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I do hereby certify on oath
and conscience, that the
accompanying thesis on
Multiple Recovites was composed
and written entirely by myself

Frederic R. W. Lyle

Multiple Hemiplegia

In the following remarks on multiple hemiplegia I have given records of three cases which have recently occurred in my practice, the occurrence of these cases first led me to look into the literature which has been published concerning their malady especially with reference to its occurrence after influenza.

These three cases appeared to me to present in a manner less marked degree the 3 clinical divisions in which it has been found convenient to group the different forms of the disease. Case 1 being characterized by the Remorse symptoms being much the most marked. Case 2, presenting Motor & Atactic symptoms, the Paresis, ones being comparatively slight, and Case 3, slight Ataxia.
and paralyzed symptoms with an almost entire absence of sensory symptoms.

Dr. James, in his work on the nervous system Vol. 1, divide the cases into 3 groups: (1) Sensory, (2) Motor, (3) Ataxia, according to the predominance of one or another set of symptoms and the case, which I am about to describe in this paper appeared to me to present some at least of the characteristic features of these classes.

Cases 1 and 2 are interesting as being examples of Post-Gripal Polynuercetic, both having been preceded by an attack of influenza. In these cases any of the other recognized causes of Polynuercetic could be excluded, as far as I could learn or see. In Case No. 3 alcohol in the form of brandy was undoubtedly the toxic agent
3.

Following the classification of Dr. Gower, in regard to the etiology of the disease (Vol. 1, p. 112), Case 1 might either be an example of what he classes as Secondary Toxemic forms. Regarding the symptoms displayed before the remittent phenomena manifested themselves, as influenza. The headache in this case however followed so rapidly on the primary manifestations, that it might almost as appropriately be included as an example of the Primary Toxemic forms. Case 2 would certainly be claimed in the Secondary Toxemic forms, being separated from the influenza attack by a distinct interval. Case 3 was the only one in which there was any history of Syphilitic infection, in the other 2 cases there was no reason to suspect
that disease or certainly no traces of it. In the alcoholic case the previous venereal affection may possibly have had some effect in producing the disease, although in my opinion it had none and this opinion was afterwards strengthened by the fact that without any anti-syphilitic treatment whatsoever he made a perfect recovery, the treatment adopted being entire removal of stimulants, general tonic treatment and massage.

The effects of syphilis in producing by its own toxins a true Hansen's chlamydiae neuritis seem to require further investigation and as far as I have been able to gather from recorded cases is only accepted as a cause by most writers on this subject in the absence of any of the other
more definite cause of Polyneuritis, but having regard to the prominent part played by diphtheria as a cause of Lakes Disease, it is probable that it may have much influence in giving rise to a disease, some cases of which bear such a strong clinical resemblance to Lakes as to have received the name of "Lakes Lakes Disease." According to the recent researches of De Girmie, Perret & others to which I shall have further occasion to refer it is often pathologically associated with it. Diphtheria being a chronic specific disease we should expect that cases of Polyneuritis produced by the toxins of its viruses would be late in showing themselves compared with the cases of the disease produced by the toxins of the Acute Rheumatic diseases. Unfortunately I have
had an opportunity of observing Polynneuritis in cases with a syphilitic history uncomplicated by Alcoholicism, but this is the impression one gets from reading the case of Polynneuritis supposed to be caused by Syphilis.

As regards the Cauation of Case I to L. I take it to have been the action of a Toxen produced by the micro organism of Influenza—recently demonstrated by Pfeiffer. Sir Saminer Strower remarks at the recent meeting of the British Medical Association are suggestive in this connection.

During the last three epidemics of Influenza we have had ample opportunities of witnessing the special affinity which the grippe to certain professions for attacking the nervous system, indeed as Althaus points out in his work on Influenza,
other disorders except Lyphle.

shows such a tendency to produce

severe symptoms & sequelae.

Without going so far as Sarnon
(Lancet 2 Jan. 1892) and regarding
all remote effects of Influenza
as produced by a remit,

affecting the Sympathetic

ganglia & nerves, the organs, and
the Sensory motor nerve trunks

still many cases recorded by
Sarnon in the above book

by others, (see Ferguson "The
African Physician" Octob. 1890
Allcohol on Influenza 1892) also
a case by (Holly: Schweinf Lizelle
his Influen Berlin 1892 Nov 14. 1880)
can be most reasonably explained
by regarding them as cases of
remote remit.

Isolated remit of nearly every nerve in the
body has been described in

patients who had previously
been gripped, and many

cases of multiple nerve affection.
have also been observed as sequel of Influenza.

The impression one gets in reading over the recorded cases of post-gingivhal paralysis is that the motor type of the disease presents itself with greater frequency than the sensory or ataxic forms. Thus D. Burdick relates a case of purely motor widespread paralysis after a slight attack of an influenzal character. Of Westphalen records 2 cases in which the sensory symptoms were extremely slight (hemianubes numb in flu). But cases showing marked sensory symptoms are by no means wanting e.g. Bransett's case of Peripheral hemiplegia after Influenza Med. J. 1891, also Dr. C. H. Mills, Trans. Phil. Med. Soc. 1892. Many other observers have related cases.
of Polynemia, following on Influenza, so that its occurrence as a sequel of that disease may be regarded as now established (see Liiehmeit "Vber Influenza Coma. Blatt f. Ges. de Vol XXI. Church Journal Amer. med. Ass. November 1890. Komen "Jenka" Su Rainville, Haft Vol 32. 12.

Drake New York med. Record 1890 Haavage Revue de Med. 1891 etc etc.

In many of the recorded cases the hands and arms appear to have been first attacked and followed by involvement of the lower extremities. The evidence of Influenza in some of the cases, e.g. those of Haavage & Buzzard, is slight and unsatisfactory but this would seem to be almost unavoidable when a disease displaying such variety in the intensity of its symptoms as Influenza is under consideration, the severity of the subsequent sequelae appears
in no way related to the severity of the quinsy attack itself, presenting in this respect a similarity to chilblain necrosis. Sometimes indeed in the case of a slight influenza attack and subsequent necrosis, especially if the necrosis is of rapid onset, it would be difficult to say very positively whether the patient had really been quinsy or whether the febrile symptoms and general pains and malaise, were not the initial manifestations of the necrosis of the so-called idiopathic variety. In case No. I I debated this question after it became clear to me that the case was one of multiple necrosis, I had however at this time 3 other patients in the same district, each presenting similar febrile symptoms, temperatures of 101 to 102.5, slight bronchial catarrh.
11.

Gastric symptoms with vague leg pains. Three of them soon recovered perfectly, and this one came alone developed severe symptoms. Medical men in the district on inquiry informed me that they had cases similar to the one mentioned above, but in none of them as far as I could learn up to the time of my inquiries, was there any nervous disorder subsequent with the exception of considerable mental depression displayed by some of the patients. Moreover, there was no other cause of recurrence either toxic or toxemica that would account for the attack. The patient had always enjoyed good health and was in no way cachectic. I was therefore in the absence of any other explanation forced to regard it as an example of Post-grippoal
Pneumonia, although the evidence of influenza was not perfectly satisfactory.

