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ORIGINAL LECTURES.

THE SYMPTOMS AND DIAGNOSIS OF MULTIPLE NEURITIS; WITH REPORTS OF CASES.

Based on a Clinical Lecture Delivered at the New York Post-Graduate Medical School, January 18, 1887.

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A very good description of multiple neuritis was given by Leyden in 1881, again by Piérsson in 1883, and by Strümpell in his "Text-book on Medicine." Dr. X. Francotti, in 1886, described it well; so also did Dr. Buzzard in the Harveian Lectures for 1885. Dr. Gowers gives a very complete account of the disease in his recently published text-book on nervous diseases. Dr. M. Allen Starr will no doubt cover the whole ground in the most thorough manner in the Middleton Goldsmith Lectures on this subject, now forthcoming.

I venture to give here some notes on the history of this new development in neuro-pathology, since it has made such a change in some of the conceptions of paralytic disorders. Dumenil, in 1864, reported¹ a case running a course of four and one-half months, and having the type of the subacute spinal paralysis of Duchenne. The autopsy showed diffuse lesion of the nerves, the brain and cord being normal. A second case,² with post-mortem examination, ran a course of several years, and started from a contusion of the sciatic. Eichhorst's case³ came next. It was like one of acute ascending paralysis, of six weeks' duration, except that there was pain and sensory disturbance. Autopsy showed acute inflammatory nerve trouble, not a simple degeneration. Then Déjerine reported a case also resembling acute ascending paralysis. He found also neuritis, including some changes in the anterior roots. Neither Eichhorst's or Déjerine's cases are very complete. Eisenlohr⁴ reported a case of subacute paralysis and autopsy. The cord was normal, the sciatic showed parenchymatous and interstitial neuritis.

Joffroy contributed a very elaborate paper to the *Arch. de Phys. Normale et Path.*, No. 2, 1879. He

describes: 1. Spontaneous parenchymatous neuritis. 2. Neuritis from lead poisoning. 3. Neuritis in the course of infectious diseases. Under the first division he describes cases of multiple degenerative neuritis, with autopsy. Lancereaux reports a case of this kind occurring in phthisis, as do Desnot and Pierret.

In 1880 Leyden⁵ summed up the work of his predecessors, and reported a case of his own, with autopsy. He gave a systematic clinical history of the disease, and one which is still quite correct. Leyden was followed by Melchert.⁶

In 1882 Scheube, and in the same year Baelz, showed that the nerve symptoms of beri-beri were due to a multiple neuritis.⁷ Koeniger,⁸ writing on this same subject in 1882 and 1884, alleged that the neuritis was only a complication. Ballet, in 1883,⁹ found atrophy of the anterior cornua in beri-beri—nerves not examined. Harada¹⁰ found neuritis and degenerative changes also in anterior nerves in dorsal and lumbar regions. M. P. Mendes¹¹ found lesions in the nuclei of the columns of Goll, and in the posterior columns, especially, of the cervical and lumbar cord there was atrophy of cells and parasites in vessels; also a neuritis as far up as the spinal ganglia. Tschowski¹² reported several cases, with autopsy in three. He found multiple neuritis, and also atrophic cells in the lumbar cord.

After Leyden's communication there were soon published a large number of cases, by Hiller,¹³ Granger Stewart,¹⁴ Eichhorst,¹⁵ Strümpell,¹⁶ Müller,¹⁷ Vierrordt.¹⁸ Piérsson wrote¹⁹ a systematic monograph on the subject in 1883.

Roth, in 1883, reported²⁰ a case of acute general paralysis, with autopsy, showing extensive parenchymatous and interstitial neuritis. Dr. S. G. Webber, of Berlin, reported some cases in the *Archives of Medicine*, Vol. xii, p. 33. See also articles by

⁵ Charité-Annalen, 1880, and in Zeit. f. klin. Med., 1880.
⁶ Inaug. Dissertation, 1881: Greifswald. Beitrag zur Diagnose der subacuten Poliomyelitis u. Multiple neuritis.

