DISSEMINATED SCLEROSIS IN GENERAL PRACTICE WITH OBSERVATIONS ON ITS DOMESTIC AND ECONOMIC MANAGEMENT.

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Thesis submitted for the Degree of M.D. (Glasgow University.).

by,

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DISSEMINATED SCLEROSIS IN GENERAL PRACTICE
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Introduction:

When I was a boy at school, I had an uncle, a favourite uncle, who was paralysed and used an invalid chair. It gave me great joy to visit him, and push his carriage along the country road near his home. It brought me much sadness, and also much thought, as to why such a fine man, so jovial and apparently unconcerned about his physical weakness, should not recover and be like other people.

After he died, I was told that he had suffered from a disease called Disseminated Sclerosis which no doctor could cure. I found this difficult to understand, but like any ordinary youth with multitudinous interests and pursuits, the favourite uncle and his incurable malady were soon forgotten.

My interest and curiosity however, were soon to be revived, when as a student in the wards, I again encountered this gross injustice to the human body. Its secrets were

gradually disclosed, and its multiple forms revealed.

With a fuller knowledge obtained from medical training and an appetite for further information concerning this disease, I have gathered to-gether my personal observations of the past twelve years, and beg to present them now in thesis form. I have been in general practice for ten years, and so can not claim originality in the Historical and Histological sections of this thesis. I have also had to depend on hospital and local authority laboratories for certain tests and analyses. By careful reading of recent literature, I have formed a consensus of opinion in these departments.In other sections, I have given my personal observations from cases which have been under my care, or which have been examined by me. I have discussed my findings with regard to my agreement or otherwise with current opinion. The importance of early diagnosis with related signs, and of sensory disturbances, has received my special attention. Further, I have related my experience of the care of sufferers from Disseminated Sclerosis in their homes: I have discussed their social and economic problems, insofar as they might interest and be useful to a young practitioner or student.

Fourteen cases are described in detail, and these are fairly representative of the condition. Forty others are presented in tabular form. Twenty-two cases

are culled from case reports, by kind permission of the Medical Superintendent of the Glasgow Corporation Hospitals. None of these cases is included in those described in detail. I have been privileged to examine a few of the remaining cases, during vacation periods and spare time, in other private practices and hospitals.

The series comprises a total of fifty-four cases, which may be a sufficient number to render valid, the statistical and other findings pertaining to this disease.

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Historical Outline.

Some little knowledge of the functions of the spinal cord existed even before the time of Galen, in the third century. In fact the ancients, such as Hippocrates the 'Father of Medicine', used the term "paraplegia" as meaning a paralysis of all parts of the body below the neck. They attributed this solely to haemorrhage in the brain. Galen had a much better understanding than this. He stated that partial paralysis could occur as a result of disease or injury to any special vertebra apart from apoplexy. Much of his information was derived from experiments on monkeys. Further knowledge was obtained by him from ancient Greeks who carried out dissections on the bodies of criminals, and who also carried out vivisections.

The views of Galen held sway for eighteen centuries, and even in 1819, it is found that John Cooke, lecturing in London, depends extensively on the findings and writings of Galen and the Greek school, to sustain his argument. Cooke states at this time, that the most frequent type of paralysis is paraplegia, dependent on a diseased state of the spine.

Fifteen years later, Carsewell, a ?Graduate (a Glasgow student ?first pathological illus. coll. London?) medical student in London, commenced drawing pathological tissues in colour. In 1837, one of these sketches showed superficial coloured plaques placed indiscriminately throughout the cord,

pons and medulla. He described this condition as a "peculiar diseased state of the cord and pons varolii, accompanied by atrophy of the discoloured portions, all of them occupying the medullary substance, which was hard, semi-transparent, and atrophied. It begins with changes on the surface of the white, and extends to the gray substance".

In I840, Cruveilheir described a case, pathologically as well as clinically, as it occured in a woman aged fifty-four years. He called the condition "induration of the cord with paraplegia". Later, he sketched the sclerotic patches in the spinal cord of his patient. This picture is probably the first illustration of the disease on record. Research workers were stimulated to increased endeavour after the publication of Cruveilheir's findings, and attempts were made to differentiate this type of paralysis from other diseases of the cord, notably Tabes and Syringomyelia.

In I849, Frerichs diagnosed clinically, a few examples of spinal sclerosis. These cases were doubtfully received at the time, but ten years later, Valentiner, one of his pupils, published their subsequent histories, with post-mortem reports, and pathological findings. The certainty of the diagnosis was thus established. Frerichs' clinical observations on the disease are interesting. He stated that,(L), the condition is produced gradually, with exacerbations

and remissions; (2), the one side of the body is affected and then the other; (3), paresis of the lower extremities appears early and reaches a high degree; (4), the disturbance of motility outweighs that of sensibility; (5), the chief seat of the disease is in the medulla, with disturbance of the 9th., IOth., and IIth., cranial nerves; (6), there are frequent psychic episodes; (7), sclerosis of the nervous system is more frequent in the young; (8), the general nutrition for a long period, remains undisturbed.

After these findings of Frerichs and Valentiner, I can trace nothing notable in the history of this disease, for the next twenty years. During the sixth and seventh decades of last century however, Charcot, Vulpian and others, studied many cases closely, and concentrated all earlier information. They then published the symptomatology and pathology of the disease very much as we know it to-day. The Charcot triad of signs,- nystagmus, intention tremor, and scanning speech, - is classic. The related immunity of the axis cylinder from the sclerotic process, was also emphasised by him.

In the early years of the present century, dissatisfaction was felt regarding Charcot's picture, and several observers took up the subject in serious effort to obtain further information. They soon added to the facts already known, but kept within the framework outlined by

Charcot. Oppenheim began to lay stress on the sensory lesions which had either been unobserved or neglected. Next, Strumpell described the condition of the reflexes, and made special mention of the disappearance of the abdominal and epigastric responses; and this was followed soon after by Uthoff's detailed descriptions of the eye disturbances, more especially retro-bulbar neuritis and scotomata.

Since then, the process of evolution has been largely on the clinical side, with the addition of factors quite important in diagnosis and symptomatology.

Thus it is seen, that for many centuries, especially during the period of cloistered learning in the Middle Ages, little advance was made. Then quite suddenly in England and in France over a hundred years ago, the glimmering of a recognition of such a disease, began: then followed a dull period of fifteen years or so, during which no new facts were established, but by gradual assimilation and concentration of the facts already known, Disseminated Sclerosis became established as a pathological entity.

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ETIOLOGY:

The exact cause of Disseminated Sclerosis is still unknown, but the majority of workers is agreed that a specific cause does exist. The fresh lesions, as found in the nervous system, are of an inflammatory nature. Whether this inflammation is brought about by bacterial infection, circulating poisons, or other unknown factors, remains a mystery.

Disseminated Sclerosis is essentially a disease of early adult life, the onset being rare before sixteen years of age, although several cases are reported in the literature as commencing much earlier.
My youngest patient was seventeen years of age when symptoms first appeared, and only four cases in my series showed early signs of the disease before the age of twenty years. The disease seldom begins after forty-five years of age. The oldest patient in my series showing symptoms of the disease for the first time, was forty-two years of age. Eighty-five per cent of my cases commenced between the ages of twenty and forty years.
Most writers say that the condition is more prevalent in females, in the ratio of 5:2, but in my series, 57% were males, and 43% were females.

Occupation seems to play little part in the etiology. I have made careful observations in this matter, and find that 46% of the cases examined, were engaged in light manual work,

(L, in tables), 28% had sedentary occupations,(S, in tables), while 22% only did heavy work, (H, in tables). The remaining patients were not in any employment. Thus it may be deduced, that the disease is not so common among heavy manual labourers, as it is among those doing lighter work.

The geographical distribution of the disease shows it to be more common in some countries than in others. So far, I have not had the opportunity to study the condition abroad, but from the literature, I find that Britain stands mid-way between Switzerland, where the incidence is the highest of all countries, and America, where the incidence is about one-third the British rate.

Disseminated Sclerosis is found to occur with about equal frequency among town and country dwellers, although agricultural workers appear to have a relative immunity. People of all social grades are equally affected.

It was suggested by Pierre Marie, the Parisian physician, that this disease might be a sequel to various infectious diseases. Typhoid fever, Malaria, Influenza, acute rheumatism, Diphtheria, Measles, and Scarletina have all been considered in this direction. In my own cases, only six had a history of infectious disease occuring within two years of the onset of symptoms of Disseminated Sclerosis. Of these, three suffered from Diphtheria, two from Scarlet Fever, and one

from Gonorrhoea. This latter case, No. 45 in the series,contracted the disease eight years previous to the onset of nervous symptoms. He was properly treated at the time, and showed no signs of late sequelae when I examined him. In none of these cases therefore, is there any justification for relating the one disease to the other, and their occurence must be regarded as purely accidental. Chronic tonsillar and dental infection, although frequently present among my cases, must be similarly considered and dismissed.

Oppenheim suggested that certain mineral substances might originate this disease, especially metallic substances like Lead, Arsenic, Tin, and Manganese. Other poisons have also been incriminated, and of these, the most discussed were Alcohol and Carbon Monoxide. Heat-stroke, lightning, and exposure to cold and wet, have also been named as causes. None of my cases presented any evidence of the above factors, as contributory causes of their illness.

Twelve cases had a history of more or less severe injury, sustained at variable periods before the onset of nervous symptoms. In view of the fact that this represents 22% of the series, I feel bound to emphasise Injury as at least an important exacerbating factor.

Pregnancy and parturition must also be considered as exacerbating or provoking factors, as exemplified in Case II of the series.

Heredity and Syphilis are not causative factors, and I am in complete agreement with this finding. During the course of my investigations, however, I have frequently noticed that one or other of the parents of a sufferer from this disease, has been abnormally excitable and nervous. Neurotic or excitable parents should, in my opinion, be included among the predisposing factors.

In recent years, several theories have been propounded, and numerous experiments carried out, with a view to determining the cause of Disseminated Sclerosis. None of these has been universally accepted as correct. Tracy J. Putnam of Boston recently suggested that minute venous thromboses may be the starting point of the characteristic lesions. He thought that clotting was brought about through the presence of abnormal ferments in the blood. Argument against this theory seems to be justifiable, since there is no apparent reason why thrombosis should not occur in other venules throughout the body. Further, there still remains to be explained away, the cause of the altered coagulability of the blood.

Experiment and research in various parts of the world, has led to the discovery of a lipolytic enzyme, named Esterase, in the blood serum of patients. This factor is thought by some, to bring about the destruction of the myelin sheath. It is present in the blood serum in greater quantity during remissions. During the acute phases, there is less of it in the blood, owing to its greater demand for the destructive process. This abnormal lipolytic activity may not

be of primary importance in the etiology of the disease: it may be simply an accompanying factor of the condition, or a by-product of some other process.

Experiments by Weil have resulted in the discovery of a heat resisting, myelinolytic agent in the urine of some patients, and Karaday has demonstrated changes in the lipases of the duodenum.

In spite of these theories and findings, the cause of this disease is far from being satisfactorily explained. I think it possible that Esterase may be active on the blood cells as well as on the myelin sheaths. In this way, Putnam's condition of thrombosis, may be brought about by the proximity of the affected venules to the nerve tissue. This is entirely a matter for conjecture, and the chances are that a much more simple explanation will be forthcoming before long. From my own observations and experience of the disease, I have formed the opinion that severe trauma, either physical or psychical, sustained by a young person of nervous temperament, may be sufficient to precipitate the condition. Further, in the continued absence of injury or mental shock, I think that the disease will fail to materialise in the majority of individuals so predisposed.

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PATHOLOGY.

Disseminated Sclerosis is characterised by the presence of grey sclerotic patches throughout the central nervous system. These patches are scattered irregularly throughout the spinal cord and brain. In the latter, they occur more commonly in the lower parts, namely the Medulla, Pons, Crura, and Thalami, and rarely in the upper part of the hemispheres. They are sometimes present in the cranial nerves and in the roots of the spinal nerves. These sclerotic patches were at first thought to be due to glial overgrowth, but the disease is now regarded as having its primary focus in the myelin sheath. Most pathologists grant that the changes are of an inflammatory or toxi-infective nature, but the evidence for this is purely of histological origin. Possible fallacies may occur in basing arguments for the infectious nature of any process, on the microscopical appearances alone. Steiner has described a spirochaete present in fresh lesions, and later, a virus has been considered responsible, but there is no consensus of opinion or good evidence available to support either of these agents.

In the majority of cases running a prolonged course, sclerotic patches predominate in the nervous tissues In cases where the disease runs a rapid course, terminating fatally in a few months, all the focal areas are found to be acute, with little or no glial overgrowth. Sometimes the course

of a chronic case is brought to an end by an acute exacerbation, and post-mortem examination reveals numerous recent plaques. On examination of sections of the brain and spinal cord, many grey, translucent areas of varying size and shape can be seen. These have a firmer consistency than the normal brain tissue, and offer an increased resistance to section. Sometimes a plaque may be found which appears rather yellowish in colour and of a softer consistency. These occur in acute, fulminating cases, or in patients who have died during an acute phase of the disease. These different naked eye appearances, the grey translucent patch, and the soft yellowish patch, are found histologically to represent the extremes of the diseased process. By means of these extremes, the story of the disease can be reconstructed.
In Weigert-Pal preparations, the soft yellow plaque is found to have lost its myelin sheaths: these are found broken up and partially destroyed by phagocytes. The latter are seen to be free in the damaged tissue, and to have a tendency to gather in the perivascular space. Here, also, may be found a few mononuclear cells, lymphocytes and plasma cells. The axis-cylinders are also involved in the destructive process, but only to a limited degree. Most of them are swollen and tortuous, but only a few are seen to be broken. The blood vessels are normal, and show no evidence of their involvement in a toxic process.

The Histological findings at this stage, therefore, show that

the disease process is predominantly one of destruction of the myelin sheaths in scattered foci. These foci are scattered in rather haphazard fashion, throughout the brain and cord. The damage to the axis-cylinders in the acute patch, is rarely so severe as to be irreparable, though the changes found probably indicate a complete loss of their function of conduction of the nervous impulse consistent with the increase in symptoms during a relapse.

A different picture presents itself when the grey, translucent plaque is examined histologically. In Weigert-Pal preparations, the plaque of demyelinisation is found to be sharply limited and in contrast to the surrounding normal tissue. The degenerated myelin is seen to become completely destroyed. In the cord, the glial fibres are arranged parallel to the surviving axis-cylinders. The findings with axis-cylinder stains are somewhat variable. In some plaques these structures are seen to have returned to normal. In others they are greatly reduced or absent. This tendency to preservation of the axis-cylinders in the lesions, accounts for the fact that ascending and descending degenerations are not a prominent feature of the disease process in the spinal cord. However, in cases which have run a long course, where the longer tracts of the cord are subjected to involvement in plaques at several levels, there is generally quite a marked amount of ascending and descending degeneration. In such gliosed plaques, the mononuclear reaction in the peri-

vascular spaces is generally found to have disappeared. The vessels themselves are often somewhat thickened and hyaline, which is rather surprising, in view of the fact that they show no appreciable signs of damage in the more acute stages. Between these two extremes, the acute destructive lesion and the completed stage of glial repair, plaques showing various intermediate stages can usually be found. Plaques are found to involve both grey and white matter, but it is surprising how little change can be detected in the ganglion cells, even when embedded in a dense glial scar.[1.]

The other viscera show no lesions which can be related to the process in the nervous tissues.

The cerebrospinal fluid is normal in over 50% of cases. In others there is sometimes a slight increase in the number of mononuclears. Chemical analysis shows that the chlorides and glucose are normal, while the protein is normal or very slightly increased. In acute stages or phases of the disease, a moderate pleocytosis may be found. This increased cell count probably indicates the formation of recent sclerotic patches. The only change of diagnostic value, is the presence of a colloidal gold curve of the paretic or luetic type. The paretic Lange reaction was present in 40% of my cases in which the test was carried out. The presence of a paretic curve, in the absence of a positive Wassermann in the blood and spinal fluid, is certainly of diagnostic value.

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SIGNS and SYMPTOMS.

