MYELITIC MANIFESTATIONS OF NEURO-LUES :

A PATHOLOGIC AND CLINICAL STUDY .

ALEXANDER F . R . DEWAR .

SEPTEMBER 1933 .

ProQuest LLC.
789 East Eisenhower Parkway
P.O. Box 1346
Ann Arbor, MI 48106 – 1346

FOREWORD.

In the course of neurological work we were struck by the variety of symptoms and signs met with in syphilis of the central nervous system. At first we had hoped to make a wider survey but the vastness of the literature forced us to restrict our attention to the more narrow sphere of syphilis of the spinal cord. We must confess to occasional repetition but this seemed inevitable in separating the subject into pathological and clinical sections. We have touched on many points;no attempt has been made at completeness. The relevant and salient features only have secured recognition. As will be seen from the references,we have availed ourselves amply of foreign and other neurological journals. Nonne's masterpiece,his Syphilis und Nervensystem, will remain for long the basis for those seeking knowledge in this branch of medicine. We have made full use of the writings of many authorities, Dejerine,Dana,Chung,Erb,Leri,Leopold,Margulis, Spiller,Oppenheim,Raymond,Williamson,and others. The pathology and symptomatology have been extensively worked out. The field for greatest scope would seem to lie in the problem of pathogenesis, and to that end the efforts of future workers will be directed.

CONTENTS.

ACUTE MYELITIS. In discussing myelitis Bassoe and Hassin draw attention to the great variety of spinal cord lesions included in that category, such as, inflammatory, degenerative, and necrobiotic changes caused by thrombosis, embolism, and endarteritis. Clinically, the picture of myelitis and myelomalacia may be identical and in a few cases the pathological findings fail to differentiate. Myelomalacia or so-called "yellow" softening of the spinal cord cannot be looked upon as a myelitis, yet inflammatory conditions ultimately may cause the softening described, for example in poliomyelitis, Pott's disease, and syphilitic cord lesions. Areas in such cases are usually small and circumscribed. The most frequent cause of softening is vascular thrombosis. (thrombotic softening of the spinal cord or "spinal thrombosis" of Singer). Mager states that 80% of cases of so-called acute myelitis are due to necrobiotic or degenerative processes, normally brought on by vascular changes, either infiltrative or proliferative. In acute syphilitic paraplegia, the meninges and cord may be affected, either alone or together. In the meninges, there may be disease of the blood vessels only, (Schmaus, Sottas, Biernacki, Moeller), or in the neighbourhood, inflammatory infiltrations round the vessels, (Goldflam, Rosin, Raymond), or a frank meningitis, (Sottas and Lancereaux), (Siemerling, Williamson), and under certain circumstances, small gummatous processes. (Lamy, Sottas and Lancereaux). The cord shews a more or less extensive vascular

destruction,irregular patches of softening,or sclerosis, (Schmaus,Dejerine,Raymond),or a larger more or less transverse patch usually in the mid-dorsal region. (Sottas et al.). Both Lancereaux and Raymond describe cases shewing a necrosis of grey matter adjacent to changes in the white substance. Siemerling mentions the possibility of a gummatous mass penetrating the white substance from the meninges. Finally there is Williamson's case of a haemmorhage into the grey substance with consequent secondary necrosis. Varying degrees of endarteritis,periarteritis,and mesoarteritis are invariably found in the vessels. Sottas found small gummata in the vessel walls. Babonneix and Widiez describe a case of amyotrophic lateral which shewed at post-mortem an aneurysm of the anterior spinal artery with diffuse inflammatory changes affecting the crossed pyramidal and Goll's tracts. Thrombosis of the smaller and larger spinal arteries are a frequent finding as are also thromboses in the endo- and peri-phlebitically diseased vessels. The patches of softening are found most usually towards the periphery of the lateral and posterior columns or in the anterior grey matter. Cole reports a case shewing vascularisation in the lumbo-sacral region. The pia-arachnoid over the first three lumbar segments was slightly fibrosed. The meningeal vessels were filled with blood,one artery shewing a recent marginal thrombus. Several arteries shewed small areas of fibrosed intima slightly distorting the lumina. In a few arteries there was marked oedema of the entire

wall with a moderately severe infiltration of lymphoid cells. Locally the periphery of the cord shewed several areas of increased neuroglia irregularly distributed. The white matter shewed a few areas of irregular sheath degeneration,not to be regarded as entire tract degeneration,but appearing more particularly in the lateral columns and affecting only slightly the anterior pyramidal tracts and columns of Burdach and Goll. Congestion was present throughout the cord,being prominent in the grey matter and particularly in that of the posterior horns. The greater part of the posterior horn almost to the emergence of the posterior roots was the seat of severe degeneration. The peri-vascular spaces were much enlarged and oedema extended into and was accompanied by marked disorganisation of the substance of the horns. The diagnosis was necessarily subacute syphilitic meningitis with acute myelitis involving particularly the posterior cornu. In Dejerine's two cases there was central myelitis of the grey substance. In one of Williamson's cases there was haematomyelia and thromboses scattered through the grey substance of the cord with accompanying arterial and phlebitic disease,syphilitic vascular damage with resultant haemorrhages. In 1920,R.Henneberg published reports of two cases of pure vascular spinal lues. In the first there was an extending ischaemic area following atypical syphilitic vascular disease shewing endarteritis and endo-phlebitis obliterans with hyaline degeneration of vessels. The second case shewed a sclerosing process rather than coftening,a condition

the sequel to the slower course of the disease. THE meninges are seldom spared;there is frequently both pachymeningitis and leptomeningitis. In many cases there ensues an ascending and descending "secondary degeneration". As Nonne points out,softening of the spinal cord substance is usually a consequence of thrombosis. The pial vessels may be diseased,in this way affecting the nourishment of the cord substance (Biernacki),or the cord vessels themselves may be involved. If the area supplied by the vasa corona is affected,the resulting picture is one of multiple patches in the periphery of the white substance of the lateral,posterior,and anterior columns;an endarteritis of the anterior spinal artery affects primarily the anterior grey matter.

Chung made a study of 34 cases of rapidly developing SYPHILITIC PARAPLEGIA and divided them clinically into three types,the one presenting either a complete loss or a reduction of all sensations and a complete motor paralysis. The cases of this group are manifestly clear-cut transverse lesions and vary only in the degree of injury which affects the components of the cord. In cases of sudden paraplegia with few or no premonitory symptoms,the meningitic process although widespread is not prominent,whereas the thrombotic lesions are confined to one or more important vessels. It follows that in cases of subacute interference in the blood supply when the clinical symptoms develop less rapidly and extend over a longer period,say one month or less,the changes

in the meninges and blood vessels correspond to those of a severe meningitis. It is in these subacute cases that a rich exudate of cells, chiefly lymphocytic, plasma, and endothelial cells, is found in the meninges as well as an extensive infiltration of round cells in and about the walls of the vessels. Periarteritis and endarteritis together with phlebitis gradually impinge on the circulation of the smaller vessels with consequent interference with the nourishment of the cord. We have already mentioned the invariable alterations in the myelon, softening and disintegration of neural elements at the site of the lesion, and ascending and descending degeneration of tracts with chromatolysis of ganglion cells in the grey matter above and below the lesion, in large measure secondary to interference with the blood supply. Collins in 1912 described three cases of acute myelitis with evidence of softening from thrombotic vessels in one case and agreed with an earlier observation of Bastian that "acute myelitis is caused not by an inflammation but by thrombosis of some of the vessels of the spinal cord". Since then, Nonne, Williamson, Schultze, Cadwalader, and Buzzard and Greenfield have voiced the same opinion., Chung demonstrated two cases with vascular lesions, the one involving especially the arteries and the other only the veins, producing identical changes in the cord in each case. In case one, at the site of the lesion at Th.2., the degeneration of the white matter was most marked peripherally; the meningeal thickening was slight except on the dorsal

aspect where there was moderate proliferation of the connective tissue elements together with some infiltration of round,plasma,and endothelial cells. In this part of the cord there was thrombosis of the arteries,in various stages of organisation from complete block to canalisation. The group of vessels, particularly the arteries just dorsal to the posterior septum was chiefly and almost solely affected. Elsewhere the vessels appeared normal. There was some hypertrophy of intimal coats with round cell infiltration; the inflammatory exudate was slight. Above the lesion with the exception of the secondary ascending degeneration of the tracts and some chromatolysis of the anterior and podterior horn cells,the cord meninges and blood vessels shewed little evidence of pathologic change. Below the lesion down to the sacral portions the fibre tracts shewed secondary degeneration. The pathology therefore included thrombosis of the posterior spinal meningeal arteries with softening and secondary degenerative changes in the cord and evidence of an acute inflammatory exudate in the meninges at and slightly below the level of the lesion especially in the posterior and dorso-lateral aspects. In the second case,microscopic examination at the level of Th.3. revealed extensive disintegration of neural elements with large areas of softening, consisting chiefly of gitter cells involving the the periphery as well as the centre of the cord. The areas appeared wedge-shaped at the periphery and more or less oval at the centre. The ganglion cells in the grey matter were reducad in number and those

remaining shewed chromatolysis and slight neuronophagia. No haemorrhages or thromboses were seen but in many of the vessels could be seen a moderate amount of infiltration with round cells. The meninges about the lesion were only slightly thickened. The pial vessels, especially the veins, on the dorsal and lateral aspects shewed thrombi and round cell infiltration in the intima of the vessel walls. The occlusion of vessels by organised fibrin cells and pigmented particles could be seen to affect chiefly the venous supply. Above the lesion in the cervical region, there was ascending degeneration in the fibre tracts and chromatolysis of the ganglion cells in the grey matter. The blood vessels in the cord and pia did not appear to be dilated or congested, with the exception of the pial veins, particularly the dorsal group, which shewed some small round cell infiltration in the thickened muscle walls. Below the lesion, at the lumbar and sacral level, there was degeneration of the descending tracts and ganglion cells and evidence of an inflammatory exudate in the meninges consisting of small lymphocytes, a few plasma and endothelial cells, some large mononuclear leucocytes, fibrin, and dilated congested blood vessels. We have here a pari- and endo-phlebitis with thrombosis of pial vessels on the dorsal and lateral aspects of Th.3 segment resulting in softening of nerve tissue at that level and secondary changes above and below the lesion. Chung believes that the degenerative process in parenchymatous tissue may be looked upon as

non-inflammatory,a myelomalacia not unlike that described by Hassin in his case of typical "yellow" softening of the cord. He considers that these bridge shaped foci of softening,consisting of gitter cells and neural detritus,when found to be non-inflammatory, might be regarded as significant of some circulatory disturbances either from emboli or thrombosis of coronal vessels of the cord. He agrees with Nonne that "specific meningitis is by far most frequently located on the posterior surface of the cord". The dorso-lateral group of vessels,being smaller in calibre than the anterior median group are perhaps more vulnerable and readily occluded in syphilitic arteritis or phlebitis. It is significant that disorganisation of the venous system can produce widespread change in the cord. In 1893 Lamy reported a case of very acute progressive myelitis shewing changes only in the veins. In 1896 he again described an early case of meningo-myelitis in which only the veins were diseased. Rieder's researches prove that the veins are seldom normal;an endo- and peri-phlebitis are almost customary accompaniments,which lead to venous sclerosis.

In the region of the softened areas there is nearly an overgrowth of glial elements, sometimes only a network of glial tissue remains leaving many vacuoles which may be filled with one or more gitter cells. In Williamson's case there was haematomyelia and thromboses scattered throughout the grey substance. Necrosis not infrequently occurs, leading to the formation of syringo-myeloid cavities.

Clinically this may cause great difficulty in the differential diagnosis. In 1897 E.Schwartz of Vienna demonstrated a case of syphilitic myelo-meningitis with cavity formation in the anterior horns of the lower cervical and middle and lower dorsal regions. A perusal of the reported cases in which pathological investigations have been carried out suggests the relative frequency of syringo-myelia secondary to syphilitic myelo-meningitis and also the probability of the co-existence of the two diseases without causal relationship. But the existence of a true primary syringo-myelia of syphilitic origin has still to await demonstration.

Nonne cites many cases of BROWN-SEQUARD'S symptom complex of syphilitic origin. It may be acute as in Armstrong's case, subacute, or chronic as in the majority of cases. The pathological process is precisely similar to that already described. Typical classical forms are much rarer than the atypical, the lesion may not amount to a complete hemi-section or else it may pass over and affect more or less the contra-lateral side of the cord. If the lesion be in the cervical or dorsal region, the distribution of motor and sensory disturbances is easily understood. In the lower lumbar and sacral cord however the separate segments are so closely pressed together that a lesion involving one lateral half of the cord allows few sensory channels to pass beneath it to the opposite side, superficial anaesthesia being present on the injured and

paralysed as well as on the uninjured side. The syndromy of syphilitic disease of the lumbo-sacral and cauda-equinal regions is extremely variable and is often associated with vascular change and thrombosis more cephalad in the cord. Occasionally the hypertrophic meningitis has given rise to symptoms of sub-arachnoid block. Chung in his two cases, observed such changes and explained the inflammatory reaction in the lower portion of the cord as due to general vaso-motor and trophic disturbances and decreased resistance. A more plausible explanation is that of Peron who says a propos syphilis of the roots of the cauda equina that the lower zone facilitates the sedimentation of inflammatory elements with consequent organisation .

Recently L.Ramond discussed a case of SPASMODIC PARAPLEGIA, probably a syphilitic menongo-myelitis. He excluded all possibility of a lesion of the peripheral neurone such as an anterior polio-myelitis or polyneuritis. As in all spasmodic paraplegias there was a lesion of the central neurone, affecting the pyramidal tract in the brain or cord. The cause of the interruption of nervous conduction was manifestly either a disseminate sclerosis or a syphilitic process. The C.S.Fluid confirmed the latter diagnosis. M.Kroll reviews the subject of specific myelitis and apoplectiform paraplegias in syphilis of the spinal cord. Chung considered thrombosis of spinal vessels chiefly responsible, particularly the dorso-lateral group. Ramond suggested an intermittent

claudication of the cord. Margulis on the contrary thought the paraplegia was due to destruction of the anterior spinal arteries which provide in part the blood supply of the lateral columns. As a result of careful anatomico-pathological research he found in certain sections evidence of miliary aneurysms. The dura was thickened with the small vessels and capillaries markedly dilated and filled with blood. Kroll was of opinion that an apoplectiform paraplegia could occur only with changes in the meninges. The dilatation of blood vessels with general stasis inclines to thrombosis. He recalls the possibility of vaso-motor and trophic disturbance and suggests the likelihood of angio-spasm in stasis,and vaso--paralysis which might finally lead to thrombosis. (Spielmeyer). Ricker drew attention to the importance of destruction of vessel innervation. In the light of Schwartz' experimental work,he conceived a functional destruction of the circulatory system,to explain apoplectiform paraplegias. We have not come across a case of spinal apoplexy of syphilitic origin but a few have been reported in the literature. Much awaits to be done in this subject but the conception of functional "Kreislaufstoerung" in Ricker and Spielmeyer's sense is extremely suggestive and worthy of further examination.

ACUTE and CHRONIC ANTERIOR POLIO)MYELITIS.

Before discussing syphilitic polio-myelitis we shall digress and note briefly certain features of the cells and cell-groups of the spinal cord. It is of great clinical importance to distinguish between "tract"cells and "root"cells. These tract cells are scattered over the dorsal horn and a large part of the ventral horn. They are mostly devoid of definite grouping,but some are united into cell complexes,the most important of which is the column of Clarke,at the base of the dorsal horn. From these cells arise the centripetal fibres of the second order,the nexus between the posterior root system and the higher centres,and comprising the dorsal and ventral spino-cerebellar tracts and the spino-thalamic tract,and the so-called inter-segmental ~~fibres. The root cells lie wholly~~ or associating tracts, which serve to connect the grey matter of successive segmentsof the cord. These "proprio-spinal", "intrinsic",or"spino-spinal"fibres are found in the ventro-lateral and dorsal columns and are composed chiefly of ascending and descending inter-segmental fibres. The root cells lie wholly in the ventral horn and are more or less definitely grouped. In the lumbar and cervical enlargements particularly one can distinguish an antero- and a poatero-lateral, an antero- and postero-mesial,and a central group. The most important are the lateral groups from which spring the anterior root fibres. The cell groups of the ventral horn enjoy a copious blood supply.

Radial branches from the vasa-corona supply the white substance and the greater part of the dorsal horns. A special arterial trunk penetrates the ventral horn,on the right side or the left,according to the level. This sulco-commissural artery forms an intricate network which ensures adequate nourishment to the root cells. Only in the most peripheral portion of ventral horn,close to the boundary of the white matter,does the vaso-coronal system share in the nourishmant of the grey matter. The existence of two arterial territories in a transverse of the cord is of very considerable moment. As one might expect, syphilis may cause acute,sub-acute,and chronic anterior polio-myelitis. Schmaus in 1889 first shewed that the clinical picture of acute anterior polio-myelitis might be produced by syphilis. At autopsy,in the grey matter of the anterior horns of the lumbar segments,were seen round cells,large pale cells,and cells which contained fat granules. In the margin of this areathere were a number of remnants of individual medullated fibres. No ganglion cells were found. In the anterior roots many medullated sheaths shewed evidence of degeneration. Elsewhere in the cord,the changes in the smaller vessels did not appear to be inflammatory. The intima was thickened and hyaline in consistency. The vessels of the posterior column shewed most change. There was degeneration of nerve fibres in the neighbourhood of the diseased vessels. The case responded to early anti-specific treatment;failure

to obtain later improvement was due to the fact that the anterior horns had already degenerated. He concluded that there were two diseases present, a non-inflammatory degeneration of the walls of the arteries in the posterior columns and an inflammatory process in the anterior horns independent of changes in the blood vessels, their only connection being syphilis as the cause. Thomas reported a case where the large ganglion cells which should occupy the ventral horns had either completely disappeared or were represented by their shrunken and degenerated remains. He concluded that the degeneration in the grey matter was secondary and dependent upon lesions in the blood vessels which were regarded as syphilitic.

Preobraschenski reported the first case which was clinically a pure example of syphilitic acute anterior polio-myelitis. Autopsy revealed an acute interstitial inflammatory process with specific syphilitic changes in the anterior grey substance and in the spinal meninges.

Eisenlohr mentions a case presenting the picture of a sub-acute anterior polio-myelitis which improved under anti-specific treatment. It shewed typical atrophic paresis of the upper and lower extremities. The diagnosis was made on clinical grounds alone, no pathological evidence being available. Goldflam in 1893 described a similar case, the paralysis supervening fifteen months after infection and improving under treatment. In Fr. Schultze's case a progressive sub-acute atrophic paralysis followed

five months after syphilitic infection, complicated by bulbar symptoms and leading ultimately to death. No pathological confirmation was possible, syphilis being considered the most probable cause.