⁷ Zeits. f. klin. Med., 1882, Bd. iv, p. 616, and Virchow's Archiv, 1884, p. 146, Bd. 95, and p. 531, Bd. 99.

⁸ Deuts. Archiv f. klin. Med., Bd. 31, pp. 141, 307; Bd. 32, p. 83; Bd. 34, p. 419.

⁹ Proc. de la Société Anatomique.

¹⁰ Neurol. Centralblatt, 1885, p. 326, two cases.

¹¹ Prog. Méd., No. 14, 1885.

¹² Neur. Centralb., 1886, p. 484.

¹³ Berlin. klin. Wochenschr., 1881, No. 41.

¹⁴ Edinburgh Med. Journal, 1881.

¹⁵ Archiv. f. Psych. u. Nervenk., Bd. xiv, p. 695. ¹⁶ Ibid., p. 339.

¹⁷ Ibid., p. 668. ¹⁸ Ibid., p. 678.

¹⁹ Ueber Polyneuritis acuta, Volkmann's Samml. klin. Vort., No. 227, 1883.

²⁰ Correspondenzbl. f. Schweiz. Aerzte, 1883, No. 13.

¹ Gaz. Hebdom., 1864, No. 13.

² Ibid., 1866, Nos. 4, 5, 6.

³ Virchow's Archiv, Vol. 69.

⁴ Centralb. für Nervenheilk., 1879, and Deut. Arch. für klin. Med.,

Bd. 27, 1880.

Eisenlohr,²¹ Strümpell,²² Moeli,²³ Hirt,²⁴ Löwenfeld,²⁵ E. Remak,²⁶ Grocco,²⁷ and Oppenheim.²⁸

The relation of alcoholic paralysis was studied by Strümpell,²⁹ by Dr. Dreschfeld,³⁰ and by Schultz.³¹ These writers give a full history of this special subject up to the date of this writing.

The relation of arsenical paralysis to neuritis was studied by the writer, and a full account of it given in *Brain*, January, 1887. In the same article the history of the relation of lead-poisoning and of diphtheria to multiple neuritis is given. In the past year a number of additions to our knowledge have been made, all of which are to be summarized and presented by Dr. Starr in the lectures referred to.

In a thesis entitled "Des Pseudo-tabes," by L. Leval-Picqueche (Paris, 1885), a very complete history of this form of multiple neuritis, from various causes, is given. Dr. Starr has also called my attention to a very complete work entitled "Paralysis Toxiques," by Broussais, of Paris.

The symptoms of multiple neuritis develop in two forms; 1st, those of atrophic motor paralysis with subsidiary sensory changes; 2d, those of ataxia and other sensory disturbances with only moderate motor paralysis. The motor form is the more common. The sensory or ataxic form occurs chiefly as the result of poisons or the infection of diphtheria.

The Atrophic Motor Form.—The symptoms generally come on rather suddenly, and are sometimes accompanied with fever, which may reach 103° to 104° F. The patient suffers from feelings of numbness, prickling or burning in the feet and ends of the fingers. These sensations gradually extend up the extremities, but rarely reach the knee or elbow. They are accompanied by sharp pains and feelings of weakness, and the paralysis steadily increases until the patient is unable to walk or use his hands. The lower extremities are oftenest and most seriously affected, the upper extremities next, and the muscles of the face and trunk least often of all. The flexors of the foot and the extensors of the hands are particularly attacked. The paralyzed muscles speedily become atrophied. The bladder and rectum are not affected. The sexual power is lessened. The pulse is generally quite rapid, owing, perhaps, to involvement of the vagus.

On making a physical examination the skin is generally found hyper-æsthetic, and yet there may be some tactile, thermal and pathic anæsthesia. Later this generally develops. Pressure along the course of the nerves and over the muscles causes much pain, as do muscular movements.