The description of the disease process which I have just given will serve to indicate the extensive variability of signs and symptoms possible in Disseminated Sclerosis. Healing of older plaques, combined with the occurence of fresh lesions, provide an ever changing picture in the same case. Some patients may show certain signs and symptoms, perhaps only a few, over long periods. Other patients may show new additional signs or symptoms, from week to week or from month to month. It is found that only about I5% of cases exhibit a typical symptom syndrome. In this ordinary, most prevalent type, there is a definite clinical picture, constant within certain limits. In this type of case, the patient probably complains of tiredness and weakness in one limb, more likely a lower limb, with unsteadiness when walking, and stiffness in the muscles. The leg, arm, and face muscles may be affected separately or to-gether. Isolated or groups of symptoms may be found all over the body, depending on, and in proportion to,the location and dissemination of the plaques.

An acute form of the disease is said to occur, commencing with headache, vertigo, generalised pain, and gastro-intestinal disturbance. Within a few days typical nerve symptoms appear, and death occurs in a few weeks. I have never seen such a case, and I am inclined to think it more related to epidemic encephalitis than to disseminated sclerosis.

The more common chronic forms may be conveniently classified, according to the predominant symptoms. In such cases there is no associated constitutional upset, the disease showing itself in so unobtrusive a manner as to cause the patient at first to belittle his complaint. He often merely states that his leg or legs feel tired, or that he has a useless sort of feeling in an arm. Frequently the patient may complain of sclaffing a foot when walking, or that he finds himself tripping or stumbling easily. Climbing stairs also proves a difficulty, owing to stiffness of the leg muscles. Bladder disturbances often accompany this type of case, the patient complaining of having to strain to pass water, or that he feels that the bladder is not empty at the end of the act. Within a short time such a patient is likely to show rigidity of the muscles, increased knee-jerks, clonus, and an extensor plantar reflex. Abnormal sensations, such as feelings of 'pins and needles', heats and cold, or even pain often accompany the motor nerve disturbances, as may be seen from my case histories. The usual picture, therefore,is that of spastic paraplegia, with bladder disturbance. Further, a history of injury, as a fall, after jumping, or severe physical or mental strain is frequently obtained. Double vision or dimness of vision is sometimes the first complaint, or may be found to have occured at some previous date. Other symptoms may be first to appear as seen in my case analysis, such as trembling of the head, arms or legs, dizziness, or involuntary laughing or crying. Thus we find

that either the brain or spinal cord may be the first part of the nervous system affected by the disease, and that the location of the plaques determines the nature of the signs and symptoms. Cases presenting isolated cerebral symptoms are less common than those with spinal symptoms. In the former, optic atrophies with scotomata, ocular paralysis, and transitory diplopia are often prominent. Muscular weaknesses with headache, nausea and vertigo, are usual accompaniments of the cerebral type of case. Rarely, other cranial nerve lesions may cause initial symptoms, and mild apoplectiform or epileptiform seizures infrequently precede other disturbances.

Seven unusual forms of this disease are mentioned in the literature,[2.] each of which might be confused with other conditions. I have not, so far, had the experience of any such types in practice. They may be summarised as follows:-

a. Cases with prominent mental symptoms, with the picture of a slowly advancing dementia.
b. Forms resembling brain tumour.
c. Hemiplegic forms due to lesions in the cerebral course of the pyramidal tracts.
d. Forms resembling pontine encephalitis.
e. Cerebellar types like paralysis agitans.
f. Those resembling neurosyphilis.
g. Others simulating transverse myelitis, progressive muscular atrophy, and amyotrophic lateral sclerosis.

I now propose to discuss the individual signs and symptoms of Disseminated Sclerosis in detail, and to give my personal views on these as they have occured in my cases. I think they are best described under the affected systems.

Most common and most prominent of all are the Motor Nerve Disturbances. Of these, loss of power or paralysis is of primary importance, and is the fundamental deficiency of the disease. It was present to a greater or lesser degree, in all of my cases, while over 80% of these patients complained first of all, of weakness in one or other of the four limbs: 90% showed paresis in the lower limbs, 62% showed paresis in the upper limbs, while 37% showed, or had a history of,weakness of the muscles of the face. The limb affected frequently depends on the occupation of the patient, insofar as it entails more use of one limb than another, as for example, the arms of painters and carpenters. Usually, I have found that large groups of muscles are simultaneously affected, especially in the arms and legs, but occasionally small muscle groups show paresis, as in the larynx of singers. The sphincters of the bladder and rectum may also be involved fairly early in the disease.
The tired, useless feeling in a limb, is a very important symptom, and may be the only complaint over a period of several years.
I have found that the paralysis may be monoplegic, hemiplegic, or paraplegic, and is indicative of lesions in the pyramidal tracts.
I have not seen any more than a very slight weakness in the muscles

of the thoracic and abdominal walls.

Inco-ordination of movements or Ataxia is also a motor nerve disturbance frequently present. It may be demonstrated clinically by asking the patient to touch the tip of the nose with the fore-finger of either hand, when the upper limbs are suspect; the lower limbs are tested by asking the patient to place either heel on the opposite patella, or, if he can walk, I often ask him to walk along the edge of a carpet. Sensory ataxia, commonly found in Tabes, differs from this type, insofar as the sense of position is also lost. If pass-pointing be exaggerated by depriving the patient of his sight, then sensory ataxia is present: it only rarely occurs in disseminated sclerosis. Romberg's sign, I regard as particularly useful, for demonstrating slight inco-ordination of the lower limbs, with the patient on tiptoe with his knees bent. 70% of my cases have shown this inco-ordination of movements, and the large majority of these had varying degrees of Rombergism. Ataxia did not appear as a first symptom in any of the cases under discussion.

I have found that Intention Tremor is one of the most striking symptoms in well established cases. Often, however, it is not seen , in early cases. It occured in 74% of these cases, and in Case 43, the first complaint was trembling and shakiness of the legs. It may be absent for several years as in cases 19, 37, and 38. Tremor is increased by effort,

especially if prolonged, and by emotional disturbances, but is not affected by closing the patient's eyes. I have found it, in most instances, to develope gradually, but infrequently it may come on quite quickly: it is often bilateral, related closely to the paresis. The arms are most often involved, then the head, chin, face, and trunk muscles. The legs are less frequently affected. In the trunk, it may cause a rocking motion, and it causes a nodding or swaying of the head. Passive tremors of the hands while at rest are also sometimes seen. The patient's handwriting may undergo gross changes when tremor is marked. I have here appended a characteristic example in the form of the signature of a patient.

The name represented above, is Daniel Duncan.

Intention Tremor is anatomically explained in text books, as being due to the presence of plaques in the cerebro-rubro-thalamic tracts.

Disturbances of gait and station occur in every case showing ataxia and muscular hypertonus of the lower limbs. The gait may be purely spastic, or, where cerebellar symptoms are present, it is spastic-ataxic. A pure ataxic gait is uncommon, while one may be almost sure that a patient will soon be bedridden, when his gait becomes spastic-paretic.

Muscular atrophy, in a strictly pathological sense, only rarely occurs: when it does, the muscles of the hands are involved. I have not seen true atrophy of muscles in disseminated sclerosis, but muscle wasting of a generalised nature in the terminal, cachetic stages, is frequently observed: this is purely nutritional.

In keeping with the spasticity and hypertonus, the tendon reflexes are increased. The upper limbs show increased triceps, biceps, and wrist jerks: the lower limbs show increased adductor, knee, and tendo-Achilles jerks. The knee-jerks were increased on one or both sides in 92% of my series. The jaw-jerk is also exaggerated in over half of these cases. Ankle-clonus is often present, (20%), patellar clonus less so,(I2%). The abdominal reflexes were altered in one way or another in all my cases. Some only showed a diminution of response, others a complete loss: some on one side, others on both sides. 72% showed a complete loss bilaterally. The cremasteric reflex was affected proportionally. An extensor plantar response, or Babinski sign was obtained in 98% of cases, on one or both sides. Like other signs in disseminated sclerosis, the skin reflexes were found to vary somewhat from time to time, but I have never seen them return after showing a complete loss.

The Sensory Phenomena of this disease were among the latest to be recognised, and are thus probably the least understood.

I have found them to be very fluctuating in character, and apt to be overlooked, or wrongly interpreted. Having given them careful consideration in the series of cases under discussion, I wish to emphasise their outstanding features. Typical objective and subjective sensory disturbances are of fairly frequent occurence. In this series, 36% of the patients complained of abnormal sensations at certain periods of their illness, while in four cases, paraesthesiae appeared as practically the first symptom. Some other sign may have been obtainable in these cases, had the opportunity for examination previously arisen.
The occurence of pain in this disease, is of primary importance to the practitioner, since it is to him that the patient will apply, for quick relief. I have found in practice, that a patient with neuralgia of any description, or in any anatomical area, will not complain of his paralysis, poor vision, or any other gross incapacity, while the pain lasts. These deficiencies in the meantime, drift into the background, until relief is obtained. The most severe type occurs, in my opinion, in the presence of a lesion in the posterior root of a spinal nerve. I have seen one case with such a lesion, where severe pain round the costal margin, was comparable to calculus colic. Trigeminal neuralgia infrequently occurs, and may also cause the patient great suffering: IO% of my cases had this symptom at one time or another, during their illness. It must, however, be borne in

mind, that this condition may occur, quite apart from the presence of sclerotic plaques in the tracts of the Fifth nerve. Other segmental pains may occur, mostly in the lower limbs, resembling the shooting pains of Tabes. These are not usually so severe as the types just mentioned above, and seem to be more easily borne. Severe pain in the extremities is uncommon. Arthritic pain more frequently occurs, and the knee-joints are most affected. Case 5 in my series, had both girdle pain and knee-joint pain, and the latter proved a great inconvenience to him since it always seemed to become worse during a remission of his other symptoms. Perhaps the mechanism is the same as that which produces aching in the muscles of an ordinary individual after performing unusual exercises. The knee-joint pain in this instance was of a stabbing character. Lesser degrees of pain are often present in different parts of the body, and may not be subject to complaint by the patient. I have not included these in my per centage rate, since they are to be found, on interrogation, in nearly every case, and take no prominence in the clinical picture. Some loss of feeling is of common occurence, and most commonly affects the soles of the feet and the finger tips. This numbness is frequently accompanied by feelings of pins and needles and prickling sensations, and may appear as a first symptom. I have found it to differ from the paraesthesiae of subacute combined degeneration, in that it is more markedly anaesthetic in nature, and is not so much

confined to the distal portions of the limbs. Tingling feelings are more pronounced following Addison's Anaemia. Response to touch and thermal stimuli is accurate in the majority of cases, but small areas may be found to be deficient, particularly in well established cases. Muscle-sense is also impaired in the later stages of the disease, especially sense of position. Pallaesthesia, or appreciation of the vibrations of a tuning fork placed over bony prominences, is characteristically absent. This is an important factor in the diagnosis of the disease. Loss of vibration sense was elicited at some point in 82% of my series. Other paraesthesiae such as heats and chills, pressure, tightness, pruritus and formication are not so commonly found. Another dysaesthesia observed in one patient, occured when the head was flexed on the chest. Tingling pains resulted from this movement, chiefly across the shoulders, but also extending to a lesser degree, down the arms to the fingers. Return of the head to the normal upright position, caused a disappearance of the sensation. I agree with R.M. Brickner, who states that sensory disturbances are more prevalent in highly neurotic patients, than in those who are more nervously stable.

Speech Disturbances were present in 34% of my cases. There is seldom any alteration in the early stages, but changes are often present when the disease is over two years duration. Scanning

speech, as described by Charcot, occured in only two of my cases in the advanced stages of the disease. Other types of speech disturbance were more common. Exaggerated mouth movements are quite common prior to the speech becoming affected. Most of the cases stuttered a little, or staccatoed their words. It is said that singers soon notice a tiredness appearing in the voice, with increasing difficulty in modulating the tone. This can reasonably be accepted as true. Dysarthria and explosive speech are also sometimes encountered in the terminal stages, often accompanied by inco-ordinated movements of the tongue. Chewing and swallowing, although reported as being rarely affected, were not altered in any of these cases.

Bladder Symptoms are said to be present in 70% to 80% of cases. In this series, only 52% had any upset in this way. The most common complaint among these, was the feeling of insufficiency, or incomplete emptying of the bladder. Others said that they had to press unduly to pass water. Complete paresis with retention was present in two cases. When the bladder was affected, I usually found the action of the bowels also deficient. Over 60% of these patients suffered from this complaint, constipation being much more common than loss of control. Bowel incontinence was present in four cases in the terminal stages.

I have not known menstruation or parturition to be affected in Disseminated Sclerosis.

Cranial Nerve Disturbances occured in over 70% of my cases, and

were usually of a transient nature.

Disturbance of the Olfactory nerves is of rare occurence. Only Case 5 in my series showed hallucination of smell. Second Nerve lesions are common, and were present in 68% of these cases. They appeared mostly as partial optic atrophies in the form of simple atrophic pallor on the temporal side of the discs: 20% of patients with Optic nerve lesions had dimness of vision, with fine optic atrophies or scotomata. Blindness, partial or complete, coming in fleeting attacks, is sometimes the initial symptom, as in Case 22, and Case 37. Oscillopsia, or oscillating vision, was found to be fairly common when nystagmus was pronounced. In the presence of this symptom, the patient complains of seeing objects, which he knows to be stationary, move backwards and forwards, or waggling from side to side. Oscillopsia usually occurs while the patient is walking, but may also occur while resting if the gaze is fixed. Nystagmus is the most characteristic eye disturbance in this disease, and was seen in 80% of my cases. Its discovery as a symptom of brain sclerosis was first pointed out by Valentiner about the year I856, but its diagnostic value was then over-estimated. True central nystagmus is not common, but nystagmoid movements on lateral motion of the eye, occur at the above rate in established cases. In cases with marked cerebellar lesions, the movements of the eyeball are rotatory in character.

These eye movements may be classified and summarised as follows:-

(a) Continued rhythmical oscillations, or true central nystagmus, analagous to the continuous movements of the head and body;

(b) Rhythmic oscillations set up on movement of the bulb in any direction, analagous to the intention tremor of the hands;

(c) Nystagmoid movements on extreme lateral or vertical movements, analagous to fatigue movements;

(d) Ataxic movements.

Of these, (c) is by far the most common.

Third Nerve Lesions in the form of ocular muscle paralysis, are fairly common, may be transitory or persisting, and are of diagnostic importance: 30% of this series had a history of such a lesion. Paralysis of convergence is the most frequent form, and double vision as a symptom, frequently occurs in early cases. Ptosis was less frequently found, and when present was one sided and incomplete. Fourth nerve paralysis was observed in Case II. Involvement of the motor branches of the Trigeminal was not seen, and only rarely occurs, but facial neuralgia had been present at one time or another in seven cases. Abducent paralysis was well marked in one case at examination, with apparent internal strabismus in the normal eye.

Weakness of the face muscles was fairly often encountered, over 30% of the patients showing this

symptom. Facial paralysis in Disseminated Sclerosis , I have found to be of a strikingly fluctuating character, and usually one sided.

Hearing is sometimes temporarily affected, as in Cases 4I and 46, and the Vestibular nerve is even more often affected, especially in its cerebellar connections: this may explain some of the more severe nystagmus. Vertigo is a prominent symptom in this disease, and was present in 58% of these cases. It occurs through involvement of the Vestibular nerve and other sensory tracts. Rotatory giddiness, with a tendency to fall, resembling Meniere's syndromy, has been said to occur in Disseminated Sclerosi but I have not experienced this type or degree of vertigo. Vagus or pneumogastric hypertonus may take the form of hyper-acidity or tachycardia, both of which were complained of in one case. Taste was unaffected in this series, but deviation of the tongue to one side, indicative of Hypoglossal paralysis, was observed in one case also.

Several other symptoms of this disease occur from time to time, but are neither common nor important. Most interesting perhaps is involuntary crying and explosive laughter, both due to brain disturbance in the cortico-bulbo-cerebellar reflex paths. One patient showed the laughing symptom, and another made the facial grimaces of weeping, without tears. Mental symptoms and associated psychiatric phenomena, are neither

common nor prominent. I had few examples of these disturbances, and these only occured in the terminal stages of the disease. They took the form of mild intellectual reduction or dementia, with mental confusion, and difficulty in thinking. Capacity for concentration and memory, were rarely seen.

