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VARIOUS

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## ORIGINAL ARTICLES.

### BRACHIAL PARALYSES.*

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WITH the permission of the Society, I wish to give a brief account of brachial paralyses, in connection with a case of paralysis of some of the muscles of the forearm.

The patient, Mrs. Keefe, came to me December 5, 1887, complaining of a loss of power in the right wrist and hand. Her history, taken at the time, is as follows:

Patient has been in general healthy. In 1880 she began to have attacks beginning with numbness and tickling in the right hand; these ran up the arm to the face, the lower lip twitched, but head and arm did not move. The tongue became thick, patient got words mixed, was sometimes unable to speak a word. There was then great pain in frontal and occipital regions, she felt nervous, occasionally vomited. Patient always knew attack was coming on by seeing lights before the eyes in spots. Occasionally had hemianopsia. Attacks came every month or so. She has had none since last February. At time of her mother's death she had hal-

* Read before the Cuyahoga County Medical Society.

lucinations in the night, thought she saw fairies and sisters of charity, but knew at the time that these were delusions. Has had some crying spells, and sometimes feeling of lump in the throat.

Last July, five or six months ago, she began to lose power in her right wrist, and this has been getting worse ever since. No tinglings, but some soreness in wrist, which came on after the paralysis.

Status Praesens—Complete paralysis of right extensor communis digitorum, extensor carpi radialis longior and brevior, and extensors of the thumb and little finger. Supinator longus acts well. No anæsthesia. No other paralyses. Sight good. Fundus oculi normal. Pupils equal and react well. Knee jerk normal.

Patient has had no colic nor constipation. No specific history; patient has never used hair dye nor face powder. Has been in habit of drinking six or eight cups of coffee a day; no alcohol. Has no lead line on gums, takes good care of her teeth. Urine contains no albumen and no casts.

Our first problem is to determine whether the disease is organic or functional; if organic, whether the lesion is situated in the brain, spinal cord, or peripheral nerves.

In looking over the history we are struck at once with the similarity of the sensory attacks described by the patient, with those which occur in Jacksonian epilepsy, which is caused by disease in the cortex of the brain. In these attacks, first described by Hughlings Jackson, the convulsion usually begins in one of the extremities, 'say the hand, it may be that one of the fingers begins to move, the tremor is communicated to the others, then crawls up the arm, the face begins to twitch, and the patient then usually loses consciousness. The convulsion may become general or remain unilateral. In like manner a sensory aura may begin in one hand and creep up the arm, and this seems to be the fact in the case under discussion. Convulsive movements, however, have been nearly or quite absent, but the patient gives a fairly clear account of aphasia immediately afterwards, which

is especially interesting, as the symptoms exactly follow the positions of the arm, face and speech centres in the cortex. The nerve storm here began in the arm centre, passed through the face centre to the tongue centre, which is located, by most authorities, then to Broca's motor speech centre. Is this a paralysis of cortical origin, due to disease of the arm centre, and to be brought into connection with the sensory symptoms just described? To determine this it is necessary to make an electrical examination, and find or exclude the reaction of degeneration.

It is found that when a nerve is cut through, a degeneration of its fibres takes place below the point of injury, and at the same time there is an organic change in the muscles which it supplies. The same thing occurs from destruction of the large motor ganglion cells in the anterior horns of the gray matter of the cord. This change causes a difference in the electrical reactions of the muscles, called the reaction of degeneration.

A normal muscle reacts to both the Faradic and galvanic current with a sharp contraction, and the contraction on cathodal closure is greater than the contraction on anodal closure. The degenerated muscle does not react to the Faradic current; its contraction on galvanization is sluggish; the contraction on cathodal closure is usually weaker than that on anodal closure. These differences are shown in this table, and it is necessary to bear them in mind.

When motor fibres are cut across in the brain or lateral columns of the cord, the descending degeneration stops at the ganglion cells in the cord; these are not involved, and the muscles give no reaction of degeneration.

Examination in this case revealed the fact that the paralyzed muscles do not respond at all to the Faradic current; their galvanic excitability is markedly decreased; to the galvanic current their contraction is sluggish and very characteristic. Cathodal closure contraction is slightly greater than anodal closure contraction. That is, although the galvanic formula is not reversed, there is unmistakable reaction of degeneration.

We are therefore certain of an organic cause, which is not cerebral, but situated either in the cervical cord or in the musculo-spiral nerve.

The sensory attacks described above have no connection with the paralysis, but were probably auræ of migraine which regularly followed them.

The diseases which occur in the cervical cord are the following:

Acute polio-myelitis, or ordinary infantile paralysis. This is an inflammatory process involving the anterior horns of gray matter. It causes atrophy and reaction of degeneration, and usually runs its course without sensory symptoms. The disease may be ushered in with constitutional symptoms—chill, fever, convulsions, headache, delirium—but these subside within a week, and the paralysis then remains stationary or improves slowly. Excluded here, as this is a process which is decidedly progressive.

Progressive muscular atrophy is a chronic disease which causes paralysis with reaction of degeneration. It is due to a probably primary degeneration of the large motor ganglion cells of the cord. There are no sensory symptoms. It is here exceedingly improbable, because in it the small muscles of the hand are regularly the first ones affected, the atrophy showing itself in the dorsal interossei. Both hands are apt to be affected at about the same time.

Amyatrophic lateral sclerosis regularly begins in the cervical cord, and consists in a chronic degenerative change in the anterior horns of gray matter and in the pyramidal tracts. The first symptoms are stiffness of the muscles and increased tendon reflexes, weakness and fibrillar twitchings—a very different clinical picture from the one presented by this patient.