Electrical examination of the muscles shows generally a partial degenerative reaction, the muscles being less irritable than normal to both galvanic and faradic currents. The contractions are sluggish, but

it is not common to get polar changes. The nerves show also diminished electrical sensibility, but furnish no positive indications of neuritis as against polio-myelitis, though perhaps their irritability is less. There are not usually any marked vaso-motor phenomena except œdema. Sometimes the secretion of sweat is profuse, and in arsenical cases I have seen the feet and hands shed the epidermis as if it had been raised by a blister.

The disease reaches its height in two or three weeks, or even less. The symptoms then gradually ameliorate, and the rule is a progressive improvement ending in cure in four to six months. In some cases the spinal cord apparently becomes involved, and the sphincters are paralyzed; diabetes and various other central symptoms appear.

Some cases of multiple neuritis run a very acute course, and present the symptoms of acute ascending spinal paralysis (Landry's paralysis). Other rare cases present a slowly progressive course lasting three or four years, e.g., Dumenil's case.

These types are different clinically and etiologically from the disease ordinarily known as subacute multiple neuritis.

The Sensory or Ataxic Form is spoken of by Gowers as rare, but I have seen quite a number of illustrations of it. Here the symptoms begin with burning, tingling sensations in the feet and hands, especially the former. The patient notices that his gait is uncertain. The tendon reflexes disappear, the limbs are partially anæsthetic, or areas of anæsthesia are found. There is a moderate degree of motor weakness and atrophy. In a few days the patients may develop nearly all the symptoms of locomotor ataxia. These cases are generally of toxic origin. They run a subacute course, about as does the motor form.

The symptoms may be summarized as follows: Prodromic period in some cases of several weeks characterized by some numbness and lumbar pains. Rather brusque onset, sometimes with pain.

Motor Symptoms: Progressive symmetrical paralysis, ascending, and affecting lower limbs most and oftenest. Paralysis flaccid, atrophic and painful. Partial degeneration-reaction. Abolition of knee-jerk as a rule, and of skin reflexes in paralyzed limbs.

Trophic Symptoms: Muscular atrophy; œdema; rarely erythema, eczema, herpes, changes in nails and skin, local asphyxia and gangrene.

Sensory. Paræsthesia; especial characteristics are burning sensations, pains of both darting and dull character, worse on movement; tenderness in limbs, and especially over course of nerves. Later some anæsthesia, general or over distribution of nerves.

Cranial Nerves: In rare cases only affected, but optic neuritis is not very rare. Rapid pulse from involvement of the vagus sometimes occurs. Sphincters not involved as a rule.

The termination is usually favorable, but death may come on unexpectedly.

Further details in the history will be brought out in the report of the following cases. These I will give here only in synopsis.

Case 1.—Alcoholic Multiple Neuritis. Sensory

²¹ Neur. Centralbl., 1884, Nos. 7, 8.

²² Ibid., 1884, No. 11.

²³ Charité-Annalen, Vol. ix, 1884.

²⁴ Neurolog. Centralbl., 1884, No. 21.

²⁵ Ibid., 1885, No. 7. ²⁶ Ibid., No. 14.

²⁷ Contrib. alla stud. clin. ed anat.-path. delle Nerv. mult. prim., 1885.

²⁸ Deutsches Archiv f. klin. Med., 1885, Bd. 36, p. 561.

²⁹ Krankheiten des Nervensystems, p. 121, and Berlin. klin. Woch., 1885, No. 32.

³⁰ Brain, 1884, July et seq.

³¹ Neurolog. Centralbl., Nov., 1885.

Form.—The patient, a bar-tender, had always been healthy, and gave no history of syphilis. In the past year he had indulged excessively in sexual intercourse, but still more excessively in alcohol, and after having been on several sprees he was taken to the hospital suffering from delirium tremens. This developed into mania, and he was confined in the asylum for several weeks. During this time he became partially paralyzed, but improved and was discharged. When seen by me soon after, he presented the symptoms of a man in the early stage of locomotor ataxia, except that he had no eye symptoms, girdle pains, bladder or marked sexual trouble. There was marked ataxia, paræsthesia, areas of anæsthesia, loss of tendon reflex, some motor weakness. I learned that he later did develop some central symptoms, which lead me to think that the cord became also involved.