In case No. 2, the patient was severely and unmistakably gripped in Paris in the spring of 1893. She was residing in a pension near the Saint Germain Boulevard, and several of the other inmates of the house suffered at the same time. This patient's health also before she was gripped had been good. It is thus a difficult matter to offer any reasonable explanation of how out of so many gripped patients such a very small number exhibited their disease as a sequel, the health of the patient before the attack of influenza does not as far as I can find seem to influence it, neither does a thrombathic disease seem to have any effect
I have seen many individuals presenting that diathesis in a very marked degree recover from influenza without a trace of nervous disturbance of any kind, and my 2 cases of it occurred in individuals who could not be included in the diathesis. Possibly defective elimination of the toxics in these individuals whose nerve fibers are attacked might account for it or excessive production of these toxics from some cause on the same principle as that in the alcoholic form the blood contains a certain amount of the toxic agent before giving rise to impairment of nerve function, in the same way the forms of the disease following in diathemic influenza etc etc there must be a certain quantity of the toxæmic agent in the
Circulation to bring about a similar result.

Dr. Charlton Bastian (Brain's Diet) 1894 supposes that in the cases, (whether following on influenza or other diseases) which develop multiple neurites "there is either some variation in the nature of the poisons finding their way into the blood, or that the ordinary poisons are favoured by the coexistence of one or more favouring conditions" by co-existing favourable circumstances I take him to mean either a neuropathic condition, or the presence of some abnormal blood condition, the suggestion of Allhau's of a deficient supply of the antitoxin which usually is present in sufficient quantity to antitoxinise the toxin, would I think fall under this abnormal blood condition i.e., the blood would
he wanting in a sufficient supply of the Antibacterin which is usually to be found in patients convalescent from influenza, according to the supposition of Althaus (Althaus on Influenza).

The cases which Barlow describes as due to "Poisons evolved during metabolism Processes occurring in some of the organs or tissues of the body" and in which he includes some of Polyneuritis occurring in diabetic as well as subjects, also cases due to after excessive muscular exercise Cachexia form one of the so-called idiopathic cases, present greater difficulty in regard to the Carcinuation and Gower calls attention to the chemical relation between Peers and Alcohol in diabetic multiple Nerviers.

It is certainly a curious fact that agents differing so widely
in chemical composition as azelphide of Carbon arsenie alcohol & the toxin of Pfeiffer, and other facts shove all under certain conditions give rise to a somewhat similar set of symptoms.

The distance of the felves from the toxicic cells although it is the only explanation given of the localised distribution of the disease as far as I have yet seen, does not seem capable of explaining that distribution satisfactory. I shall have further occasion to refer to this in connection with Dr. Sedgley Martin's work on the subject.

It may be convenient if I insert here my records of the cases to which I have referred, they are secured in Enfield and the suburban of London.
Case I.

Mrs. Bateman. Age 68.

Residence: Avenue Pavilion. She works in the post office. Her husband was postmaster. She assisted in the stationers shop attached to the post office. She was in this way somewhat exposed to cold and draughts.

History. Family. Father died age 65 of consumption. Mother age 79 cause unknown. 3 brothers all living. 4 sisters. 3 deceased. 1 in childhood, cause unknown. 1 of decline age 30. 1 died at 60 cause unknown. 1 sister alone 75 in good health.

Personal. Has always had good health except for periodic attacks of rheumatic of considerable severity occurring about once a year.

I first saw her on 21 June 1894, she had been feeling unwell for about a week with headache, pain in back.
children & a feeling of nausea.  
caused with slight expectoration.  
Her habits as to food & drink  
were unimportant, usually  
taking 2 glasses of all per  
dian Beldom or never more.  
Her temperature when first  
seen was 102.7 pulse 90  
fever & rales were heard over  
the front of the lungs, the  
 bases were clear.  
She was kept in bed but  
rectal applications to the chest  
an empyema mixture &  
valine enemata were ordered.  
23-24 June.  
Ear 101 pulse 90  
Respiration 26.  
patient had  
learned a fairly good night  
of no pain.  
this evening she  
had a severe attack of  
vomiting which lasted 2 hours  
the vomited matter prevented  
nothing noteworthy.  
23-24 June.  
 improvement  
Bronchitis & fevers few. 
thick 100-67.
25th June. Patient complained of tingling numbness with pain in both feet and legs. The  
feet on inspection showed no abnormality, slight pain and tenderness on handling  
and moving the feet.  
Sac. Sal. 3 1/2 g. k. was  
ordered and continued for  
24 hours without any effect  
in relieving the pain.  
26th June. 98 1/2. Pain in feet  
& legs worse & there was some  
slight oedema of both ankles.  
The pain disturbed patient  
so much that warm applications  
were ordered for the parts, the  
Sac. Sal. was discontinued & 1/4  
grain Camphor. 3rd pill twice  
daily was substituted.  
28th June. Temp. 100. Pulse 88  
pain still severe. On this date  
I first noticed motor weakness  
in the Extensor muscles of both legs.  
29th June. Morning Temp 96.2  evening 99.
30 June. Patient's condition was unchanged. Spontaneous pain, severe tenderness on touching and handling feet and legs, slight oedema of both feet. Temp 100.6
morning, 101 evening. Feeling that there was some diabetic derangement than had been before examined out. The urine contained a large deposit of urea. No albumen or sugar.

The other systems presented nothing remarkable.

Neurous System - Spontaneous pains in feet and legs described as sometimes shooting at other dull aching, always worse at night, numbness of tingling not complained of at this stage. Irregular anaesthetic patches on dorsum of feet, front of legs and to a less extent on back of legs. Tactile sensibility was lost altogether on these patches.
The tactile sensibility of the corneal surface of these parts seemed impaired for although a firm touch was perceived a light one was not, the impression was delayed in perception & the localization of it imperfect.

Sensibility to pain & tested by pricking & Faradic current showed a certain amount of Analgesia in these patches.

Sensibility to Heat & Cold -

Impression, delayed but correctly interpreted excepting in the Anæsthetic areas.

The pupils reacted normally to light and distance.

Hearing had been slightly impaired for some years otherwise unchanged.

Coordination this was difficult to ascertain satisfactorily owing to the motor weakness, the patient was quite unable to stand or walk & I therefore cannot
say whether or not it was at all affected
Motor Symptoms. The tendinous point
of the Sartorius and Pectoralis muscles
in both legs, inability to extend
the toes or approximate the
foot to the front of the leg;
drooping of the feet, the flexor
muscles of the legs also appeared
to be slightly affected; all attempts
to move the feet or legs against
resistance being exceedingly
feeble. The thigh muscles did
not suffer, nor at their time
did any of the other muscles.
Reflexes: Skin - Plantar absent
in both feet. Deep, Patellar
reflex absent in both legs.
No ankle clonus could be
obtained. Organic Reflexes were
unimpaired.
Electrical Conditions. The
batteries used were for Faradism
one of Forster's dry cell batteries
for Constant Current Stokes' a,
supplied by Toronto & Lisset's man
London. Facadeum over the
net pah. Hence with a mild
current gave no contraction
in Extension of either leg, a
tracine current produced
very slight contraction, to a
strong current decided contraction.
The result in the muscle was
similar. The muscle of the
left leg responded if anything
greater more readily than the
right side, but the difference
was slight. Sallvanic Current
applied to net pah. Hence gave
no result. In the Extension muscle
of both legs, Sallvanic reaction
was increased and Ace = C.C.