The hypertrophic form of pachymeningitis occurring in the cervical region of the cord, was first described by Charcot. It eventually leads to paralysis from pressure on the motor tracts; but this is preceded by local pain and tenderness, a prominent symptom also in affections of the meninges of the brain, and by paræsthesiæ and anæsthesiæ from interference

with the sensory posterior nerve roots. None of these symptoms are present.

Syringo-myelia regularly begins in the cervical cord and consists in the formation of a cavity around the central canal, from the breaking down of a new formation much like a glioma. Sensory symptoms are prominent, there being patches of anæsthesia, especially to heat and cold.

Tumors and abscesses of the cord cause by pressure sensory symptoms, and are apt to cause also a spastic condition such as is found in lateral sclerosis.

There remains the chronic form of polio-myelitis anterior. This is a progressive affection, without sensory symptoms, and gives rise to paralysis with atrophy and the reaction of degeneration. It usually begins in the lower extremities, and constitutes one form of the so-called ascending paralysis. It is theoretically possible that the paralysis in this patient is caused by such an inflammatory process, but the localization is not a typical one, and there is a form of peripheral disease which better explains the symptoms, as we shall see later.

Among the peripheral nerves the musculo-spiral is very often diseased, usually as a result of injury or the selective action of poisons. Traumatic paralysis may be caused by the pressure of a crutch in the axilla, in which case the triceps is paralyzed as well as the supinator longus and extensors; by pressure from the patient leaning on his arm when he is asleep, when the injury usually occurs half-way down the arm, between the triceps and biceps, and the triceps is usually spared; by a blow or cut. In all these cases the sensory as well as the motor filaments of the nerve are affected, and there is anæsthesia of the integument supplied by the radial, *i. e.*, the back of the radial side of the hand, of the thumb, and of the first two fingers as far as the second phalanges. In our patient this is not the case, and, furthermore, she gives absolutely no history of injury.

Multiple neuritis is a disease which has attracted much attention of late. It is an inflammation occurring along nerve trunks, and causes paralysis with atrophy. But the paralyses are irregularly distributed, and sensory symptoms

are always present and are apt to predominate. The same may be said of paralysis resulting from pressure on a nerve by tumor or aneurism, and of the variety caused by the so-called rheumatic neuritis.

There remain the paralyses of toxic origin, and of these, that caused by lead is the most frequent. This is an affection which begins insidiously with weakness in the extensor communis digitorum muscle, and the neighboring extensors soon become involved. There is, then, as in this case, atrophy with the reaction of degeneration, very rarely pain, tingling or anæsthesia. There is another peculiarity of lead palsy which is much relied on to distinguish it from that due to injury of the nerve, namely, the exemption of the supinators. The supinator brevis is not accessible. In order to see whether the supinator longus is paralyzed or not, it is simply necessary to cause the patient to flex the forearm on the arm, with the hand in the position of pronation. *Thus.* You see at once that this patient's supinator longus acts well.

For both these reasons we exclude nerve injury and ordinary forms of neuritis. But we have already excluded functional and cerebral troubles, and all spinal diseases except one, namely, chronic polio-myelitis anterior. But that affection, as pointed out, is very rare; it usually begins in the lower extremities; it may affect a group of muscles which habitually act together, but rarely, if ever, an isolated group of muscles supplied by one nerve. It is a much less violent supposition to consider this paralysis as toxic, due to the ingestion of lead.

Inquiry has as yet failed to find out the source of the poison. A visit to the home of the patient brought out the fact that she has used tin and granite-ware coffee and teapots, some of which, she stated, turned the coffee black. Chemical tests applied to these in a way suggested by Professor Morley, have failed to show any lead in their composition, and in the absence of other testimony we must consider, either that there has been sufficient lead to cause poisoning in the drinking water, which patient states comes through a lead pipe, or that the cause is one which has

evaded our observation, but which we may later bring to light. In regard to poisoning from lead pipes, there are a few observations of this kind on record, although they are rare. The patient states that she has not been in the habit of letting the water run for a time before using it, but she has now been instructed to do so.

No examination of the urine for lead has been made, owing to the inconveniences which attach to a sulphuretted hydrogen apparatus.

The absence of the lead line on the gums is the rule with patients who are in the habit of brushing their teeth.

Although it is the rule for both arms to be affected, cases are not rare in which both legs, or one arm alone, or an arm and a leg suffer. Buzzard records a case in which the right arm and left leg were the only seats of paralysis, and from this curious arrangement the diagnosis of hysteria had been made. Electrical examination showed the presence of the reaction of degeneration in the affected muscles. The same patient had no lead line and no colic.

The pathology of lead paralysis is still a moot point. All observers are agreed that there is an organic lesion, and that the muscle fibres undergo atrophy in the same way that they do after section of a nerve. The muscle fibres become smaller in calibre, there is increase in the number of nuclei, in fat and in connective tissue.

Changes in the spinal cord have been found by some observers. The latest researches are those of Schultze of Heidelberg. He found in a number of cases the spinal cord healthy, the musculo-spiral nerve the seat of a well-marked inflammation. How and why the inflammatory process attacks only the motor fibres is unexplained, as is the selective action of the poison in affecting only the radial nerve. This fact has its analogue in neuralgia, especially that affecting the sciatic nerve, caused by a neuritis which affects only the sensory filaments.

The treatment which has been followed in this case is the one familiar to you all. Potassium iodide has been given internally to hasten the elimination of the poison; the affected