually congenital. The tumors occur in all the nerves of the body. One of the most remarkable is that described by Prudden, the specimens of which are in the medical museum of Columbia College, New York. There were over 1,182 distinct tumors distributed on the nerves of the body. Prudden † has collected forty-one cases from the literature, in a majority of which the peripheral nerves were affected.

Neuromata rarely cause symptoms, except the subcutaneous painful tumor or those in the amputation stump. Here they may be very painful and cause great distress. Motor symptoms are sometimes present, particularly a constant twitching. Epilepsy has sometimes been associated, and relief has followed removal of the growths.

The only available treatment is excision. The subcutaneous painful tumor does not return, and excision completely relieves the symptoms. On the other hand, the amputation neuromata may recur.

### III. DISEASES OF THE CRANIAL NERVES.

#### I. OLFATORY NERVE.

The functions of this nerve may be disturbed at its peripheral ending, at the bulb, in the course of the nerve, or at the central origin in the brain. The disturbances may be manifested in subjective sensations of smell, complete loss of the sense, and occasionally in hyperæsthesia.

(a) *Subjective Sensations; Parosmia*.—Hallucinations of this kind are found in the insane and in epilepsy. The aura may be represented by an

\* Journal of Anatomy and Physiology, vol. xv.

† American Journal of the Medical Sciences, vol. lxxx.

unpleasant odor, described as resembling chloride of lime, burning rags, or feathers. In a few cases with these subjective sensations tumors have been found in the hippocampal lobules. In rare instances, after injury of the head the sense is perverted—odors of the most different character may be alike, or the odor may be changed, as in a patient noted by Morell Mackenzie, who for some time could not touch cooked meat, as it smelt to her exactly like stinking fish.

(b) *Increased sensitiveness, or hyperosmia*, occurs chiefly in nervous, hysterical women, in whom it may sometimes be developed so greatly that, like a dog, they can recognize the difference between individuals by the odor alone.

(c) *Anosmia; Loss of the Sense of Smell*.—This may be produced by: (1) Affections of the termination of the nerve in the mucous membrane, which is perhaps the most frequent cause. It is by no means uncommon in association with chronic nasal catarrh and polypi. In paralysis of the fifth nerve, the sense of smell may be lost on the affected side, owing to interference with the secretion.

It is doubtful whether the cases of loss of smell following the inhalations of very foul or strong odors should come under this or under the central division.

(2) The lesions of the bulb or of the nerves. In falls or blows, in caries of the bones, and in meningitis or tumor, the bulbs or the nerve trunks may be involved. After an injury to the head the loss of smell may be the only symptom. Mackenzie notes a case of a surgeon who was thrown from his gig and lighted on his head. The injury was slight, but the anosmia which followed was persistent. In locomotor ataxia the sense of smell may be lost, due possibly to atrophy of the nerves.

(3) Lesions of the olfactory centre. There are congenital cases in which the nerve structures have not been developed. Cases have been reported by Beevor, Hughlings Jackson, and others, in which this symptom has been associated with disease in the hemisphere. The centre for the sense of smell is placed by Ferrier in the uncinate gyrus.

To test the sense of smell the pungent bodies, such as ammonia, which act upon the fifth nerve, should not be used, but such substances as cloves, peppermint, and musk. This sense is readily tested as a routine matter in brain cases by having two or three bottles containing the essential oils. In all instances a rhinoscopical examination should be made, as the condition may be due to local, not central causes. The *treatment* is unsatisfactory even in the cases due to local lesions in the nostrils.

#### II. OPTIC NERVE AND TRACT.

##### (1) *Lesions of the Retina*.

These are of importance to the physician, and information of the greatest value may be obtained by a systematic examination of the eye-