No motion or trouble. No tendency
to bed-ness, edema of both
ankles & dorsum of feet, with
a glossy appearance of the skin
in these regions.
The Cerebral & mental functions
were as good as ever they had been
24th.
Sleep was much interrupted by the pain in the foot & leg.
Percussion of spine and hot sponge test gave negative result. No girdle pain.
Sdaily. Temp. 98.7. No spontaneous pain. For the first 14 days patient improved.
There was less spontaneous pain although the feet were still very tender on examination. I ordered Pot. Iodid. goats time twice daily, the patient remaining in bed and the feet protected from cold by wrapping in wool. She took nourishment freely. The pain persisted unchanged and the Pain phenomenon was still absent.
22nd June. Patient began to suffer from tingling & numbness in her hands and forearms, this tingling shortly gave place to severe pain similar to what had been
experienced in the legs, and this was followed in 2 days by motor weakness in the hands and arms. At this time I was absent on holiday for a month and Dr. Means of Daling looked after the patient in my absence; he informed me that on 31st July there was a great exacerbation of the pain in the arms, trunk, and on the following day a large rash of herpes vesicles appeared on the extensor aspects of the hands and forearms also on the forehead and cheeks, having been preceded in these parts also by pain of a neuralgic character. On 15th 19 August she had exacerbations of pain in both legs and arms, and from the chart I see the temp rose on these occasions to 102°
On my return I again examined the patient on 5 September. I need not repeat the description of the condition of the feet and legs. The sensory symptoms were the same, motor weakness was greater, the drooping of the feet was more marked. It required a strong faradic current to produce muscular contractions at all. There was increased response to Salvane. Current and contraction occurred with equal readiness at either pole. In the arm, the sensory symptoms were very similar to those in the leg. The muscular weakness giving rise to slight wrist drop, the grasp was feeble, muscular sense diminished, and much fibrillating twitching of the muscles. The skin on the dorsum of the hands was smooth and shining.
27.

The muscles of both forearms responded only to strong faradic currents. Salvarionic current applied to metacarpal bone above the joint. Candles gave no contraction. There was increased response in all the muscles of forearms, and 2 cc = 2.5 cc, but was not greater as far as I observed. Organic reflexes still intact.

20 Sept. Patient became very much worse: temp 102.7, pulse 120, severe frontal headache in the evening or second crop of encephalitis. Vesicles appeared on extensor aspects of hands, forearm, over the malar prominences of cheek, with 2 vesicles on the soft palate.


No signs of involvement of the diaphragm in the paralysis.
Patient gradually lost strength and died on the 26th.

No. Autopsy was permitted.

Case 2. Mrs. Viola, age 43
Residence: Huntington Road, Easting
Examined: 10 June 1874.

General Surroundings: good
Precautions taken: nothing

Present Illness: When in Paris in the spring of 1883 she had a severe attack of influenza. During convalescence she began to have feelings of numbness and tingling in her feet and legs. This was followed by loss of power which gradually increased until she was quite unable to move the legs. Patient cannot give me exact date, but she thinks it was about a month from the commencement of the giddiness.
attack before she felt the loss of power in her limbs. 
The complete loss continued for 6 months after which she gradually began to regain power. She was treated in Paris by massage & electricity. 
She returned to England in November 1893 & consulted Dr. Russell Reynolds, who considered the paralysis to have a peripheral origin & gave a hopeful prognosis. 
Patient came to reside in Rome in February 1894. 
There was nothing remarkable about patient's appearance excepting her gait, which will be presently referred to. 
The nervous system was the only one that presented any abnormality of note. 
Nervous system. No numbness tingling or pain or tenderness on handling the parts at this time.
Sensibility to touch - that affected do. Pain, that affected do. Heat, cold not affected.

Pupil reacted normally to light of distance.

Organic reflexes had not been at all affected.

Motor functions Plantar reflex absent in both feet. Patellar reflex absent in both legs.

There was considerable atrophy of all muscles below the knee in both legs

Attempts to move the leg against resistance were feeble and the patient could not stand or walk without a great deal of support.

The muscles of the lower extremities were the only ones affected.

Coordination - great incoordination of legs, the feet and legs were thrown about in great confusion when patient attempted
to walk, in a word, her movements in this respect were tabetic. No incoordination in the upper extremities.

Electrical Condition. The strongest faradic current that would be borne by the patient produced only feeble contractions. In an normal muscle, this current would have produced violent contraction. Both legs were equally insensitive. Feeble contractions were produced by Salvarsan, and the Odoral

recalled the Cathodal Clamping contraction. Nutritive. Great paleness, and wasting of flesh, of both legs. No joint affection. There had never been any tendency to become tachy in the nature of lightening pains & no past in the other Cases.

Treatment: Massage & Salvarsan.

Patient continues slowly better.
perceptibly to improve. The incoordination of their state (Jan 1895) is not so great & motor power is slowly increasing.

Case 3.
Residence:umberland Road. Islington.
Complaints: Tingling, freezing and slight pain in fingers of toes, inability to walk any distance, a weakness in legs causing him to feel a tendency to fall, palpitation of heart and flatulence.

General Surroundings: In fairly good surroundings, small tradesman class, premises in which he lives hygienic.

History: Bred by his father, notable except that one brother has fits and is very alcoholic.

Habits. He has been taking brandy in admitted excess for some months past, quantity varying
Personal History. Rheumatic disease accompanied by a rash at age 23. No other previous disorders. Married 3 children, all healthy. Wife never miscarried. Describes himself as having always been of a nervous temperament, easily startled and frightened. Two years ago he suffered from freezing and tingling feelings in his fingers and toes with gastric symptoms. Previous to this attack he had been drinking freely but, on the recommendation of a medical man, he gave it up, and the symptoms subsided. He cannot remember how long they took to subside.

Present Illness. Following some months' indulgence in brandy, he again began to suffer from the old freezing and tingling in his fingers and toes in April last, accompanied by
Weakness in the legs, and a tendency to fall, especially if his eyes were closed, he was also troubled by starting and jerking of the muscles of the legs and arms, and inability to execute the finer movements of the fingers required in his trade, so that he had to give up all the fine work he was also subject to slight starting pains in the toes & fingers at times.

The patient is a well developed man, with no obvious marked appearance, excepting a notably degree of Anaemia.

I first examined him 3 June 1894
Temp. 98.6 Pulse 80.


Circulatory System. Occasional slight pain in preaceondia, occasional feelings of faintness
frequent palpitation & dyspnea. Absolute Cardiac dulness was not increased. A soft middling diastolic sound was heard in the apex & axilla, but not audible at Scapular angle. Second pulmonary sound was accentuated.

Urine. No albumen or sugar.