Case 2.—Alcoholic Multiple Neuritis. Motor and Atrophic Form.—This patient I saw at Bellevue Hospital, while visiting for Dr. Tuttle, and have shown him several times to my class. The patient was a young man aged 23 years, who for over a year had indulged nightly in sexual intercourse and very excessively in whiskey-drinking. While playing ball one day he noticed a weakness in his legs. This increased in a few days, and he was now obliged to take to his bed. When seen by me he presented the usual symptoms of alcoholic paralysis. His lower limbs were alone affected, while here the flexors of the foot were especially attacked. The feet dropped, and at one time could scarcely be moved at all. There was partial degeneration-reaction. The legs were much atrophied and there was some anæsthesia, but not much pain. There was no ataxia. The knee-jerk was abolished. The sphincters were normal. The patient got worse for several weeks, then began slowly to recover. At the end of six months he could barely walk with help. His mind remained clear.

Case 3.—Multiple Neuritis from Arsenical Poisoning. Sensory or Ataxia Form.—I have shown the class two patients who suffered from multiple neuritis of this type (published in full in *Brain*, January, 1887). In one the patient, a young man of 25, took a poisonous dose of Paris green. Within a week he had developed very characteristic symptoms of pseudo-tabes, or arsenical ataxia. The symptoms were burning in the feet and hands, extreme ataxia, some tactile anæsthesia of limbs, combined with extreme hyperæsthesia and tenderness, especially over the course of the nerves; peeling of the skin of feet and hands; pains in the legs, especially on movement; optic neuritis; partial degeneration-reaction; moderate paresis and atrophy; no girdle pains, no sphincter paralysis, no involvement of face or eye muscles. Mind clear, but patient nervous and hysterical. There was gradual improvement up to a certain point, then the symptoms remained stationary for many months.

Case 4.—In the second case the patient, a man of 45, was taking Fowler's solution in daily doses of 5iiss, and he gradually developed a pseudo-tabes similar to that above recorded, but milder in type. His symptoms, too, improved, and he was finally left

with simply an annoying numbness of the feet and finger tips. Two other cases have since come under my notice.

Case 5.—Multiple Neuritis from an Infection of Beri-beri.—This case occurred in the service of Dr. Roosevelt at Bellevue Hospital, and through his kindness I was able to show it to my class. The case has been reported in full by Dr. Roosevelt, and will soon be published; so I will not give the details here. The patient, a sailor, after suffering several weeks from the anæmia and anasarca characteristic of the "wet form" of beri-beri, gradually developed a paralysis of the lower extremities. The flexors of the foot were most affected. The paralysis gradually involved the muscles of both legs, but to a much less degree those of the thigh. There was some pain and paræsthesia, but little other disturbance of sensibility. The muscles atrophied and showed in one leg partial, in the other complete, typical degeneration reaction. The paralysis progressed very slowly for a few weeks, then slowly improved. Judging from some of the recorded histories, beri-beri may also produce a sensory or pseudo-tabetic form of the disease, but it appears to me that the multiple neuritis is not the disease, but is only one of the symptoms, or in some cases one of the sequelæ.

Case 6.—Multiple Neuritis from Rheumatic Cause.—There is no doubt that multiple neuritis may be caused by what is known as rheumatic influences; and the following case seems to illustrate it, although I confess that the diagnosis can not be made positively:

M. D. G., a Jewish ex-Rabbi, age 29, married, of Russian birth, had always been healthy, but had greatly overtaxed himself a few years ago by study. His present occupation is that of a peddler. About two months before he was seen by me he had been much exposed to cold and wet. He felt one day a great weakness coming on in the lower extremities, and to a less extent in the arms. He felt also numbness, prickling, and dead, heavy sensations in the extremities. All this increased until in a day or two he could hardly walk, and could not go up stairs. About this time he had severe pains in one knee, which lasted only a night. His symptoms did not improve, and he finally came to me. There was then the same condition of paresis of the extremities with paræsthesia; the knee-jerk was abolished; there were areas of anæsthesia over the external surface of the legs; there was considerable static and locomotor ataxia. The eye (he had but one) was normal, and he had no girdle pains or sphincter troubles. No degeneration-reaction. Urine 1026, acid, rather dark, no albumin or sugar. Under treatment he gradually improved. Nine months later I saw him again; the knee-jerk had returned, and all his symptoms had disappeared.