Mild apoplectiform and epileptiform attacks, with transient disturbances of consciousness, are said to occur in about 20% of cases, but neither of these symptoms occured in my experience. Vasomotor and trophic signs, such as angiospasm with cyanosis, vessel spasms with oedema, and syringomyelia-like lesions, only very rarely occur.
Hypertrichosis occured chiefly over the back, in one case in the late stages. This is probably caused by the presence of a plaque in the hypophysis, producing secondary changes in the suprarenal functions. Dermographia was present in a few cases, over small isolated areas only, and bed-sores and other skin ulceration was only present for short periods before death.

Laboratory Analysis of blood and cerebrospinal fluid was carried out in the majority of these cases, but nothing peculiar to the condition resulted. The fluid was obtained by lumbar puncture, and in each case was clear and colourless, and not excreted under any pressure. The cell count of the C.S.F. never exceeded five. Taking the high normal limit for Total Protein at 40 mgms. per 100 c.c., I find that fully half of the specimens examined,

showed a protein increase of a slight degree. The highest was 80 mgms., per cent, and the lowest 22mgms. per cent. Globulin was absent in every case but three, and these gave only an indistinct deposit, in contra-distinction to the dense deposit commonly seen in G.P.I. The Wassermann reaction was negative in every case, both for blood and spinal fluid. Lang's Colloidal Gold Curve was of the mild paretic type in over 50% of the specimens examined: a few were more of the luetic variety, while the remainder gave negative reactions. The highest Chloride determination was 746, and the lowest 662. Normal reduction of Fehling's solution was obtained in all cases, but I could not obtain quantitative figures.
No characteristic abnormalities were found in the blood of any of the cases examined.

The Diagnosis of Disseminated Sclerosis may be a comparatively simple matter, if the Charcot triad or symptoms closely approaching it ,be present. But this combination of symptoms is found only in a small proportion of cases, and then only when the disease is established. On the other hand, great difficulty in making a diagnosis might arise, for example in a case without multiple foci, ushered in by apoplectiform attacks. Such an occurence is certainly rare, and I have not yet seen one, but in view of the multitudinous forms which this disease may assume, these unusual beginnings must ever be borne in mind. Speaking

generally, I would say that the presence of disseminated sclerosis should be suspected in all cases of non-syphilitic adults, between twenty and forty-five years of age, who show multiple lesions of the central nervous system, with remissions.

Because of the widespread dissemination of the lesions in this disease, there is scarcely a combination of symptoms which can not occur. From the family doctor's point of view, the commonest early manifestations of the disease are of greatest importance. An early symptom may be very easily overlooked or belittled, and with its subsequent disappearance, as is typical of the condition, the possibility of organic disease of the nervous system being present, may be neglected. Such an oversight may have unfortunate repercussions, for, when the second attack comes along in one or two years time, the first symptoms may be recalled by the patient or his relatives. Another opinion may be sought, and the true nature of the condition revealed. This does not reflect credit on the practitioner who failed to recognise the first fleeting signs of the disease, and who contented himself with an apparent disappearance and cure. The onus, therefore, lies with the family doctor in the ordinary pursuit of his many duties, to keep a sharp look out for certain early manifestations of the disease, not only for the benefaction of his patient, but also for the maintainance of his own good reputation. I have found myself in the past, sitting

on both sides of the fence, if I may put it that way, and must state that it is much more pleasant to be in the right than in the wrong.

What then, are the early signs and symptoms of value in arriving at the diagnosis? Further, how are they to be recognised as belonging to this disease?

From my experience, I have formed the opinion, that acute retro-bulbar neuritis, with temporal pallor of the optic disc and transitory changes of vision, is the most common early sign. The recognition of this sign only involves the use of the ophthalmoscope by the direct method, and the observing of paleness of the disc as compared to normal. Secondly, loss of strength in a limb is an important early symptom, and is present early in about 80% of all cases. Thirdly, vertigo, although not so commonly present, is of value if occuring early. Fourthly, and if accompanied by muscular weakness in a limb, loss of sensation to the vibrations of a low pitched tuning fork placed over the bony prominences, is almost diagnostic of early disseminated sclerosis. One or more of these signs may appear very early, and be unaccompanied by other signs for long periods. Later, the onset of muscular hypertonus with increased reflexes in the affected limb, diminution or loss of the abdominal reflexes, ataxia, speech disturbances, bladder upset, and nystagmus, are diagnostic of the condition.

The most outstanding feature of the disease is the peculiar advancing and receding course which it pursues, so constituting the remissions. During these quiescent periods, the entire clinical picture becomes changed, so that a patient may move about in almost a normal manner, apparently fit and well. Because of the scattered plaques throughout the nervous system, many symptoms arise, and thus other organic and functional diseases may be simulated. The following are the most important of these disorders, which may require careful differentiation:-

Hysteria, Neuro-syphilis, Amyotrophic lateral sclerosis, Encephalitis lethargica, Tumour of the brain, Chronic hydrocephalus, Arteriosclerosis, Syringomyelia, Spinal cord tumour, Paralysis agitans, Friedreich's ataxia, Other familial ataxias, Subacute combined degeneration, Meniere's disease, Chronic metallic poisoning, especially lead and mercury.

The chief pitfall in diagnosis occurs in cases of Hysteria, which mostly occurs in young adult women. In this condition there occurs an almost daily fluctuation of symptoms, most of which are of a sensory nature. Euphorism is a recognised concomitant in the majority of disseminated sclerosis cases, and through this, optimism on the part of the doctor often enables the patient to obtain better control of his weaknesses. This results in a similarity to the changeable symptoms of hysteria being established.

Frequently during the routine work of my practice, hysterical symptoms, not accompanied by signs of organic nervous disease, crop up. These are usually readily amenable to treatment. It is sufficient to remember that in such conditions, the abdominal reflexes are unaltered, and there are no pathological reflexes like Babinski's sign, optical changes, or nystagmus.

In any case, it is of paramount importance to differentiate from syphilis of the nervous system, which like disseminated sclerosis, may occur in middle life. Neurosyphilis however is more common in men over forty years of age. In both diseases we may be confronted by patients who have, in the course of a few months or possibly a few years, developed from slight beginnings, a spastic weakness of the extremities with exaggerated deep reflexes. In both, remissions and exacerbations may occur. Headache, giddiness, and mental deteriorations, with transient cranial nerve paralysis, give presumptive evidence of syphilis. If a history of a syphilitic lesion, and a positive Wassermann reaction in the blood and spinal fluid is obtained, the diagnosis is certain. I agree with the general opinion, that no matter how closely the two groups approach each other, there always remains a few differential signs of much significance and importance. My experience of neurosyphilis is somewhat confined, but sufficient for me to state that in disseminated sclerosis, the reactions of the pupils are almost constantly normal, whereas in neurosyphilis the pupils

are almost universally fixed. Irregularity in the contour of the pupils also, gives corroborative evidence of the presence of syphilis. The eye muscle paralysis in syphilis are of early appearance and usually complete: in disseminated sclerosis, they are only partial and incomplete, and of a transitory nature. Disseminated sclerosis gives almost constantly negative serological findings, thus further differing from syphilis. There is one other symptom to which I attach much significance, and that is that the spasticity in syphilis is more intensive than that of disseminated sclerosis, unless in very advanced stages of the latter. In the early stages of disseminated sclerosis, there is obvious muscular weakness, and usually only a slight degree of spasticity, while in syphilis there is marked spasticity, and relatively less weakness. The mental symptoms in disseminated sclerosis are not prominent, and never reach the intensity to suggest general paralysis, where dementia with delusions of grandeur are commonly present. Also, the tremor of general paralysis is neither constant nor of the intention type. The speech in G.P.I. is more slurring and tremulous, and the repetition of spoken words is difficult. In tabes dorsalis, the pains in the extremities and trunk, with other marked sensory disturbances, absence of knee-jerks, and primary optic atrophy, are distinguishing features.

Paralysis agitans occurs more commonly

in men, and is seldom seen in individuals under forty years of age. The tremor found in this disease may resemble that of disseminated sclerosis, and it has been reported in the literature that several cases of the latter have shown a festinant gait. I have at present four cases of paralysis agitans in my practice, three men and one woman, and from the clinical picture presented by these cases, I am of the opinion that, the age of the patient, the rigidity and fixity of the face muscles, and the absence of ocular changes, are sufficient for the purpose of differential diagnosis.

In Amyotrophic lateral sclerosis, which affects the sexes equally, there are no sensory disturbances, intention tremor or nystagmus.

Encephalitis lethargica also affects the sexes equally, and may occur at any age. Somnolence or drowsiness, is usually the most prominent sign, and is adequate for differential purposes.

I have had three cases of Meniere's disease in my practice during the past few years. They were all adult males. Nystagmus, which was present in two of these cases, resembled that of disseminated sclerosis, but the sudden onset, intense giddiness, tinnitus, and gastric upset, were sufficient for the purpose of differential diagnosis.

Friedreich's ataxia, a family disease, comes on about the age of puberty, and affects brothers and sisters alike. Cerebellar ataxia is also hereditary

and familial, but does not commence till after puberty, and sometimes late in life. Both of these conditions are rare, and their course is steadily progressive, with no remissions: in the former, scoliosis and pes cavus often accompany the nervous disorder. Multiple neoplastic lesions, and scattered areas of cerebral softening, may cause a little similarity to disseminated sclerosis, but these are usually found in elderly people. Increased intra-cranial pressure, or intra-spinal pressure, with evidence of primary tumour elsewhere, will make the diagnosis clear in the first instance. In the second place, there will be signs of hyperpiesis and arterio-sclerosis. In Syringomyelia, which chiefly affects males under thirty years of age, skeletal deformities such as kyphosis and scoliosis,asymmetry of the skull, and occasionally spina bifida, are present. Subacute combined degeneration of the cord, a late sequel of Addison's anaemia, is seen more frequently in general practice than the above mentioned conditions. Ataxic-spastic paraplegia, with paraesthesiae of the fingers, may closely simulate disseminated sclerosis. Blood examination, however, always leads to the correct diagnosis. In isolated cases, like one female case of which I had charge,the paraplegic symptoms may precede any suspicion or evidence of blood disease.

In cases of chronic metallic poisoning mistakes in diagnosis are unlikely to occur, if a careful

history of the illness is obtained. In plumbism, or chronic lead poisoning, a history of lead colic some weeks or months before, may often be obtained. The so called lead palsy, affecting the extensor muscles of the wrists and fingers, is the commonest nerve lesion: this results in wrist-drop, which may be accompanied by a progressive wasting of the small muscles of the hands. In less characteristic cases, tremors, cramps, and shooting pains in the limbs may also be present. In chronic mercurial poisoning, the diagnosis may be made from the presence of mercury in the urine.

In the differential diagnosis of disseminated sclerosis, therefore, it is my opinion that neurosyphilis and the sclerosis in the cord following Addison's anaemia, present the greatest difficulty. All the other conditions I have discussed, have one or more outstanding or typical symptom or sign. It is interesting to note, at this point, that Sachs and Steiner[3.] have devised a complement fixation test for disseminated sclerosis, comparable to the Wassermann test for syphilis. If this test becomes reliable, the diagnosis of disseminated sclerosis will be greatly facilitated. I cannot, so far , obtain any good corroborative evidence of the validity of this test.

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PROGNOSIS.

In my experience in practice, there are few diseases presenting so many difficulties in prognosis as disseminated sclerosis. I have already discussed the repercussions of a faulty diagnosis in the earliest days of the illness: I feel justified in adopting the same attitude towards the prognosis. In hospital and consulting practice, the same difficulties do not arise, as those which confront the family doctor. It is a matter for great conflict of mind, as to whether it is better to tell a parent of the hopelessness of condition as it may affect one of his family, or to withhold such information, in the hope that a long remission of symptoms may follow the initial attack. I have found it best to be rather optimistic than otherwise, in view of the fact that many of these patients continue to perform their usual duties, for several years after the onset of the disease. At the same time, one must use some restraint as to the ultimate outlook of such cases. I have found it best to qualify the hopeful opinion, by warning the most mentally stable relative, of possible recurrence and progression of the condition.
It is my experience that a large majority of cases are more or less progressive, and that many of them die within ten years of the onset of symptoms. Other cases, which have enjoyed long remissions, may run a course of twenty years or more. In more acutely progressive cases, death may supervene in from six months

to two years after the first symptom. Remissions have been known to last almost a lifetime, and I have seen periods of three, four and five years elapse, before a second exacerbation occured. When patients have such long periods free from symptoms, the disease seems to have been cured: but since healing does not take place without fresh lesions appearing, no patient is ever totally free of signs of the disease, once it has commenced. The disease will always make a reappearance if the patient lives and escapes other ailments. Not only is the course of the disease notoriously variable, making prognosis almost an individual problem, but also, the data upon which a definite prognosis could be made, are not available. No single observer has the opportunity of watching a large number of cases, from beginning to end of their illness. Estimates of the duration of the illness, depend entirely on the care taken with the history of the case. The patient may often fail to mention or recognise early symptoms, unless specific enquiry is made for them. Temporary dimness of vision, or diplopia occuring some years ago, is not associated by him with the weakness of his legs, of which he is now complaining. The duration of the illness from the time the patient first comes under observation, is of interest, although it yields only limited information about the total duration of the disease. In my tables, it may be seen that the longest duration was I8 years, in a female aged thirty-eight years. The average duration of the

condition before examination varies with the type of practice of the medical man. In hospital practice, Bierly and Dudgeon in I92I, found the average to be four years, while Brain, in consulting practice, found the average from fifty patients, to be five to six years. These latter included eleven who were seen within six months of the onset of symptoms. Seventeen cases had been affected for five years or more. In one case the duration was twenty years, while in another it was twenty-nine years. B.Bramwell made estimations of the duration of the disease by ascertaining the average period which had elapsed between the date of the first symptom recognised by the patient, and the date of death. A group of thirty-six patients, gave an average duration of about eight years, but this figure is below other findings. In general practice, the duration of symptoms before examination varies usually from a few days to a few weeks, but I have found that sometimes the patient does not consult his doctor because of the first early symptoms, but more often it is their second appearance that causes him to seek advice. Because of this, the duration of the disease, even in general practice, may be one year or more. I agree with the general opinion, that ten years may be reasonably accepted as the average duration of the disease in fatal cases.

I have not had experience of the acute form of disseminated sclerosis, so do not propose to discuss it.

Sufficient be it for me to state that such a type does exist, and widespread and rapidly progressive involvement of the nervous system takes place. From available literature on this acute form, I gather that it is ushered in by marked constitutional upset, with failure of speech and swallowing, indicating early involvement of the brainstem. Death is said to occur in from three to six months.

The classical form of the disease, with nystagmus, scanning speech, and intention tremor, with spastic paraplegia, is most often seen in young adults. This type usually progresses fairly rapidly, and incapacitates the patient in three or four years. I find that when spinal symptoms predominate, the progress of the disease process is slow, and that the patient may remain in a stationary pathological state for several years; also, this course is more common in people contracting the disease after thirty years of age. As would naturally be expected, the most benign form is that inwhich long remissions occur, during which the patient enjoys normal health; remissions may last weeks, months, or even years. Thus a remission lasting over seven or more years, may simulate a complete recovery.

The acuteness and severity of the onset, do not necessarily mean a bad prognosis, since I have found in my cases that an acute single lesion is often followed by a good recovery and a long remission. Many subacute

lesions usually indicate a more rapidly progressive course. In general, I find that the younger the patient, the more rapidly progressive is the disease, although there are exceptions to this, since an early onset may sometimes followed by a long remission: relatively acute cases also, may commence in middle age.
Certain extraneous conditions, I have found to provoke acute exacerbations. Most important of these is pregnancy and partur - ition, as exemplified in Case II of this series. Other provoking factors are the infectious diseases, particularly streptococcal infections and influenza, Injuries, which I have already discussed, surgical operations,and teeth extractions.
The terminal stages of the disease are only to be seen in general practice or in Poor Law hospitals. I have seen patients die of this disease under both of these administrations. The late stages are marked by ever increasing disability. Exaggeration of the paralysis, ataxia, and spasticity of the lower limbs ultimately confine the patient to bed. Tremor and ataxia of the upper limbs make it more and more difficult for him to attend to his needs. The speech sometimes becomes so affected by the disease, as to be almost unintelligible, and the knowledge that this is so, may cause great emotional upset to the patient. In spite of advanced optic atrophy, it is unusual for the sight to be severely affected. Occasionally there is mild impairment of the intellect. Death may occur from pneumonia, nephritis or pyelitis, tuberculosis,

self-imposed starvation, or other intercurrent disease.