Hypertensive System. Tingling & numbness in fingers and toes. Slight spontaneous pain sometimes in the same parts more frequent at night. Did not complain of any tenderness on handling the parts. Sensibility, a firm touch was distinctly felt. A light touch was not distinctly felt or accurately located on the fingers or toes or on the dorsum of the feet. Heat and cold were correctly interpreted. The mucous membrane appeared to be unaffected. The pupils reacted to light & distance.
Motor Functions. The organic reflexes were all intact. The man's muscles were poorly developed, but there was no obvious wasting. No twitching of any kind in the muscles. Marked excess of the plantar and cremasteric reflexes, others skin reflexes normal. The Bice and wrist phenomena were greatly exaggerated, and there was greatly modified myotatic irritability of all the muscle of the legs and forearms, a slight tap producing marked contractions. Voluntary, although latent complained of motor weakness in the legs, his resistance to movements did not appear to be diminished, and the different movements of the legs were performed without apparent difficulty. The grasp was strong. Coordination. In the legs was affected. Romberg's symptom present.
and if left to himself, he would have fallen. In the upper extremities coordination was not affected, beyond the difficulty before mentioned with his fine work, which I regarded as due to sensory disorder only; he touched any part of his face with both hands without hesitation.

Electrical Conditions. There was marked increase of faradic irritability in all the muscles of the legs and forearms. Vasmotor and nutritive nothing abnormal.

Cerebral and Mental: Unchanged. Hot sponge test & Percussion of Shins gave negative results. Diagnosis: Commencing Alcoholic Polyneuritis.

Treatment and Progress

Alcohol was stopped, Anaemia treated by iron and arsenic. The local treatment consisted
only in massage or rather, simple rubbing of the affected parts, carried out by the patient himself. The patient to have thoroughly carried out the directions about Alcohol, as he soon began to improve, and in 3 weeks had lost all symptoms, although he still complained of slight weakness in the leg. In 5 weeks he said he felt as well as before the attack. The Cardiac bruit could not be heard. Since this patient from time to time and the assume me he is free from symptoms.

Multiple hematomas except as a manifestation of alcoholism, or as a sequel of diphtheria, is I think a malady which is not of common occurrence, at least in general practice it has not been my experience to meet
with many cases of it. These two cases of partial and
polyneuritis [one presenting involvement of the sensory
fibers and the other of the
motor fibers] primarily drew
my attention to the value of
Gowers' classification of the
Clinical Varieties of the Disease
Fig. 1: A sensory (2) A motor and
(3) An Ataxic form. This
classification is clear, simple
and useful, and although one
may not find cases which
conform exactly to one or other
types it still acts as a most
useful guide in studying the
symptoms of disease which
is notorious for the diversity
of its manifestations; their
multiplicity of symptoms is well
seen in Dr. Buzzard's little book
on peripheral neuritis.

Previews to dominant
publications in the Gazette Hebdomadaire.
in 1864 and 1866, one finds records of cases in all probability due to peripheral nerve disease but the idea of a peripheral origin does not seem to have suggested itself to those recorders (Alloisio, L'artea des maladies de la moelle épinière, 3me Edit. Tome II. Obi 76-77 Paris (1837) also (Duchenne, L'Électrization Paralysée p. 610 Paris 1855) in Edinburgh in 1870 Benjamin Bell describes a case of paralysis following anterior fever, probably peripheral without offering any opinion as to the seat of the lesion, although the observation of the Ravan physicians occurring four years earlier would probably be known to them. It is remarkable that Francis of Dublin in describing the épidémie de Paris in 1828 mentions the affection as at least having nothing to do with the brain.
on spinal manœuvr (Graham Clin. Lect. on Pract. of Med. Rev. 1884, Vol. 1, p. 578). In this country Grainger Stewart was undoubtedly the first not only to make a diagnosis of multiple neuritis, but also to confirm his diagnosis by post mortem examination.

(Edin. Med. Journal April 1881) In most cases of the disease it seems to be agreed that there are premonitory symptoms of some kind. Legge (Princip & Pract London 1881) describes it as beginning abruptly and running a rapidly irremediable course (Vol. II, 1424). One of Grainger Stewart's cases showed premonitory symptoms for three, another for six weeks, and the third for 12 days only. Swellings of fingers and toes seem to be among the most common prodromata, and
these symptoms according to Charlton Bastian (Deans Oct 1874) may precede the loss of power even for months. So the symptoms, bronchial, cardiac, and general rheumatic symptom according to Gowers are some of the symptoms complained of while the disease is developing especially in its alcoholic form.

An acute onset Gowers regards as rare, "unless excited by some coherent influence." This is readily understood as the effects of the poison must first be felt disturbing the function of the nerves before impairing their nutrition. In the alcoholic case described in this paper the disturbed function was felt for some 9 to 10 weeks before the onset of the slight ataxic and motor symptoms which he afterwards displayed. The patient's attention having
been particularly dreaded to them, as they interfered with his acceleration. In other two cases were preceded by what I took to be, and had described to me in one case, ordinary attacks of influenza. Case 1 the palsy developed rapidly motor symptoms showing themselves on the 9th day from the time I saw her. In Case 2 motor symptoms were observed about 3 weeks from the commencement of patient’s illness.

The alcoholic case exhibited the symptom of increased mental instability which was the first time I had had an opportunity of observing this symptom in the disease. Its occurrence in the early stage of multiple recurrent is mentioned by many writers on the subject e.g. James & Raro.
In Case No. 1 there was marked petrullary tenchening in the muscle of both forearms, but I have never had an opportunity of witnessing the violent jutting on attempted voluntary movements, described by Ross and which he believes were frequently mistaken for Ataxia (Ross & Berg, Penumb. Memoir, p. 24). In this same case the biceps of the forearms were considerably flattened, and had lost their roundness of contour. The wasting of the interossei on the dorsum of the hands was also observable, the thenar & hypothenar eminences did not appear to have wasted to any great extent. Dr. Ross and Berg in their work mentioned above have described and figured a great many deformities produced by the muscular
wasting which occurs in this disease. Chief amongst these are the characteristic foot and wrist deformities, also the so-called 'main on galle' and amongst the minor deformities, narrowing between the metacarpal bones, flattening of the thenar and hypothenar eminences, and the back of the forearm. Distortions of the fingers are mentioned by these authors as of importance to an early recognition of the disease e.g. the weakness of the opponens pollicis permitting the metacarpal bone of the thumb to be drawn back so as to lie in the same plane as that of the fingers, also an inability of patients in the early stages of the disease to flex the thumb into the palm without at the same true flexing the distal phalanx.
and which is manifested by asking the patient to touch the tip of his little finger with his thumb. The distortion in the lower exanthem is when affected present similar characters

In Case No. 1, the eruptions of pemphigus which occurred during the course of the disease were doubtless produced by disturbed innervation, the trophic nerve strands having become affected in the medullary masses. These eruptions both were distributed on the backs of the forearms and hands on the forehead, malar regions, scalp, and chin. The eruption was in each instance preceded by severe neuralgic pain and occupied the regions on the face which are supplied by the supra orbital and supra orbital orbital branches,
of the facial nerve, and some mental twinges of the trigeminal. There was no paralysis of the oculi or frontalis, or any muscle of the eye or face as I made a careful examination to ascertain if there were changes in this region were accompanied or followed by any motor weakness.