Nonne has seen clinically five cases, one female and four males, of progressive chronic anterior polio-myelitis. In all five only the upper extremities were affected. He refers also to a case of congenital syphilis shewing evidence of a chronic anterior polio-myelitis. In J.Hoffmann's case we see the development of an acute motor spinal paralysis in a congenital luetic. In 1911, Zadieck published two cases of luetic anterior polio-myelitis in adults. In the one the affection was chronic, the sequel to infection two years previously while the other was acute, supervening some nine years after initial infection. In none of these cases however was detailed pathological confirmation available of what clinically appeared to be typical polio-myelitis. It is highly probable that the pathological picture would be one of meningo-myelitis with vascular disease.
Merle reports a case where there was muscular atrophy of the four limbs progressing during a period of fifteen years. At post-mortem many of the cells of the anterior horns were wanting; small collections of round cells were found in addition to slight thickening of vessels. The process was considered to be syphilitic.
Vix presented a case with the clinical picture of progressive spinal muscular atrophy. Meningitis played the chief role and caused degeneration of the

anterior roots. The disappearance of the anterior horn cells was secondary. Syphilis seems to have been the cause of the meningitis. Vix refers to some of the literature on the relation of syphilis to progressive spinal muscular atrophy and concludes that the number of cases is too small for a decision as to which process is more common,a primary degeneration of the anterior horn cells or meningitis with implication of the anterior roots.

Spiller in 1909,described a case of anterior polio-myelitis,microscopical examination of which shewed the lesion to be a thrombosis affecting the anterior spinal artery and the branches issuing therefrom in the 8th cervical and 1st thoracic segments. The vessels were much thickened and some were almost entirely occluded. The softening was intense in the affected segments,as shewn by the presence of numerous fatty granular cells and minute haemorrhages. The anterior horns were softened above these regions as high as the 4th cervical segment. The lesion implicated the anterior horns,the cord area anterior to the crossed pyramidal tracts,and the extreme anterior part of the posterior columns. The pyramidal tracts were partially degenerated. The round cell infiltration of the pia, together with the proliferation of the intima of the anterior spinal artery and its branches in the lower part of the cervical swelling,indicated syphilis,as the cause of the lesions. Spiller notes the importance of the case as shewing the occurrence of

thrombosis in a very limited distribution of spinal vessels. He suggests that spinal thrombosis is not a rare condition and is probably the cause of most of the apoplectiform palsies that occur in myelitis. Then the occlusion is usually widespread,or at least implicates most of the vessels in any one transverse region of the cord. Batten in 1904 emphasised thrombosis as the possible cause of the symptoms in acute anterior polio-myelitis. It may be that this disease is caused by a primary thrombosis of the anterior spinal artery or its bramches induced by many and various forms of infection,and not necessarily a specific infection.

Margulis as recently as 1930 discussed exhaustively the clinical and pathological picture of acute syphilitic thrombotic softening of the spinal cord. He finds that the ischaemic softening in thrombosis of the anterior spinal artery extends not only into the central grey but but into the white substance of the anterior columns. He believes that the blood supply of the anterior and inner-ventral parts of the lateral columns is provided chiefly by the anterior spinal artery. There results from arterial blockage ischaemic and degenerative changes and local necrosis in the white substance of the anterior columns.As a result of theese changes there develops secondary degeneration of the long tracts of the cord. His findings,clinically and pathologically,are those of many distinguished predecessors; his record is interesting in the emphasis he puts on the source of blood supply to

the anatomical components of the spinal cord. It is evident then, that there does exist an entity which can be legitimately termed acute anterior polioMmyelitis of syphilitic origin. But the process rarely limits itself to the spinal grey matter. Even in Preobraschenski's case there was evidence of specific syphilitic change in the spinal meninges. And Raymond in 1893 made a study of a cord where he found not merely a degeneration of the anterior horn cells but also a diffuse meningo-myelitis with advanced arteritis; he thought the degeneration of the anterior horn cells was possibly secondary to vascular disease.

Leri in 1903 described two cases where there obtained a diffuse vascular meningo-myelitis. In no respect could the diseasebe considered "systemic". All the elements of the cord were involved and the findings were consistent with syphilis being the sole cause. In 1922 Lari stated that syphilis was the most frequent cause of lesions of the spinal cord that result in progressive amyotrophy. On the pathological side he re-iterated his verdict that the disease was not a systemic anterior polio-myelitis but a diffuse vascular meningo-myelitis. The essential lesion of the anterior horn cells was a pigmentary atrophy with a lymphocytic meningeal infiltration which forms especially in the region of the anterior roots minute nodules that resemble gummata. The vessels entering the cord are surrounded by lymphocytes and may shew syphilitic changes.

The cord itself may shew changes in its posterior tracts resembling tabes,or in the pyramidal tracts, resembling amyotrophic lateral sclerosis.

Winkelman as recently as 1932 discussed the subject of chronic syphilitic polio-myelitis and criticised the stress laid by French writers on the role of syphilis. He suggested that mild round cell infiltration in the meninges did not spell syphilis but was more indicative of degeneration.

Nik.A.Popow in 1929 discussing atrophic syphilitic spinal paralysis,describes the pathology as a chronic and chiefly degenerative process, leading to atrophy of the ganglion cells of the anterior horns and to degeneration of the pyramidal tracts with changes in the glia and to a less extent in the vessels. He states that the vascular change suggests a primary(toxic)affection of the parenchymatous elements of the cord. All the changes suggest a toxic origin dependent on the syphilitic virus and one can talk of a systemic disease. ("systemerkrankung"). Theories of pathogenesis will be discussed at a later stage. Suffice it to say for the moment that the existence of a primary affection of the grey substance of the cord of syphilitic origin has not been demonstrated indubitably in spite of such a possibility as for example in amyotrophic tabes mentioned by Raymond and Wilson. Finally it is questionable if a true syphilitic anterior polio-myelitis has ever been described. We feel however that the future will furnish evidence which will establish the fact of a primary toxic annihilation of neurones as wellas

of nerve fibres. We pass now to the consideration of the pathology of amyotrophic meningo-myelitis, often called spinal progressive muscular atrophy of syphilitic origin,or atrophic syphilitic spinal paralysis and with that we devote some thought to the problem of syphilis as a possible cause of systemic degeneration of the motor tract.

AMYOTROPHIC MENINGO-MYELITIS or

SPINAL PROGRESSIVE MUSCULAR ATROPHY.

The boundary of luetic affections of the nervous system is almost inexhaustible.(shier unerschoepflich.Nonne). A perusal of the literature reveals the amazing extent to which the cord can be damaged in neuro-lues. Certainly in cases of chronic syphilitic disease of the myelon there is almost invariably a degree of meningeal thickening and widespread endarteritis with round cell infiltration affecting to some extent the peripheral white matter. The ascending centripetal tracts particularly the spino-cerebellar tracts and later the spino-thalamic would be affected early as would also the anterior pyramidal,vestibulo-spinal and tecto-spinal tracts among the descending or centrifugal fibres. The lateral pyramidal and rubro-spinal tracts should from their topography be affected later. In actual fact the crossed pyramidals are more frequently involved than any lateral column tract; no doubt this may be due in some measure to the extreme ease with which we can detect such defect. In our experience a

primary sclerosis of the crossed pyramidal tracts is a far from rare manifestation of late neuro-lues. In dealing with chronic anterior polio-myelitis, progressive spinal muscular atrophy,and amyotrophic meningo-myelitis we must expect to find evidence of specific disease in the lateral columns as well as in the anterior columns. The resulting condition will be in many cases an amyotrophic lateral sclerosis. By Marchi's method changes in the crossed pyramidal tracts have been described in some of the cases,for instance those of Bielschowsky,Moleen and Spiller, Aoyama,Grunow and others. In many cases the antero-lateral ground bundle is affected.(Dubil-charcot, Struempell,Oppenheim,Moleen and Spiller,Cassirer and Maas). Spiller considers these cases as progressive muscular atrophy but as marking a transitional stage to amyotrophic lateral sclerosis. In Hammond's case the posterior columns were normal but the direct and crossed pyramidal tracts and Gower's tract were degenerated. Many would consider this case one of combined sclerosis. In Dreschfeld's case there was marked atrophy of the crossed pyramidal fibres but mostly in the cervical region. Grunow's case shewed an isolated sclerotic area in the crossed pyramidal tracts. Sainton in 1899 reported a case where the columns of Burdach and Goll shewed marked sclerosis;the anterior horn cells were degenerated together with slight sclerosis of the direct and crossed pyramidal tracts.

Raymond and Riklin in 1900 reported

several transitional forms. Other cases are on record in which degeneration has been noted in the columns of Goll and Burdach and in the direct pyramidal tract. In this connection the case reported by Vix is of unusual interest. Clinically it was a progressive spinal muscular atrophy. There was chronic fibrous thickening of the pia with round cell infiltration especially in the anterior portion of the cord and most intense in the cervical region. The pial septa and larger vessels of the cord shewed round cell infiltration. This chronic meningitis had caught the anterior spinal,roots causing atrophy of the anterior horn cells. There was degeneration of white matter in the posterior columns and along the entire periphery. Some degeneration was noted in one of the crossed pyramidal tracts in the cervical region. The case was one of syphilitic meningo-myelitis with amyotrophy. He quotes a case of Oppenheimer in which the clinical and pathological pictures were similar to those in his case.
In 1912 S.Leopold reported a case of progressive muscular atrophy probably of syphilitic origin. Distinct degeneration and sclerosis were noted in the lateral columns at all levels of the cord, more especially marked in the lumbar and cervical regions. The sclerosis was systemic in character. The anterior roots shewed a similar sclerosis. Nissl's stain shewed intense involvement of the ganglion cells of the anterior horns with marked reduction in number. He noted very slight thickening of the meninges and some mild sclerosis in the vessel

walls. The blood vessels were congested particularly at the cervical level. There was moderate round cell infiltration round some vessels and some peri-vascular oedema with glial proliferation in the crossed pyramidals areas. Raymond in 1893 pointed out the relation of syphilis and amyotrophic lateral sclerosis and recently Hoffmann,Leri,and Dana have emphasised this.

A recent valuable study of amyotrophic meningo-myelitis by Martin appeared in 1925. The following were the pathological findings in his case; a chronic lepto-meningitis throughout the whole length of the cord and medulla;a severe ependymitis; an intense glial reaction towards both surfaces of brain stem and marked degeneration of white matter round the margin of the cord. There was a less degree of degeneration of the more central white matter,the columns of Burdach being the least affected;this tract degeneration was most advanced in the middle of the cervical enlargement. The degeneration of anterior horn cells and cells of Clarkes column was almost absolute in the cervical enlargement,much less but still very definite throughout the remainder of the cord. The type of cell degeneration in the cervical enlargement appeared to be different from the type which predominated in the lower portions. He noted a moderate degree of arteritis chiefly affecting the intimal and adventitial coats of the vessels; the smaller vessels were increased in number. Throughout the cord there was a moderate amount of

peri-vascular infiltration with lymphocytes. The whole process was plainly syphilitic. The meningitis and arteritis were characteristic. The type of cellular degeneration found in the cervical enlargement has been described and illustrated by Lapinsky and others in tabes and by Berger in general paralysis. He states that the meningitis with marginal degeneration of the cord, and the cellular destruction in the grey matter and with diffuse tract degeneration are never completely dissociated but they do show some degree of variation.

Raymond and Cestan under the title "meningo-myelite marginale progressive" described cases in which the cellular destruction took a minor place and the marginal degeneration was the chief pathological change. It is noteworthy that in nearly all cases the site of maximum cellular destruction has been in the cervical enlargement. Somewhat odd is the fact that the lumbar enlargement from Th.9. to Th.12. is apparently unaffected. There is frequently glial overgrowth within the degenerated cord; this may break down and lead to the formation of syringo-myeloid cavities; such necrosis occurred in the case of Alquier and Touchard, small cavities being present in the upper cervical cord and medulla. With a pronounced endo- and peri-arteritis it is certain we are dealing with an inflammatory condition, a typical diffuse meningo-myelitis. There is no question here of a primary degeneration of the ventral horn cells, the changes being most definitely the result of the diffuse meningo-myelitis. The destruction of the nerve elements would seem to be secondary to vascular disease and not due to any

direct action of the syphilitic toxin. It may be noticed in passing that softening is most frequent in the dorsal region on account of the greater poverty of vascular anastomosis.

TT.Falkiewicz in 1924 discuss the subject of amyotrophic spinal lues. In one case examination at the level of the 5th lumbar segmentshewed a fibrosed pia with lymphocytic infltration. The pial vessels shewed no evidence of endothelial change but there was thickening of media and adventitia,both arteries and veins being affected. The anterior horn cells were fairly well preserved,the medial and lateral groups being a little poor in cells. The large-celled medial anterior group was preserved;the small-celled medial posterior group was relatively poor-celled. Dorsally there was an excessive hyperaemia with dilated vessels filled with blood and an increase of lymphoidal cellular elements in the meninges. He noted signs of inflammation in Clarkes column and in the poaterior columns. Laterally,towards the periphery,there was some sxlerosis suggesting more a peripheral degeneration. In the cervical swelling there were small haemorrhages with some sclerosis of the posterior columns towards the periphery. There were considerable inflammatory changes in the dorsal region,typical of a chronic productive meningitis,shewing marked degeneration of the anterior root fibres. The changes in the ganglion cells were not quite those of an amyotrophic lateral sclerosis but of a progressive atrophy. In case two,

at the level of the lumbar region,the meninges were a trifle fibrosed with thickening of vessel walls. There was marked lipodystrophy and atrophy of the anterior horn ganglion cells with corpora amylacea in the roots. In the mid-dorsal region,the pia was fibrosed with lymphocytic infiltration of the meninges. The cervical region shewed thickening of meninges,marked changes in the vessels with sclerosis of the walls. The motor region of the brain shewed some meningitis with mild diffuse encephalitis. Falkiewicz agrees with Margulis' observations that the disintegration of the anterior horn cells is the consequence of meningeal involvement, with lipoidosis the sequel to vascular change. Case two is in many respects similar to that of Martin's. Case one,on the contrary,suggests the involvement to a greater or less degree of all three columns. N.A.Popow in 1929 discussed the pathological changes in a case of chronic amyotrophic spinal lues in a female aged 57. There were changes in the motor cells of the anterior horns and to a lesser extent in the cells of Clarkes' columns. He found also degeneration of the pyramidal tracts. He concludes there is a distinct form of amyotrophic spinal syphilis,(metasyphilis) the pathological picture of which is a chronic primary chiefly degenerative process in the cells of the anterior horns of the grey substance of the cord with extension to the pyramidal tracts. He agrees with Margulis that this case is one in the uninterrupted chain of symptom--complexes of late neuro-lues,with tabes on the one hand,and on the other,Erb's spastic syphilitic paralysis with internediate forms of combined tabes and combined lateral sclerosis.

Spiller in 1912 expressed the opinion that on a priori grounds there could be no objection in accepting syphilis as a possible cause of the various diseases of the motor system. It was agreed that in para-syphilis there could be a degeneration of the afferent cord fibres then why should there not be a degeneration of the central motor tracts and of the ventral horn cells or a combination of this type with degeneration of the posterior root fibres? Prior to this period, many had looked upon these diseases as abiotrophies, following the suggestion of Gowers. Marie and Leri have considered syphilis in an aetiological relationship to progressive spinal muscular atrophy. Aran, MacDonald, Thonvenet, and Gharcot have found syphilis in the history of amyotrophic patients. Hammond, Niepce, Fournier, and Misserbi have observed the relation of cause and effect between the two disorders. Spiller cites a number of cases of disease of the motor tract, and attempts to determine in what proportion a lymphocytic infiltration in slight or moderate degree could be found in the pia. Such a finding is not proof of syphilis but is at least the most common picture. In Dercum and Spiller's case the clinical and pathological findings were those of amyotrophic lateral sclerosis. There was degeneration of the cells of the anterior horns of the cervical and lumbar regions; degeneration of the crossed and direct pyramidal tracts in the former extending beyond the area of the tracts and as high as the pons, with some slight sclerosis of the

posterior columns in the lower cervical and upper thoracic regions. They noted very slight round cell infiltration of anterior septal pia. In 1908, Spiller described a case of amyotrophic lateral sclerosis with degeneration extending to the motor cortex. The nerve cells of the anterior horns were degenerated especially in the cervical region. The degeneration of motor tracts extended from the cerebral cortex to the lumbar region. In another case the findings were those of degeneration of the pyramidal tracts and of the nerve cells of the anterior horns and medulla oblongata with only very slight round cell infiltration of spinal pia of the anterior septum in the lumbar region. Mills and Spiller reported a case of progressively developing paralysis at first of the hemiplegic type, from from primary degeneration of the pyramidal tracts. There was intense and long-standing degeneration of the right crossed and left direct pyramidal tracts extending into the pons with comparatively recent degeneration of the left crossed and of right direct pyramidal tracts traced by Marchi's method into the lower part of the right internal capsule. They considered the case to be one of primary degeneration of the motor tracts. Cellular infiltration of lymphocytic character of moderate intensity was found in the pia about the cerebral peduncles and pons. Some of the vessels were thickened. Spiller mentions three other cases, one of amyotrophic lateral sclerosis and the other two of primary degeneration of pyramidal tracts.

It is to be noted that in no case was there appreciable thickening of vessel walls. Spiller considered these cases as primary degenerations, and held the syphilitic toxin responsible for the destruction of motor tracts and for the marked lymphocytic infiltration of the pia. He thought it improbable that the degeneration of the pyramidal tracts would be caused by the lymphocytic infiltration.

The question of COMBINED COLUMN DISEASE is one of cardinal moment and of relatively common frequency. In Dana's two cases there was the combination of posterior and lateral column symptoms. He supposed that these were due to specific disease of the blood vessels of the posterior and lateral columns of the cord. Renner published a case which shewed some posterior column degeneration in the cervical region with pyramidal tract degeneration extending throughout the rest of the cord. Very many varieties of clinical and pathological pictures are described. Nonne mentions a case suggestive of multiple sclerosis or combined column disease of syphilitic origin. It improved markedly under anti-syphilitic treatment. Yet another combination is that of lateral column with a rudimentary form of tabes.

We must mention the subject of AMYOTROPHY in TABES, on which a vast amount of diligent research has been done. Raymond, Dejerine, Lapinsky, Preobraschenski, and many others have discussed the topic. It is outside the scope of this paper to deal with it clinically and we propose to mention the

main pathological features in order to complete the survey. S.A.K.Wilson in 1911 studied the Aran-Duchenne type of muscular atrophy occurring with tabes. He believed that among the types of tabetic macular atrophy was one which by its progress,nature,and functional distribution was definitely of central origin and analogous to the Aran-Duchenne type. It seems that while some cases especially those in which true tabetic symptoms are not prominent,are occasioned by a syphilitic meningitis,there are others where the amotrophy is the result of a chronic process affecting the anterior horn cells more or less directly, that is,the accompanying vascular,meningeal,or peripheral changes are not sufficient to have produced it. In such cases it would seem justifiable to conclude that the syphilitic toxin has been the cause,mare particularly since the lesions are widespread,diffuse and irregular. Spiller applies this opinion to the expression of the view that if this be true of Aran-Duchenne atrophy occurring with tabes we must assume it is true of this form of atrophy occurring with spinal syphilis,and if the atrophy be due to a chronic process affecting the anterior horn cells,the evidence of spinal syphilis may be very slight. We have already alluded to the subject of a primary chronic neurotropic process affecting the ventral horn cells and expressed an opinion of doubt as to its existence. Short of final proof however we are justified in assuming the existence of such a process,since without it we fail altogether of an explanation of a fairly common pathological picture.