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TREATMENT.

It is my intention, in this section, to discuss the various forms of treatment employed in this disease, their advantages and disadvantages, partial successes,and failures . I desire further, for the purpose of this thesis, to give my own opinion of their respective values,if carried out in general practice.

It has to be borne in mind at the outset, that the cause of disseminated sclerosis is still unknown. Because of this, any form of treatment used, is bound to be empirical and lacking in rationale. Further, the occurence of spontaneous remissions of symptoms, apart from the effects of therapeutic measures, creates difficulty in estimating the true effect of any treatment used. Still further difficulties arise as a result of the almost daily changes of symptoms which occur naturally throughout the course of the disease.

During acute exacerbations, rest in bed is imperative, and this should include rest of the eyes. In the periods of remission, I find it best to encourage the patient to live a normal life, and to continue his or her employment if at all possible. It is well to advise at all times, the avoidance of

of over-exertion and fatigue. The chief aim in treatment, should be to postpone as long as possible, the ultimately inevitable bed-ridden state. It is my view, that suggestion and encouragement go a long way towards helping this.

It is not good practice to form hard and fast rules or routines as to treatment, as each case must be assessed on its own merits or requirements.
In practice, oral treatment is most easily carried out, and for this reason, Arsenic, given as Fowler's solution, is the most useful drug available. The rationale of this treatment is not only because arsenic can be conveniently given by mouth, but also because of the possible relationship of the disease to a spirochaetal origin. It is best to commence giving Fowler's solution in three minim doses, three times daily: then increase the dose by one minim daily, until fifteen minims are taken thrice daily. Thereafter, the dose is similarly reduced, until the original dose is arrived at. I find it best, after this course is completed, to withhold further treatment for three weeks, and then repeat. This routine may be continued almost indefinitely, and is best suited to patients with single early symptoms. I think that the first remission is definitely prolonged, when this method of treatment is commenced early, and continued over one year at least. In certain cases, where a speedy remission is not obtained, the oral treatment may be successfully supplemented by the

intravenous injection of one or other of the arsenical preparations, such as silver salvarsan, sulfarsenol, novarsenobillon, etc. Sulfarsenol is given weekly for four to six weeks, either into the veins or muscles, in doses of 0.45 gm. to a male, and 0.3 gm., to a female. Salvarsan is given intravenously in doses of 0.2 gm., for a male, and 0.I5 gm., for a female, at the same intervals. From my own experience of these preparations, the benefits resulting from their administration are variable. No one of them can claim superiority over another.

Quinine therapy in disseminated sclerosis, has received more support in America and elsewhere, than in Britain. I have tried giving quinine hydrochloride or sulphate in three to five grain doses, thrice daily, during acute exacerbations, and have found it frequently beneficial and efficient in its action.

Liver treatment has also enjoyed periods of popularity, but I think the results obtained are over-rated. The equivalent of twelve ounces of liver should be recommended daily, over an indefinite period. In general practice I find , that even for the treatment of Addison's anaemia, liver is hardly obtainable nowadays, and the cost of concentrated extracts is so high that the ordinary insured patient can not afford to buy them. The most economical way of giving liver in practice, is by weekly injections of Campolon, (Bayer),in 2 cc. ampoules, a box of five

of which can be purchased wholesale, for less than five shillings. But again, the value of this treatment in organic nervous disease is a very doubtful quantity.

4. I have not tried injections of antimony, but Brain states that if given as 'Fuadin', in graded doses, the remissions tend to be prolonged.

A useful mixture which may be given during acute attacks, is potass. iodid; grs. 7½, with liqu. hydrarg. perchlor. I fl. drm., three times daily.

The only remaining therapy falling for discussion from a purely medical point of view, is artificial pyrexia. Various methods are employed in order to induce an artificial fever in organic nervous diseases, and the resulting shock to the nervous system is said to stem the progress of the disease process. The artificial fever is frequently followed by a striking temporary improvement, and I have found it to be most beneficial in progressive cases, in young adults.

T.A.B. and malaria, in sterile suspension, are the vaccines most commonly used to induce fever. Like the arsenical preparations, the results are of equal value. A B.coli vaccine called Pyrifer, is recently receiving more favour. It is put up in graduated ampoules by Yarrow Ltd. of London, and is given intravenously on alternate days. The patient's temperature rises quickly after the injection,

and often reaches 104 degrees F. This may be maintained for three to six hours, after which it gradually falls to normal. It may be purely imaginative, but I feel that the safety of the patient is more secure when the B, coli vaccine is used. Certainly, in practice, one can not take the same liberties and risks, as can be taken with a patient in hospital or nursing home.

Various electrical appliances are now in use for the purpose of inducing fever. Their use, however, are not at the present time, looked upon favourably, since just recently, one patient so treated, was afterwards the subject of a coroner's inquest. I think fear played a big part in this mishap, and would not therefore, advise the use of such apparatus in 'nervous' patients.

A discussion of the treatment of disseminated sclerosis would not be complete, without reference to the surgical efforts at present being made to this end. Acting on Putnam's vascular hypothesis, which I have 5.
already described, Royle in Australia, and T.S.Wetherell in Syracuse have been performing the operation of bi-lateral cervico-dorsal sympathectomy, with a view to improving the ciculation, and decreasing the irritation in the central nervous system. Good results are now being claimed by these men, in several cases. Being also of the belief that a local vascular abnormality is the cause of the disease, Koch and de Savitsch have performed sympathectomy and ganglionectomy by the anterior approach, in

a number of cases. They have described the results obtained from fifteen such operations, in the British Medical Journal, June IIth. I938. It is their opinion that further trial of this therapeutic measure in advanced cases, is desirable.

Finally, with regard to the treatment of individual disturbances in this disease, I find that incontinence of urine is effectively controlled by giving a pill containing a half grain of dry extract of belladonna, three times daily. This is a distressing symptom in practice, and demands efficient treatment. Cascara, with or without enemata will deal effectively with constipation, while if the gait be ataxic, Fraenkel excercises or other suitable re-educational methods, are often beneficial. Pain of any nervous origin peculiar to this disease, is usually controlled by giving any of the barbitone group. I find that Luminal in ½ gr. doses, or Adalin in 5 gr. doses, three times daily, will nullify most of the dysaesthesiae in disseminated sclerosis.

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CLINICAL and DISCUSSION.

I have selected the following fourteen cases, from the thirty-two which I have examined or had charge of, for the purpose of detailed description. Good and reliable histories were obtained from these patients, and although they nearly all have some symptoms in common, each one has one or more symptom, not obtainable in the others. As a whole, they are representative of the disease in its numerous varieties.

Following these descriptive cases, I have included forty others in abstract form.

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Case I.

D.R.; aged 28 years.; male; married; quarryworker.

Complaint:

This man complained of loss of strength in both legs, disturbance of the sight in the right eye, and a prickling feeling in the fingers of the left hand. Sometimes this prickling sensation was replaced by lack of feeling in the fingers. He also complained of difficulty in passing water, and that this was sometimes worse than at other times.

Duration.

It was two years previously that he first noticed weakness in the lower limbs.

Family History:

Both parents were in good health, and two sisters and three brothers were also well. He was the youngest of the family, and apart from the fact that his mother was a 'very nervous woman', there was no history of hereditary disease of any kind.

Personal History:

He lived in a two apartment house in modest circumstances, was an abstainer, and smoked about fifteen cigarettes per day. He had always enjoyed good health until this illness commenced, and at no time had he been exposed to poisonous fumes or gases, or any of the heavy metals. He had never sustained any injuries through accident, nor was there any history of venereal infection. The first idea he had that something was wrong was two years ago, when he noticed a feeling of'pins and needles' in the left lower limb, followed soon after by a weakness in the knees when going up or down stairs. This was accompanied by a temporary blurring of the sight in the right eye, which he did not associate with his other symptoms. To these symptoms was added a tendency to stagger while walking. His doctor told him he was suffering from a nervous trouble which

would probably require some time and treatment before improvement occured. Much to his delight, however, his condition was so much better in four weeks, that he was able to return to work. There remained a slight dizziness, especially when he closed his eyes, and a little weakness in the left leg. Two months later, he again became incapacitated, his disabilities returning even worse than before. At this time also, he was greatly disturbed by a persistent itching of the nose, cheeks, and upper lip. He felt that his memory for long past events was becoming poor, and that his speech was becoming slow, causing him some difficulty in getting out the word he wished to say. His words were inclined to be slurred. He now had little feeling in the left hand, and could not fasten his buttons, or lace his shoes. He noticed that his left hand was more clumsy and awkward than his right. Spectacles were purchased, but the blurring in the right eye persisted. He had no headaches, but slept poorly. He often wakened during the night, and could not fall over to sleep again. He had difficulty in starting the flow of urine, and a feeling that more should come after the flow had ceased. Constipation troubled him a great deal, and he had had enemata on several occasions. He had always maintained a bright outlook on things in general, in spite of the fact that his illness handicapped him considerably. He had been given different medicines by his doctor. In this condition he came into hospital, under my care.

Present State:

On admission, his pulse, temperature and respirations were normal: B.P. I28/80: he was sparely built but fairly well nourished: the lips and mucous membranes were well coloured, and the heart and lungs were healthy: the urine was amber in shade, alkaline, S.G. I0I8, and was negative for albumin, blood, and sugar; it contained a thick deposit, which, on examination, was seen to consist chiefly of pus cells. There were no glandular swellings, and neither the liver nor the spleen were palpable. There was slight nasal obstruction owing to deflection of the septum.

Neurological Examination:

This patient was euphoric,of normal intelligence, and collaborated well in the history of his illness, except in the recalling of remote events. He had no hallucinations or delusions. His speech was slow, with a tendency to scanning, and I noticed that some words were unintentionally spoken louder than others.

There was neither anosmia nor parosmia in spite of the nasal obstruction, but examination of the optic nerve heads by direct ophthalmoscopy, showed pallor of the entire disc on the right side, and on the temoral side of the left. Horizontal nystagmus in both eyes was present, but no eye muscle paralysis was seen. The pupils were equal in size, circular in shape, and

reacted to light and accommodation, directly and consensually. There was some loss of sensation, and abnormal sensations, on the face above the mouth, indicating implication of the first and second divisions of the sensory root of the fifth cranial nerve. The sense of taste on the anterior two-thirds of the tongue was normal. He had no neuralgia, the prickling feeling aforementioned being the only paraesthesia. The seventh nerve function was normal, and in the absence of eye muscle pareses, the vertigo was attributed to a lesion of the vestibular nerve. Ninth, tenth, eleventh and twelfth nerves were tested and found to be normal.

Examination of the motor functions, showed decided weakness in the muscles of the lower limbs, particularly the left, while the trunk and arm muscles appeared to be strong. Inco-ordination of movement in the lower limbs was marked. He could not walk along a straight line, and his efforts in the heel to knee test were poor. The left upper limb showed definite pass-pointing, but the right was almost normal. A positive Romberg sign was also obtained. The lower limb muscles were hypertonic, but there was no wasting.

The paraesthesiae of the left upper and lower extremities was corroborated by examination. There was impaired sense of touch, pain, and temperature, in the fingers of the left hand, and in certain small areas below the

left knee. There was astereognosis of the left hand, and loss of vibration sense, in both legs, and left arm.

Examination of the reflexes gave the following results:-

/ is present and normal; // is exaggerated; - is for absent:

	RIGHT.	LEFT.
Plantars (or)	Ext.	Ext.
Epigastric	-	-
Abdominals	-	-
Cremasteric	-	-
Knee-jerks	//	//
Ankle-jerks	/	//
Adductor-jerks	/	//
Triceps	//	//
Biceps	/	//
Wrist	/	/
Jaw	/	-
Ankle clonus	/	/
Knee clonus	/	/

Deglutition	Normal
Defaecation	Difficulty
Micturition	Difficulty.

Trophic changes were absent, except that the skin appeared glossy, and there were no bone or joint lesions.

Laboratory Findings:

Blood counts, including differential counts were normal, and the blood Wassermann reaction negative. The urine contained abundant pus cells. The C.S.F. was obtained by lumbar puncture, and was ejected as a clear, colourless fluid, without abnormal pressure: the cell count was five, total protein 30 mgms. per cent, chlorides 720 mgms. per 100 c.cm., and a normal reduction of Fehling's solution was obtained. The Wassermann reaction was negative, and the fluid culture was sterile. The colloidal gold curve was not obtained in this case.

Chief Diagnostic Features:

1. Motor weakness of the lower limbs, with Babinski's sign, exaggerated deep reflexes, loss of vibration sense, and paraesthesiae.
2. Disturbances of vision, speech, and bladder.
3. Nystagmus, vertigo, and cranial nerve lesions, particularly optic neuritis.
4. The age of the patient, history of the onset with remission of symptoms.

Treatment:

This was commenced by giving Fowler's solution in three minim doses, combined with protein shock therapy, by means of weekly intramuscular injections of 20 c.cs. of sterilised

milk, a method not now in much favour. Nevertheless, a remission of symptoms followed this treatment, but continued for only about seven weeks. A further and more severe attack came on, and was not successfully treated by artificial fever, for the reason that the temperature could not be raised high enough, to have any beneficial effect afterwards. This man's condition became progressively worse, and contractures of the legs,(paraplegia in flexion) and retention of urine took place. He died after six weeks in this condition, from pyelitis, nephritis, and uraemia. Autopsy was not obtained.

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Case 2.

D.D.; male; 33 years; miner; married.

Complaint:

This man first sought my advice regarding attacks of seeing double. He also complained of pain in the nape of the neck and across the shoulders on bending the head forwards, and stated that on two occasions, once in church, he had laughed out loud without apparent reason.

Duration:

It was only about three weeks previously that he saw double for the first time.

Family History:

His father was killed in a pit accident, ten years previously, and his mother was a chronic invalid, suffering from heart trouble. He had one brother and one sister, both in good health. There was no history of hereditary disease, and no relation was invalid from any cause.

Personal History:

The patient lived in a three apartment house, in moderately good circumstances, and was temperate in his habits with regard to alcohol and tobacco. His previous illnesses were only those common to childhood, and he had never sustained serious injury. He had worked as a miner for more than ten years, and had

always been accustomed to heavy work. There was no history of venereal disease. His present illness began about three weeks ago, when on cycling home from his work, he began to see double, and toppled off his machine. He thought that a particle had gone into his eye, and he continued his journey home on foot. About three hours later, his vision had almost returned to normal, and next day it was quite normal. A few days later, while in church, he commenced laughing for no apparent reason, and could not stop, in spite of protests from his wife. He was taken home by his wife, but the laughing ceased as soonas they had left the church. During the following week, he had one further attack of laughing, while sitting at the fireside with his family. He said he did not know what brought on this fit of laughing, but that he had no inclination to do so while he was busy at his work. Each time it had occurred, he had been sitting quiet, doing nothing at all. At this time also, pain at the back of the neck and over the shoulders into the arms, had appeared. He felt this only when the head was bent forward, as when tying his boot laces, or taking food. On return of the head to the upright position, the pain went away. The persistence of this pain had forced him to seek medicaladvice, the other symptoms being merely mentioned casually during interrogation.

Present State:

No constitutional upset was present: the temperature

pulse and respiration rates were normal: B.P. 138/84: he was tall and thin, but strong physically: the lips and mucosae were well coloured, and the heart and lungs healthy. The urine was amber, acid to litmus, S.G. 1020, and negative for albumin, sugar, blood, and pus. Neither the liver nor the spleen was palpable, and there were no other glandular swellings or changes in the joints. The skin was of smooth texture and elastic.

Neurological Examination:

This man was right-handed, intelligent, had a good memory, and co-operated well in the history of his illness. He slept well without dreams, and had no hallucinations or delusions. He was euphoric, and did not appear unduly upset about his condition. In conversation there was a slight halting in his speech which was not quite normal.