In regard to multiple hereditaries and bullous exanthems there is one case of pemphigus mentioned in Bassano's little book on Peripheral dermatitis. But the most remarkable instances of traffic influence in the disease are I think to be found in these cases where the fumes of carbon monoxide were the toxic agent, one of the most striking being that mentioned by Hare (Pflüger's Zeit N.S. Bd. II p. 35. 1869).
in which the whole surface of the body was covered with a pemphigous eruption. Several cases of herpetic eruptions are recorded by Bendit in his description of Pemphigal skin disorders due to carbon (Archives Générales de Médecine, Tome VII p. 516, 1865, Paris) also a case by Lütte (Deut. Wochenschrift No. 5, 1899). Bullous eruptions seem also to be a fairly common accompaniment of Arsenical paralytic (Bowers, Durian, System Vol. III). In all the 3 cases which I have given here the organic reflexes remained intact.

For Raw and Bury (Pemphigal, second ed. p. 159) report 10 cases in which the sphincters were involved in the paralysis. Fagge (1891 edition) describes the sphincters as being rarely involved.
and in Bizzards book on peripheral nerves. Several cases in which the organic reflexes were involved are recorded. Gowers and Bates (Brain Dec. 1894) regard involvement of the organic reflex as pointing to central changes. Gowers mentions the nerves of the alimentary viscera as seeming always to escape. In connection with the question of visceral reflexes there is an interesting paper by Ferguson (Alcohol and Neurologist) in which he describes a case showing implication of the bladder and other abdominal viscera. Gowers regards the immunity of Dr. Williamson of the lung nerves as more doubtful and Dr. Williamson has since found degenerative change in the pulmonary branches of the Vagus and in the terminal
mucous membrane of the Phrenic in a case of alcoholic neuritis (Ross & Bucy page 145)

Implication of the organic reflexes as a rule then would seem to justify one in regarding the cord itself as suffering in addition to the peripheral disease. In the ten cases mentioned by Ross & Bucy in which the sphincters were involved, there is no record of any autopsy; had there been it would in all probability have revealed some degenerative change in the cord.

Granger Glendevi's second case (Edin. Med. Jour April 1881) presented girdle pain and involvement of the bladder as symptoms, and in the 37th case of that series secondary degenerative changes were demonstrated occupying the usual sites of these secondary changes viz. the columns of Gall and Sweet-Aubert-rect.
Regarding the ataxia of Peripheral nerves, I am of opinion that cases 2 and 3 recorded here presented true ataxia, unmistakable for some muscular weakness. I shall have further opportunity of referring to the subject of ataxia in this disease when discussing its pathology. In case No. 1, the power of performing combined movements was imperfect, but this I attributed to the great muscular weakness in both upper and lower extremities, and owing to this weakness there was some difficulty in estimating whether or not true ataxia did exist. My conclusions on this case however were that it did not present any real ataxic symptoms. Case No. 2 on the other hand was a marked example of nervous
takes peripherical, the legs
being jerked about and
brought forcibly down in
quite a Tetanic fashion. As
far as I have been able to judge
the high stepping gait and
dropped toes which have
been described as so character-
istic of this disorder, do not
present characters less suffi-
ciently different from the Faked
gait, to help one much in
the differential diagnosis
of the two maladies, most
peoples would in case No I
had they attempted a diagnosis
by the gait alone would I
think have said it was a
case of Fakes Dorsali.
Dr. Pen (Ran and Bury)
seemed to doubt the occurrence
of true Ataxia in Polyanesthia.
especially in the Alcoholler
and Diphtherite forms, but
his colleague Dr. Berry has
since seen cases which have caused them to alter that opinion which he expressed in common with Dr. Ross.
Such observers as Leyden relate a case of ataxia of the lower extremities attributable to a poisons caused by exposure to hemp and coal (Leyden Uber einen Cent.
Ataxie Zeitscher f. Klin. Med. 1891 page 7676). It seems impossible to say at present what proportion of cases of this disease present atactic symptoms, but as regards the past general manifestations of the disease in reading over the recorded cases to which I have had access the predominance of motor and ataxic men
sory forms certainly struck one as being somewhat marked, whether the greek No Toreno
has a selective action picking out the motor fibres and these concerned in co-ordination (if any such exist) can only be determined by the study of a great many more cases than have as yet been recorded of multiple necrotic occurring after influenza; but having regard to the known selective action of lead for motor nerve fibres and of such toxicemic agents as the products of Löffler's bacillus in bringing about a paralysis which is chiefly motor a similar selective action by the products of Pfeiffer's influenza bacillus is very probable.

The organic reflexes in all 3 of my cases were unaffected during the course of the disease, in the case which proved fatal there was no retraction or falling in of the epigastrium pointing to involvement of the Chilblain.
the cause of death in this case was I believe exhaustion owing to the extensive effusion of the pemphigus bullae. These bullae corresponded to the ulcerations of the follicular variety of pemphigus: for 3 days prior to death there was a considerable discharge of purulent matter tinged with blood from the bowels, from which I presumed the presence of bullae in some part of the bowel, probably the rectum, there was no tendency to the formation of beds more at any period of the case.

In the fatal case I had unfortunately no opportunity of fortifying my diagnosis by an autopsy, permission to remove any part of nerves being refused. The general account of the appearances in the nerves when there are
sufficiently escape to give rise to naked eye appearances, are reddness and swelling, with minute haemorrhages, with loss of contour and want of lustre in the nerve affected, in the later stage, wakening and in some instances, reduction of the nerve to mere connective tissue strands (Gowers Vol II p. 131. Bastian in 'Luzius', Dict. 1874). According to these observers, the naked eye appearances would seem largely to depend on the extent to which the nerve terminals of connective tissue were involved. Sidney Martin (Journal of Path and Bact. Feb 1873) points out that the degeneration occurring in the affected nerves differs at first from the Wallerian degeneration, that degeneration being secondary to another change.
which constitute a local and partial Injury to the nerve. According to S. Martin, certain regions of the affected nerve trunk, when treated by Osmin Acid, were observed not to take on the stain, these unstained portions extending certain across the branch or only in bundles of fibres, the white substance being attenuated in these regions while above and below the staining showed the presence of the white substance. The axio cylinder of these unstained parts then becomes attenuated and finally substances taking on the Osmin Acid stain. The rest of the process according to S. Martin then proceeds as in the ordinary Wallerian degeneration of nerve fibres from the site
of reaction to the muscle. Dr. Martin also points out the interrupted degeneration that sometimes occurs. These observations then show that the nerve fibre does not degenerate to the periphery until the axis cylinder has thickened. Dr. Martin found the process in alcoholic and ethylated cases to be essentially the same. In the same paper Dr. Martin rejects the theory of trophic influence as accounting for the peripheral distribution of Polyneuritis, pointing out that a nerve when cut off from its trophic centre dies from above downwards, not from below upwards. He however offers no explanation in place of the trophic one, nor have I as yet seen any advanced. Gowers noted post mortem
a selective distribution of the nerve degeneration case which presented motor and atactic symptoms during life showing when the mixed fibers were examined many healthy fibers mingled with the degenerated ones, then one might assume to be unaffected sensory fibers, and conversely where sensory symptoms were most marked we might look upon the unaffected fibers as motor.