Spinal Syphilis and Multiple Sclerosis.

Spinal Gummata.

Various writers have described the association of SPINAL SYPHILIS with MULTIPLE SCLEROSIS and have ventured to suggest the possibility of a syphilitic disseminate sclerosis. Schuster mentioned such a case in 1885. The symptoms improved ubder anti-syphilitic therapy. The case was a doubtful oneof multiple sclerosis and no pathological evidence was forthcoming to prove or disprove the contention of syphilitic aetiology. Wohlwill in 1912 examined the problem of their inter-relationship and concluded that in spite of their clinical and anatomical similarity, there was no aetiological nexus, and that one could have a chronic multiple sclerosis with a chronic syphilitic infection sui generis. The existence of a disseminate of syphilitic origin still awaits proof.

The occurrence of GUMMATA in the cord must always be considered in differential diagnosis, in the Brown-Sequard syndromy, in compression of the cord or in circumscribed sub-acute anterior poliomyelitis. Cases have been described by Osler, Orlowsky, Gowers, Rosenthal, and Williamson. The pathology of formations is identical with tertiary lesions elsewhere in the body. Localised gummatous disease may be either diffuse or circumscribed in small or large tumours. In Pick's case there were extensive miliary gummata on the inner surface of the spinal dura and arachnoid, illustrative of the miliary form of gummatous disease. There may be isolated syphilitic disease of the lepto-meninges extending into the septa evidenced by small-celles infiltration with or without gummata.

Circumscribed serous meningitis.

In the neighbourhood of gummata, there may exist softening of the cord either through arterial disease or perhaps also through the toxic action of the specific noxa on the nerve cells. Rumpf differentiates between diffuse infiltrating gummata and circumscribed gummatous tumours; usually there is a localised infiltration of the meninges and their lymph spaces with the newly formed gummatous tissue, with occasionally definite tumour formation. Oppenheim considered these circumscribed gummata in the spinal cord substance secondary to meningitis. Boettiger on the contrary thought the gummatous tumours were primary because they appeared to be of older standing than the meningitis. In passing we draw brief attention to the condition of CIRCUMSCRIBED SEROUS MENINGITIS occurring most frequently in the cervical region. Lepto-meningitis may be exudative or indurative; if the meningitic exudate be sufficient, the sequel may be a circumscribed serous meningitis giving all the signs of a compression myelitis, anf through compression a general transverse ischaemia. The clinical picture is one of paraplegia with vesical disturbance and most usually symptoms of posterior root involvement. There may be evidence of sympathetic affection if C.8. and Th.1. be irritated in a syphilitic radiculitis, a syndrome often overlooked or attributed to some other aetiological agent.

The researches of Charcot, Ross, Rumpf, Leyden, and others, suggested that the most frequent form of spinal syphilis was an incomplete transverse dorsal myelitis. In 1892 Erb drew special attention to the symptom picture of "SYPHILITISCHEN SPINAL-PARABYSE" which he characterised as follows: "mehere Jahre nach einer syphilitischen Infektion langsame und allmaehliche Entwicklung von spastischen Paresen der unteren Extremitaeten, spastischer Gang, eigentliche motorische Laehmung gering, Muskelspannungen gering, Steigerung der Sehnenreflexe, Sensibilitaets-stoerungen gering oder fehlend, leichte oder staerkere Blasenschwaeche, die Intelligenz, die Hirnnreven, die Pupillen, die Psyche, ebenso die oberen Extremitaeten normal". Oppenheim and Leyden-Goldscheider expressed the view that Erb's symptom complex was only a stage of the well-known meningo-myelitis syphilitica. The case published in 1909 by Collins and Taylor with details of post-mortem investigation, shewed it to be a subacute incomplete dorsal myelitis. In 1893 Marie in his text-book took the view that Erb's syphilitic spinal paralysis was the expression of a transverse myelitis. Trachtenberg in 1894 considered it a post-syphilitic toxic systemic disease analogous to the effects induced by Ergotin, Pellagra, and Lathyra poisoning. Muchin also viewed it as a post-syphilitic manifestation. Nonne after a lengthy citation and review of authorities expresses the following conclusions. There are cases which present the symptom somplex of Erb. The symptom complex is not a syndrome but a manifestation of the disease itself. Anatomically it

is certain that this disease is either a primary combined column disease of the posterior and lateral columns without specific syphilitic disease of the spinal cord,or a chronic myelitis involving the columns like an intra-funicular myelitis. It seems unnecessary to give Erb's paralysis the clinical status he intended it to have. With very slight variations in the symptom-complex the differential diagnosis might be a matter of extreme difficulty,multiple sclerosis,extra-meddullary tumour,and sub-acute combined degeneration of the cord among others would require consideration.

We note in passing the possibility of a "PARALYSIE AIGUE ASCENDANTE" of syphlitic origin. Zambaco,Leon Gros,and Lancereaux described a syphilitic form of Landry's paralysis. Landry himself considered such a form. Fischer,Jaffe, and Nonne among others have reported cases. This acute ascending spinal paralysis can begin late but more usually it appears early in the course of the second year after infection. It affects first the lower limbs,then the upper,and finally the trunk muscles. The paralysis appears to spread from segment to segment and may be descending in type,commencing in the face,then upper and lower extremities. The chief changes are in the cells of the anterior horns and Clarkes columns,in the nature of chromatolysis and eccentricity of nucleus. There are no mental,sphincter,or sensibility disturbances. G.L.Lambridght in 1925 reported a case of acute syphilitic myelitis with fatal ascending paralysis. It is in no sense a Landry's paralysis butinteresting

on account of the unusual termination. The posterior and lateral columns were affected with paralysis of sphincters. Autopsy revealed numerous areas of softening with creamy exudate throughout the cord, more marked in the dorsal and lumbar regions. Microscopic examination shewed acute degeneration of the anterior and lateral tracts with considerable swelling of the axis cylinders.(Spasticity of limbs gave way to flaccidity and lost reflexes as cord inflammation progressed). There were no heamorrhages but considerable peri-vascular round cell infiltration.

We have already made reference to the variety of syphilitic cord lesions. we close this brief pathological survey with the SYNDROME of GUILLAN-THAON,a review of five cases of which was recently published by Nayrac and Lassure. Guillan and Thaon described it as "une syndrome de syphilis diffuse du névraxe associant tabes,paralyse générale et myélite syphilitique". They cited ten cases. Nayrac and Lassure found the classical meningo-encephalitic lesions of general paralysis;opalescent meninges.degeneration of myelinated fibres of the cord, new vascular formation,plasmo-lymphocytosis,and alterations in the pyramidal cells. In short we find the lesions of diffuse specific myelitis. In case one,there was degeneration of posterior columns and discolouration of dorsal cerebellar tracts and the cord appeared demyelinated. In cases two and five,section shewed degeneration

of the posterior half of the white medullary substance with the lesions mostly in the tracts of Goll. Case three shewed more or less normal posterior tracts with demyelination of the direct and crossed pyramidals. In the four cases without exception there was demyelination of the posterior columns and disappearance of the fibres which radiate from the cornu-radicularis in Clarkes column and tractus intermedio-lateralis. Lesions of the pia mater were most intense, there was evidence of hypertrophic meningitis with a tendency to sclerosis in addition to generalised congestionand plasmo-lymphocytic infiltration. The meningitis predominated at the edge of the posterior columns but existed all round the cord as far as the anterior median fissure. It was their opinion that the meningitis was in large measure responsible for the cord degeneration.

PATHOGENESIS.

We have already alluded to the subject of pathogenesis, but we now propose to discuss it in slightly greater detail. There are three main theories in regard to pathogenesis;

1. That the condition consists of an infection of the lepto-meninges with syphilitic organisms; as a result the matted arachnoid and pia become adherent to the cord and the toxins from the meninges probably invade the peripheral margin of the cord and destroy it fibre by fibre, the theory of destruction per continuitatem.
2. The theory that the syphilitic toxin passes through the lymph channels.
3. The theory that destruction is associated with vascular disease.

As Martin says several circumstances point to the destructive influence reaching the nervous tissues mainly by diffusion and infiltration from the surface of the cord and brain stem. Wasting in the majority of cases appears first in the intrinsics of the hands, those muscles which are represented in the outermost parts of the cervical anterior horns, and next attacks the extensors of the wrist and fingers which have the next outermost representation, (Winkler). It might be surmised that the first stage of the infection process is an infection of the lepto-meninges with syphilitic organisms. In consequence of the resulting inflammation the matted arachnoid and pia become adherent to the cord and the physiological moat having thus been bridged,

toxins from the meninges probably invade the outlying margin of the cord and destroy it fibre by fibre.(Martin). It is a well known fact that the W.R.in the C.S.Fluid in many cases becomes quickly negative upon vigorous anti-syphilitic treatment and from this fact alone some neurologists think it probable that the site of the infection is in the meninges and not in the nervous parenchyma. Further,the meninges may be attacked by syphilis at any of its stages. Read describes a case of acute syphilitic meningitis fifteen days after the appearance of the chancre and there are other cases described in the literature. The majority of cases occur during the cutaneous stage of the disease. Wile and Stokes consider that probably in every case of syphilis attaining the second stage a more or less profound involvement of the central nervous system takes place. As far as we are aware no worker has reported spirochaetes in the earliest lesions of central nervous system disease, although this fact of a negative finding means little or nothing as Klippel and Dainsille have pointed out. It is always wise in case of doubt to have recourse to intra-testicular innoculation of the questionable fluid into rabbits in view of the success of such measures at the Pasteur Institute. These vacts seem to suggest that the site of the infection is in the meninges and that toxins may be responsible for the meningeal changes and destruction rather than the spirochaete itself. Again,the cellular destruction and the marginal degeneration shew great relative

quantitative variation from case to case,it is therefore likely that the former is brought about in a somewhat different way from that suggested for the latter. It may be that the whole cord is permeated per continuitatem by toxins from its surface,but this explanation is not adequate in which white matter between the margin and the degenerated cells has apparently not suffered, neither does it explain the relative escape of the posterior columns in all cases of progressive spinal muscular atrophy.

Another hypothesis,and one which finds considerable favour,is that the destructive influence is directly associated with the vascular disease. In many cases recorded the changes in the vessels are not of sufficient severity to kill the cells and tracts by deprivation,nor in the less affected portions could any relation of cellular damage to particular vessels be made out. If,instead, we suppose that the part of the vessels in the destructive process is the transmission of a toxic substance into the central nervous tissue from the blood,we encounter the same objections as have already been raised against total permeation of the cord from its surface. It is extremely difficult to relate the cellular damage to particular vessels;the indefiniteness of regional blood supply and the compensatory power of capillaries and venules make accurate assessment a matter of difficulty if not impossibility. S.A.K.Wilson in discussing the

Aran-Duchenne type of muscular atrophy suggests there are cases where the amyotrophy is the result of a chronic process affecting the anterior horn cells more or less directly, that is, the accompanying changes in the meninges and vessels are not sufficient to have produced it. He thinks it justifiable to conclude that the syphilitic toxin has been the cause more particularly since the lesions are widespread, diffuse and irregular. Further, Spiller has frequently found a degeneration of the pyramidal tracts of the spinal cord, apparently primary, with marked lymphocytic infiltration of the pia. He thinks it unlikely that such infiltration causes this degeneration but that both probably have a common cause, the syphilitic poison. If the motor fibres are susceptible to the syphilitic poison, it is natural to, infer that the motor cells likewise are susceptible and not unreasonable to suppose that cases may exist in which the syphilitic poison exerts its influence chiefly on the motor system and produces little lymphocytic infiltration[1]. Wilson's and Spiller's ideas, while reasonable, lack microscopic and physiological proof. Struempell gave it as his opinion that the spirochaete produced a chemical poison, affecting the nerve tissue primarily, such tissue being replaced by neuroglia, followed by changes in the blood vessels. Martin mentions that the W.R. in the blood is negative in one third of all cases and concludes that while therefore vascular disease is usually present and must exert an adverse influence on the well-being of the nervous elements, it cannot be held wholly accountable for their death.

Finally it is possible that the syphilitic toxin, to reach the central parts of the cord, makes use of the lymph channels. This is by far the most satisfying hypothesis, since it explains the sparing of the posterior columns in syphilitic amyotrophies. They are known to have a lymph system separate from that of the remainder of the cord; they may therefore escape when the rest of the cord is affected from the lymphatic stream, or conversely they may be affected alone. In possibly the great majority of cases of cerebro-spinal syphilis this explanation may fit the facts, but there still remain many clinical and pathological pictures for the elucidation of which this theory is scarcely adequate. It must be assumed that the cell bodies in the gery matter are more easily overcome than the tracts of the white matter. This is to be expected from their lack of a myelin covering and from the analogy of diseases such as acute anterior polio-myelitis. ~~or amyotrophy.~~ This explains reasonably a syphilitic anterior polio-myelitis or amyotrophy, but still fails to account for the frequency of a meningo-myelitis affecting the lateral columns and producing clinically a specific lateral sclerosis. Other suggestions advanced to account for the cellular degeneration in amyotrophies are that it may be due to disease of the peripheral nerves or anterior roots or of the isolation of the cells by the interruption of the reflex and pyramidal paths. As Martin says, with so much other destruction

within the cord it seems unnecessary to,look outside it for the source of the cellular change,and in any case the type of cell degeneration is not that which results from a reaction a distance. Without doubt,there is some truth in all three theories; many other factors, of which at present we know little,must determine the preponderance or combination of causes producing the symptom pictures.

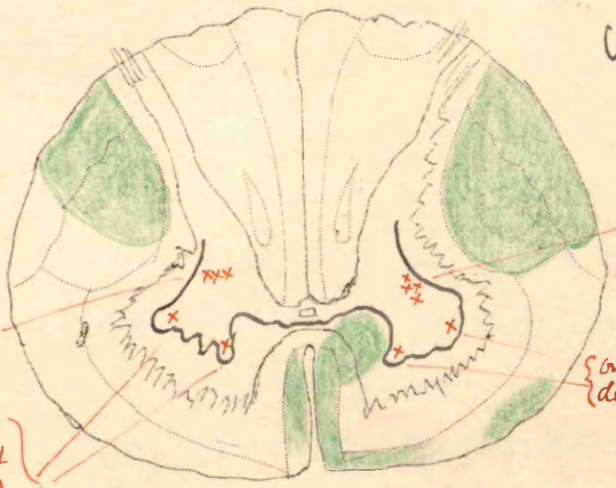
Upper part of Cervical Enlargement.

Dorn Aaron.

membranes thickened and adherent to surface of cord with thickened vessels. White matter very spongy and open all round surface beneath them.

Demyelination shown in green.

Upper part of Cervical Enlargement.



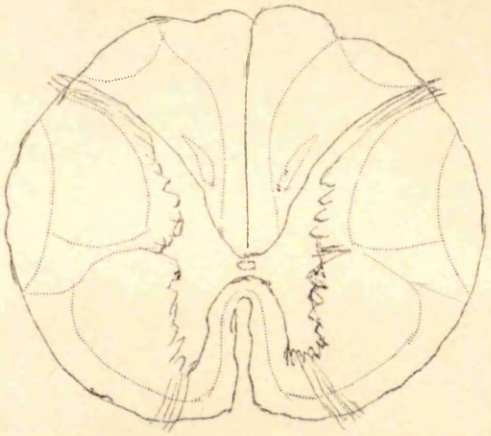
Cells numerous and fairly well preserved.

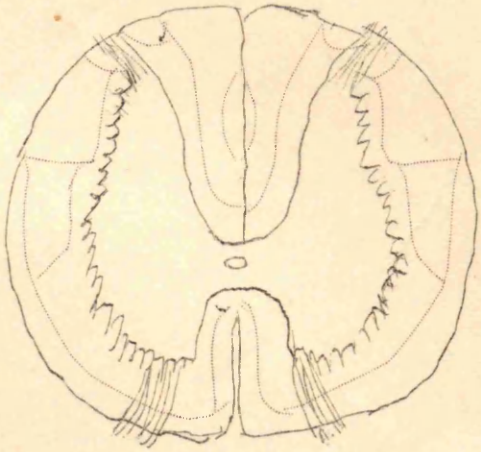
Anterior horns much shrunken.

Only occasional shrunken and degenerated Nerve cell surviving.

Cells fewer and rather more degenerated than on opposite side

Cells scanty and degenerated: small vessels thickened and with small celled infiltration round them and some small haemorrhages round them.



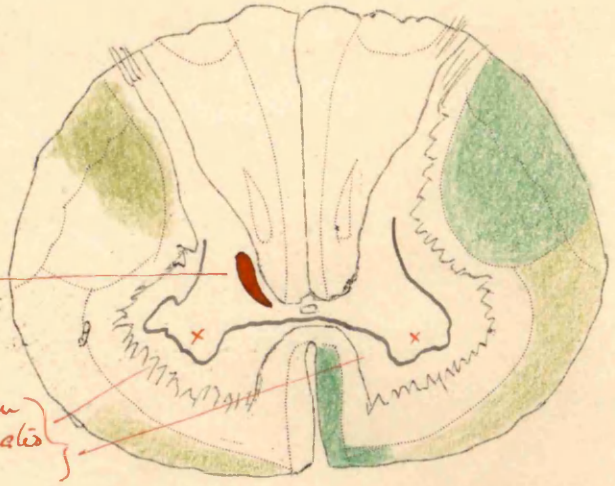


PATHOLOGICAL REPORT OF AARON DONN.

A case of amyotrophic lateral sclerosis of syphilitic origin.

Macroscopically, the spinal cord was somewhat soft throughout, but there was a distinct and much more marked softening of the cervical enlargement, suggestive of the acute transverse softening of a "myelitis". Microscopically, sections of the upper and lower parts of the cervical enlargement shew thickening, matting, and adhesion of the surface membranes to the cord, the peripheral margin of which immediately beneath them shews a spongy and open appearance of the white matter. There was well marked demyelination of the direct and crossed pyramidal tracts, distinctly more evident on the right side (see diagram, areas of demyelination in green, in degrees as shewn by colour). This change is mostly of old standing, but there is a moderate degree of staining of some of the myelin sheaths with osmic acid in nerve fibres scattered throughout these affected areas, again most marked on the right side. The anterior horns are both much shrunken and the cells of the anterior median and antero-lateral groups are greatly diminished in numbers, while those surviving are shrunken and degenerated, the cells of the postero-lateral group being also affected but to a distinctly less degree (see diagram). The vessels of the grey matter are thickened and shew small round celled peri-vascular infiltration with here and there minute haemorrhages extending from them, the largest

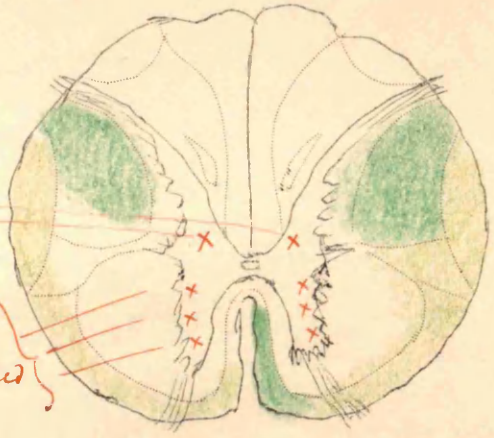
Dorn Aaron.