I proceeded to examine his cranial nerve functions, but found only abnormality of the Optic nerve heads. There was pallor of both discs, the right being more pronounced, and chiefly on the temporal side. There was no diplopia at examination, and no eye muscle paralysis. The pupils were circular and equal; they reacted to light and accommodation, directly and consensually. Neither nystagmus nor squint was seen. The motor system was functioning normally, there being no loss of power, inco-ordination of movements, tremor, or muscle wasting in the limbs.

The reflexes responded as follows:-

	Right.	Left.
Plantars	Flex.	Flex.
Epigastric	-	-
Abdominals	-	-
Cremasteric	✓	✓
Knee-jerks	✓	✓
Ankle-jerks	✓	✓
Adductor-jerks	✓	✓
Triceps	✓✓	✓✓
Biceps	✓✓	✓✓
Wrist	✓✓	✓
Jaw	✓	
Ankle-clonus	-	-
Knee-clonus	-	-
Swallowing		
Defaecation	All normal.	
Micturition		

The only disturbance of the sensory system was the dysaesthesia on the back of the neck, across the shoulders, and extending down the arms to the thumb and fore-finger, this corresponding to a lesion implicating the posterior roots of the 3rd. 4th. 5th. and 6th. spinal segments. Further, he could not appreciate the vibrations of a tuning fork, placed over the bones of the arms.

No trophic changes were present.

Laboratory Findings:

Blood counts and films were normal: Hgn. 106: blood Wassermann negative; the C.S.F. was later obtained by lumbar puncture, and was ejected without pressure as a clear, colourless fluid, with no coagulum; the cell count gave no polymorphs, and one lymphocyte per c.mm.; total protein 20 mgms. per 100 c.cm.; chlorides 730 mgms. per 100 c.cm.; normal reduction of Fehling's solution; Wassermann negative; colloidal gold curve mildly paretic; no micro-organisms were found.

Chief Diagnostic Features:

The provisional diagnosis of disseminated sclerosis was based on the following:-

1. Bi-temporal pallor of the Optic discs following retro-bulbar neuritis.
2. Transient diplopia.
3. Loss of abdominal and epigastric reflexes.
4. Increased tendon reflexes in the upper limbs, with loss of vibration sense, and slight hypertonus.
5. Dysaesthesia from cervical segments of the cord.

Treatment:

I commenced treatment of this case with 3 minim doses of Liqu. Arsenicalis, thrice daily, and increased the dose by 2 minims every week, until he was taking 15 minims three times daily. The dose was then diminished weekly by 2 minims, until the

original dose was reached. Arsenic was then stopped, a remission of symptoms having occured. The course was repeated after a rest of three weeks. Six months later a further acute attack occured, with signs of motor weakness and tremor in all four limbs. This time I tried Quin. Hydrochlor. grs. 5, thrice daily for three weeks without benefit, and then gave him a course of T.A.B. vaccine by weekly intravenous injections: I gave 20, 50, I00, 200, 400, and 500 millions over a period of six weeks. A good reaction was obtained after each dose, and the progress of the disease was temporarily delayed.

The history of this case extends from that time , until six years later, when I had to remove him to Poor Law Hospital; ultimately he became paraplegic with gross ataxia, tremor, and bladder incontinence, and was unable to leave his bed. He is still alive in Thrashbush Home, but still bedridden.

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Case 3.

N.W.; aged 25 years.; Female; single; teacher.

Complaint;

This girl has been under my care and observation for over four years. She complained initially of a 'silly' feeling in the left leg, which caused some loss of control of its movements while walking. Sometimes the left foot 'sclaffed' on the pavement. Further complaint of 'prickling' sensations in the same limb was made, and she stated that sometimes stationary objects danced in front of her eyes while walking. After interrogation, she admitted that on two occasions she had seen double, but only for a very short time.

Duration:

It was four weeks previously that she felt the unusual feelings in the left leg, the 'prickling' sensation being the first symptom.

Family History:

Both parents are alive and well. There are two sisters and one brother, all in good health. No history of hereditary disease was obtained, and no relation was suffering from any illness.

Personal History:

This young woman was strong and of the athletic

outdoor type. She played hockey and danced in the winter-time, and was fond of walking, golf and tennis, in the summer. At the age of twelve years, she had scarlet fever, and made an uninterupted recovery, but had not otherwise suffered any serious illness, or injury. Her home circumstances were excellent and her habits exemplary. The present illness began about four weeks previously, with the abnormal feelings in the left lower limb, followed after a week or so, by the lack of power and control in the same limb. At the end of the first week, an attack of double vision came on in the classroom, but this passed off after a few minutes: a second attack occured ten days later, and lasted about the same time. During the third week, she had felt better, but the silly feeling in the leg remained about the same. She was afraid to seek advice on the matter, as she did not wish to be off duty until the end of the term.

Present State:

The pulse and respiration rates were normal, and the temperature was also normal: B.P. II8/70: her general appearance denoted a strong, healthy life: the lips and mucous membranes were well coloured, and the heart and lungs were sound: the urine was acid, amber, S.G. IOI4, and was negative for albumin, sugar, blood and pus: there were no glandular swellings, and the liver and spleen were not palpable.

Neurological Examination:

The patient was right-handed, intelligent,

had a good memory, and slept well without dreams. She was euphoric, and inclined to giggle or laugh nervously while recounting her story, but on the whole, she co-operated very well. The speech was jerky or spasmodic, with the result that her words were often jumbled and difficult to interpret.

I proceeded to examine the functions of the cranial nerves, and recorded two abnormalities. Firstly, external ophthalmoplegia was present on the right side, showing implication of the sixth nerve. This inability to move the right eye outwards, caused an apparent internal strabismus on the left side, with resulting diplopia. Secondly, there was slight ptosis on both sides. Examination of the eyes showed the pupils to be equal, circular, but both a little dilated: they reacted to light, directly and consensually, but gave little reaction to accommodation. There was a fine lateral nystagmus in both eyes, and positive Rombergism.

Examination of the motor nerve functions revealed paresis of the left lower limb. Strength in the right limb was normal. There was marked hypertonicity of muscles in the left leg, and the muscles of the right were suspect. The trunk and upper limb muscles were normal. Inco-ordination of movement was apparent in the left leg, but the heel to knee test was fairly well done on the right side. It was only with great difficulty that she managed to walk along a straight line, the

left foot being swung outwards before being put down rather deliberately, on the floor. There was no ataxia, pass-pointing, or tremor inthe upper limbs.

I then tested the responses of the sensory nerves, and found loss of vibration sense over various bony prominences in all four limbs. Paraesthesiae were most prominent in the left lower limb. The sensibility to pain and thermal stimuli appeared normal, but the sense of touch was impaired in certain small areas below knee-level on the left. The tactile discrimination in these areas, was never less than 3 cms. Sensation of joint movements and postures, and kinetic and stereognostic senses were normal. There were no changes in the bones or joints, and no evidence of trophic disturbances.

Laboratory Findings:

Blood counts and films were normal: Hgn.90%: blood Wassermann negative: The C.S.F. was later obtained by lumbar puncture and sent for analysis: it was clear and colourless, expressed without pressure: cell count nil: total protein 24 mgms. per cent: colloidal gold reaction - 2221110000; Wassermann negative bacterial culture sterile.

State of the Reflexes:

		Right		Left.
Plantars	----------	Flex.	---------	Exten.
Epigastric	---	-		-

	Right		Left.
Abdominals	-		-
Knee jerks	✓		✓✓
Ankle jerks	✓		✓✓
Adductor jerks	✓		✓✓
Triceps	✓		✓
Biceps	✓		✓
Wrist	✓		✓
Jaw		✓	
Ankle clonus	-		✓
Knee clonus	-		✓

Deglutition

Defaecation All normal

Micturition

Menstruation was normal.

Chief Diagnostic Features:

1. Paresis and paraesthesiae of left lower limb.
2. Lesions of the third and sixth cranial nerves.
3. Disturbance of speech, nystagmus, diplopia, absence of syphilis, and the age of the patient.
4. Loss of abdominal reflexes and vibration sense.

Treatment:

I commenced treatment in this case with potass. iodid. grs. 7½, and liqu. hydrarg. perchlor. m. 30, three times daily, for four weeks. This was followed by a course of silver salvarsan intravenously (.I5 gm.) given weekly for six weeks. This course of treatment has been repeated twice during the past three years when symptoms became acute. During remissions, this patient has been almost normal, and has not been incapacitated for work except while receiving injection treatment. The weakness in the lower limb is now more pronounced during an acute attack, and she further complains of numbness in the fingers of both hands.

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Case 4.

D.N.; 26 yrs.; single; motor mechanic.

Complaint:

This young man complained first of all, of some loss of power in the right leg, with stiffness in the muscles of that limb, especially when running or going up stairs.

Duration:

The complaint was only of ten days duration.

Family History:

His mother was alive and well, but his father had died of cancer of the stomach. He had three sisters all alive and well. There was no history of hereditary disease or of any relation in ill health.

Personal History:

He lived in a four apartment house, in good circumstances, and his personal habits were always without fault. His work has never been injurious to his health at any time, and he was never exposed to noxious fumes or gases, or to any of the heavy metals. No history of previous illnesses or injuries of a serious nature, was obtained, nor was there any indication of venereal infection. This was the first time that he had felt anything wrong with him, and he recalled that two weeks before seeing me, he had jumped a ditch in order to retrieve a ball

with which some other mechanics and himself were playing, at the lunch hour. Two or three days after this, his leg began to trouble him, and he blamed the jumping of the ditch.

Present State:

At examination, the temperature, pulse, and respirations were normal; B.P. 124/76; he was strong, looked in robust health, and was well nourished; the lips and mucosae were well coloured, and the heart and lungs were in perfect condition; the urine was free of all abnormalities, S.G.1016; the abdominal organs were normal, and there were no glandular swellings or joint changes. Examination of the ankle joint showed no signs of injury whatever.

Neurological Examination:

I then proceeded to examine the nervous system, and found the patient to be right handed, intelligent, and possessed of a good memory. He co-operated well in the story of his illness, slept well, and had no fears or delusions; the speech was quite normal.

I next examined his cranial nerve functions, and found by direct ophthalmoscopic examination, temporal pallor of the right optic disc, with a normal left disc. There were no other ocular lesions or palsies, and the pupils were circular, equal, and reacted directly and consensually to light and to accommodation. The vision was normal for acuity,

and field. No other cranial nerve disturbance was found. Examination of the motor system showed muscle weakness of the right lower limb, with spasticity, ataxia, and mild muscular tremor. The left leg seemed strong, and normal in every way. The muscles of the trunk, head, and upper limbs were unaffected. No muscle wasting was present.

Response to touch, weight, pain, and temperature was normal, except for an area of hypoaesthesia above the right external malleolus, extending almost to the knee, and an area of paraesthesia on the inner side of the right thigh, where touch with cotton wool was registered as slightly painful. Loss of vibration sense was present in both lower limbs. There were no trophic lesions or bony abnormalities.

The state of the reflexes was as follows:-

	RIGHT	LEFT
Plantars	Ext.	Doubtful.
Epigastric	-	✓
Abdominals	-	-
Cremasteric	✓	✓
Knee jerks	✓✓✓	✓✓
Ankle jerks	✓✓	✓
Adductor jerks	✓✓	✓
Triceps jerks	✓	✓

	RIGHT.		LEFT.
Biceps jerk	✓		✓
Wrist jerk	✓		✓
Jaw		✓	
Ankle clonus	✓		-
Knee clonus	✓		-

Swallowing

Defaecation — All normal.

Micturition

There were no trophic lesions, or changes in the bones or joints.

Laboratory Findings:

Blood counts and films were normal; Hgn. 102% blood Wassermann was negative; the C.S.F. was afterwards taken by the lumbar route, and was found to be clear, colourless, and not under pressure; cell count showed absence of polymorphs, and 5 lymphocytes per c.mm.; total protein 32 mgms. per cent; normal reduction of Fehling's solution; chlorides 750 mgms. per cent; Wassermann negative; culture sterile.

Chief Diagnostic Features:

1. Paresis, ataxia, and tremor of the right lower limb, in an otherwise healthy man of twenty-six years.
2. Extensor plantar response, indicating lesion of the pyramidal tracts.

3. Paraesthesia and hypoaesthesia of the affected limb.

4. Temporal pallor of the right optic disc.

5. After history of remissions.

Treatment:

This man has continued to work since his trouble began four years ago. There have been a few minor exacerbations of symptoms, followed by remissions, but these have never been sufficient to incapacitate him. No further disturbances were recorded during these attacks. I have given him several courses of Fowler's solution, commencing with three minim doses, thrice daily, gradually increasing to I5 m.. This course was followed on each occasion by a rest period of three or four weeks. Since he has been able to carry on with his work all the time, and being his mother's sole support, I have not yet had the opportunity of trying induced pyrexia.

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Case 5.

E.M.; 39 yrs.; male; married; company director.

Complaint:

This man complained of weakness and unsteadiness in the lower limbs, shaking of the hands with numbness and tingling in the fingers, pains in both knee-joints and around the lower border of the ribs, and of having to strain to pass water.

Duration:

He first complained six years previously, of weakness of the legs.

Family History:

Both parents died in old age, and one brother and two sisters older than himself were alive and well. There was no history of hereditary disease, or of venereal infection. No relative was ill from any cause.

Personal History:

His home circumstances were excellent, he was an abstainer, and smoked cigarettes and cigars in moderate numbers. Towards the close of the Great War, he sustained a shrapnel wound in the lumbar region of the back, close to the vertebral column. He was at that time twenty-eight years of age. He enjoyed normal health for about two years after leaving the army. His present illness then began with weakness in the left leg, and pains in the left knee-joint. He had some loss of control

in this limb, but after a few weeks, this weakness passed off. He had been able to continue his work during this period, his duties being mainly secretarial. During the past three years, he had continued his work at home, since his legs had become so bad that he could not walk about alone, without stumbling, and falling; his chief complaint now, was loss of power and numbness in the fingers, which prevented him writing up his books. Contrary to the more prevalent euphorism in this disease, this patient was mentally depressed, and foresaw no betterment of his condition in the future. He had suffered no other illness, operation, or accident, apart from the shrapnel wound in the back: this wound involved only the soft tissues, although of a fairly extensive nature; the vertebral column was not injured.

Present State:

The temperature, pulse and respirations were normal; B.P. I30/76; he was tall, well built, and in a good state of nutrition; the lips and mucosae were well coloured, and the heart and lungs in very good condition; the urine contained no abnormalities, was acid, amber, and S.G. I0I4; the liver and spleen were not palpable; constipation and straining to pass urine were pronounced.

Neurological Examination:

He was right-handed, highly intelligent, and co-operated well in the history of his illness. Mental depression was evident, but this I reckoned as wholly due to his

state of health. His memory was excellent, but sleep was often interrupted by nightmares, with strong emotional fears , usually pertaining to his war experiences. Speech was staccatoed, with exagerrated face movements.

I examined the functions of the cranial nerves on many occasions, and from time to time observed various disturbances. There was almost constantly present, a parosmia of a malodorous variety, although the nose and throat were apparently healthy. Direct ophthalmoscopy showed distinct pallor of both optic discs, following retro-bulbar neuritis. The pupils were circular, the right smaller than the left, and they reacted directly and consensually to light, but the accommodation reflex was intermittently lost. There was ptosis of the right eye, with enophthalmos, indicating cervical sympathetic paresis. This finding was not constant, but improved from time to time, so as to be almost unrecognisable. Oculomotor disturbances were singularly absent during examinations. Nystagmus on all planes was frequently observed, and was sometimes markedly rotatory in character during exacerbations, when vertigo and ataxia were also pronounced. Fourth, fifth, sixth, and seventh nerve lesions were never elicited. Tinnitus on both sides, and vertigo, were often prominent symptoms during acute attacks. Dyspeptic symptoms as heartburn and flatulent eructations with water-brash, were frequently a source of upset to the patient's comfort. On two occasions, I observed the tongue to be deviated to the right.