Case 7.—Neuritis Complicating Locomotor Ataxia.—I have shown to the class on several occasions a patient who had had for a year some slight ataxic symptoms, but had been able to do his work, that of a brakeman, every day, and who believed himself to be suffering only from a paralysis of the left third nerve, which was of several years duration. One

day, not feeling very well, he took a Turkish bath, and next day was in bed with an intensely painful paralysis of the lower extremities. There was exquisite tenderness along the course of the nerves, burning feet, pronounced ataxia, and abolition of knee-jerk. The symptoms seemed to point to the development of a neuritis complicating the original posterior sclerosis, and this is an accident which I believe may sometimes occur in true locomotor ataxia.

The diagnosis of subacute multiple neuritis cannot always be made with certainty. It must be based, 1st, upon the etiology; 2d, on the symptoms; and 3d, upon the course of the disease.

Diagnosis from the Etiology.—Painful symmetrical progressive paralysis, with atrophy coming on after an attack of rheumatism, is probably neuritic.

The same may be said regarding attacks coming on in alcoholic patients. The post mortem evidence collected by Dreschfeld, Schultz, Francotte and Bernhardt shows that almost all cases of alcoholic paralysis are of peripheral origin. Some are, however, I believe, myelo-neuritic.

The paralysis from arsenical poisoning, bi-sulphide and oxide of carbon, and in some cases after the use of lead, are peripheral. The pseudo-tabes after diphtheria and in diabetes is neuritic, but glycosuria may occur in true tabes.

The paralysis and pseudo-ataxia after infective diseases, diphtheria, variola, pneumonia (*Virchow's Archiv*, Vol. 68), measles, typhoid, and occurring in the course of acrodynia and beri-beri, are neuritic.

It is not yet demonstrated that multiple neuritis occurs in children except from beri-beri, although Webber and Chapin have reported cases which appear to belong to this category.

Diagnosis from the Symptoms.—Rather sudden onset with marked pains in legs and arms, and sometimes fever, followed by rapid symmetrical atrophic paralysis, are the main characteristics of subacute multiple neuritis. Hyperæsthesia is present at first, with great tenderness of muscles and along the course of the nerves. Later tactile and thermic anæsthesia of moderate type develops.

The specially characteristic symptoms in detail are: Symmetrical ascending paralysis affecting the lower extremities most, and especially the flexors of feet and extensors of arms.

Diagnostic Electrical Reactions.—E. Remak reports a case of generalized neuritis with marked electrical alterations of non-paralyzed nerves and muscles. It was in a woman of 30 years, and followed acute rheumatism. This was followed by burning pains and numbness of the legs and arms, with intention-tremor and some atrophy of hand muscles.

Clonic movements, intention-tremor or athetosis. Muscles of face and extremities, though not paralyzed, only react to severest electrical currents and give polar changes.

Erb noted the degeneration-reaction (milder form) in non-paralyzed muscles—head paralysis (*Arch. f. Psych.*, 1885, v, p. 445). Also Bernhardt (*Berlin. klin. Wochenschr.*, 1878, Nos. 18 and 19), and Buzard (*Brain*, 1878, vol. 1, p. 121).

Kahler and Pick found the severe form of degeneration in lead paralysis. *Beiträge zur Path. u. Path. Anat. des Centralnervensystems*; Leipzig: 1879; p. 131. So did Kast (*Centralb. f. Nervenheilk.*, No. 8, p. 137).

Kahler and Pick (op. cit.) also found these changes in a case diagnosed at the time as polio-myelitis anterior subacuta, but which gives much such a history as that of Remak's case. Bernhardt has also described such a case (*Virchow's Archiv*, 1879, Bd. 78, p. 274). In Schultz's case of alcoholic neuritis marked diminution of E. I., but no qualitative changes.