Lower part of cervical enlargement.

Semi-recent haemorrhage

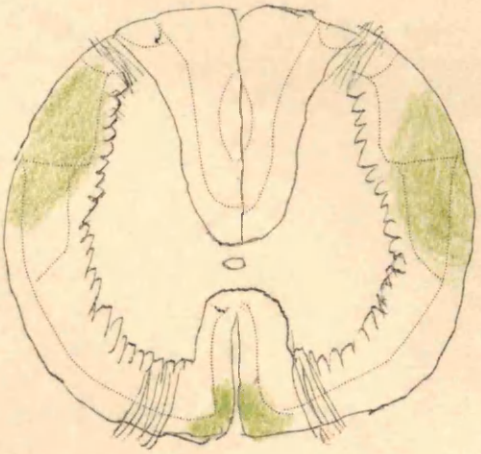
Anterior horns much shrunken with very few and much degenerated nerve cells.



Mid-Dorsal.

Cells fairly numerous but most showing degenerative changes

Cells moderately diminished in numbers and most of them showing moderate to fairly-marked degeneration on both sides.



Lumbar Enlargement.

Anterior horns not so shrunken as higher up: cells in not very well-defined groups, but mostly rather small and many degenerated. A more patchy change than in cervical and dorsal regions.

Areas of demyelination in green - affecting especially crossed and direct pyramidal tracts in degrees as shown by colour.

of which was found om the left side in the lower part of the cervical enlargement(see diagram). In the dorsal region the cells of Clarkes column are fairly numerous but mostly shew degenerative changes, whilst those of the intermedio-lateral,poatero-medial, and antero-medial groups were moderately diminished in numbers,with the majority of the cells shewing a moderate but fairly wellmarked degree of degeneration on both sides. In the lumbar region, the sclerosis of the crossed pyramidal tracts is still fairly well marked,especially on the right, with moderate change in the anterior parts of the direct pyramidal tracts. The anterior horns in this region are much less shrunken than higher up in the cord,and the cells though somewhat small in size,shew a patchy degeneration,some of the surviving cells staining rather better than elsewhere, but many also being distinctly degenerated. Transverse and longitudinal sections of representative muscles from the arm shew considerable degeneration in all stages up to complete disappearance,the fibrils still surviving being mostly much diminished in size,but a few are swollen and coalescing with one another,though in a very few they are still discrete. Only a small proportion shew faint cross striation. The sarcolemmal cells are greatly proliferated and fibrosis of the degenerated muscles is taking place. Similar sections of muscles of the leg shew much earlier stages, many of the fibres being swollen,but almost all

are still shewing cross striation,with early proliferation of sarcolemmal cells,and only a slight to moderate degree of fibrosis.

Spiller cites a number of cases of amyotrophic lateral sclerosis. In one there was very slight round cell infiltration of the pia of the anterior septum in the cervical region,apart from degeneration of the anterior horn cells and pyramidal tracts,with no thickening of vessels. In another case the degeneration extended to the motor cortex from the lumbar region.

Our case belonged to the same category as did Spiller's except that the damage did not extend higher than the cervical region,so frequently the site of greatest destruction. The degree of lymphoidal infiltration would suggest moderate duration.

CLINICAL SECTION.

AFFECTION OF ROOTS. Having recently had a a case of a syphilitic neuritis of the right peroneal nerve we had hoped to develop the subject in this paper but the vastness of the literature forced us to curtail our attention to the spinal cord and its coverings. Nevertheless it is important that we should refer to the topic of multiple peripheral neuritis, in so far as its clinical picture may simulate an acute anterior polio-myelitis. Syphilis as a cause of polyneuritis is usually included in the cachectic group but it occasionally undoubtedly acts as an acute infective toxaemia. W.Harris deals with the casebof a man aet.50 who three months after acquiring syphilis developed some ataxia and numbness of feet. He recovered but within the year, the symptoms returned with rapid onset of weakness of legs and arms, but again he almost completely recovered under injections of salicylate of mercury. A few months later, weakness and wasting of feet and hands began, which steadily progressed to complete paralysis of all muscles below the knees, with bilateral finger-drop and wasting of forearm muscles. All deep reflexes were lost, but without anaesthesia. Gradually the power returned in the gastrocnemii and hands. The knee jerks returned but there was no trace of power in any of the anterior tibial muscles nor any reaction in them to either form of electrical current. Harris suggests that the correct interpretation would probably be a polyneuritis associated with considerable anterior horn cell damage in the fifth lumbar region. In the differential it might be said that asymmetry

of paralysis does not exclude polyneuritis and that wasting of intrinsic musculature of the hands is common in polyneuritis and retention of knee jerks by no means rare. Stanley Barnes sought to distinguish toxic degeneration of the lower neurones from polyneuritis by the great atrophy of intrinsic hand muscles, the comparative slightness of sensory change, the absence as a rule of contractures, and the integrity of the psychical state. All these points are comparatively common in cases which clinically resemble and should be classed as polyneuritis. Harris regards polyneuritis and polio-myelitis as essentially the same process affecting different portions of the same neurone, and states that occasionally mixed cases are met with of permanent damage associated with polyneuritic symptoms which recover. Recently Harvier reported a case of pseudo-myopathic form of chronic syphilitic anterior polio-myelitis; he believed it was the topography and not the nature of the lesions that decides the morphology of the myopathies. Friedmann in his case of progressive muscular dystrophy believed that the lost knee jerks were either due to a lesion at the neuro-muscular junction or to posterior sclerosis of the spinal cord. Oppenheim cites a few cases where changes were found in the spinal cord but these were atypical and complicated cases.

Trabaud discusses a case illustrating the syndrome of Guillan and Barré, a polyradiculo-neuritis with dissociation albumino-cytologique. With the meningeal reaction were motor troubles with

abolition of tendon reflexes and sensory disorders. In the differential a myelopathic muscular atrophy, (type Claude and Schaeffer),an acute ascending myelitis,(type Barth and Leri),an acute anterior polio-myelitis,(type Touchard and Meaux-Saint-Marc), or finally the polyneuritic type of Landry's disease would have to be considered. Many factors which we need not relate suggest a specific direct action on the roots. In the same broad category is a case of Draganesco's,a radiculite sensitivo-motrice d'origine syphilitique. The syndrome was characterised by paresis of the superior extremities and a flaccid paraplegia with discrete muscular atrophy and sphincter trouble. The tetraplegia was accompanied by abolition of deep and superficial reflexes,muscular hypotonia,and objective sensory disturbances for touch and temperature. The C.S.Fluid shewed dissociation albumino-cytologique. The case was not one of tabes because of motor paralysis and absence of A-R pupils. A specific polyneuritis was excluded by the total absence of modification of electrical reactions. This case simulates Kahler's syndrome of multiple syphilitische Wurzelneuritis,examples of which have been described by Buttersack,Eisenlohr, Oppenheim,and Nonne. Mention must be made also of a syphilitic radiculitis of the cervical and upper dorsal cord,giving rise to a special symptom-complex known by the name of Horner,a combination of oculo-pupillary symptoms with vaso-motor secretory symptoms.

MENINGITIS.

Syphilitic meningitis of the encephalon in children of parents with luetic history is fairly common and usually overlooked. Rodrigo records five cases. The shifting clinical picture is seldom typical and may resemble that of meningism or of serous or tuberculous meningitis except that changes in character are almost constant. Convulsions are more frequent the younger the child. Leredde ascribes to meningitis about 75% of the average 1180 deaths of syphilitic children under five at Paris each year. Guido Milani mentions the case of a recently infected young woman who developed acute and severe syphilitic lepto-meningitis. She recovered promptly under specific treatment except that the strabismus persisted for a few months. Here we are concerned with the incidence of spirochaetal disease on the spinal meninges. They shew a peculiar vulnerability to the toxin and are liable to more or less gross change from a few days after initial infection till late in the tertiary stage. In Read's case an acute meningitis developed fifteen days after the chancre. The spinal coverings are usually affected together, the pia and arachnoid being matted and adherent to the dura and substance of the cord. The meningitis may be diffuse or localised in smaller or greater patches which may be single or multiple; they are more frequently multiple, circumscribed fibro-gummatous meningeal exudations of variable localisation. (Sezari). The acute form of meningeal syphilis consists of a

diffuse small-celled infiltration,as a rule localised round the small vessels. Gilbert and Lion have differentiated a form hyperémique and nécrobiotique, a form diffuse embryonnaire,a form gommeuse and diffuse scléreuse. Little advantage is gained from such elaborate sub-division,since they are all expressions of the same underlying inflammatory-infiltrative process. These processes can encircle the cord completely,or involve only the anterior or posterior columns. Most frequently the syphilitic meningitis is restricted to the posterior region. This holds for all the meningitides and in spite of the assurance of certain authors it is in no way typical of spirochaetal meningitis. One can only say of the cauda equina that its meninges are subject to a specific meningitis as was proved in Eisenlohr's case.

The general meningitic symptoms are in essence those of any other meningitis;pains in the neck,between the scapulae,in the back or over the sacro-lumbar region;paraesthesiae,painful sensations and attacks of pain,radiating in the upper or lower extremities or back,and hyperaesthesiae in the territory of pain distribution. There may later be spasticity of musculature . The superficial and deep reflexes are brisk and may be pathologically increased. Stiffness of the back and a diffuse throbbing sensation is frequently complained of. Charcot and other French authors consider the nocturnal pains,their "rachialgie nocturne",typical of the specific condition. Nonne suggests that the evidence is not in favour of this contention.

Various other changes soon appear; the spinal roots may be affected with regional hyperaesthesia and anaesthesia, vaso-motor effects particularly below the level of the lesion, irritative symptoms, and perhaps a mild dysuria. Fournier speaks of an "analgésie syphilitique secondaire", a regional symmetrical analgesia frequently seen in the secondary period. There is little doubt that meningeal symptoms should be looked for as early as the secondary stage of syphilitic infection. They may be almost trivial yet their distribution is indicative of a segmental lesion and not a fibrositis or "neuritis". The meningitis may be acute, sub-acute, or chronic. In the acute stage there is usually marked vascular change with perivascular infiltration, with overflow of symptoms into the cord, particularly a mild spasticity and slight sphincteric disability. The chronic picture shews somewhat widespread irritative symptoms, frequetly of a root character, and may if the diseased meninges take the form of a greater or smaller circumscribed gummatous envelope, suggest mild compression.
In middle life the meninges are often considerably thickened but the outline of the cord is still perfectly clear; the changes while primarily specific in origin are also in part due to a degenerative arterio-sclerosis. This curtailment of blood supply causes in turn a mild ischaemia of the posterior and lateral columns, a common happening in the later periods of life.

AMYOTROPHIC MENINGO-MYELITIS.

The existence of a syphilitic amyotrophic meningo-myelitis has long been recognised. Nonne discusses the topic fully in his text-book. Later authorities have suggested that wasting occurs in one of three situations and that the disease moves from one group of muscles to another on the basis of pathological spread from the periphery towards the centre. The pathogenesis has yet to be clarified;the anterior horn cells may be affected from the meningitic process in the form of a circular peripheral sclerosis,or as a result of a proliferating pial septal meningitis. The clinical effects would naturally be entirely different in the two cases. The muscular atrophy is "atonic" in type with loss of reflexes,is apparently of spinal origin,and is as a rule unattended by any sensory loss. Some such cases may be due to chronic saturnine poisoning, a few to other causes,but the majority are due to syphilis. Leri is the protagonist of this view and has claimed that a particular clinical type is nearly always syphilitic. In discussing this problem we will adopt Charcot's division of cases into amyotrophic lateral sclerosis and progressive muscular atrophy although in fact the combined lesion nearly always exists. Reviewing the literature we find that the average period between infection and the onset of wasting is 17 years, though it varies from 4 to 37,in Vizioli's case as high as 44 years. Nonne has recordeda few cases

in hereditary syphilitic subjects. Males are usually affected, the average age incidence being 48. Martin in 1925 dealt in very great detail with the question and classified the cases into three groups, one with wasting beginning in the hands, two in the shoulder muscles, three in the legs. Severe pain heralds the onset of disease; it may continue for several weeks and be referred to the shoulder or half of back on which later wasting will appear. Raymond and Cestan's case began with spasticity. Weakness ensues with atrophy. Pain, weakness, atrophy, affect in this order the entire musculature. The wasting is atonic, there is atrophy with loss of reflexes. It nearly always begins in one or other of above three situations, small muscles of the hand, shoulder muscles, or muscles on the outer side of the leg. Spiller mentions a case in which the neck muscles were weak. The atrophy may be noticed first in the thenar eminence, with loss of adductive power and inability to appose thumb, with possibly hollowing of interosseous spaces. The weakness is felt in the extensor group of the forearm and not in the flexor as one would expect. There is fibrillation in the extensors and loss of supinator jerk. The supinator longus muscle with its higher segmental supply, C4, C5, may escape. Martin states that the disease jumps from forearm to deltoid and views these as very characteristic, associated with the fact that the degeneration within the cord probably spreads from without inwards more than from segment to segment, and secondly with a certain "patchiness" which is apparent in the cell destruction in the spinal cord.

Both upper limbs may be affected simultaneously,or one limb may be entirely withered and its fellow intact. The wasting may begin in the shoulder muscles and from there jump to the extensors of the forearm,or the wasting may begin in the peronei. There is often some spasticity and perhaps sphincter trouble;when spasticity occurs,wasting of the lower limbs does not often seem to follow. The subject has recently been discussed in the Proceedings of the Royal Society of Medicine. Fouche mentions the case of a male,aet.37. There was diplopia,generalised wasting and atrophy of all skeletal muscles. The pupils were fixed to light and accommodation;extreme bilateral ptosis,right greater than left. The external movements were limited with no lateral movement and only slight range on looking up or down. There was weakness and wasting of all muscles of mastication,with weakness of left lower face and complete frontalis paralysis. The palate was immobile. The tongue protruded to the left and shewed a certain amount of fibrillary twitching. Sensory system normal. The deep reflexes were all absent,and the sphincters normal. The blood W.R. was strongly positive. The C.S.Fluid shewed 10 cells per cmm.,total protein 0.08%, negative Lange and W.R.. No pathological evidence was available but the findings suggest some bulbar involvement,common in the idiopathic amyotrophic lateral sclerosis but rare in the specific variety. Grainger Stewart mentions a case of syphilitic amyotrophy(?congenital lues) a male,aet.49., complaining of weakness ~~of right feet~~ with paraesthesiae

of four years duration commencing in the right foot. No ocular or sphincter trouble. Fibrillation in muscles of shoulder girdle;legs were both weak. The deep reflexes were very brisk;slight defect of position sense in both legs. The C.S.Fluid shewed moderate lymphocytosis with a paretic Lange;the W.R. was negative in the blood and C.S.Fluid. The shoulder girdle as the site of maximum defect was suggestive; the brisk reflexes and defect of position sense betoken mild affection of the lateral and posterior columns respectively. Stone in discussing amyotrophic syphilitic meningo-myelitis,atates that fibrillation is less constant than in idiopathic cases of amyotrophic lateral scleresis. The sensation was usually normal and when change occurred it was the vibration sense only. Nonne reports having observed five cases of anterior chronic progressive polio-myelitis of syphilitic origin. There were four males and one female. In all five cases the upper extremities only were affected. In the female ,the muscles of the hands, forearms,ans arms were paralysed and atrophied. One case shewed atrophic paralysis of the shoulder girdle, and in part of the upper and lower arm musculature; in another the upper extremities were uniformly affected and in yet another the musculature of the hands and extensor aspects of the forearms. In all these cases signs of amyotrophic lateral sclerosis were lacking. Dana in 1906 published a report of 130 cases of spinal atrophy,examples of chronic polio-myelitis,Aran-Duchenne progressive muscular atrophy, and amyotrophic lateral sclerosis. Ellinghaus in

1916 reported acase of sub-acute polio-myelitis in a 24 year old man, six months after initial infection. The upper extremitiesw were mainly affected, the inferior extremities shewing isolated patellar areflexia. The symptoms regressed on treatment. It is evident that a pure syphilitic progressive muscular atrophy is rare. It may be , that the origin of such lesions is seldom peripheral, as was formerly thought, but usually spinal in the anterior horns. Schaffer and Lapinsky in their researches on tabetics with flaccid myopareses believed the damage to be primary in the ventral horn cells. It may quite possibly be a direct effect of the syphilitic toxin on the ventral cells. It must be remembered that the sequel to vascular damage can be an uncomplicated picture of clinical anterior polio-myelitis caused by pathological change in some of the branches of the anterior spinal artery. If all the branches are affected then almost certainly defect is observable in the surrounding white matter, demonstrated clearly in Preobraschenski's case.

Winkelmann recently discussed chronic syphilitic polio-myelitis and extressed the opinion that syphilis played a role in the atypical rather than in the typical cases of atrophic conditions. In his case the atrophic manifestations were most marked in the muscles of the neck with failure to hold the head erect; the upper limbs were affected much less. Thomas, Harris, and Hoffmann have reported casesof acute syphilitic polio-myelitis. S.A.K.Wilson studied the A[R]an-Duchenne type of muscular atrophy

occurring with tabes and believed that among the types of tabetic muscular atrophy is one which by its progressive nature and its functional distribution is definitely of central origin and analogous to the Aran-Duchenne type. He stated that while some cases of this sort especially some in which true tabetic symptoms were not prominent,were occasioned by a syphilitic meningitis,there were others where the amyotrophy was the result of a chronic process affecting the anterior horn cells more or less directly, i.e.the accompanying vascular meningeal,or peripheral changes were not sufficient to have produced it. He thuoght it justifiable to conclude that the syphilitic toxin had been the cause more particularly since the lesions were widespread,diffusenand irregular. If this be true of the Aran-Duchenne atrophy occurring with tabes,he assumed it was true of this form of atrophy occurring with spinal syphilis,and if the atrophy be due to a chronic process affecting the anterior horn cells,the evidence of spinal syphilis may be very slight. An immense literature has grown up in connection with this subject of tabetic amyotrophy;we merely mention it in so far as the anterior horn cells are involved. Martin intentionally used the term amyotrophic meningo-myelitis as suitable for describing a spinal progressive muscular atrophy of syphilitic origin from inception to termination. He clearly felt that in most cases there was an overflow of pathological change into the lateral columns thus involving the crossed pyramidal and other tracts.