Investigation of the motor system, revealed gross weakness in the muscles of the lower limbs, but the upper limbs were strong, and the hand-clasp vice like. All four limbs showed inco-ordination of movements, spasticity and muscle tremor of the intention variety. There was no muscle wasting. Tactile sensation and two point discrimination, were impaired in large surface areas, in both the trunk and the limbs; pain and thermal sensibility were only affected in the fingers and toes. Sensation of joint movement and posture in all four limbs, was poor, and when objects of varying shape and weight were put on the hands with the eyes closed, there was often great difficulty in recognising them. Vibration sense was lost over both tibiae, and over the bones of the fingers and toes. Pass-pointing was marked in both arms, and the heel-to-knee tests were badly performed. He was quite unable to walk along the edge of a carpet during exacerbations. Rombergism was almost constantly present. Libido and potentia were unaffected. Dysaesthesiae inthe form of joint pains and pain along the costal margin from posterior root involvement, were frequently prominent, and sometimes dominated the clinical picture.

No decided vasomotor or trophic changes were observed. The state of the reflexes was fairly constant during the time I had this patient under my care, and was as follows:-

	RIGHT.		LEFT.
Plantars	Ext.		Ext.
Epigastric	-		-
Abdominals	-		-
Cremasteric	✓		-
Knee jerks	✓✓✓		✓✓✓
Ankle jerks	✓✓		✓✓
Adductor jerks	✓✓		✓✓
Triceps	✓		✓
Biceps	✓✓		✓
Wrist	✓✓		✓
Jaw		✓✓	
Ankle clonus	✓		✓
Knee clonus	✓		✓

Swallowing	Normal
Defaecation	Impaired.
Micturition	Impaired.

Laboratory Findings:

Blood films and counts were normal; Hgn. 98%; Wassermann reaction was negative; lumbar puncture had previously been performed, and gave negative findings.

Chief Diagnostic Features:

This was an advanced case of the

disease. The diagnosis is made in virtue of the spastic paraplegia, ataxia, nystagmus, intention tremor, bladder and speech disturbance, and remissions.

Treatment:

The condition was already well established, and treatment by salvarsan injections had previously been carried out on two occasions, with but little success. Arsenical mixtures, iodide mixtures, and bromide mixtures, had been repeatedly given. As an alternative therapeutic measure, I gave him a course of T.A.B. vaccine intravenously, by weekly injections, for six weeks. The doses given were 20, 40, 80, I50, 300, and 600 million germs. The patient responded well to this treatment, an induced fever resulting from each dose. A fairly good remission of symptoms followed, during which he was able to do his writing and book-keeping continuously for almost six months, and was able to walk about the house with the aid of a walking stick. At this juncture, my personal observations on this case ended, but I know that the patient is still alive, the duration of the disease now being sixteen years.

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Case 6.

J.B.; aged 30; domestic servant; single.

Complaint:

This woman complained of being unable to walk, even with the aid of a walking machine which she had previously used with some success, and that her general condition was becoming gradually worse. She further complained of tingling and numbness in all four limbs, and of occasional pains in the legs.

Duration:

It was six years since the present illness began, but it was only during the past five months that she had been unable to walk a little.

Family History:

Both parents were alive and well. Two sisters were also in good health. There was no history of hereditary disease, or of relations suffering from any illness.

Personal History:

This patient was always of good habits, and her home surroundings had always been satisfactory. She had her appendix removed when eighteen years of age, and made an uninterrupted recovery: otherwise her health had been good. Her present illness began at twenty-four years of age, and the first symptom was a dragging of the right leg when walking, followed soon after

by trembling in both hands when grasping any object: there was also a prickling sensation in both legs. This passed off in about two months, but about fifteen months later, she sometimes saw double, and the leg weakness returned more pronounced than before. There was also difficulty in commencing the flow of urine at this time. Hospital treatment was then carried out, and she was given a course of injections which made her very fevered. She was sent home much improved, after seven weeks in bed. This remission of symptoms lasted three years, during which time, however, she was unable to follow her usual employment. Once again an acute attack came on, and she had not been really well since. Until five months ago, she was able to walk a little unaided, but staggered and felt dizzy: she moved about more freely with a mechanical support. During the past five months she had been wholly unable to walk, and was bedridden.

Present State:

The general condition of this patient, and her state of nutrition, were much below average: the cheeks, lips, and mucous membranes were pale: B.P. II2/70: the pulse, temperature, and respirations were normal: the heart was healthy, but there was cough and a bronchial sputum. The urine was amber, acid to litmus, S.G. I0I6, and contained no abnormalities. The skin was dry, with a tendency to flake, and there was a healing bed-sore over the sacrum. The lower limbs showed commencing contractures.

Neurological Examination:

The patient was euphoric, intelligent, had a good memory, and co-operated well in the history of her illness. She did not sleep well, and often had bad dreams, but no hallucinations or delusions. The speech was of the scanning type, and articulation was fairly good.

I proceeded to examine the functions of the cranial nerves, and found the olfactory intact. Direct ophthalmoscopic examination, revealed fine optic atrophy of both nerve heads, especially the right. The acuity of vision was poor, there were no scotomata present, and the field of vision was unrestricted. From time to time the oculomotor system showed fourth and sixth nerve lesions on the right side, with consequent diplopia on looking in that direction, and internal strabismus on the left. I saw no evidence of a supra-nuclear lesion at any time. The power of downward movement was also impaired in the right eye, and its exercise also caused double vision. On two occasions the left eye showed slight ptosis, with loss of the accomodation reflex, lasting only a few days each time. The pupils were circular, the right usually slightly smaller than the left, and reacted to light, directly and consensually. There was a suggestion of smoothness and lack of mobility on the right side of the face, which was a constant feature of the case.

I next investigated the motor functions, and found according to the patient's complaints. There was muscular paralysis in all four limbs, present to a marked degree in the legs, and accompanied by spasticity and intention tremor. Inco-ordination of movements was pronounced, and pass-pointing in the upper limbs was present, and was not increased by closing the eyes. Sensibility to touch and its discrimination by two-point test, was impaired in scattered areas in all four extremities. The reaction to pain and temperature varied considerably, even when identical areas were tested. Muscle sense was extremely deficient in the lower limbs, but the patient could identify with due consideration, ordinary articles of everyday use. Pallaesthesia was absent over both external malleoli. Dysaesthesiae of the legs were prominent in the form of arthritic pains in the knees and ankles, and shooting pains particularly in the right leg.

The state of the reflexes was as follows:-

	Right.	Left.
Plantars	Ext.	Ext.
Epigastric	-	-
Abdominals	-	-
Knee-jerks	+++	++
Ankle-jerks	++	++
Adductor-jerks	++	++

Triceps	≠		≠
Biceps	≠		≠
Wrist	≠		≠
Ankle clonus	≠		≠
Knee clonus	-		-
Jaw		≠	

Swallowing	Normal
Defaecation	Difficulty
Micturition	Difficulty.

Menstruation was irregular and scanty.

Laboratory Findings:

Blood counts and films showed a typical hypochromic anaemia: Haemoglobin 40% ; Wassermann reaction was negative ; lumbar puncture was performed, and the fluid was colourless, without coagulum, and not under pressure; the cell count showed an absence of polymorphs, and three lymphocytes per c.mm. The total protein was 60 mgms. per cent, and Pandy's test showed a haze: there was normal reduction of Fehling's solution, and the chlorides were normal: the colloidal gold curve was II22200000 ; the culture was sterile.

Diagnosis:

This was a well established case of disseminated sclerosis, with typical pyramidal lesions and cranial nerve

disturbances.

Treatment:

The anaemia required treatment, so I prescribed the following mixture -

Ferri. Ammon. Cit.		1 oz.
Magnes. Sulph.		1 oz.
Liqu. Arsenicalis		2 fl. drms.
Aquam	ad	6 fl. ozs.

Sig: 2 fl. drms. t.d.s. ex aqu.

I also gave her weekly injections of T.A.B. vaccine into the veins, in the following doses:- 10, 30, 60, 150, 250, 500, and 750 million organisms.

There was no reaction to the first two doses beyond a moderate increase in the pulse rate. After the third dose, she said she felt difficulty in speaking, and had cramps in the legs. The reaction to the fourth dose was similar, with little increase of temperature and pulse rate. After the fifth dose, vomiting and salivation occurred, with an increase of temperature to 102°F. The patient felt fairly exhausted for three hours, and then gradually recovered.
A similar reaction followed the other two doses, and the course of treatment was stopped. Four weeks later, a slight remission of symptoms occurred, but still she could not walk, even when supported. Her condition became gradually worse thereafter, and she died a few months later of Bronchitis and Pulmonary Congestion.

----oOo----

Case 7.

O.S. ; aged 27 years ; clerkess ; single.

Complaint:

This young woman complained of mistiness of sight in the right eye, increasing gradually over a period of four weeks, and loss of strength in the left arm.

Duration:

It was four weeks since the eye condition began, and just over two weeks since the arm weakness became manifest.

Family History:

Both parents were alive and well, and she had one sister who was also in good health. There was no history of hereditary disease, venereal infection, or of any relation suffering from a similar condition.

Personal History:

The patient had always enjoyed good health, and her habits were excellent. Her home circumstances were also very satisfactory. The present illness commenced four weeks previous to consulting me, when she felt a dimness in the right eye while writing. On closing the left eye, she could still read her own writing, but it appeared as in a poorer light as compared to reading with the left eye only. She had not at any time seen double. The initial dimness lasted only a few days, and then improved. This improvement led her to think that it

was due to constitutional upset, so she did not think any more about it. About a week later, the mistiness returned, and after persisting for a few days, she consulted an optician who provided her with spectacles. The glasses did not help, and this caused her much concern. Added to the eye weakness, a useless kind of feeling had presented itself in the left arm, so she decided to seek medical advice.

Present State:

My examination showed her to have a normal pulse, temperature, and respiration rate. The blood pressure reading was I24/76. She was well built and otherwise in robust,good health. The heart, lungs, abdominal organs, and urine, were normal in every respect.

Neurological Examination:

The patient was intelligent and pleasant natured, but euphorism was absent. She had a good memory, and co-operated well in the history of her illness. She answered questions smartly and without hesitation, and slept well without dreams or nightmares: there were no hallucinations or delusions. The speech was quite normal, and she had not noticed any change in the singing voice.

Examination of the cranial nerve functions, revealed only a lesion of the second, on the right side. The pupils were circular, and the right was a little larger than the left: they reacted to light and accomm-

odation, but the reaction on the right was sluggish as compared to the left. Lateral nystagmus was present in both eyes. Rough tests with a white headed hat pin revealed deficiency in the field of vision of the right eye, with central scotoma, and direct ophthalmoscopic examination, showed pallor of the right optic disc. The left disc was normal in appearance. Pain was present in and around the right orbit, especially when ocular movements were carried out: this I took to be indicative of retro-bulbar neuritis. The media were clear, and there were no signs of macroscopic eye lesions, nor were there any lesions of the oculomotor system.

I now examined the motor system, and found all the muscles well developed and in a good state of nutrition. There was no gross weakness in the left upper limb, but a fine tremor of the intention type was present, which reached its maximum as an object was about to be grasped. Pass-pointing on the left was also present, not increased by closing the eyes. No inco-ordination of movements was present in the lower limbs. There was also a suggestion of fine muscular tremor in the head. Objective and subjective sensory disturbances were absent in all four limbs.

Menstruation was unaffected.

The state of the reflexes was as follows:-

	RIGHT.	LEFT.
Plantars	Ext.	Flex.

	RIGHT.		LEFT.
Epigastric	-		-
Abdominals	-		-
Knee jerks	✓		✓
Ankle jerks	✓		✓
Adductor jerks	✓		✓✓
Triceps	✓		✓✓
Biceps	✓		✓✓
Wrist	✓		✓✓
Jaw		✓✓	
Ankle clonus	-		-
Knee clonus	-		-

Swallowing

Defaecation All normal.

Micturition

Laboratory Findings:

The blood Wassermann was negative, and films and counts were normal. I had not the opportunity to have the spinal fluid analysed in this case.

Chief Diagnostic Features:

This was a case of early disseminated sclerosis, diagnosed in virtue of the fleeting attacks of dimness of vision, with pallor of the disc and central scotoma,

evidence of a lower motor neurone lesion affecting the left upper limb, altered abdominal reflexes, and positive Babinski sign on the right side.

Treatment:

I prescribed Fowler's solution in this case, giving three minim doses, thrice daily, and increasing by one minim daily, until fifteen minims were taken: the dosage was then diminished by the same method, until the original dose was arrived at. A rest period of two weeks was then allowed, and the course repeated.

The sight fluctuated a good deal during treatment, and then settled to a constant degree of dimness, which was not so pronounced as originally. The left arm also improved, and there has been no acute attack now for over two years. During this time she has continued her employment.

----oOo----

Case 8.

W.B. ; 40 years old ; male ; married ; shopkeeper.

Complaint:

This man complained of a tired feeling and shakiness in the legs, and of having to strain to pass water.

Duration:

It was four months previously that he felt the legs becoming weak.

Family History:

Both of his parents were dead, his father having died of cerebral haemorrhage, and his mother of senile decay. He had two sisters older than himself, and one brother younger, all alive and in good health. There was no history of hereditary or venereal disease, or of any relation suffering from a like condition.

Personal History:

The patient lived in a four apartment house, in comfortable circumstances, was a total abstainer, and smoked about fifteen cigarettes daily. He served two years in France during the last war, suffered the effects of poison gas on two occasions, and sustained a bullet wound in the right shoulder. From these mishaps he had made a good recovery, and had good health during the past eight years or so. His present trouble

commenced about four months previous to my seeing him, and the first warning he had that all was not well, was the weariness in the lower limbs, particularly the right. He noticed further, that his legs were shaky, and that sometimes the right foot 'sclaffed' the floor or footpath while he was walking. Once or twice he had missed his step and almost fallen, when ascending a small step-ladder in his shop. He did not look upon his present condition as serious, because some days he felt better than others especially if he found time to sit down a while to rest. For a few weeks past, he had noticed that he was worse on Saturday evenings, after a long day standing in his shop. He further complained of difficulty in starting the flow of urine, but did not associate this in any way with the weakness in the legs. While relating his story, I noticed that his eyes filled with tears, which he quickly removed with a handkerchief. I asked him if he was easily moved to tears, and he told me that during the past few days, he had wept once or twice for no obvious reason, but that previously he had not ever wept easily. He had no business worries, nor was he sufficiently concerned about his present state of health to precipitate the condition. He had always been of a happy disposition and contented mind.

Present State:

This man was of moderately good physique, 5 ft., 9 inches in height, and a stationary weight of II½ stones. He looked quite well, although of a sallow complexion. There

were no glandular swellings, and no cough: the appetite was good, the bowels regular in action, and no digestive upset was recorded; the heart and lungs were healthy; B.P. I30/84; the urine was acid, amber, S.G. I020, and contained no deposit or abnormal ingredients the skin was in a healthy state.

Neurological Examination:

The patient was intelligent, had a good memory, and co-operated well in the story of his illness. He did not sleep well, but had no nightmares, delusions, or hallucinations. He spoke above the average speed, but articulated properly, and without impediment. He had noticed no change in the speech or in his handwriting.

I tested the functions of the cranial nerves, and found no abnormalities. The acuity and field of vision were normal, and ophthalmoscopic examination was negative. Horizontal nystagmus was seen on both sides, but no oscillating vision. He had never seen double.

Examination of the motor system showed distinct loss of power in both lower limbs, and the muscles were hypertonic. There was no sign of wasting. Inco-ordination of movements was evident in the heel to knee test, and intention tremor could also be seen in both legs. The muscles of the trunk, head and upper limbs appeared normal in every way.

He responded well to painful, tactile and thermal

stimuli, and there was no astereognosis. Vibration sense was absent over both malleoli. No paraesthesiae or dysaesthesiae were present.

The state of the reflexes was as follows:-

	RIGHT.	LEFT.
Pupils	o	o
Light	✓	✓
Accommodation	✓	✓
Plantars	Ext.	Ext.
Epigastric	-	✓
Abdominals	-	-
Cremasteric	✓	-
Knee jerks	✓✓	✓✓
Ankle jerks	✓✓	✓
Adductor jerks	✓✓	✓✓
Triceps	✓	✓
Biceps	✓	✓
Wrist	✓	✓
Jaw	✓	
Ankle clonus	✓✓	✓
Knee clonus	✓✓	✓

Swallowing	Normal
Defaecation	Normal
Micturition	Difficulty.

Libido and potentia were not impaired. There were no trophic changes, or lesions in bones or joints.