Rapidly developing atrophy of muscles. Partial degeneration-reaction.

The usual formula being—

Faradic current over muscles—E, sensibility lessened; E, irritability lessened or absent.

Galvanic current over muscles—E, sensibility lessened or absent; E, irritability lessened, contractions sluggish. Generally in reversal of poles, but KaCC > AnCC.

Edema is often marked, without albuminuria. The pulse-rate is high. Sphincters not involved; no decubitus. The face and eye muscles are rarely involved, but occasionally there is optic neuritis. Optic neuritis has been noted by Eichhorst (*Virch. Archiv*, 1876, Bd. 69, p. 69); Strümpell (*Archiv f. Psych.*, Bd. 14 p. 339); Löwenfeld (*Ueber Multiple Neuritis*, 1885, p. 15); E. Remak (*Neurol. Centralbl.*, No. 14, 1885); Dana (*Brain*, June, 1887).

The burning hands and feet, the peeling of the skin of the extremities, the sometimes sharp demarcation of the paræsthesia of the extremities, are diagnostic points to be noted.

Favorable termination favors the diagnosis of multiple neuritis. The presence of degeneration-reaction with retention of voluntary movement favors polio-myelitis. Localization of paralysis in physiological groups of muscles favors polio-myelitis, symmetrical ascending paralysis or poly-neuritis.

The diagnostic points in a case of Schulz's seen by Strümpell were: The multiplicity of the processes attacking the extremity nerves; high-grade atrophic paralysis, with highest grade diminution of electrical irritability, without quantitative changes; the slight paræsthesia without anæsthesia; the joint affection; the absence of the tendon-reflex with presence of skin reflexes; tenderness over the great nerve trunks; pains on starting to walk; the eventual return of the tendon-reflexes and normal electrical irritability. The bladder and rectum were, however, somewhat affected.

Diagnostic signs given by L. Löwenfeld in a case of rheumatic multiple neuritis with athetosis were: Motor weakness with fever and sensory disturbance limited to certain nerves; girdle sensations and great sensitiveness of the back muscles; absence of pupillary, bladder, rectal and sexual symptoms; girdle sensations about the extremities; electrical reactions were those of partial degeneration-reaction; there was striking diminution of electrical irritability to both currents, with slight polar changes. This has been found by Brenner and Bernhardt also.

The ordinary course of an acute poly-neuritis is, according to Roger:³² Sharp attack, with severe pains in extremities, oftenest in lower; often a fever; then paralysis, without contractures, with rapid atrophy. Hyperæsthesia is followed by anæsthesia; tenderness; electrical degeneration reaction.

Initial pains, early, marked sensory disturbances, combined with degenerative muscular atrophy and paresis of extremities, *following acute articular rheumatism*, point to subacute multiple neuritis.³³

Some particulars may be given regarding the diagnosis of the different forms of multiple neuritis:

1. *Alcoholic neuritis* runs a less acute course, is less progressive, and the paralysis is more confined to the extensor groups. The legs alone are often affected. There is a characteristic hyperæsthesia and hyperalgesia. Cerebral symptoms are frequent. The gait in the pseudo-tabetic cases is modified from that of true tabes by the fact that, the extensors of the feet being paretic, the foot is brought down flat.

The diagnostic signs of *alcoholic neuritis*, as given by Oetlinger (loc. cit.), are: The symmetrical paralysis, almost always beginning in the lower extremities, and affecting especially the common extensors of the foot and the extensor hallucis. Bladder, rectal, facial and eye paralyses are not present. Ataxia is not constant, nor are contractures. In chronic forms there is hyperæsthesia; in acute forms analgesia. Generally there there is œdema and trophic disturbances of the skin. Tendon reflex always absent.

There are three forms: mild, chronic and acute. The prevalence in women was not observed by Oetlinger. The ataxia was static only in a case described by Lilienfeld.