Clinically it is not always a matter of ease deciding if the lateral columns are implicated. Several observers reported the preservation or slight increase of tendon reflexes;were these cases of amyotrophic lateral sclerosis? Oppenheim does not regard exaggeration of tendon reflexes as sufficient evidence to make a case of progressive muscular atrophy one of amyotrophic lateral sclerosis. Dana ignores even a little spasticity because he limits the term amyotrophic lateral sclerosis to those cases which from the beginning and dominantly shew the spastic and contracting type of muscular atrophy. Most to-day consider chronic anterior polio-myelitis and progressive spinal muscular atrophy as synonymous terms and separate the amyotrophic lateral sclerosis. Others,(Marie,Gowers)would group all under the same heading. This difference of opinion may be attributed to the point of view, some emphasising the clinical and others the pathological features. The dual lesion of anterior horn cell and crossed pyramidal tract will now be briefly noticed.

ATROPHIC SYPHILITIC SPINAL PARALYSIS WITH PYRAMIDAL TRACT INVOLVEMENT.

The great majority of specific spinal amyotrophies shew pronounced vascular changes in the cord and meninges,manifestly definite evidence of an inflammatory condition. All the elements of a diffuse meningo-myelitis are present,the degeneration of anterior horn cells being the outstanding feature of an extensively diseased cord. Robertson recently reported a case of a female aet.31. The disease commenced four years ago,when she became aware of an intermittent sensation of tingling over the tips of the fingers of the left hand,with progressive decrease of power. The hand was blue and cold. Earlier in the illness she complained of pains over the left cervical region and a burning sensation over the spinas of the 6th and 7th cervical vertebrae. An examination shewed a left-sided cervical palsy, with slight diminution of sensation over the left side of the face. Left claw hand with marked wasting of all intrinsics of hand and flexor pronator group in forearm and slight wasting of upper arm muscles. Tendon reflexes were brisk,exaggerated on the left side. The knee jerks were equal and brisk,with slight spasticity of calf muscles. The plantar responses were extensor on both sides. C.S.Fluid shewed total protein of 0.04%,24 cells,85% lymphocytes,5%large mononuclears,10%polymorphs. W.R.in C.S.F. and blood was positive. A point of interest in this case was the exaggerated reflexes. Most cases are atonic with

areflexia and wasted limbs. Occasionally there may be difficulty in differentiating from syringo-myelia; in the latter the arm reflexes are invariably absent. Falkiewicz first case illustrates the genesis of disease with pain and loss of power in the shoulder girdle and hands,followed by atrophy;sphincter trouble in the form of retention. The tendon reflexes were present with fibrillary contractions in the musculature of the upper extremities. At autopsy there was pyramidal area sclerosis,with signs of inflammation in Clarkes and the posterior columns. Laterally towards the periphery,there was sclerosis,the syphilitic "halo". In addition there were small haemorrhages in the cervical swelling. In his second case,that of a male aet.55,there was pain over the sacrum with numbness over the left leg,spasticity of muscles but no bladder or bowel disturbance. He shewed marked atrophy of the shoulder musculature and small muscles of the hand with a low grade atrophy of the lower limb musculature. The knee and ankle jerks were exaggerated with a right extensor response. There was no ataxy or sensory disturbance. In case one,the clinical picture is the sequel to a chronic productive meningitis,affecting marked degeneration of the anterior root fibres. Popow mentions the case of a female aet.57,with painful sensations in the legs, paraesthesiae,weakness in the lower extremities with little vesical dysfunction. The ankle jerks were absent and the patellar reflexes just present. Autopsy revealed changes in the pyramidal tracts and motor cells of the

anterior horns. The paraesthetic sensations were due to meningeal involvement. Frequently the blood and C.S.Fluid may shew nothing. In his second case,a male aet.57,there was weakness of lower extremities with ultimately complete loss of power;no ataxia;coition and sphincters normal. Power in the upper limbs was good with no atrophy but complete areflexia. In the lower limbs active movements were poor;dropped feet, hypotonia but no ataxia. Areflexia in the left leg with a low grade patellar response on the right. Babinski response absent. There was atrophy with marked fibrillary twitchings;some muscle joint and vibration sense loss in the left leg. There was evidence of diffuse muscle atrophy. The noteworthy points in this case were the complete upper extremity areflexia with retention of movement and power and absence of sensory disturbance,of ataxia,or amyotrophy.
Popow sees in this fact the amyotrophic process assuming an ascending character. In the upper extremities we have a type of spinal progressive muscular atrophy beginning;in the lower limbs we see the typical appearances of a flaccid paralysis.
The cases of Merle with atrophy of all four extremities,Oppenheimer,Nonne,Leopold, and Vizioli are of this type. The case of Bochroch and Gordon in 1902 shewed an amyotrophic process affecting only the lower extremities without changes in sensation but with sphincter disturbance and absent knee jerks on the one side,and bilateral extensor responses. This case had the character of a disseminated affection and began with paralysis of the cerebral

nerves,ptosis,diplopia,etc. Margulis considers these cases as units in an uninterrupted chain of symptom-complexes of late neuro-lues,with tabes on the one hand and Erb's spastic syphilitic paraplegia on the other hand,with combined tabes and combined lateral sclerosis occupying intermediate positions. Leopold's case is important in that it represents a transitional stage between progressive muscular atrophy and amyotrophic lateral sclerosis. Clinically it shewed the picture of a progressive spinal muscular atrophy,while pathologically the lesions were those of an amyotrophic lateral sclerosis in mild form. The patient,a male,aet.50,complained of difficulty in opening the lid of his watch,with weakness in the fingers of the left hand some eighteen months later. There followed weakness and inability to use arms and hands with some weakness in the left leg. No pain;no bladder or rectal trouble. The tongue shewed fibrillary tremors. There was generalised wasting of musculature,shoulder girdle,hands and arms with marked fibrillary twitchings. Patellar reflexes if anything were diminished. Babinski absent on each side.No spasticity,some power in muscles of lower limbs. The supinators were still active. Necropsy shewed marked atrophy and loss of ganglion cells in the anterior horns with moderate distinct sclerosis of the crossed pyramidal tracts. We have dealt then with pure cases of syphilitic spinal amyotrophy,two cases of which were reported by Dejerine in which no portion of the white matter shewed any change. In nearly all cases the antero-lateral ground bundle shews a greater

or less degree of degeneration,as in Dubil-Charcot's, Striuempell's,Oppenheim's,Moleen and Spiller's, Cassirer and Maas' ets. The clinical features of such cases would be amyotrophy with evidence of destruction of so-called inter-segmental or associating fibres. In the majority of cases there is some interference with the crossed pyramidal tracts;these may represent a transitional stage to amyotrophic lateral sclerosis,or as in Hammond's case the posterior columns may be normal with evidence of degenerative change in the direct and crossed pyramidal and Gowers' tracts;many consider this case one of combined sclerosis. Grunow's case shewed an isolated sclerotic area in the crossed pyramidal tracts. Sainton's case carries the process a stage further. Atrophy commenced in the upper extremities,finally involving the legs and feet. Necropsy shewed marked sclerosis of the columns of Burdach and Goll,degenerated anterior horn cells,and slight sclerosis of direct and crossed pyramidal tracts. Raymond and Riklin in 1900 reported several transitional forms. Other cases are on record in which degenerative change was noted in the tracts of Goll and Burdach and in the direct and crossed pyramidals. Dercum and Spiller reported a case with spastic lower limbs and hyper-reflexia;weakness and rigidity of legs and bulbar speech. At autopsy the anterior horn cells of cervical and lumbar regions were degenerated as were also the crossed and direct pyramidals with some slight sclerosis of the posterior columns in the cervical and upper thoracic regions. Laterally,the degeneration extended as high as the pons.

Finally,cases are frequently met with shewing a spastic paraplegia due to primary degeneration of the pyramidal tracts of syphilitic origin. There may be evidence of overflow of pathological change into the adjacent tracts or columns but the major change is concentrated in the crossed pyramidal tracts with a resulting picture of a pure primary lateral sclerosis. Spiller mentions such a case in a male aet.42. He shewed spastic paralysis of the lower limbs without pain or atrophy but contractures,and with bilateral extensor plantar responses. Sensation for touch and pain were normal in the upper extremities. Post-mortem findings were those of primary degeneration of the pyramidal tracts with very slight round cell infiltration of spinal pia in the cervical and lumbar regions and no thickening of vessels. The nerve cells of the anterior horn were little if at all altered. It seems evident from Spiller's case that degenerative processes can extend up to the medulla affecting the bulbar nuclei,particularly the nuclei hypoglossi,pons, cerebral peduncles,and the motor cortex. It is impossible to say if the neuronal degeneration is primary in the cell elements or secondary to pial lymphocytic cellular infiltration and endarteritis, a frequent finding in the region of the pons and cerebral peduncles.

MENINGO-MYELITIS: MYELITIS.

Meningo-myelitis is the commonest form in which syphilis of the spinal cord manifests itself. The posterior and anterior roots possess their own arterial branches,localised disease of which may cause root symptoms. A diffuse gummatous change or excessive meningitic exudate may induce a compression myelitis and through compression of larger vessels create a general transverse ischaemia giving rise to softenings (formerly called myelitis) or sclerosed patches and so leading to secondary degeneration in ascending or descending directions. A meningitis with associated myelitis is commonest in the dorsal region of the cord on account of its being most slender at that point, and also on account of the greater poverty of vascular anastomosis. Premonitory symptoms are extremely common,tiredness,boot pains,twinges of pain in the arms or legs,paraesthesiae,or perhaps a feeling of weakness in the lower limbs. In proportion to the extent to which the periphery is affected so will symptoms be present. Sensation is generally affected. All sensation may go,especially in lumbar cord involvement, or,as sometimes in derangement of dorsal cord,sensation alone may be disturbed. Thermanalgesia alone may be destroyed. The Brown-Sequard complex is met with sometimes. An interesting feature is the not infrequent presence of oedema of the lower limbs,due to loss of vaso-motor control. Bed sores appear and spread rapidly. The temperature sense is often implicated; touch;localisation;pressure and pain sense may be unimpaired. Sometimes the temperature sense is preserved

with loss of the others. The pain sense alone,or pain and temperature senses can be destroyed. The tendon reflexes are mostly raised;the superficial reflexes can be increased or diminished. Frequently some disturbance of vesical function is the earliest symptom. In the chronic lumbar myelitis,vesical dysfunction may take the form of detrusor paralysis or mere sphincteric weakness in the form of incontinence; a combination of both with a picture of "ischuria paradoxa"is not unusual. Meningo-myelitis is usually chronic though iccasionally it may be more or less acute. Acute transverse myelitis unaccompanied by gross meningeal change is a slightly rarer form of spinal syphilis. It may be very acute,coming on in hours or days with almost complete and absolute paralysis. Some place the symptoms of acute syphilitic transverse myelitis in two groups,the process either developing in a few hours,or taking several days to a week or more to appear fully;the onset may be as sudden as a spinal haemorrhage,as in one of Williamson's patients. Sensibility is severely affected though variously;sensory symptoms are prominent; paraesthesiae,cutting,lancinating,boring pains in the lower extremities,in the hips. Charcot long ago laid great stress on spinal irritability,paraesthesiae, girdle sensations,shooting pains,tiredness in the limbs,and muscles twitchings. Pain in the back is a common prodromal symptom. In three of Williamson's cases,retention of urine was the first premonitory sign and its significance needs emphasis. Involvement of the sphincter with retention and later absolute loss of control is usually early. The first sign may be

incontinence,while in some patients premonitory constipation has been noted. Paralysis may come on catastrophically and absolutely in the course of a few days. The legs usually suffer and occasionally the arms. Convulsive mevements and spasms in the toes, feet,and legs with weakness may precede paralysis. Oppenheim,Rosin, and Siemerling claim that that a variable patellar reflex is an early valuable sign. The reflexes may persist and may even be hyperactive with a positive Babinski response and flexing and contracting of limbs;this is seen in some cases with involvement of dorsal cord. If the lesion be in the lumbar cord,all reflexes are lost. The differential diagnosis is sometimes a problem of great difficulty. One has to consider an acute transverse paraplegia and spinal caries with tuberculous exudate. A malign tumour of the vertebral column may press on the cord or meninges and lead to softening;a sarcoma invading the vertebral bodies gives a picture which frequently strongly simulates an acute meningo-myelitis. An intra-medullary tumour may cause an acute paraplegia as may also but more rarely an atypical disseminate sclerosis. Landry's paralysis and syringo-myelia must be excluded. Finally an extra-medullary cysticercus cyst must be considered,a case of which we have in our ward at the moment. When the onset is very acyte,one must suspect haemorrhage or haemato-myelia;some authorities speak of a"spontaneous" spinal apoplexy. An intra-medullary thrombpsis or embolus may give rise to apoplectic signs and symptoms.

Lesions of this nature are in the cord as they are common in the cerebrum. Spiller mentions an interesting case, where four years prior to the paralysis, the patient had pain between the shoulders. On admission, he complained of numbness and weakness in the upper extremities and later similar symptoms in the lower limbs. There was impaired temperature and pain sensation in the upper part of the thighs and over the trunk as high as the first or second rib and in both upper limbs. He had incontinence of bladder and rectum. The arms were much wasted and almost completely paralysed. Haemato-myelia of the cervical enlargement and first thoracic segment was diagnosed but haemorrhage was thought of instead of thrombosis. Necropsy proved the lesion to be one of anterior spinal artery occlusion affecting C.8. and Th.1. segments. A thrombosis is more likely to occur in the lumbar region owing to the blood supply of this portion of the cord being at a point more distal from the heart and to the long course of the re-inforcing arteries.

Ghung has an interesting paper on thrombosis of the spinal vessels in sudden syphilitic paraplegia. He describes a man aet.35, complaining of of paralysis of the lower limbs and retention of urine which developed suddenly a week before admission. Examination revealed the loss of all forms of sensation below Th.7., and a flaccid type of paralysis, with superficial and deep areflexia and no Babinski or clonus. Between Th.4-7, a zone of hyperaesthesia. The sphincters were spastic. The blood and C.S.F. W.R. was positive. The chief pathological change was thrombosis

of the posterior spinal and meningeal arteries with direct softenings and secondary degenerative changes in the cord. Chung states that in the acute cases or types in which the lesion was thought to be purely thrombotic, the period of flaccidity was considerably shorter (as judged by the return of reflexes and spasticity in recovered cases) than in sub-acute or chronic lesions in which it extends over a much longer period without any sign of supervening spasticity.

Chung's second case was a male aet.26, complaining of inability to walk and pass urine of sudden onset. Examination revealed a zone of hyper-aesthesia corresponding to Th.8,9, and 10 levels., but from the 11th down to sacral level, there was complete anaesthesia to all forms of sensation. Both lower limbs were paralysed with areflexia. No Babinski sign or clonus; sphincters were spastic. The C.S.F. shewed a cell count of 40 small lymphocytes;. Autopsy revealed a periphlebitis and endophlebitis with thrombosis of the pial vessels in the dorsal and lateral aspects of Th.3 segment, resulting in softening of nerve tissue at that level, and secondary changes above and below the lesion. In these cases there was little or no meningitis or myelitis; there was evidence of meningeal reaction below the lesion in the form of mild inflammatory exudate; the apoplectic paraplegia was caused by the venous changes with consequent softening. The frequency of involvement of the posterior aspect of the cord as compared with the anterior may be accounted for by the relative size of the blood vessels and their vulnerability to the

to the attack of the syphilitic virus. Chung in a detailed study of 34 cases of rapidly developing paraplegia divides them into three clinical types according to the symptoms;firstly,a form with either a complete loss or a reduction of all sensations and a complete motor paralysis,these being clear-cut transverse lesions and vary only in the degree of injury which affects the components of the cord; secondly,a form with a dissiciated sensory disturbance and paraplegia suggesting syringo-myelia but more likely to be due to some vascular catastrophe. (Spiller and Chung);thirdly,a form with no sensory involvement and only disturbance of motor function. Except for the nature and onset,these cases Chung thinks,closely resemble the class of case first described by Erb in 1892 and later by Kuh. Chung discusses the symptomatology of the acute and sub-acute groups of syphilitic paraplegia. The acute form is characterised by few or no prodromal symptoms,a complete flaccid paralysis of both lower limbs, invariable sensory disturbance,loss of sphincter control,and trophic changes. The sub-acute type shews premonitory symptoms extending over some time,up to three weeks,with either a spastic or flaccid paralysis, some sensory loss,sphincter trouble and trophic changes. The sensory manifestations varied considerably;practically in every case subjective paraesthesiae were present;the sensory findings apparently depended on the degree of stoppage and the particular areas in cross section supplied by the diseased vessels,the syphilitic virus having no

particular predilection for any group of vessels. The motor component was always involved, the paralysis being usually of the flaccid type. In such cases the complete areflexia and loss of plantar response with faecal and urinary retention corresponded to the condition of so-called "spinal Shock" the duration of which is very variable.

L. Ramond recently dealt with the the subject of SPASMODIC PARAPLEGIA. He reported the case of a female aet.53, who suffered from spasmodic paralysis of the lower extremities with hyper-reflexia, positive Babinski's, retention of urine, and some disturbance of sensibility more marked on the right. The spasmodic character of the paralysis with hyper-reflexia removed all doubt as to the site of the lesion; a temporary abrogation of function of the central motor neurone, that is, the pyramidal tract in the brain or cord. In adults a spasmodic paraplegia of cerebral origin is rare; lumbar pain and vesical dysfunction are both in favour of the medullary origin of the paraplegia. The cause of the interruption of nervous conduction could be compression or inflammation. In Ramond's case, the onset was slow, hence acute myelitis could be excluded. He mentions two disorders which might give rise to intermittent claudication of the cord, a disseminated sclerosis or medullary tumour. The age, intensity of motor-sensory change, importance of urinary symptoms, with absence of ocular change all contra-indicated a sclerosis. The blood and C.S.F. W.R. were strongly positive, the C.S.F. shewing 10 lumphocytes per cmm.,

and 0.1% of total protein. Later the superior extremities were affected which suggested the possibility of cerebral or intra-medullary tumour. The evidence pointed preponderatingly to the spasmodic paraplegia being the result of a syphilitic meningo-myelitis. Reference has already been made to Kroll's work on apoplectiform paraplegias in syphilis of the cord. He thought that changes in the meninges such as thickening might give rise to apoplectiform paraplegias. Such clinical catastrophes could readily be understood as the sequel to some functional disturbance of the circulation. Ricker stressed the importance of the destruction of the perivascular nerve elements. Spielmeyer conceived a process of angiospasm in stasis and vaso-paralysis, the sequel to which would be thrombosis. Relatively few cases of spinal apoplexy have been recorded in the literature and current views are largely the product of experimental work.