Laboratory Findings:

The blood and cerebro-spinal fluid were both fully examined, but normals were recorded in every detail. The Wassermann reactions in both fluids were negative.

Diagnostic Features:

This was a case of early disseminated sclerosis, the diagnostic features being:-

1. Altered abdominal reflexes and Babinski sign, giving evidence of a pyramidal tract lesion, affecting the lower limbs.
2. Intention tremor, spastic muscles, ataxia, and loss of vibration sense.
3. Involuntary weeping, indicative of brain disturbance.
4. Occurence of a remission of symptoms.

Treatment:

I prescribed Arsenic in the form of Fowler's solution, commencing with a small dose, as in previous cases, and increasing daily by one drop, until fifteen drops thrice daily were being taken: the process was then reversed, until the original dose was reached. A rest period of two weeks was then given, and the course of treatment repeated. During the second rest period, a remission of symptoms occured, in which he could walk about

almost quite normally; the act of micturition was much less troublesome, and the weeping turns ceased. At this stage the case passed out of my charge, having been six months under my care and observation. I have recently been informed, that this man still carries on his business, as a newsagent and tobacconist, but that he now has to use an invalid chair when his legs are bad. He is now fifty years of age, his trouble having lasted ten years to even date.

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Case 9.

H.T. ; AET. 22 years; warehouseman; male; single.

Complaint:

When this young man came under my care, he stated that he first complained of shakiness and loss of strength in the right leg, followed soon after by a similar feeling in the left. Four months later he felt his legs so weak, that he could not walk, and was forced to go to bed. Soon after this, shakiness appeared in the arms and head, and he felt very dizzy, even when lying down.

Duration:

It was one year previously, that the leg weakness first appeared.

Family History:

Both parents were alive and in good health. The patient had no brothers or sisters. No relative was suffering from any serious illness, and there was no history of hereditary or venereal disease.

Personal History:

The patient lived in a good home in the country, and was of excellent personal behaviour. He had scarlet fever, and whooping cough as a schoolboy, and made good recoveries. When seventeen years of age, he fell from a pony which had got

out of control, and sustained a fractured clavicle and bruises on the back. After six weeks incapacity, he returned to his duties, feeling perfectly fit, and continued at his work for four years without further interruption. His present illness then commenced. Superadded symptoms soon appeared after he was confined to bed, in the form of difficulty in articulating his words, so that his speech became drawling in character. Further, three weeks before I first examined him, he experienced for the first time, difficulty in passing water. This symptom had been constant until one week before I saw him, when he contracted an acute retention of urine, and required the aid of catheters. In spite of the utmost care being taken, cystitis occured, and pus and albumin appeared in the urine. Surgical opinion was then obtained, and a permanent supra-pubic cystostomy was performed. A box-belt was thereafter applied.

He had previously received a course of arsenical injections, without benefit.

Present State:

His general appearance was a true reflection of the distressing illness he had suffered. He was pale, and anxious in facial expression, and looked ten years older than his actual age. He was thin, and had a poor appetite for food. There were no glandular swellings, respiratory disturbances, or cough: the heart was in fairly good condition, and no abdominal abnormalities apart from the bladder condition, were found. The urine was amber,

alkaline, S.G. I022, with a deposit of phosphates and pus: albumin was also present, but no sugar or blood. The skin was in good order except for small inflammatory pressure areas over both buttocks and sacrum.

Neurological Examination:

The patient was euphoric, intelligent, possessed of a good memory, and co-operated well in the history of his illness. He slept fairly well, and had no hallucinations or delusions. The speech was of the scanning type. I proceeded to examine the cranial nerve functions, and found by ophthalmoscopic examination, bi-temporal pallor of the optic discs, with normal fundi. There was no alteration in the sight, and diplopia had never occured. There was no other cranial nerve lesion at the first examination, but I later observed the tongue to be pushed to the right side, and to be very tremulous. Horizontal and rotatory nystagmus was a constant feature of the case.

I then examined the motor system, and found marked loss of power in both lower limbs, with spasticity, spontaneous tremor, and gross ataxia. Both upper limbs were affected in a like manner, but to a lesser degree. There was also decided muscular tremor in the head and neck. Loss of vibration sense was found over nearly all bony prominences, and on the posterior aspects of both legs, response to touch and temperature was deficient. Dysaesthesiae were absent.

The state of the reflexes was as follows:-

	RIGHT.	LEFT.
Pupils	o	o
Light	≠	≠
Accommodation	≠	≠
Plantars	Ext.	Ext.
Epigastric	-	-
Abdominals	-	-
Cremasteric	-	-
Knee jerks	≠≠≠	≠≠
Ankle kerks	≠≠	≠≠
Adductor jerks	≠≠	≠≠
Triceps	≠	≠≠
Biceps	≠≠	≠
Wrist	≠≠	≠
Jaw	≠≠	
Ankle clonus	≠	≠
Knee clonus	≠	≠ (sustained)

Swallowing	Normal
Defaecation	Difficulty
Micturition	Gross difficulty.

There were no trophic disturbances, or changes in the joints.

Laboratory Findings:

Blood counts and films showed nothing pathological: Hgn. 90%; B.P. I20/72 ; Wassermann reaction was negative ; lumbar puncture was performed, and a colourless fluid without coagulum, was emitted; it was not under pressure; the cell count was normal, and there was no increase in protein, chlorides, or sugar: the culture was sterile, and Wassermann reaction negative.

Chief Diagnostic Features:

This was a case of progressive disseminated sclerosis, of a fairly acute type. The diagnosis was made in virtue of the following:-

1. Spastic paraplegia at the onset, in a youth with robust health.
2. Consequent paresis of the upper limbs.
3. Nystagmus, speech, and bladder affection.
4. Bi-lateral Babinski, loss of abdominal reflexes, exaggerated tendon responses, loss of vibration sense, and optic nerve head pallor on the temporal halves.

Treatment:

The first course of arsenical injections had not produced any alleviations of symptoms, nor had a spontaneous remission occured. The bladder condition was at first treated by the oral administration of Pyridium tablets, and bladder

lavage was done on alternate days, with a I in 500 solution of oxycyanide of mercury. This helped the condition greatly, and in my opinion should have been continued. My superiors at that time thought otherwise, and cystostomy was performed. After the operation, the cystitis cleared up well, and the pus disappeared. Albuminuria persisted, and in fact, became slowly worse.
I felt, however, that something else might be tried to stay the progress of the disease, and I was allowed to give the patient a course of T.A.B. vaccine. I gave this into the veins, in doses of 25, 50, I00, 200, 400, 800, and I600 million organisms at ten day intervals. He did not react to the first and second dose, but artificial fever was obtained with each subsequent dose. I failed, however, to obtain a remission of symptoms, and in view of the fact that the condition seemed to be progressive, nothing else was attempted. Paraplegia in flexion supervened, the sight began to fail, incontinence of bowel occured, and , with increasing albuminuria and renal insufficiency, dropsy became prominent. The patient died six months after the injections were stopped, the cause of death being disseminated sclerosis with toxaemia consequent on renal failure. Autopsy was not granted. The total duration of the disease was just over two years, and the period under personal observation, one year.

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Case IO.

J.S.; aged 34; labourer; single.

Complaint:

This man came under my care and observation, complaining of unsteadiness when walking, shakiness of the head and of all four limbs, and dimness of vision.

Duration:

The condition was of four years duration.

Family History:

His father died of heart trouble at sixty-six years of age, and his mother was still alive and aged seventy. He had one brother and three sisters, all in average good health. No relative suffered from similar complaints, and there was no history of hereditary or venereal disease.

Personal History:

This patient belonged to a poor working class home, but had always had good food and regular meals. He was a heavy cigarette smoker, but was abstemious in the consumption of alcohol. No previous illnesses, accidents, or operations, were recorded. The present illness commenced four years ago, with trembling of the hands, followed soon after by the same feeling in the legs, and some blurring of the sight. Twice, during the past four years, he had had good periods, lasting

several months each time, and during these periods, he had felt really quite well. Following these remissions, the symptoms had returned, if anything worse than before. He had noticed that since the last remission of symptoms, his speech had become altered, and that he was now inclined to drawl his words. The chief complaint now is weakness and shakiness in the legs, which are preventing him from walking without support: this is the first time he has not been able to walk about by himself. He had been in hospital for several weeks about three years previously, and had received massage and physical exercises, in addition to a course of injections. The improvement which followed this treatment was sufficient to allow him to do some light work, at which he continued until six months previous to my seeing him. At that time the walking became very difficult, and he was forced to give up his job. A short spell of improvement again followed, but only lasted a few weeks this time.

Present State:

He looked a strong healthy type of man, with no signs of recent loss of weight. The heart and lungs were healthy, although he was suffering from a slight bronchial catarrh at the time of my first examination: B.P. 126/76: no abnormalities were present in the urine, the specific gravity of which was 1014: The tongue was moist and tremulous, the digestion good, and the bowels regular. The skin was dry and scaly, but free from any

pigmentation. There were no glandular swellings or changes in the joints.

Neurological Examination:

This patient was euphoric, but not very intelligent, and the application of leading questions was necessary more often than one would desire. His memory was good, and this helped substantially in obtaining the story of the illness. He suffered greatly from insomnia, and stated that he had never slept for longer than two hours at a time, for the past two years. The speech was of the scanning type.

I now proceeded to examine the functions of the cranial nerves, and found only a lesion of the second. Ophthalmoscopic examination showed pallor of both discs, following retro-bulbar neuritis. The acuity of vision was diminished in both eyes, but particularly in the right, where a restriction of the field, in the form of a central scotoma was determined. There was no ocular paresis, and he had never seen double. The pupils were circular and equal, and reacted quickly to light and accommodation. A fine lateral nystagmus was seen in both eyes.

Examination of the motor system showed marked paralysis in all four limbs, the lower being more affected than the upper. Pass-pointing was present on both sides, and the heel to patella test was poorly performed by both lower limbs. Romberg's sign was positive. Intention tremor was present

in all limbs, and also in the head, neck, and tongue.

His response to the sensations of touch, pain and temperature was normal, except for slight anaesthesia in the fingers of both hands. Astereognosis was present in the lower limbs, but absent in the upper. Vibration sense was lost over most bony prominences, and dysaesthesiae were absent.

The state of the nerve reflexes was as follows:-

	RIGHT.	LEFT.
Plantars	Ext.	Ext.
Epigastric	-	-
Abdominals	-	-
Cremasteric	-	-
Knee jerks	++++	++
Ankle jerks	++	++
Adductor jerks	++++ spasm	++
Triceps	+	++
Biceps	+	+
Wrist	+	++
Jaw	++	
Ankle clonus	+ sustained	+ sustained
Knee clonus	+ "	+

Swallowing
Defaecation — All normal.
Micturition

Laboratory Findings:

Blood films and counts were normal, and haemoglobin I00%; Wassermann reaction was negative; lumbar puncture was performed, and a clear fluid without colour or coagulum was expressed without pressure; the cell count was I lymphocyte per c.mm.,and the total protein was 35 mg. per c.cm; chlorides were 746 mg. per cent, and there was normal reduction of Fehling's solution; the colloidal gold curve was 5554442I0 or of the paretic variety; the Wassermann was negative, and no micro-organisms were present.

Diagnostic Features:

This was an established case of Disseminated Sclerosis, and the diagnostic features were the spastic para-plegia with remissions, dimness of vision, intention tremor, ataxia, loss of abdominal reflexes with Babinski sign, and loss of sensibility to the vibrations of a tuning fork.

Treatment:

I gave this man T.A.B. vaccine intravenously, for eight consecutive weeks, in the following dosage:- 20, 40, 80, I50, 300, 600, I200, and 2000 million organisms. A sharp reaction was obtained after each injection, and he was prostrated after the last two doses, for three days. This treatment was followed and supplemented by co-ordinated exercises, massage, and passive movements of the limbs, which materially reduced the ataxic gait, and spasticity of the muscles.

A further remission of symptoms was obtained, during which the patient could dress and feed himself, tie his own boot laces, and brush his hair. He could walk much better and alone, with only the aid of a walking stick. How much of the improvement was due to the treatment, and how much to the change of environ - ment and rest in hospital, remains a matter for conjecture.

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Case II.

E.M.K.; aged 24 yrs.; female; married.

Complaint:

This woman complained of dimness of vision, of seeing double on looking downwards, and of stiffness and tiredness in the legs.

Duration:

The complaint was only of two days duration.

Family History:

Both of her parents were alive and in good health. There was also one sister in good health. No relative was in ill health from any cause, and no history of hereditary or venereal disease was given.

Personal History:

The patient was admitted to the obstetrical wards in labour. She was a primipara, and came from a working class home on the north side of the city. Spontaneous delivery of a full-time male child took place five hours after admission, and the confinement was normal in every respect. On the second day of the puerperium, she complained of dimness of vision, and told me that she saw double on looking down towards the foot of the bed: this did not occur when the patient looked straight in front, or upwards. She further stated that the legs felt stiff, and even in bed they felt lacking in strength.

On the day following delivery, her condition was evidently normal, the eye disturbance having come on overnight. Before admission she had been feeling quite well, apart from the usual functional upsets of late pregnancy. Two years previously, she remembered seeing double intermittently, for a few days, and at that time the right foot seemed to get out of control while she was walking. She attributed this to going'over on her heel'in the street, but did not think anything further about it, since it went away in about a week. She had scarlet fever when twelve years of age, and was in hospital for six weeks. No other illnesses, accidents, or operations were recorded, nor was there any history of dangerous employment.

Present State:

The general state of the patient was below par, but this could be attributed to loss of appetite, and gastric upset during pregnancy: the heart and lungs were healthy, and the B.P. I22/80; the urine was clear of albumin, and no oedema was present; there were no other abnormal ingredients. The puerperium was proceeding normally, without temperature or pulse rate increase.

Neurological Examination:

The patient was euphoric, intelligent, had a good memory, and co-operated well in the history of her illness. She slept well in ordinary circumstances, and had no delusions or hallucinations. Under the strain of examination,

I noticed that her speech was sometimes explosive in character. I investigated the functions of the cranial nerves, and found pallor of the whole of the right optic nerve head, and of the temporal half of the left. Acuity of vision was impaired in both eyes, but otherwise the fundi were normal. The third and sixth nerves were functioning properly, but paralysis of the fourth, on the right side was obvious. Loss of the restraining influence of the superior oblique muscle, was causing the eyeball to be rotated inwards by the overaction of the internal oblique muscle. This lesion was the cause of the double vision, with accompanying internal squint. Implication of the fourth nerve was further corroborated by the fact that the symptom only occured when the plane of vision fell below the horizontal, as when she looked down at the foot of the bed, There were no further cranial nerve disturbances seen. The pupils were equal and circular; they reacted directly and consensually to light and to accommodation; horizontal nystagmus was present, particularly on looking to the left.

I proceeded to examine the motor system, but found difficulty in stating whether or not, muscle paresis was present. There was difficulty in carrying out the heel to knee test on the right side, but not on the left, and there was no pass pointing in the upper limbs. Slight hypertonus was present in both lower limbs, more so on the right, but there was no muscle tremor.

Vibration sense was absent over both malleoli and tibiae, but there was no other sensory loss or perversion.

The state of the reflexes was as follows:-

	RIGHT.	LEFT.
Plantars	Ext.	Ext.
Epigastric	-	≠
Abdominals	-	-
Knee jerks	≠≠	≠≠
Ankle jerks	≠≠	≠
Adductor jerks	≠≠	≠
Triceps	≠	≠
Biceps	≠	≠
Wrist	≠	≠
Jaw	≠	
Ankle clonus	≠	-
Knee clonus	≠	-

Swallowing

Defaecation All normal.

Micturition

The lochia were normal in quantity and quality, and there were no trophic disturbances, or lesions of the bones or joints.

Laboratory Findings:

Blood counts and films were normal, and the

Wassermann reaction negative; Hgn. 90%; the spinal fluid was not obtained, owing to the patient's condition.

Diagnostic Features:

1. History of first attack two years previously.
2. Remission lasting that time.
3. Acute attack following parturition, with spastic paraplegia, ataxia, nystagmus, diplopia, and dimness of vision, from altered discs.
4. Signs of pyramidal lesion, affecting the legs.