Most writers on the subject of multiple heretics seem agreed that the Anterior Tibial in the leg and the Musculo Spinal in the arms are usually the first and chief to suffer, and their would appear to apply more especially to the Alkalied forms. Descriptions of degeneration in the Basi and
Phrenic nerves are recorded in Ross and Bury p. 1144. Gowers describes the nerves of the elementary viscera as seeming always to crowd, but gives no reason why it should be so; with all regard to Gowers' authority on this subject, I am inclined to attribute the haemorrhages peculiar on the soft palate and presumably in the retina, which occurred in one of my cases to implication of the trophic nerves of these parts.

The pathological changes in the muscles in this disease would appear to be principally an increase in the interstitial tissue, especially of its nuclear elements, (Power, Vol II p. 134) and in acute case paralysis, metastatic change and loss of
normal stratum is added; these parenchymatous changes in the muscles have been found principally in dephrenetic cases, but there are some recorded in which they were present in cases which were not dephrenetic in origin. There are 4 cases mentioned by Hackhauer and quoted by Gowers (Vol II p.192) in which the muscles only were affected forming an acute myoarthritis, the nerves and eccitities in these cases being found healthy. These cases are most remarkable, greater intensity of the toxemia being insufficient to explain the excuse of the nerves.

S. Martin in the article quoted above describes two alcoholic cases in which the muscles showed more or less fatty degeneration, the muscle fibers
which presented a normal appearance being supplied by undegenerated nerve fibers. In a case of post-deahthracic neuritis described by the same observer the faulty degeneration was most marked in the muscles of the soft palate and cleathraign, the heart muscle and the vortic also showing some faulty fibers.

The spinal cord has been found healthy in a majority of cases of Poliomyelitis especially those of a non alcalaloe origin. Grainger Stewart at an early period in the recognition of the disease pointed out secondary degeneration in Gallio's columns and the direct cerebello-tect tract in a case which was not alcalaloe in origin. Powers mentions irregular disseminated areas of mepletic in occurring.
also Chronic inflammation of
the pia mater sometimes
involving the peripheral
layers of the cord, also a
general increase of connective
tissue throughout the cord,
especially marked in the
posterior columns. This
meningo-myelitis occurring
in alcoholic cases would
appear however to be a
separate effect altogether
of alcohol and to be in no
way related to the presence
of peripheral lesions, I
have been unable to find
any record of its occurrence
in other than alcoholic
cases, although in saying
this I must add that I
do not include Landrey's
paralysis, as post mortem
examination in that disease
has in some cases shown changes
of the nature of a Meningo-myelitis.
Such a large amount of attention has in recent years been devoted to diseases of the peripheral nerves, and lessons of these themes have been demonstrated and recorded so thoroughly upon so many diseases, that a consideration of the pathology of multiple neuretias, involves to a certain extent the study of all pathological conditions of which it may prove a sequel; it has been found to occur after nearly all the fever...
Diphtheria, Influenza, Pertussis (Brit med. Jour. 25 Aug. 1891)
SYPHILIS, PNEUMONIA, TUBERCLE, MALARIA, DIABETES, SEPTICAEMIA
been here (in itself a form of the affection) typany, larynx
Hagnands disease etc. besides
the degenerative forms and the
toxic forms (metabolic and non metabolic). Each of these
different causes seem to
bring about under certain
unknown conditions, a similar
degeneration of nerve fibres,
and give rise to set of
associated symptoms and
results constituting a disease
asso distinct from a mere
pathological process; under
these circumstances it might
be well to give up the term of
Alcoholic Pneumonia or denomi-
native paralysis substituting
Amyotonia for paralysis, a conveying
a more definite idea of the pathology.
of the conditions. Rosenheim (Archiv f. Psych. VIII 3) was the first to suggest the chemical products of the various organisms as producing the resulting nerve affection, a view which is now very generally accepted and would seem to explain the common action of so many different micro organisms. Jameson (Vol i p. 137) further points out that alcohol is known to be a frequent result of the growth of certain organisms. & kidney martin in his researches into the chemical pathology of anthrax (Paul- otomi's lectures 1882) succeeded in separating three toxins from the tissues of patients dead of anthrax. These consisted of two albumenases chiefly. Another albumenase associated with an organic acid, these albumenases when injected into
rabbits. Dr. Martin found pain
rise to a definite symptom
ly Paroxysms of muscles, which
was always general and
progressive, often less constant
effects were produced which
do not directly concern my
subject.

The hypothesis is advanced
by some that the changes in
the nerves are due to
impairment of their nutrition
by primary functional changes
in the spinal tracts enter,
only seems to involve the
subject, and it is much
simpler and certainly more
in accordance with the
morbid conditions found in
the nerves, to suppose the
Cause acting on the fibres
themselves. Dr. Charlton Bax-
nows out a further objection
to their theory in that it does
not hold in the least to explain
the peripheral distribution of
the nerve degeneration.
If then multiple neuritis is
always due to a parasod
blood state we can explain
its asymmetrical distribution.
the distance from the trachea
spinal centres has hitherto
been regarded as accounting
for its peripheral distribution.
although D. Sydney menton
does not regard this as being
capable of explaining on a
satisfactory manner the involvement
of peripheral nerves. But in
what way are we to account
for the electrice action of the
toxines on the different acts
of nerve fibres, sensory and
motor constituting a reflex or
on the nerve fibres pulling
a certain group of muscles, the
alkalalic form usually selecting
first and chiefly the extensor
groups of muscles, the debaristi
the soft palate and ocular muscles; and we frequently see what we take to be the same toxins in one case selecting the sensory or the motor fibers principally, it is possible that in this latter case the choice is determined by varying acceptability of the different fibers in different individuals, or attributable to some slight of the medium in which the toxins is produced.