In connection with the subject of myelitis we would mention the syndrome of Guillain and Thaon, characterised by them as a "syndrome de syphilis diffuse du névraxe associant tabes, paralyse générale et myélite syphilitique". Men are more f frequently affected, according to Nayrac and Lassure in the proportion of four to one. They may shew true ataxia with Rombergism. Guillain found the reflexes exaggerated; they may be normal, diminished or abolished. The Babinski response and Argyll-Robertson pupil are absolute constants. Disorders of subjective sensibility are common enough but less intense. Objective disorders

are constant. Sphincter troubles are frequent and dysarthria of the general paralytic type very common. The mental state is more like the general paralytic with indifference and euphonia. Lancinating pains in the lower limbs often add to the catalogue of symptoms. There exist the meningo-encephalitic lesions of the general paralytic, the cord changes constituting a diffuse specific myelitis.

Dejerine in 1913 described a sensory medullary syndrome under the name, "syndrome des fibres radiculaires longues des cordons posterieurs". The characteristic feature of this syndrome is the considerable alteration of deep sensibility, leading almost to abolition with integrity of tactile, pain, and thermal sensation. The long posterior fibres are solely affected. Porot mentions an illustrative case, of spinal syphilis in course of regression in which the syndrome existed in its purity, and representing a phase or stage in the evolution of specific lesions of the cord. No attempt is made to discuss the payhology of the condition nor is any reason advanced to account for the peculiar vulnerability of these root fibres. The syphilitic toxin is only one of many agents liable to produce this syndrome.

The possibility of a LANDRY&S PARALYSIS must be kept in mind. In the acute myelitides, the syphilitic change in the cord gives rise to ascending and descending degeneration; doubtless the acute variety commences at that stage. Nonne published a typical case in a 35 year old man who developed an acute ascending paralysis four months after initial infection. Within four days the bulbar region was involved in the form of paresis of muscles of deglutition, mastication, and respiration. Typical C.S.F. reactions were obtained. Vigorous treatment effected cessation of spread with recession of paralysis and ultimate cure of patient. The history, C.S.F., and response to treatment proved the case to be a "Landry" of specific origin. It has alraedy been remarked that the sequel to thrombosis is not infrequently necrosis with consequent formation of syringo-myeloid cavities. In 1902, Dekeyser described syringo-myelic symptoms in a luetic and believed it to be a true case of syringo-myelia of syphilitic origin. Oppenheim says that syringo-myelia is frequently met with in syphilitics. Petren in 1909 mentioned a case shewing a combination of syringo-myelic and pachymeningitic symptoms; he concludedthat if symptoms of a spinal meningitis appeared in a syringo-myelic, one could reasonably suspect a syphilitic basis. The subject is of extreme interest, and a profitable one on which to speculate but we agree with Nonne when he says,

Multiple Sclerosis of syphilitic origin.

Gummatous Tumours.
Brown-Sequard complex.

"man muss sagen,dass heute das Thema'Syringo-myelie und Syphilis' noch nicht spruchreif ist". Much the same relationship obtains between idiopathic disseminated sclerosis and symptoms of multiple sclerosis of syphilitic origin. The pathological pictures may be almost identical yet the clinical course in the two diseases is totally different. Scanning speech,true rotatory nystagmus,intention tremor,and the general nodding shaking of back and head are not seen in cerebro-spinal syphilis.(Nonne) Becterew has described a disseminated syphilitic cerebro-spinal sclerosis,but he differentiated very clearly his clinical picture from the well-known classical form of multiple sclerosis.

Allusion has been made to the possibility of gummatous tumour formation. The symptoms vary within wide limits;they may be due to gummatous meningeal proliferation,a chronic gummatous myelo-meningitis,circumscribed gummatous tumours, an intra- or extra-medullary gumma with a greater or less degree of involvement of the meninges and roots. Examination of the cerebro-spinal fluid is of cardinal importance in these cases. With typical serological reactions and suggestive Lange curves there is usually an increased protein content with definite lymphocytosis and possibly slight excess of globulin. The response to energetic treatment is a useful empirical sign of correctness of diagnosis.

Finally we refer in conclusion to the frequency of the BROWN-SEQUARD SYNDROME in syphilis

of the cord. Here again the typical symptoms may be due to a vascular softening,meningitic overgrowth, gummatous tumour formation,or a circumscribed meningo-myelitic process. Most cases are chronic although Armstrong reports an acute case. The clinical picture is not always clear-cut and pure;frequently sensibility may be slightly diminished in the paresed or paralysed limb and vice versa. The syndrome would appear to be in many cases an element in the pathologic-clinical complex of a dorsal meningo-myelitis,with greatest incidence in the thoracic region of the spinal cord.

We make no claim to have discussed the subject of spinal cord syphilis exhaustively, but we have attempted to portray a few of its protean manifestations,and in conclusion we present 24 case histories illustrative of our findings arrived at as a result of a survey of the literature and our own necessarily restricted experience.

CASES. PRIMARY SYPHILITIC DEGENERATION OF THE CROSSED PYRAMIDAL TRACTS.

J.B. Male. aet. 54. Complained of difficulty in walking,unsteadiness,pain on moving the neck, and headache. There is a history of syphilitic infection 30 years ago. Twelve years ago he first noticed some heaviness in the lower limbs. The condition has not progressed very much since then; he is still able to get about with difficulty. His Blood Pressure is 160/98. The cranials are normal. The right pupil is slightly smaller than the left; both react to light and accommodation. The right external rectus is weak; there is slight nystagmoid movement to the right. The power in the upper limbs is normal and well preserved. The lower limbs shew definite weakness. The sensory system is normal. The arm reflexes are normal and equal. The abdominals are present. The lower limb reflexes are greatly exaggerated and equal with slight bilateral ankle clonus and an extensor plantar response on both sides. The organic reflexes areunaffected. His gait is spastic and unsteady. The Blood and C.S.Fluid are positive. The damage is almost exclusively restricted to the pyramidal tracts; it seems reasonable to conclude that this case is one of syphilitic spastic paraplegia from primary degeneration of the pyramidal tracts.

Primary syphilitic degeneration of crossed pyramidal tracts.

G.S. Male. aet. 50. Complained of difficulty in walking of sudden onset one year prior to admission. He began to drag his legs and finally was unable to rise from his chair. He had incontinence of urine. His condition improved slightly and he was able to walk with difficulty. There was a history of syphilitic infection 30 years ago. The cranials were normal. The pupils were small, equal, sluggish to light but normal to accommodation.There was evidence of analgesia in the left leg below the knee,and some deep pressure loss in the same area. Vibration and sense of position were dulled in the left leg. In the lower limbs ,power was much reduced.THere was marked hyper-reflexia in both legs with ankle clonus and bilateral plantar extensor responses. The legs were very stiff and spastic.The abdominals were absent,with incontinence of urine. The Blood Pressure was 140/108. The Blood W.R.was strongly positive,over 10 M.H.D. The C.S.Fluid W.R. was positive,over 7 M.H.D.,a cell count of 11 per cmm., practically all small lymphocytes,with occasional endothelial,polymorphs,and plasma cells. The total protein was 0.04%, a trace in excess of globulin, and a Lange curve 1232210000. This is a case of syphilitic menongo-myelitis with definite involvement of both crossed pyramidal tracts. It is a spastic paraplegia from primary degeneration of the pyramidal tracts with some degree of meningeal thickening.

ACUTE SYPHILITIC TRANSVERSE MYELITIS.

A.O. Male. aet. 46. On admission the patient complained of paralysis of both legs. The morning before admission he woke up and found himself unable to move his legs and with retention of urine. Examination revealed small unequal pupils, the right slightly larger than the left; they both reacted sluggishly to light and normally to accommodation. The cranials nerves were normal. Pain, thermal and tactile sensations were lost below the level of Th.9. At the level of Th.8 there was definite hyperaesthesia. The day after the paralysis, the knee and ankle jerks were absent on both sides. Power in the lower limbs was almost completely abolished, some very feeble contraction of both quadriceps femoris was noticed, and slight dorsiflexion of toes on both sideswas effected. A week after the catastrophe, voluntary power in the legs shewed some signs of returning, more so in the right than in the left leg. The knee and ankle jerks in both lower limbs were greatly exaggerated and equal, while the plantar responses were strongly extensor. The abdominals were absent in all four quadrants. The patient had retention of urine. The heart was fibrillating and the blood pressure was 110/80. The other systems were normal. The C.S.Fluid was clear, colourless, and under normal pressure, shewed 98 cells per cmm., nearly all of which were small lymphocytes, with an occasionalsmall endothelial cell, plasma cell, and polymorph. The total protein was 0.08%, globulin in

well-marked excess,The W.R. strongly positive,over 10 M.H.D.,and a Lange curve 555554421. The Blood W.R.was a weak positive, 3 M.H.D. $T_H$is is a typical case of acute syphilitic transverse myelitis. Nonne and Orlowsky have mentioned cases of such sudden onset. The suddenness would almost suggest a severe spinal haemorrhage. There were no promonitory signs,suchas the fairly typical prodromal pain in the back,the complete absence of paraesthesiae,girdle sensations, shooting pains,tiredness in the limbs,or muscle twitchings. Nor was there any variable patellar reflex, claimed to be a valuable sign by Oppenheim and others. The reflexes in this case most definitely returned, hence the lesion must have been in the lower thoracic region.

INCOMPLETE DORSAL MYELITIS.

G.D. Male. aet. 53. Complained of dragging his feet after walking a shortb distance; sometimes he loses controlof the legs, and occasionally has a numb feeling in the right hand. The trouble commenced one year ago when his feet would tend to drop;he had no pain,but his legs tired more easily than before. Eight months ago he felt as if he were walking on a pad; this however has disappeared. His Blood Pressure is 160/98.The cranials were normal. The pupils were unequal,the right being greater than the left,they reacted to light and accommodation. There appeared to be some slight diminution to all forms of sensation over the trunk and lower limbs especially the vibration sense,below the level of Th.6. The upper limbs were normal. There was some degree of spasticity in both legs. The arm reflexes were normal and equal. Those of the lower limbs were markedly exaggerated with bilateral ankle clonus. The plantars were both extensor. Definite weakness of vesical sphincter. The gait was spastic. The Blood and C.S.Fluid were positive. This is a case of a syphilitic meningo-myelitis affecting the crossed pyramidal tracts. The symptoms suggest the incomplete dorsal meningo-myelitis rather than a primary degeberation of the motor tracts,in other words,the process is a progressive peripheral sclerotic menongo-myelitis. The specific toxin would appear to be invading and affecting the motor tracts by permeation from the periphery and not acting on the tracts directly. It is impossible to state dogmatically the precise mode of pyramidal involvement.

MENINGO-RADICULITIS AFFECTING C5.6.7.8. AND Th.1.

J.B. Male. aet. 39. On admission the patient complained of insomnia, "pins and needles" in the left arm, left side and left leg, and loss of fine movement in the left hand, all of some six months duration. One year ago he complained of pain in the left elbow and some three months later the left arm became useless; this improved although he was still unable to pick up small articles. There was a history of syphilitic infection some 18 years ago. The cranials were normal. the pupils were equal and reacted to light and accomm-odation. There was no nystagmus; speech and hearing were normal. Examination shewed distinct diminution of thermal, pain, and tactile sensation below the left elbow. Marked hyper-reflexia was noted in the left arm, and exaggeration of both knee jerks; the other reflexes were normal. Co-ordination was poor in the left hand. Rombergism was absent. The organic reflexes were normal. His Blood Pressure was 145/98. Other systems were normal. The Blood W.R. was positive. The C.S.Fluid W.R. was also positive and shewed 130 cells per cmm., mainly small lymphocytes, with occasional endothelial cells, plasma cells and polymorphs. The total protein and globulin were unfortunately not estimated. In this case there was no muscle wasting or fibrillary tremors. The diagnosis was necessarily one of a meningoradiculitis affecting C.5.6.7.8. and Th.1.

SYPHILITIC AMYOTROPHIC MENINGO-MYELITIS, WITH SYRINGOMYELOID CAVITATION.

B.A. Male. aet 39. On admission patient complains of inability to move the arms, difficulty in moving the legs. He has headaches in strong light and scalds without knowing it. Symptoms began eleven years ago with severe frontal headaches. A year later the right arm became weak with wasting between the right fore-finger and thumb; three months later the right leg began to drag; the wasting in the right arm spread until the whole limb was involved. In the left arm the wasting appeared at the top of the shoulder about eighteen months ago. In each case twitching appeared before the wasting. About two years ago he noticed insensibility to heat and pain first in the right hand then in the left. The cranials are normal. The pupils are regular but unequal, the right being smaller than the left; they react to light and accommodation; the discs and fields are normal. There is numbness in the right arm, the right side of body and the right leg and to some extent in the left arm. Epicritic sensation is normal. Both hands shew pain and temperature loss. The vibration sense is diminished over the right foot. Some suspicion of astereognosis in right hand. Both arms are spastic. All the muscles of the right arm are flabby as are the muscles of the left shoulder girdle, anterior compartments of left arm and forearm, and intrinsics of left hand. The reflexes in the right arm are absent; they are just present in the left arm. All movements in both arms are greatly diminished. The supinating power is definitely lost in both arms.

The musculature of the lower limbs is definitely and uniformly wasted. Movements at all the joints are greatly restricted. The knee and ankle jerks on the right side are much exaggerated. Those on the left side are only just present. The plantar responses are strongly extensor on both sides. The gait is stiff and spastic; the patient requires support. The organic reflexes shew no defect. The Blood W.R. is strongly positive. The C.S.Fluid W.R. is negative, there are 3 small lymphocytes per cmm.,a total protein of 0.03%,and globulin is within normal limits. The pathological picture is an interesting and elaborate one. The case is a syphilitic amyotrophic meningo-myelitis with bilateral syringomyeloid cavitation and chronic cervical hypertrophic meningitis. The syringomyelic symptoms may be due to a haematomyelia or to a spreading gummatous infiltration. The destruction is affecting primarily the ventral horn cells and also the crossed pyramidal tracts among others.

SYPHILITIC DORSAL MENINGO-MYELITIS AFFECTING PRIMARILYTHE LEFT MOTOR TRACT AND SLIGHTLY THE ANTERIOR HORNS.

J.J.B. Male. aet. 55. A history that 9½ years ago he began walking badly,both legs being affected. Some three months later there was stiffness in both calves. The specific nature of the malady was recognised and he was treated accordingly with the result that the strength returned to the right leg but not to the left. The right lower extremity maintained its strength well until a year ago. The left arm became weak,and complained of pain in both hands. Now the left arm is useless;the legs are very weak,movements are very restricted and he is troubled with urinary incontinence. There is a history of gonorrhoea at twelve and syphilis at 21. The pupils are small and irregular,the left larger than the right;they react sluggishly to light but normally to accommodation.The diecs are rather pale and clear-cut but otherwise normal. The cranial nerves are normal. The motor power is good in the right arm,but very weak for all movements,especially flexor,in the left arm. There is some spasticity in the left arm. There is no wasting in the upper limbs. The supinator jerks are present,the left greater than the right. The biceps and triceps jerks are just present.The right knee and ankle jerks are sluggish;the left being present and slightly greater than the right. The right plantar is flexor,while the left is definitely extensor. Speech and hearing are normal. Vibratory sense is absent in the lower limbs. Other systems are normal. The Blood Pressure is 145/105. The C.S.Fluid was clear., under normalpressure, and shewed three small lymphocytes per cmm.,a total protein content of 0.03%, no excess of

globulin,a negative W.R. and a Lange curve 0112100000. The Blood W.R.was likewise negative, the sequel to earlier and vigorous treatment.

This case is a syphilitic dorsal meningo-myelitis, affecting primarily the left motor tract and to a less extent the anterior horns in spite of the presence of the sluggish reflexes. The clinical comdition would quickly develop into an amyotrophic lateral sclerosis. The vibratory loss would suggest an early thickening of the spinal meninges with later peripheral sclerosis.

DORSAL MENINGO-MYELITIS AFFECTING COLUMNS OF GOLL AND LATERAL COLUMNS.

L.G. Male. aet. 39. Admitted complaining of weakness of four limbs and "twitching feelings" in the legs. A year ago he had "flu", since when he has had weakness in the limbs, most noticeable on exertion and particularly in the legs; he is unable to walk far. For the past year he has felt occasional twitchings in the legs. The pupils reacted to light and accommodation normally, the right being slightly larger than the left. The cranials were normal. To pain and temperature, sensation was diminished and erratic over the right upper arm and both legs, the feet being normal apparently. Vibratory sense was absent over both legs and feet. Arm reflexes were brisk and equal with moderate power. The lower limb reflexes were not exaggerated, nor was there any spasticity; the tone and power were good. The abdominals were absent. The plantar responses were extensor in type. The gait was unsteady, with some stamping. The heel-knee tests shewed some inco-ordination. Other systems were unaffected. The Blood Pressure was 105/70. The Blood and C.S.Fluid shewed a weak positive, 3 M.H.D. This patient's pressure was extremely low, suggesting a thrombotic lesion. The condition would appear to be a dorsal meningo-myelitis affecting the lateral columns chiefly and also to some extent the columns of Goll. There would be evidence of vascular change, an endarteritis obliterans and endo- and periphlebitic destruction. The periphery of the cord would shew sclerosis. In this case the organic reflexes were normal and there was no evidence of wasting.

INCOMPLETE DORSAL MENINGO-MYELITIS.

A.B. Male. aet 61. Admitted complaining of pains and twitching in the left leg,pain in the right foot, and incontinence of urine. His gait is unsteady. Symptoms began some two years ago with unsteadiness of gait,since when they have become gradually worse. The pupils were normal,in shape and reaction. The cranials shewed no defect. In both ears,air conduction appeared to be slightly diminished; bone conduction was normal. There seemed to be some diminution to pain over and indefinite areas on both thighs and left leg. The upper limb reflexes were brisk and equal. The lower extremity reflexes were exaggerated and equal,with moderate spasticityin both limbs. The plantars were extensor. There was incontinence of urine. His C.S.Fluid shewed 18 cells per cmm., chiefly small lymphocytes with an odd small endothelial cell and rarely a polymorph; total protein of 0.06%, globulin in slight excess,and a W.R. positive to 8 M.H.D.,with Lange curve 2343210000. The Blood W.R. was negative. The BLood Pressure was 145//75. The other systems were normal. In his gait there was just a suspicion of an ataxic element. This case would be considered an incomplete dorsal meningo-myelitis probably with some marginal sclerosis with thickened spinal meninges.

INCOMPLETE DORSAL MENINGO-MYELITIS WITH MAIN INCIDENCE ON RIGHT CROSSED PYRAMIDAL TRACT.