Besides the points given, the alcoholic patient may suffer from central scotoma; tabetics have a more general narrowing of the visual field (Bernhardt).

Optic neuritis is seen in acute myelitis, multiple sclerosis, multiple neuritis, and alcoholic neuritis, but never in true tabes.

There are mental disturbances in alcoholic cases, and women are oftener affected.

The degeneration-reactions are temporary, and only noted at the height of the disease. The ataxia is static only in some cases. There may be rapid pulse and hyperidrosis.

In *arsenical neuritis*, a characteristic trophic disturbance is the entire shedding of the epidermis of the feet and hands. A hysterical mental condition is sometimes present. The paralysis may be motor and atrophic, or may take a pseudo-tabetic form, the last being rare.

A multiple neuritis of the sensory or ataxic form is differentiated easily from locomotor ataxia usually, by the changed electrical reactions (high grade diminution of E. irritability, with or without qualitative changes). In tabes, at the beginning, there may be an increased E. I., which later decreases without any degeneration-reaction.

There is an ataxic neuritis seen in early atrophic paralysis.

The pupillary symptoms, girdle-pains, bladder and rectal disturbances are, as a rule, absent in neuritis.

There may be slight pelvic symptoms in ataxic neuritis.

The tendon reflex, though apparently absent, may be brought out sometimes by Jendressik's method in neuritis; never in tabes (Moeli).

In *post-diphtheritic multiple neuritis* there are generally paralyses of the soft palate first, then general muscular feebleness, ataxia and loss of tendon-reflex. In a few cases the external eye muscles are affected, causing exophthalmoplegia externa.³⁴

Subacute multiple degeneration neuritis is to be distinguished clinically from *acute ascending spinal paralysis* (Landry's). In the latter disease there is, it is true, a multiple neuritis, in some cases at least, but its cause and course, as well as the anatomical changes in the nerves, are different.

Myelo neuritis.—Multiple neuritis may be combined with a polio-myelitis, and then symptoms of both diseases may be present. Leyden, Rosenthal and Déjerine have found cases running a course like that of multiple neuritis, with, on autopsy, evidences of both neuritis and myelitis. Eisenlohr has reported³⁵ a case that is particularly striking. J. J. Putnam reports two cases of painful myelo-neuritis.

Central spinal lesions predispose to neuritis. Déjerine reports³⁶ two cases of tabes with extensive degenerative neuritis.

Illustrations of symmetrical ascending neuritis and myelitis are given by Grainger Stewart,³⁷ who reports three cases. The patients showed the signs of multiple neuritis with ascending paralysis, beginning in all four extremities. Post-mortem examination showed secondary degeneration of the columns of Goll and of the direct cerebellar tract.

TUBERCULOSIS OF BONES AND JOINTS.

Read before the Northwestern Ohio Medical Association, at Lima, Ohio, December 9, 1886.

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(Continued from page 262.)

Cases of tuberculous dropsy of the joint should, as soon as diagnosis is possible, be operated upon, as all other means will have little effect. This operation generally consists in making incisions into the joint, removing the swelled synovialis. After having washed out the joint with disinfecting fluid, dust it over with iodoform, put in drainage-tubes, etc. The idea of preventing the spread of tuberculosis from an affected joint is less an indication for operative measures than the local condition itself. Such measures, amputation especially, may sometimes be called for to cut off the dangers of septicæmia, pyæmia and amyloid degeneration of the kidney and other organs; it is indeed surprising to see the rapid general improvement of the patient following amputation.

³⁴ For bibliography see P. Meyer, Virchow's Archiv, Bd. 85, p. 214. Also Mendre, Neurol. Centralbl., 1885, No. 6.

³⁵ Neur. Centralbl., 1884.

³⁶ Brain, 1884, p. 558.

³⁷ Journ. of Nerv. and Ment. Disease, 1882, p. 212.

³² L'Encéphale, 1885, No. 2.

³³ Kast, Archiv. f. Psych., 1881, Bd. xii, p. 266; and F. C. Müller, Ibid., Bd. xiv, p. 669.