That type of multiple toxins in which the patients present symptoms of incoordination, and which have received the name of pseudo-takes, or nervous takes peripherica are of some importance as regards their differential diagnosis from Takes, Cerealis. Whichers explanation of Ataxia one accepts be it the reasoning and
Reflex theory, or the motor theory around which so much discussion has raged on whether we admit the existence of nerve fibres especially concerned in coordination; as a rule there is not much difficulty in recognising the symptoms when seen, the irregularity of the associated muscular movements is generally sufficiently obvious, and it is only in these cases associated with considerable weakness that doubt is likely to occur, and it is this difficulty occurring in Polynœmati that has caused some observers e.g. Dr. Rams (Pentheral Neurotic Rams & Burg) to altogether doubt the existence of true atonia in this disease. In connection with true Tarses and multiple nerves.
the investigations of Meithkal, Dejonyms and Tolley (Arch. de med. Exper. et d'anat. path. 1899. I. 25-1. and The Revue de med. 1889. N. 81. 208 and 284) have demonstrated disease of the peripheral nerves as frequently coexisting with that of the posterior columns; Sandrow's recent researches into the condition of the Cerebral Cortex in the same disease and what bearing it has on the question of Ataxia. I have unfortunately no means of being acquainted with at present. Notwithstanding the great authority of Dr. Rocro on this subject I think if we search the recorded cases of Multiple Neuromatis we find sufficient evidence that disease of apparent fibrous in itself capable of giving rise to incoordination
Among the competent observers who have described ataxia with peripheral lesions only are De Fornie (Revue de Med. 1887, Revue Méd. 1889, Revue Méd. 1896, and Revue Méd. 1899) and Deman (Zeitschrift für Neurologie und Psychiatrie). In 1870, these two cases of ataxia were described by Gowers and Stewart, in both there case the atactic symptoms were very prominent, and in one of them the muscular power and cutaneous sensitivity were unimpaired (Edin. med. Journal, May 1870). In the same number of the Edin. Journal there is a case by the late Benjamin Bell "On a peculiar paralytic condition of the lower limbs following gastric fever, in
Atactic symptoms are described and which was doubtless an example of Polynematics following Enteric Fever.

The second case which I have described here was for some time most puzzling on account of the very atactic character of the ataxia, but the motor weakness, the rapid onset of the symptoms, and altered electrical condition decided in favour of a peripheral lesion, and the progress of the case is justifying that diagnosis. Cases of peripheral nerve disease presenting chiefly sensory and atactic symptoms, would present the greatest difficulty in diagnosing from true tabes, especially as it is said to occur occasionally in tabes there was an acute onset and muscular wasting. Electrical changes in
such cases might prove
useful in coming to a
decision, but in some cases
of the peripheral affection
they are said to be unaltered
or so slightly changed as
to give no guidance in diagnosis.

The conclusions of T. J.
Bury (Ross and Bury, Peripheral
sensitiveness) on the subject of the
Ataxia of Nervetti are that
"true Ataxia is a comparatively
rare symptom in multiple remittent
even when the Cutaneous and
Muscular sensitiveness are nearly
affected, but that it does occur
when signs of Muscular weakness
or diminished Cutaneous
sensitiveness are slight or absent."

The passive motion and trophic
changes occurring in the skin
and Subcutaneous tissues
in lesions of the peripheral
nerves present numerous
points of interest, from the
well known glancey orien seen
in the disease. They range in
intensity to changes which
are sometimes almost gangrenous.
The case mentioned and
illustrated in Grainger Stewart
Introduction to Diseases of the
Human System appears a
very probable example of
peripheral nerve disease
producing gangrene in the
hands and feet. There would
also appear to be grounds for
regarding some at least of the
local lymphatic asphyxias
and gangrene described by
Raynaud as due to peripheral
nerve lesion. Balhmaninoff
(Revue de Med. 1892 p. 321) Weges-
worth (Brit. Med. Jour. 1887 p. 57
and 60) and Apple R (Brit
Med Journd 1886 p. 1269) have
observed degeneration of the
peripheral nerves in patients in
whom gangrene of the
fingers and toes had occurred. Raymond himself regards the oedema in these cases as an epiphenomenon. This explanation of Raymond's disease would of course preserve the existence of special vasomotor nerve fibres which were picked out by this form of neuritis: and in the future should the existence of such fibres be established there might be reason for changing one's views as to the pathology of Raymond's disease, until these fibres are proved to exist one cannot: continue to regard that disease as produced by the imitation of morbidly active vasomotor centres in the brain and spinal cord.

I have already referred to the two outbreaks of Pemphigus which occurred in one of the cases.
recorded in this paper. The first eruption manifested itself simultaneously with an marked increase in the intensity of the sensory symptoms in the arms, and having the distribution described in the case, these bullae disappeared leaving the usual pigmentation. The second eruption also occurred with an exacerbation of the spontaneous pain and much fibrillary twitching of the extensor muscles of the forearms. The distribution of this second eruption was more extensive than the first more symmetrical in arrangement and consisted of larger bullae. These bullae rapidly became purulent spreading at the periphery as the bullae formed at their centres. Between the two eruptions the patient presented from time to time white patches on the inner side.
of the cheeks, and on the soft palate, these white patches are described by Parker and Sampson (Quain's Diet. 1874) as being characteristic of the variety P. Vegetans, thought to be of a syphilitic origin. I can offer no explanation as to how the lower extremities, which were just attacked by the new ulcers, were altogether exempted from the eruption. I attributed the immediate cause of death in this case to nephraemic absorption from the numerous large bullae.

The question as to whether Sandry's paralysis is to be regarded as a form of peripheral nerve disease, at present seems as far from being settled as was D. Baro's in his book on Peripheral Nervous; he collected 93 cases, said to have been cases of Sandry's paralysis, and after
an elaborate analysis of these cases he is led to regard the
hitherto mysterious malady as a form of peripheral neuritis.
It is interesting and not a little difficult to see by what
means O. Ross has arrived at
this opinion from an analysis
of the 93 Cases. Only of the cases
proved fatal. I do not know what
the percentage of deaths in
multiple neurites is, but anyhow
it is very small. I presume
therefore that O. Ross would
give this by regarding
Acute Ascending paralysis as
an extremely intense form
of multiple neuritis, but if
this were so surely the Autopsies
would reveal some result of
it in the nerve fibers. There
were 45 Autopsies and in only
6 were changes demonstrated in
the peripheral nerves, whether
the nerves were or were not
examined, in the other 39 cases it is not definitely stated; in a few it is expressly remarked that they were not examined.
In 18 out of the 46 marked changes of different kinds were found in the brain and spinal cord, or in both.
As regards the etiology of the 93 cases, 71 have some cause assigned, in 22 the cause is unknown; alcohol is only made accountable for 5 cases in one complicated with syphilis. 12 cases are set down to syphilis which Gowers regards as mainly giving rise to an adventitious isolated meningitis, except in its tabetic manifestations when it may cause a true parenchymatous meningitis (Gowers Vol. I p. 113) there 12 cases are not mentioned as having shown any tabetic symptoms. Next in the etiology come
a long series of 20 cases in which the disease occurred during convalescence from acute specific diseases, typhus, typhoid, diathema Pertussis, etc etc; exposure to cold is accountable for the onset of 11 cases. I occurred during the premenstruum: 1902, 1 rheumatic: and so on through all the 71 cases. (Parks and Bury: Perihedral necrotic reaction on Lady's pancy.)

Looking at the etiology as a whole one is struck by the small number of cases attributed to alcohol, such a potent factor in the production of polyureter. The authors seem to feel this themselves and have resorted to what are really only suppositions to restore alcohol to its acknowledged place as a cause of multiple necrosis.
(Ran and Bury. "Memorial to the late Dr. F.") The metallic
agents are represented as
the administration of 3 cases, 2 by
mercuric chloride, and 1 by
the tincture of antimony.
This last being employed
medicinally in the treatment
of pneumonia from which
the patient was suffering. This
caution is I think hardly likely
It would occupy too much
space to detail the means by
which Dr. Burns converts 12 cases
with no history of alcoholism,
into examples of alcoholic neuritis.
That however is the result he
arrives at.