J.H. Male. aet. 56. Patient complained of weakness of right leg and pain in the left leg and lumbar region of back. The weakness in the right came on gradually, commencing some five years ago. Five weeks ago he complained of stabbing pain along outer border of left thigh and difficulty in micturition. Examination revealed normal cranial nerves. The pupils were small, the right smaller than the left, they reacted to light and accommodation. There appeared to be some postural sense defect over the right foot and some vibratory loss over both legs. The upper limb reflexes were normal; definite hyper-reflexia in the lower extremities with the right leg particularly spastic. The right plantar was extensor, the left flexor. There was difficulty with micturition. The gait was unsteady with a spastic right lower limb. The patient contracted syphilis some six years ago, and was treated at a genito-urinary hospital. The C.S.F. shewed 2 cells per cmm., all of lymphocytic variety, a total protein of 0.03%, globulin within normal limits, a negative W.R. and a Lange curve of 0111000000. The Blood W.R. was negative. His Blood Pressure was 115/68. This case is an incomplete dorsal meningo-myelitis with the main pathological incidence on the right crossed pyramidal tract. The condition is a progressive one and will ultimately affect the left motor tract .

LUES CONGENITA WITH SPASTIC PARESIS OF LOWER EXTREMITIES ? OR PRIMARY DEGENERATION OF PYRAMIDAL TRACTS WITH EXTENSION INTO THE BRAIN STEM.

P.S. Male. aet. 25. Complained of numbness and weakness of both legs. Fifteen months ago the right leg became weak and felt numb; on several occasions it gave way; it improved however and he worked until a month ago when the left leg became weak and numb. Now he cannot stand up. The arms are unaffected. No headache or vertigo. No disorder of micturition. The right pupil is larger than the left; there are large lamellar posterior nuclear cataracts in both both lenses.The vision is 6/54,he is unable to distinguish fingers at two feet. The visual fields are considerably diminished. Hearing is diminished in both ears,air and bone conduction being affected. There is some evidence of slight left facial weakness, and the tongue on protrusion deviates slightly to the right. There is weakness and numbness of both legs. There appeared to be a suspicion of diminished sensation to pain below the level of Th.4. Other forms of sensation unaffected. No wasting of upper limb musculature, no spasticity, and no loss of tone or power. The reflexes were normal and equal. The lower extremities shewed no wasting but marked hyper-reflexia, considerable spasticity and sustained ankle and patellar clonus. The abdominals were present in the upper quadrants. The plantar responses were definitely extensor. There was some fine tremor of both hands, more marked in the right. The organic reflexes shewed no defect. The mental state was normal; the family history was not obtainable. All other systems were normal.

The C.S.Fluid was clear and under normal pressure, shewed 27 cells, chiefly small lymphocytes with a few endothelial cells, an occasional larger endothelial cell,and a very odd polymorph; no plasma cells were seen. Total protein,0.1%, globulin in moderate excess,The W.R.strongly positive,over 10 M.H.D. The Lange curve was 5555543100. The BLood W.R. was strongly positive, over 10 M.H.D. In this case one must take into account the possibility of heredo-lues. Mingazzini's case, published in 1921 (quoted by Nonne) is suggestive in that one of the children of a tabetic developed bilateral cataract with optic atrophy,together with a spastic paresis of the lower extremities,a condition from which a younger member of the family suffered later. This case therefore might be one of Lues Congenita, or a primary degeneration of the pyramidal tracts with extension into the brain stem. It simulates the case reported by Koenigstein in 1910, a case of lues congenita with spastic paresis of the lower extremities and bilateral cataract. We are inclined to group our case in the same category.

PROGRESSIVE MARGINAL SCLEROTIC MENINGO-MYELITIS WITH SCLEROSIS OF PYRAMIDAL TRACTS.

A.H. Male. aet. 59. On admission complained of "jumping of legs",chiefly right,and neuralgic pains in both arms and legs,more marked on the right. Two years ago he complained of "jerking movements" and"Dragging sensations" in the right leg,especailly in the evening when tired. For the past three weeks he has had pain in the left forearm. All symptoms have become worse during the last six months. No headaches and no vertigo. The patient gives a history of syphilitic infection some twenty years ago. The cranials were normal; speech and hearing unaffected. The pupils were small,the right smaller than the left; they reacted sluggishly to light and normally to accommodation; ocular movements were normal. The sensory system was unaffected.The upper limbs were normal in tone,power,and shewed no evidence of wasting. Arm reflexes were normal;lower limb reflexes were exaggerated,the right more than the left. The plantars were strongly extensor in type. The gait was normal. There was occasional precipitancy of micturition. His Blood Pressure was 150/100. The C.S.F.shewed 13 cells per cmm.,mostly small lymphocytes with on occasional small endothelial cell and very occasional polymorph;total protein,0.1%, globulin in moderate excess,W.R.strongly positive over 10M.H.D.,lange curve 2454421000. The Blood W.R.was strongly positive. This case would come under the category on an Erb's spastic paraplegia; we prefer to consider it an incomplete dorsal myelitis, or following Raymond and Cestan,a progressive marginal sclerotic meningo-myelitis, with marked involvement of the crossed pyramidal tracts.

COMBINED POSTERO-LATERAL SCLEROSIS OF SYPHILITIC ORIGIN.

W.S. Male. aet.43. Twelve months ago complained of "neuritis" on the right side of head, attacks of pain in the knees and legs,more marked on the left side,and numbness of the little and ring fingers of the right hand. The pains shoot down the legs,are of sudden onset,last a few seconds,and recur every 3-4 minutes. He has had headaches recently; his sight is good. There is no vertigo and no frequency. He has difficulty in initiating the act of micturition. The cranial nerves are normal. The pupils are dilated, the right a little irregular.The right pupil is inactive to light,but reacts slightly consensually. The left reacts slightly to light,better consensually, and both accommodate normally. There is definite temporal pallor of right disc with the lamina visible. The veins are distended. The left disc is normal. The motor power in upper and lower limbs is moderate. Examination of the sensory system elicits vibration loss in the lower extremities. The upper limb reflexes were present and equal. The abdominals were absent in all four quadrants. The right knee and ankle jerks were present. The left lower limb jerks were absent. The plantar responses were extensor on both sides. Co-ordination was good;no tremor or tics. The gait was ataxic and Rombergism was present. His speech shewed no defect; his tongue was smooth. All other systems were normal. His Blood pressure was130/80. The C.S.Fluid was clear,colourless,under normal pressure,contained 17 cells per cmm.,practically all small lymphocytes except for an occasional

small endothelial cell. The protein content was 0.04%,with a trace in excess of globulin, a W.R. positive to 7 M.H.D.,and a Lange 2454310000. The Blood W.R. shewed a weak partial positive, 3 M.H.D. We would suggest in this case there exists a lesion of the posterior and lateral columns. The signs are indicative of an incipient tabes,with involvement of the crossed pyramidal tracts. The combination of combined posterior column and anterior horn destruction is of frequent occurence, hence there semms no reason to doubt a primary motor tract degeneration with change in the columns of Göll.

AMYOTROPHIC MENINGO-MYELITIS WITH POSTERIOR COLUMN SCLEROSIS.

J.C. Male. aet. 50. Patient gave a history of pain in the back of the neck of some six months duration. Two months ago he lost the use of the left arm, commencing with slight weakness and becoming gradually and progressively worse. At the same time the left leg became weak and began to drag when walking. The right arm and right leg are shewing signs of weakness. Some slight stammering of speech one week ago. Severe continuous occipital headache. No vomiting and no vertigo. No sphincter disability. He complained of "pins and needles" and "weak" feeling in the left hand and to a less extent in both legs and right hand. Patient gave a history of venereal disease and penile sore some 20 years ago, for which he received no treatment.
The right pupil was larger than the left, both reacted sluggishly to light and normally on accommodation. The discs and visual fields shewed no defect. There was slight involvement of the left trapezius and some weakness of both sternomastoids. The motor system was extensively affected. On the left side the neck muscles and erector spinae were weak; the pectoralis major, deltoid, flexors and extensors of elbow, and flexors and extensors of wrist and fingers, shewed mild to severe grades of weakness. The trunk muscles were unaffected. The flexors and extensors of hip shewed slight diminution in power, these of knee moderate diminution, and those of the ankle and toes considerable diminution. All the muscles of the left side were hypotonic, more marked as one proceeded

down the extremities except in the left arm which shewed moderate spasticity. The range of movement was correspondingly diminished in the weakened muscles. On the right side, the power tone and range were slightly diminished as was the nutrition of all the muscles but to a much lesser degree than the left. The sensory system was likewise markedly affected; to touch there was an area of anaesthesia corresponding to part of C.4 skin segment over the left shoulder anteriorly, otherwise unimpaired; no hyperaesthesia. To pain there was some hyperalgesia from the wrist to the finger tips in the left arm. There was hyperal-gesia over the whole of the left half of the trunk, anterior and posterior from the level of Th.4. to L.1. The right arm and both legs were normal. We noted exaggerated sensations to heat and cold over the left hand corresponding to the hyperalgesic area; loss of sensation over C.4. Normal elsewhere. The vibratory sense was diminished at the left wrist; elsewhere unimpaired. There was marked hyper-reflexia in both upper and lower limbs, being more marked in the left leg than the right. An interesting feature of this case was the definite vasomotor instability over the whole trunk from the level of Th.4 down to L.1., anter. and posterior. The left hand was moist, warm and red. His gait was spastic with tendency to drag left leg. All other systems were normal. His Blood Pressure was 155/85. The patient's condition steadily became worse. The left pupil was much smaller than the right,

with evident enophthalmos suggesting involvement of cervical sympathetic at level of C.8. The power on the right side was becoming gradually less.The thenars, hypothenars and interossei shewed marked wasting,more obvious on the left than on the right. Incoordination was marked on both sides,but more so on the left. The plantar responses as before were definitely extensor in type. The sensory system became progressively more affected,there being loss of pressure sense,temperature, pain,vibratory and postural sense extending to the hands and forearms,the left being more defective than the right. The sense of touch was unimpaired. The weakness extended to the right side. At a later period still,the sense of touch became impaired in conjunction with the other sensory loss from the level of C.4. to Th.12. The weakness and wasting finally affected the shoulder girdles; the upper limbs became markedly spastic; the reflexes were still very active;the rectal and vesical sphincter control was lost.
The Blood W.R. was strongly positive,over 10 M.H.D. Likewise the C.S.Fluid W.R. was strongly positive, over 10 M.H.D. The C.S.F. shewed 11 cells per cmm., the majority being small lymphocytes, a few small endothelial cells, an odd plasma cell, and an occasional polymorph. Chlorides and sugar were normal. The total protein was 0.1%,with globulin in only slight excess as compared with the high protein.
This case is interesting in many respects. The gradual and asymmetrical evolution of the clinical picture is somewhat unusual. Further, for some time

there was evidence of dissociated anaesthesia suggesting some syringo-myelosis. The enophthalmos and miosis were indicative of interruption of the pupil dilating fibres and are relatively common in true syringo-myelia. They occur also in a radiculitis of syphilitic origin. Another feature is the involvement of the left column of Burdach with later that of the right, and only partial destruction of the columns of Goll, the area in contiguity with Burdach's columns. The pyramidal tracts were early and definitely affected. The ventral horn cells on the left were quickly involved with later spread to the cells of the right. The C.S.Fluid shewed typical dissociation albumino-cytologique, frequently alluded to by French writers. The pathological changes were widespread and it is not easy to decide which column was affected first. The case would appear to be an amyotrophic meningo-myelitis or progressive spinal muscular atrophy of syphilitic origin, with extension to the columns of Burdach and Goll.
No doubt the spinal meninges were thickened, and mild marginal sclerosis would add to the pathological picture.

AMYOTROPHIC MENINGO-MYELITIS OF SYPHILITIC ORIGIN.

OR PROGRESSIVE SPINAL SYPHILITIC MUSCULAR ATROPHY.

E.Y. Male. aet.61. Admitted complaining of loss of power in both arms,in right leg,and partly in the left leg. About 18 months ago he suffered acute cramp-like pain in calf muscles of right leg which lasted some 6months. During the past year patient has noticed he was rapidly loosing flesh and that the power in his right arm and leg was "going". The arms became pract--ically useless 5 months ago. Now the right leg is only just able to support him. His past history and family history present nothing of interest. Mental state normal. Cranial nerves normal. Partial right ptosis;pupils small,irregular but central;partial direct reaction to light in both eyes;consensual reaction is normal and equal;reaction to accommod--ation is and equal in both eyes. The sensory system is normal except for occasional cramp-like pain in all limbs. There is marked wasting in thenars and hypothenars of both hands with early "claw" hands. Wasting of all scapular muscles on both sides;the right biceps is absent,the left is present but very weak;supination and pronation much diminished in both right and left arm;the musculature of the lower limbs is uniformly and equally wasted;there is advanced wasting of tongue with evidence of fibrillary tremor; fibrillary twitching was present in all degenerating muscles. The right supinator jerk was just present, the left being normal. Both biceps jerks were absent. The triceps jerks were equal and normal. The lower limbs exhibited marked and equal hyper-reflexia with ankle clonus and bilateral extensor plantar response.

The abdominal reflexes were absent in all four quadrants. The organic reflexes were normal. The gait was spastic;the patient could only just shuffle along. The heart shewed left ventricular hypertrophy, the Blood Pressure being 220/140. There was slight leukoplakia of left cheek,pathological examination of tissue from which revealed chiefly hyperkeratosis. The remaining systems were normal.

The C.S.Fluid was clear,under normal pressure,and shewed one cell per cmm. of the small lymphocytic variety,0.03% of total protein,no excess of globulin, normal sugar and chloride content,with a negative Wassermann and Kahn Reaction and a Lange 0111000000. The Blood count was normal. The Wassermann Reaction in the Blood was positive to 7 M.H.D's; the Kahn Reaction was strongly positive.

The incidence of disease was mainly in the cervical cord from C.4 to Th.1 and to some extent affected the lower lumbar and upper sacral regions. The bilateral extensors indicated involvement of both crossed pyramidal tracts. The case was an amyotrophic meningo--myelitis or progressive spinal muscular atrophy of syphilitic origin. The pains suggest the possibility of posterior meningeal thickening;there was no evidence of involvement of the columns of Goll and Burdach.

SYPHILITIC AMYOTROPHIC LATERAL SCLEROSIS.

A.D. Male. aet.55. Admitted complaining of lancinating pains across lumbar region and down arms with compleete loss of power in both legs and hands,all of about one year's duration and of gradual onset. Patient gives a history of being quite well until a year ago when he experienced acute pain in the back, in periodic attacks for the first few months becoming more frequent in the latter months. He complained of severe "cramps" in both legs,especially in the left,synchronising with the attacks. At first the left leg became stiff with pain in the right lower limb. Both legs are now stiff and wasted. Occasional headache.
On admission he complains of "cramps" in the arms, and difficulty in moving the fingers with "quivering" in the muscles. Both legs are stiff,weak,and wasted, the left more than the right. He has no sphincter disability. Complains of feelings like "needles" in the tongue and hands. The legs feel cold.
The cranial nerves shewed no defect. The right pupil was small to moderate,central but irregular,did not react directly or consensually to light,but reacted to accommodation. The left pupil was small,central, and irregular, accommodated and reacted to light as did the right. Examination of the sensory system revealed apparently some diminution of deep pressure sensibility in the right leg as compared with the left. Otherwise sensation was normal in the upper and lower limbs and trunk. The musculo-motor system shewed the most marked defect. Power and movements in upper limbs were fairly good; movements in left hand were somewhat

weak,with only slight weakness in the right hand. Definite contracture in the left hand; slight contracture in the right hand;fingers of both hands maintained in position of slight flexion. Marked wasting of thenars,hypothenars,and interossei of the left hand with slight wasting of left forearm muscles. Similar wasting was evident in the right hand and forearm although definitely to a less degree. The tone was much decreased with flaccidity in above muscles especially in the left hand. Marked hyper-reflexia in upper limbs.

Both right and left lower extremities affected. All movements were very weak; only flexion and extension of hips and flexion of knees possible to any degree. Wasting was most definite in the anterior groups of leg,dorsum,and sole of foot,and in front and back of thigh. Great decrease in tone with flaccidity in above groups; bilateral foot drop with pes cavus. Rapid contractions of individual groups of muscle fibres in the upper limbs and thighs,most marked in the forearm musculature. Slight defect in co-ordination was noted in the upper limbs; tests impossible in lower limbs on account of muscular weakness.

The knee jerks were just present and equal;the ankle jerks were not elicited. The abdominals were absent in all four quadrants. The plantar responses were both extensor in type. The organic reflexes were normal, there being no vesical or rectal dysfunction.n All other systema were normal. Blood pressure 120/95.

The blood W.R.was negative. The W.R. in the C.S.Fluid was positive to 4 M.H.D. On two occasions the C.S.F. gave a cell count of 2 small lymphocytes per cmm., no excess of globulin,a total protein of 0.03%,with normal sugar and chloride content. The C.S.Fluid shewed the following Lange 01210000000.

The pathological picture has been dealt with previously, and we now briefly correlate it with the clinical findings. Pathological change was evident in the descending cortico-spinal tracts,the lateral and anterior pyramidals,throughout the entire length of the cord. The ventral horn cells shewed marked diminution in number and obvious degenerative change. The spinal membranes were thickened and adherent to the cord surface with definite endarteritic change in the small vessels and perivascular small celled infiltration with evidence of small semi recent haemorrhages. Microscopic examination revealed mild sclerosis of ascending tracts (spino-cerebellar) in the right mid-dorsal region,with possibly some change in the descending vestibulo-spinal tracts in the same region. This case is typical clinically and pathologically of amyotrophic lateral sclerosis of syphilitic origin . This patients speech and mental state were normal but there was wasting and fibrillary tremors of tongue. A careful examination of the cerebrum might quite possibly reveal a degeneration extending to the motor cortex as in Spillers case reported in 1900.

PRIMARY SYPHILITIC DEGENERATION OF THE CROSSED PYRAMIDAL TRACTS.

H.H.P. Male. aet.36. On examination one year ago,he complained of loss of power in the left leg of two months duration. The sensory system was normal,and there was no evidence of amyotrophy. The upper limb reflexes were active,those of the lower extremities being markedly exaggerated,the right rather less than the left. The plantar responses were indefinite. The gaitn was somewhat spastic; no Rombergism; co-ordination in arms and legs good. The cranial nerves were normal. The pupils were equal and regular, and reacted to light and accommodation. The blood W.R. was negative. The C.S.Fluid was clear,under normal pressure,contained 17 small lymphocytes per cmm., a faintly positive globulin reaction,0.05% of protein, and a positive W.R.up to $\frac{1}{2}$dilution.
On re-examination a year later,patient dragged the left leg badly and complained of stiffness in the right lower limb. The arm reflexes were greatly exaggerated and equal. There was marked hyper-reflexia in the lower limbs with patellar and ankle clonus. The plantar responses were both definitely extensor. Abdominals and epigastric reflexes were absent. Organic reflexes were normal,there being no dysfunction of vesical or rectal sphincters. There was no sensory loss. The gait was very spastic with fair muscular power. The cranial nerves were normal;no history of ocular disturbance. This was a case of syphilitic spastic paraplegia from primary degeneration of the pyramidal tracts. There was no clinical evidence of anterior horn cell destruction nor involvement of the posterior columns.

AMYOTROPHIC MENINGO-MYELITIS OF SYPHILITIC ORIGIN.