Nearly all the writers on
Acute ascending paralysis
describe unaltered electrical reaction
as one important sign in the
differential diagnosis of the
aforementioned. In 35 of the cases
tabulated by Burns the electrical
Reactions were noted. In 11 of these the reactions were charged or lost, and in 24 cases they were normal. In 12 cases the nature of the current employed is not stated; in 11 cases Faradism alone was employed, and in 9 cases both Faradism and Galvanism were noted. Of these last 9 cases, 3 showed reaction of degeneration completely and one partial R. D. 1 case showed diminution to Galvanism no R. D. and normal Faradis reactions. One case gave in the peroneal muscles only ACC7CCE often regions normal. Out of the 12 cases showing R. D. 3 recovered completely, one dying of bronchitis, presumably independent of the nerve disease. These 3 cases exhibiting R. D. and ending in recovery might very probably have been cases
of ordinary multiple neuritis, if they were cases of Lander's paralysis, then the commonly accepted idea of the electrical reactions remaining unchanged in that disease must be altogether misleading; on the other hand unaltered electrical reactions do not exclude neuritis as evidenced by one of Dr. Buzzard's cases, and one mentioned by Yagge (Vol. i. p. 1224) and indeed in many cases of slight paralysis from peripheral lesions, the Faradic and Galvanic irritability are either normal or so slightly altered as to give little or no help in diagnosis.

Nothing instructive is to be gained from the records of the cutaneous reflexes and the myotatic irritability in the series of cases tabulated by Ross and Bury; they appear
to have been lost in all the cases where they are mentioned at all. Definite loss of sensation is not regarded as occurring in typical cases of Landry's paralysia (Barnes' disease, 1894) and this writer regards sensibility as scarcely if at all affected, nor are pains complained of in the paralyzed parts.

Sower (Vol. 1, p. 382) remarks "that the tingling and formication has in some cases been followed by hypersensitiveness of the skin and tenderness of the muscles, but in such cases it is probable there has been multiple neuritis." In Park's 91 cases 71 had sensory symptoms, not only in the monitory stage, but also in the stage of paralysia, these sensory disorders were formication
numbness, and other paraesthesiae and in many cases severe spontaneous pain. Lastly as regards the motor symptoms in the 93 cases the order in which the muscles were attacked by paralysis varied much, in many the feet and legs rubbered first and then the arms, the trunk muscles being the last involved. In 10 cases the bulbar nerves were first attacked, and in 3 cases there were implicated secondarily to the lower extremities. Landry's description of the disease gives the following as the order in which the various muscles are attacked:

1. F E and foot muscle
2. Leg, thigh and pelvis muscles
3. Fingers, hand, arm and scapular muscles
4. Trunk muscles
5. Respiratory muscles
6. Tongue, pharynx and oesophageal
muscles. The usually accepted order of the muscles attacked in Landry's paralysis, as given in most standard works on the nervous system is (1) leg muscles (2) pelvic loin and abdominal muscles (3) thoracic muscles (4) muscles of arms either elbow or forearms (5) diaphragm and neck muscles (6) muscle of the palate.

In 15 out of the 93 cases the functions of the bladder and rectum were impaired in one case from weakness of the abdominal muscles.

The question therefore as to whether Landry's paralysis is to be regarded as related to multiple neuritis, does not appear to me to have gained anything at all approaching decisive evidence from Dr. Par's collection of cases and analysis.
Thereof, and my impressions in reading the section on Sandys' paralysis in this book on peripheral neuritis do not lead to an adoption of his views in regarding Acute Ascending paralysis as a form of multiple neuritis.

The diagnosis of neuritis from neuralgia is usually a matter of no great difficulty. It is only in the case of fixed neuralgias and some of the slighter forms of neuritis that any difficulty would be experienced; the pain of neuritis is truly neuralgic in character and affords no balm in distinguishing the two maladies, and moreover alcoholic patients are frequently the subjects of both diseases. The absence of motor weakness and the excess of the sensory pain in neuralgia
together with the absence of the signs of neuritis, such as tenderness of muscles, he would decide the diagnosis. Acute poliomyelitis anterior occurring in adults may sometimes closely simulate cases of multiple neuritis with an acute onset, but here the history of the case, the mode of onset and the gradual development of the paralysis in the spinal medulla, along with the absence of marked sensory symptoms, would furnish important diagnostic points, if a case showing the symptoms of poliomyelitis anterior acute showed pain along the course of the nerves and tenderness of the muscles, there would probably be peripheral changes in addition to the spinal lesion. Difficulties however
in distinguishing these two diseases are only likely to arise in the early stages of both, as the case progressed, the peripheral affection would be marked by advancing palsy and a subsequent interval before any improvement set in, the spinal malady on the other hand would usually be recognized by the simultaneous and widespread paralysis, subsiding in some of the parts, first attacked. But according to Gowers in all cases of multiple neuritis due to autogenous poisons, which are also the common cause of acute atrophie paralysis, considerable doubt must exist for some time, in the absence of well marked sensory symptoms as to the seat of the lesion.
The subacute and chronic forms of poliomyelitis present much greater difficulty than the acute forms in diagnosing from disorders of the nerves, and this is especially the case in the motor forms of poliomyelitis, and the difficulty would appear to be further increased by the probability that changes in the anterior horns and in the motor fibres of peripheral nerves frequently occur together. This is pointed out by Sowers, and he takes the present available facts in regard to these diseases to point to common implication of central and peripheral structures of common function (Sowers Vol. 1 p. 380). In the diagnosis of such cases the distribution of the palsy would be a most
important guide, a symmetrical distribution suggesting a peripheral origin, and the converse. Such cases would appear to be rather more theoretical than practical, as it would only be where the characteristic sensory symptoms were absent in the peripheral disease that error would be likely to arise. Gowers divides these subacute and Chronic Spinal paraplegia into 3 groups, **none differing from the Acute Atrophy** form only in its less rapid development, and distinguished from polyneuropathy by the same features as distinguish the Acute Atrophy Variety. (2) A progressive group many cases of which he believes to be examples of progressive muscular atrophy with a
subacute onset the affection beginning as a subacute atonic palsy to distinguished this from polyneuritis Dowers believes that it is necessary to wait for signs of slower wasting in other parts than those first affected (Dowers Vol. p.p. 495-483. 379) in this form the degenerative electrical reaction in often absent

(3) the third group of these palsy according to Dowers are frequently cases of polyneuritis, and he also states the possibility of mistaking the peripheral for the spinal disease. The more perfect symmetry of the peripheral affection, he takes as the Chief diagnostic point, along with tenderness of the nerve trunks, and the distribution to which they are distributed absence of
of these two last points would however be no quick in peripheral cases where fibres of motor function only were suffering.

The symmetrical wasting and paralysis of the extensors of the forearm in Chronic Muscular Atrophy at the outset of that disease, might sometimes suggest Plummer's neuritis.

Careful search would in such a case be necessary to exclude any possible source of lead poisoning, and in the case of poisoning by that mineral some of the other symptoms would in all likelihood be present.

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