OR PROGRESSIVE SPINAL SYPHILITIC MUSCULAR ATROPHY.

J.F. aet.39 On admission complained of weakness and wasting of the right hand, the left hand, and of both legs of 4years, 2years, and 6months duration respectively. His past history and family history revealed nothing of note. Mental state normal. Cranial nerves normal. Pupils were equal, central, and circular, with normal reaction to light and accommodation. Oculomotor power and conjugate movement normal. Vision, rt.6/6, lt.6/6. Sensory system was normal. The motor system shewed marked defect; rt.grip 25, lt.grip 20; weakness and wasting of biceps and triceps of right arm, with defect--ive power of supination and pronation of rt. forearm. Power in the left arm was fair with weakness of lt. supinators and pronators. There was marked wasting of thenars, hypothenars, and interossei of both hands. The legs were somewhat spastic, the right more than the left, with of flexors and extensors of both lower limbs. Fibrillary twitchings were observed in the affected muscles. Marked hyper-reflexia in both arms and legs, the right leg being greater than the left with an extensor plantar response. The gait was spastic with no ataxic element. The organic reflexes were normal. The C.S.Fluid shewed 7 small lymphocytes per cmm., 0.04% of total protein with normal chlorides sugar and globulin. The Wassermann Reaction was strongly positive in the C.S.F. and Blood.

The lesion could be localised chiefly in the cervical enlargement extending from C5 to Th.1., the case being an amyotrophic meningo-myelitis or progressive spinal muscular atrophy of syphilitic origin.

PRIMARY SYPHILITIC DEGENERATION OF THE CROSSED PYRAMIDAL TRACTS.

R.F. Male. aet. 41. Patient noticed he was walking badly and complained of weakness of right thigh and leg, some seven years ago. The blood W.R. was positive, and he improved on vigorous anti-syphilitic treatment. Four years later he complained of weakness in the left leg; the condition did not respond satisfactorily to treatment. On careful re-examination, his mental state was normal, as were also the cranial nerves. The pupils were irregular and unequal, the right being larger than the left; they accommodated normally, but did not react to light. The sensory system shewed no defect. There was no evidence of muscular wasting but movement and tone were considerably diminished in the lower extremities which were too weak to allow him to get about. The arm reflexes were normal and equal; the lower limb reflexes were markedly exaggerated and equal. The plantar responses were both extensor in type. The gait suggested an element of ataxia but there was no Rombergism. The posterior columns were apparently intact. The blood W.R. was negative; the C.S.Fluid W.R. was positive up to a $\frac{1}{2}$ dilution; the fluid was clear, under normal pressure, shewed 22 small lymphocytes per cmm., a trace in excess of globulin and 0.05% of albumin with normal content of sugar and chloride. All other systems were normal. The organic reflexes shewed no defect. We look upon this case as a paraplegia due to primary degeneration of the pyramidal tracts with some evidence of secondary syphilitic change in more distant parts of the nervous system.

PRIMARY SYPHILITIC DEGENERATION OF THE CROSSED PYRAMIDAL TRACTS.

R.H.F. Male. aet. 42. Patient admitted complaining of stiffness and loss of power in both upper and lower extremities of some 3½ years duration. There was a history of venereal infection 19 years ago. Three years prior to admission he complained of a burning sensation in the left shoulder and arm, with progressive weakness of inferior extremities. At the time the condition was recognised as specific and treateed accordingly. On examination the cranials were normal. The pupils were moderate, the right greater than the left and somewhat sluggish to light but both reacted normally on accommodation. There was no nystagmus and no history of diplopia. The sensory system was intact. Power was diminished throughout with no complete loss however. There was definite general atrophy of musculature, more marked on the right than on the left; the left arm was slightly spastic. The upper limb reflexes were normal, the left slightly stronger than the right. The abdominals and epigastrics were present and equal. Definite hyper-reflexia in lower limbs with marked left patellar clonus. The organic reflrxes were normal. Plantar responses were extensor on both sides. The other systems were normal. Patient had had frequent treatment and the blood and C.S.fluid W.R. was negative with no pleocytosis. Fibrillary twitching was not noticed. The incidence of disease in this case was strikingly restricted to the lateral columns. There was a primary syphilitic degeneration of the pyramidal tracts with possibly mild involvement of some ventral horn cells where amyotrophy was present. The case was a primary lateral sclerosis.

SYPHILITIC SUB-ACUTE MENINGO-MYELITIS WITH SCLEROSIS OF LATERAL COLUMNS.

A.C. Male. aet. 26. Admitted complaining of weakness of legs. Well until one month ago when this weakness developed, the right leg more than the left, and incontinence of urine. No history of diplopia, pains or paraesthesiae. His mental state was normal. The cranial nerves were intact. The pupils were equal, central, circular, and regular; they reacted to light and accommodation normally. Visions and discs were normal. Speech normal. The sensory system shewed no abnormality. Power was normal in upper limbs and much diminished in the lower extremities, with marked spasticity. The arm reflexes were all brisk and equal. The leg reflexes were markedly exaggerated with patellar and ankle clonus. The plantars were bilaterally extensor. The abdominals were absent. The gait was typically spastic. He complained of incontinence of urine. All other systems were normal. Blood Pressure 125/85. The C.S.Fluid shewed 25 cells per cmm., chiefly small lymphocytes with an occasional small endothelial and very occasional large lymphocyte. No polymorphs or plasma cells were seen. Total protein, 0.06%, Globulin in slight excess, W.R. strongly positive, over 10 M.H.D. and a Lange 5544321000. The Blood W.R. was strongly positive, over 10 M.H.D. This is a case of sub-acute meningo myelitis with the incidence of disease in the lower dorsal region and affecting the lateral columns causing a degeneration of the pyramidal tracts. We have no evidence of anterior horn or posterior column involvement.

PROGRESSIVE MARGINAL SCLEROTIC MENINGO-MYELITIS OF SYPHILITIC ORIGIN.

J.B. Male. aet.46. This patient complained of "staggering" when walking. Some three to four years ago he could not walk straight but tended to stagger and to sway forwards and backwards. He never actually fell. The left leg was worse than the right. His grip was less powerful than formerly; liable to drop things. He was more inclined to fall when eyes closed. No vertigo; mo headache, and no diplopia. His mental state was normal.
The cranial nerves are normal. The pupils are small, central and pear-shaped;right cataract removed,left cataract still in situ. The pupils react slightly to light,directly and consensually,and to accommodation. Vision in both eyes is much diminished. The visual fields are full. There is no objective sensory defect,but he complains of occasional "pins and needles" in the fingers and toes, and "cramps" in the feet especially when in bed. Co-ordination is good in upper and lower limbs. A very slight tendency to fall backwards on closing the eyes but there is no true Rombergism. He complains of difficulty in micturition and occasional incontinence. The arm reflexes are equal and normal. The arms shew no spasticity,wasting or diminution in range or power of movement. The lower limbs are somewhat spastic, not wasted and possesing moderate tone. There is marked hyper-reflexia with definite patellar clonus. The plantars are extensor on both sides. The abdominals are absent in all four quadrants.

His gait is spastic with a superimposed ataxic element. The right foot scrapes the ground while the left tends to be flung out. The cardio-vascular system is normal. The Blood Pressure is 115/80.

The blood Wassermann and Kahn Reactions were negative. The C.S.Fluid was clear,colourless,and under normal pressure, with 23 cells percmm.,principally small lymphocytes with an occasional small endothelial cell. The fluid contained 0.1% of total protein,and globulin in moderate excess. The W.R. was strongly positive, over 10 M.H.D. and the Lange 1232100000.

This case is a primary lateral syphilitic sclerosis. While not identical in clinical features with Erb's syphilitic paraplegia it might reasonably be included in his category. From what slight subjective sensations there were in this patient we would be inclined to suggest some meningeal thickening and possibly vascular changes. The posterior roots appear to be free from defect as are the posterior columns. From the clinical evidence we are disposed to consider this case a progressive marginal sclerotic meningo-myelitis of syphilitic origin.

SCLEROSIS OF CROSSED PYRAMIDAL TRACTS OF SYPHILITICORIGIN.

W.I. Male. aet.53. On admission patient complained of loss of power in the legs which commenced twelve previously by wasting and weakness of the left leg, with involvement of the right leg some seven years later entailing gradual loss of power. The specific nature of the condition was recognised and he was treated by intravenous and intrathecal injection. On examination the cranials are intact; the pupils are normal in outline and reaction. Speech normal. The sensory system shews no defect. There is marked spasticity in the lower limbs with weakness and some wasting but no indubitable evidence of fibrillary twitching. The arm reflexes were equal and exaggerated; the abdominals were just present; the lower limb reflexes were greatly exaggerated, the right more than the left, with definite bilateral ankle clonus and bilateral extensor plantar response. The gait is spastic and unsteady. The organic reflexes shewed no defect. There was some left cardiac ventricular enlargement with marked second aortic accentuation and a blood pressure of 170/90. The blood W.R. as a result of treatment was negative. The C.S.Fluid shewed one small lymphocyte per cmm., a total protein content of 0.1%, some four times the normal, globulin in moderate excess, a negative W.R., and a Lange curve 0012332L00. This case is an example of a primary degeneration of the pyramidal tracts with no evidence of extension to the hind or mid brain.

SCEROSIS OF CROSSED PYRAMIDAL TRACTS OF SYPHILITICORIGIN.

W.C. Male. aet.38. Patient admitted complaining of weakness of both hands, weakness of left leg,and bilateral wrist drop of some 4½ years duration. The condition has become gradually and progreeeively worse. He gave a history of having worked with lead but has always taken adequate precautions and clinically he shewed no evidence of chronic saturnine poisoning. His mental state was normal. The cranial nerves shewed no defect. The speech was natural; the pupils were moderate,central,equal,and regular and reacted directly and consensually to light and to accommodation. No contraction of visual fields. Occasional nystagmoid movement laterally. The sensory system appeared normal. Examination of the motor system revealed marked weakness of flexors and extensors of both forearms; double wrist drop and grips of 15(Rt) and 20(Lt). There was general weakness of the left leg, but no marked wasting. Very definite spasticity was noted in both lower limbs,more evident in the right. The biceps,triceps,supinator,and pronator jerks were present and equal. Thelower limbs shewed marked hyper-reflexia with ankle clonus on the right. The gait was very spastic with dragging of left foot. There was no Rombergism. The cardiovascular system was normal,with a blood pressure of 136/92. The blood picture for evidence of lead poisoning was completely negative. The blood W.R. was strongly positive. The C.S.Fluid was clear,under normal pressure,and shewed 7 cells per cmm.,the cells being chiefly small lymphocytes,with a few plasma cells,and an occasional

endothelial cell.The W.R. was strongly positive.The fluid contained 0.01% of total protein and globulin in well-marked excess. Sugar and chloride content were normal. It is relevant to note in this case that evidence of an Aran-Duchenne type of atrophy was lacking. Nor were we able to recognise any gross anterior horn cell involvement. The posterior columns shewed no signs of disease. Further,in this case the organic reflexes were intact,there being no history of sphincter trouble. This case is quite evidently a primary degeneration of the motor system, a sclerosis of the crossed pyramidal tracts of syphilitic origin. There seems no reason to doubt the primary nature of the process,a special susceptibility of the motor fibres to the syphilitic poison. It is probable that pathological examination would reveal marked lymphocytic infiltration of the pia but as remarked before, such infiltration is the result of toxic influence rather than the cause of the fibre degeneration.

REFERENCES.

Alquier et Touchard, L'Encephalie. 1909. 4. 404.
Aoyama, D.Z.f.N. 1904. Bd.26.
Babonneix,L, et Widiez,A, R.N. 37.I. 1214-1217. 1930.
Bassoe,P, and Hassin,G.B., A.N.P. 6. 1921. 32.
Bastian,H,C., Lancet. 2. 1531. 1910.
Batten, Brain. 376. 1904.
Berger, M.f.P.N. 3. 1. 1898.
Bing, Compendium.
Bochroch and Gordon, J.N.M.D. 209. 1902.
Bulschowsky, Z.f.kl.M. Bd.37. 1899.
Buzzard and Greenfield, Path.of the Nervous System.
Cadwalader,W.B.,A.N.P. 6. 541. Nov. 1921.
Cassirer, Klinische Wochenschrift. Berlin. 1911.
Cassirer und Maas, M.f.P.N. Bd.24. 1908.
Charcot,J.B., Arch.de Med.experimentale. 441. 1895.
Charcot et Dutil, Progres Medicale. 1894.
Chrisman,W.W., A.J.Syph. 16. 308. 1932.
Chung,M.F., A.N.P. 16. 761. 1926.
Chung,M.F., Archiv.of Dermat.and Syph. 14. No.2.
Cole,H.N., Archiv.of Dermat.and Syph. 9.102-113. 1924.
Collins,J., R.N.P. 10. 157. 1901.
Cossa,P., R.N. 38.II 112. 1931.
Dana, J.N.M.D. 81. 1906.
Dejerine, Compt.Rendus Series 10-11. 1895.
Dejerine, Revue de medicine. 60-78. 1884.
Dreshfield, Brain 1885.
Dercum and Spiller, J.N.M.D. Feb. 1899.
Dereux,M.J., R.N. 39.I. 689. 1932.
Erb, N.Z. Nr.6. 1892.
Erb, Lancet. 969-974. Oct.4. 1902
Falkiewicz, D.Z.f.N. 89. 1926.
Fouche, Proc.R.S.M. Vol.24. No.3. 312. Jan.1931.
Gowers, Diseases of the Nervous System.
Grunow, D.Z.f.N. 17. 1901.
Hammond, N.Y.Med.Jnl. Jan.1894.
Harris,W., Proc.R.S.M. 1909-1910.
Harris,W., Proc.R.S.M. Vol.16. 1922-1923.
Harvier,P., Bull.Soc.Med.Hop. Paris III.47. 604-610. 1931.
Harvier,P.,et al.,Bull.et mem.soc.med.d'hop.de Paris. 47. 1931.
Hassin,G.B.,et al., J.N.M.D. 72. 1930.
Hoffmann,J., N.Z. Nr. 28. 1909.
Jahnel,F., Fort.der Neurologie. 4. 19. 1932.
Kuh,S., D.Z.f.N. 6. 359-426. 1893.
Kroll,M., Zeit.f.d.ges.N.und P. 629-640. 1930.
Leri, Sem.Med. 1903.
Leri, Presse Med. 1910.
Leri, R.N. 1920.
Leri et Leronge, Gaz.des Hop. Vol.86. 885. 1913.
Leri, Cong.des alienistes et neurologistes. Bruxelles.
Leri, Questions d'actualite. 259. 1922. Paris.
Lannois,N.Iconogr.de la Salp. 1905.
Leopold,S., J.N.M.D. Vol.39. No.8. 1912.
Leopold,S., J.N.M.D. 38. 9. 1912.
Lapinsky, A.f.P.u.N.40. 602 and 752. 1905.
Margulis,M.S., D.Z.f.N. 113. 113-145. 1930.
Margulis,M.S., D.Z.f.N. 86. 1926.
Margulis,M.S., Syphilis u.Nervensystem. Berlin. 1924.
Martin,J.P., Brain. 48. 153. 1925.
Merle, R.N. 877. 1909.
Mills and Spiller, J.N.M.D. 385. 1903.
Moleen and Spiller, Amer.Jnl.of med. sci. Dec. 1905.
Marie, Tr.de Med. 340. Vol.6.
Mott, Brain. 1895.
Lambridght, J.A.M.A. 84. 1925.

Nonne,M., Syphilis und Nervensystem. Berlin. 1924.
Nonne,M., D.Z.f.N. Vol.43. Nr.3-6.
Nayrac et Lassure, R.N. 39.I. 1447. 1932.
Oppenheim. Lehrbuch. 1923.
Oppenheimer, Dissert.Wuerzburg. 1899. quoted by Vix.
Orlowsky, Ann.de Dermat.et Syph. 7. 139-151. 1896.
Popow,N.A., D.Z.f.N. 110. 117. 1929.
Porot et al. R.N. 33.II. 1926.
Preobraschenski,P.A., Neur. Zentralbl. 27. 1069-1074. 1908.
Raymond, Cliques des Maladies du System Nerveux. 1903.
Raymond, Sem.Med. Vol.13. 56. 1893.
Raymond et Cestan., R.N. 1905.
Raymond et Ricklin. Paris Congress. 1900.
Raymond et Rendu. R.N. 762. 1907.
Raymond et Cestan. L'Encephalie. 4. 1. 1909.
Ramond,L., Presse med. 251. No.16.
Robertson,E.G., Proc.R.S.M. 851. Vol.126. 1933.
Sainton, Nouv.Icon.de la Salp. 12. 317. 1899.
Schmaus, D.A.f.kl.M. 44. 244-264. 1889.
Schultze,F., D.Z.f.N. 68. 1. 1921.
Singer,H.D., Brain. 25. 332. 1902.
Spiller,W.G., J.N.M.D. 39. 589. 1912.
Spiller, Contrib. from the WM.Pepper Lab. of clin.med. 1900.
Spiller, J.N.M.D. 265. 1902.
Spiller, J.N.M.D. 36. 601. 1909.
Spiller, A.N.and P. 13. 471. Apl.1925.
Stewart,T.G. and Carmichael,E.A. Proc.R.S.Med. VOL.17. 1923-1924.
Stone,T.T., J.N.M.D. 66.
Struempell, D.Z.f.N. Bd.3. 1893.
Thomas,H.M., Johns Hopkins Hosp.Bull. 2. 369-392. 1891.
Touchard et Meaux-Saint-Marc, Ann.de mal.ven.Paris.8. 191-200.1913
Trabaud,C.J., R.N. 36.I. 808.1929.
Vix, A.f.P.u.N. VOL.47. Nr.3. 1212.
Vizioli, Congres de Moscow. 1897.
Wechsler,J.S., J.A.M.A. 77. 1728-1729. Nov.26. 1921.
Williamson, Syph.Dis. of Spinal Cord. 1899.
Williamson, Lancet. 1901.
Wilson,S.A.K., Rev. of Neur.and Psychiat. 401.Aug.1911.
Wilson, Sem.Med. 616. 1908.
Winkelmann,N.W. A.N.and P. 28. 150. July.1932,
Winkler. Manuel de Neurologie. Haarlem. 1918. 1. 160.

6

ABBREVIATIONS.

A.f.P.u.N.	Archiv für Psychiatrie und Nervenkrankheiten.
D.Z.f.N.	Deutsche Zeitschrift für Nervenheilkunde.
R.N.	Revue neurologique.
M.f.P.N.	Monatschrift für Psychiatrie und Neurologie.
J.N.M.D.	Jnl.of Nervous and Mental Diseases.
Z.f.kl.M.	Zeitschrift für klinische Medizin.
A.N.P.	Archives of Neurology and Psychiatry.
N.Z.	Neurologisches Zentralblatt.
Proc.R.S.M.	Proceedings of the Royal Society of Medicine.
Z.f.d.g.N.u.P.	Zeitschrift für die gesamte Neur.und Psychiatrie.