Treatment:

The chief difficulty was finding a treatment which suited the lying-in period. I gave her quin. sulph. grs. 3, t.d.s. and increased the dose to 5 grs., during the second week. At the end of the third week, I allowed her out of bed, but she still showed signs of a spastic ataxic paraplegia, with a tendency to fall towards the right. The diplopia had gone, but the nystagmus persisted. The state of the reflexes remained as before. I took her over to the medical wards during the fourth week, and gave her a course of Neokharsivan intravenously, in doses of .3, .45, .6, .6, .6gm., at weekly intervals. Some improvement followed, and she went home, able to walk fairly well. The sight had not improved very much, but the diplopia had not returned.

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Case I2.

R.F.; 29 yrs.; male; single; shopkeeper.

Complaint:

This man consulted me regarding a feeling of stiffness and heaviness in both legs which affected his walking, with numbness and tingling in the soles of the feet and toes.

Duration:

It was two weeks since he first noticed the heavy, tired feeling in the legs.

Family History:

There was no history of hereditary disease, and both parents and one brother were alive and in good health. He had never been exposed to venereal infection.

Personal History:

Just over two weeks previously, he had gone down to Birmingham one day, and driven back a new delivery van for his business, on the same night. He stopped only once for a few minutes on the way home, and it was on the following day that he felt something wrong with his legs. He blamed the long journey for this, but was a little perturbed because he had often driven even longer journeys without the tired, heavy feeling in the legs from which he now suffered. During the past fortnight, he had been better some days than others, but for two days now,

the stiffness in the legs had been rather worse, and he had also felt a tingling alternating with numbness, on the soles of the feet and in the toes.

He had the appendix removed eleven years previously, and had measles and whooping-cough in childhood. There was no evidence from examination, of venereal disease, and he had never sustained any serious injury, nor been engaged in any dangerous trade. His habits were good and home surroundings excellent.

Present State:

The pulse, temperature and respirations were normal; B.P. I24/82; the heart, lungs, and abdominal organs were healthy, and the urine was normal in every respect; no cough, glandular swellings, or joint changes were present.

Neurological Examination:

The patient was intelligent, had a good memory, and co-operated well in the story of his illness. He slept well without dreams, and had no hallucinations or delusions. The speech was normal and without change.

I proceeded to test the cranial nerve functions, and the only change discovered was temporal pallor of the right optic disc, while the left was quite normal. He had not complained of any visual changes. The pupils were equal and circular, and reacted directly and

consensually to light and to accommodation. Bi-lateral nystagmus on looking to the right was seen.

Both lower limbs showed a fair degree of loss of power, with muscular spasticity and incoordination of movements when the heel to knee test was applied. The muscles of the trunk, head and upper limbs, were normal. Neither intention nor spontaneous tremor was present.

Dysaesthesiae and paraesthesiae , in the form of tingling and prickling in the soles of the feet and numbness in the toes were present, and the calf of the right leg showed impaired sense of pain and touch: the two-point discrimination test was also badly responded to in this area: the stereognostic sense was intact, but vibration sense was lost over most bony points.

The state of the reflexes was as follows:-

	RIGHT.	LEFT.
Plantars	Ext.	Ext.
Epigastric	-	✓
Abdominals	-	-
Cremasteric	✓	✓
Knee jerks	✓✓✓	✓✓
Ankle jerks	✓✓	✓✓
Adductor jerks	✓✓	✓
Triceps	✓	✓
Biceps	✓	✓

Wrist	/		/
Jaw		/	
Ankle clonus	//		/
Knee clonus	/		-

Swallowing, Defaecation, and Micturition were intact.

No trophic disturbances or joint changes were discovered.

Laboratory Findings:

Blood counts and films were normal, and the Wassermann reaction negative; Hgn. I04%; spinal fluid was not taken in this case.

Diagnostic Features:

The case was one of early disseminated sclerosis, diagnosed in virtue of the presence of signs of lesions involving the pyramidal tracts, with partial atrophy of the right optic disc, nystagmus, ataxia, and sensory disturbances.

Treatment:

Arsenic was selected as being the most suitable form of treatment. I prescribed Fowler's solution, and arranged the dosage as I have described in previous cases. The course was repeated twice, and a remission occurred soon afterwards. This remission lasted four years, when a second attack came on, similar to the first. The case at this stage was out of my care.

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Case I3.

J.A.; 48yrs.; male; labourer; married.

Complaint:

This patient complained of inability to walk, through weakness in the legs, loss of power in the hands and arms, dimness of sight, and loss of control of the bowels and bladder. He further complained of pains in the back and legs, and of loss of feeling in the fingers of both hands.

Duration:

He had been invalid, off and on, for about eighteen years.

Family History:

Both parents were deceased from causes unknown, and one brother was killed during the Great War. Two sisters were alive, and in good health. There was no history of hereditary disease, and he could not recollect any relative having suffered from a similar condition.

Personal History:

Prior to the onset of the present illness, the patient did not remember having had any serious illness, or any accident. About eighteen years previously, the present illness began with loss of power and trembling in the legs, which troubled him for about six months. He was in hospital about that time, for several weeks, and felt much better on dismissal.

He returned to work, and was able to continue for three years, when he suffered a fresh attack of weakiness in the legs. This attack was accompanied by shakiness in the hands, and dimness of vision. Again some improvement took place after a few weeks, but not sufficient to allow of his return to his usual duties. He had not been fit for remunerative employment for fourteen years, and had been entirely confined to bed during the past three months. Since he became confined to bed, he had not been able to control the urine, and several times, control of the bowels had been lost. He was in hospital on four different occasions, for periods varying from five weeks to four months, and had improved with treatment each time. He had taken ' a good amount of medicine in his day', and had received treatment by injections on two occasions.

Present State:

This man had been three times in a Poor Law Hospital, but in view of the fact that his condition was becoming progressively worse, and there being little hope of improvement, his wife desired to have him at home. I had thus the opportunity of attending him in the terminal stages of the disease, over a period of two months, until he died. When I first examined him, his general condition was poor. He was tall in stature, but very thin and almost emaciated: I could almost delineate every bone in his body. There was an overgrowth of hair of a downy texture,

extending downwards from the nape of the neck to the lumbar region, and laterally,as far as the mid-scapular line in the dorsal region: the hair was void of pigment, although on other parts, the original colour was retained. Serous effusions were present in both knee joints, but articular pain was absent. The heart sounds were pure, but of feeble quality, and showed occasional extra systoles. He had occasionally suffered from attacks of palpitation, but not during the past few weeks. The B.P. reading was II2/78; the lungs were apparently healthy, and there was no cough; the urine was acid, S.G. IOI8, contained a deposit of urates and mucus, and a haze of albumin. The skin was dry with a tendency to flake off when rubbed.

Neurological Examination:

The patient's intelligence had deteriorated considerably during the past six months, and it was only with patience and perseverance that a coherent story of the illness was obtained. The memory was poor, both for recent and remote events, and his deficiencies in this respect had to be supplemented by statements from his wife. He slept badly, and frequently required a sedative at night. Cerebration was inclined to be slow, and the speech was of the scanning type. He was emotional, and subject to weeping turns. I examined the cranial nerve functions, and found the olfactory intact. Acuity of vision was greatly diminished, and the field also restricted. Bi-lateral

optic atrophy and scotomata were present. External macroscopic changes in the eyes were absent, and the pupils were circular, equal, and reacted directly and consensually to light, and to accommodation: these reflexes were sluggish: oculomotor lesions had been present, since his wife stated that she had previously noticed the right eye turned in, and that he had complained of seeing double on more than one occasion. There was bi-lateral horizontal nystagmus at examination. The 7th, 8th, and 9th,nerves were working normally, but a lesion of the I0th. on the left side was apparent: the uvula became deviated to the right when I asked him to say 'AH'. There was no difficulty in swallowing, and no regugitation of fluids by the nasal passages. The IIth., and I2th. nerves were intact.

Paralysis was almost complete in the lower limbs, and the muscles showed a degree of atrophy from disuse, with generalised spontaneous tremor. The upper limbs also showed marked paresis, and tremor of the intention type. Although he could not feed himself, he succeeded with difficulty, in carrying out the tests for inco-ordination of movements. Pass-pointing was pronounced on each side. Spasticity was present in all four limbs, and contractures in the lower limbs maintained a position of paraplegia in flexion. The sensory reactions were poor, particularly in the lower limbs. Sensibility to touch, pain and temperature was impaired in scattered areas, below each knee, and over a fairly large area anteriorly, in the right thigh.

The responses on the left thigh were more accurate. Muscle sense was also seriously restricted in the lower limbs, although the hypertonus and contractures did not permit a large range of passive movements to be carried out. Dysaesthesiae were absent, as was also vibration sense. The fingers of the right hand were anaesthetic, as were also the thumb and forefinger of the left. The sensory response to stimuli applied to the upper limbs, thorax and abdomen was accurately recorded. Some loss of sensation was noticed on each side of the face, front of the neck, and occipital region. The state of the reflexes was as follows:-

	RIGHT.	LEFT.
Babinski Oppenheim Plantars. Gordon's	Ext.	Ext.
Epigastric	-	-
Abdominals	-	-
Cremasteric	-	-
Knee jerks	++	++
Ankle jerks	++	++
Adductor jerks	++	++
Triceps	++	++
Biceps	+	++
Wrist	+	++
Jaw	++	
Ankle clonus	+	+
Knee clonus	Effusion interfered with test.	

Of the organic reflexes, deglutition alone was intact. Libido and potentia were lost.

Laboratory Findings:

While in hospital, the blood and spinal fluid were examined and analysed. Films and counts had been normal, and Hgn. I02%; Wassermann reaction was negative; C.S.F. was clear, colourless, and not under pressure; cell count showed no polymorphs, and 3 lymphocytes per c.mm.; no protein increase, and the chlorides and glucose was normal; colloidal gold curve 3322222000.

Diagnostic Features:

This was an established case of disseminated sclerosis, in the terminal stages. The significant features and diagnostic signs were:-

1. General signs of a disease involving the pyramidal tracts, with remissions and a long course.
2. Cranial nerve disturbances, altered speech, intention tremor, nystagmus, loss of sphincter control, and paraesthesiae.
3. Mental deterioration and emotional upset in the late stages.

Treatment:

In view of the utter hopelessness of this patient's condition, purely palliative treatment was employed. I gave him ½ gr. pills of ext. bell. sicc., t.d.s., with a view to lessening

the output of urine; this was a distinct help. The remainder of the treatment consisted of relieving pain and discomfort, as the occasion arose. I found Luminal and Veronal in small doses, given twice or three times daily, most useful. These were ultimately displaced by the opiates, and Nepenthe in 8 to I0 min. doses was very effective. Tolerance was soon established, however, and much larger doses were demanded, before death occured. Loss of all hope of recovery, with subsequent mental depression and refusal of nourishment, hastened the end.

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Case I4.

J.W.R.; 33 yrs.; postman; married.

Complaint:

This man consulted me two years ago, regarding weakness of the legs, and dimness of sight in the left eye.

Duration:

The complaint was of three weeks duration.

Family History:

His father was alive and in fairly good health, but his mother had died of cancer six years previously. He had no brothers or sisters. No relative suffered from a similar condition, and there was no history of hereditary disease.

Personal History:

The patient lived in a three apartment house, in good circumstances and surroundings. His work as a postman did not expose him to any injurious factors, apart from inclement weather. He had always been engaged in the postal service, acting in earlier years as a telegraph boy. He took moderate physical exercise, chiefly on the bowling green and through the medium of his employment, was a total abstainer, and smoked only a little. He had suffered from a mild attack of influenza four weeks previously, and it was on returning to his work, that he felt the weakness in the legs, and became aware of dimness in the left eye. There was no history of any other illness or accident,

and he had never been exposed to the risk of venereal disease.

Present State:

The temperature, pulse, and respiration rate was normal; B.P. I24/76; he was of good build, average height, and the state of nutrition was satisfactory; the lips and mucosae were well coloured, and the heart, lungs, and urine, were normal in every respect; the abdominal organs were also healthy.

Neurological Examination:

The patient was right handed, intelligent and co-operated well in the history of his illness. The memory was good, and he slept well without dreams. He had no delusions or hallucinations. The speech was normal. Investigation of the cranial nerve functions, revealed only a lesion of the left optic nerve. Ophthalmoscopic examination showed pallor of the temporal half of the left disc, while the right was quite well and evenly coloured. The fundi were otherwise normal, and the media were clear. There was no restriction of the field of vision, but acuity was abnormal in the left eye. By using Snellen's test type card, I recorded his vision as R. 6/6, and L. 6/I8. I next examined the motor nerve functions, and found some loss of power in both lower limbs. The muscles were well developed, and hypertonic. Inco-ordination, more marked on the right, was demonstrable by the heel to knee test, and slight intentional tremor was present in both limbs: this was also seen in the head. His gait and station were slightly ataxic and

spastic. No abnormalities were found in the muscles of the trunk, or upper limbs. Sensory disturbances were entirely absent, and there was no lesion of the bladder or bowel. The pupils were were normal in every respect, and bi-lateral horizontal nystagmus was present. The state of the reflexes was as follows:-

	Right.	Left.
Plantars	Ext.	Ext.
Epigastric	-	-
Abdominals	-	-
Cremasteric	-	-
Knee jerks	++	++
Ankle jerks	++	+++
Adductor jerks	++	++
Triceps	+	+
Biceps	+	+
Wrist	+	+
Jaw	++	
Knee clonus	+	+
Ankle clonus	+	+

The organic reflexes were all intact.

Laboratory Findings:

Blood counts and differential counts showed no abnormalities; Hgn. I06%; Wassermann reaction negative; the C.S.F. as obtained by lumbar puncture, was clear and colour-

and not under pressure; the cell count was 5 lymphocytes per c.mm. and no polymorphs; the total protein was 40 mg. per I00 c.cm.; Pandy's test showed a haze; chlorides and glucose were normal; colloidal gold curve 5555544II0; Wassermann reaction negative; no micro-organisms were found.

Diagnostic Features:

This was a case of early disseminated sclerosi diagnosed in virtue of:-

1. General signs of pyramidal tract lesions, as for example, spastic paraplegia, Babinski sign, increased knee jerks, clonus, and loss of abdominal reflexes.
2. Partial atrophy of the left optic disc, of the post-retro-bulbar neuritic type.
3. Intention tremor, and nystagmus.

Treatment:

As this man was unfit to continue his duties, I took the opportunity of putting him to bed, and commencing a course of artificial pyrexia without delay. I gave him intravenous doses of the B.coli vaccine Pyrifer, six injections on alternate days. He maintained an artificial fever almost throughout the course, the temperature reaching a maximum, usually just under I04° F., between four and five hours after the injection; it thereafter gradually subsided, and was normal on each occasion before the next dose was administered. This treatment seemed rather severe

on the patient, and his friends rather doubted my sanity for carrying it out, but all was well at the end of the course, and he gained a sufficient return of power in the lower limbs to enable him to return to work. The partial blindness was also much improved. Acting on the belief that liver therapy is beneficial in these cases, I have included this in his diet, to be continued indefinitely. There has been no acute exacerbation in this case, the remission having lasted about eighteen months.

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I have incorporated and discussed the outstanding features of these cases, and their clinical significance as factors in this disease, in the preceding sections of this thesis. I beg now, to present the remainder of my series of cases, in tabular form.

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Signs and Symptoms etc.	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30
1. Age.	24	25	23	43	25	26	27	32	44	43	25	27	32	24	30	47.
2. Sex.	F	M	F	M	F	M	F	M	M	F	F	M	M	F	M	M
3. Type of Work.	S	L	L	L	S	S	S	H	H	L	L	H	L	S	S	S
4. Duration of illness.	½	2	2	12	½	¼	2	3	5	2	¼	1½	2	7	6	10
5. First Symptom.	35	35	36	35	35	38	35	19	36	38	35	35	35	43	35	36
6. Chief Symptom now.	35	35	35	30	35	38	35	35	37	35	35	30	37	35	34	35
7. Previous Infectious Diseases.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
8. Hereditary Disease.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
9. Venereal Disease.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
10. Wassermann test.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
11. Blood counts etc.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
12. History of Injury.	0	✓	0	0	0	0	0	0	✓	0	0	0	0	0	✓	0
13. Speech Disturbance.	0	0	0	✓	0	0	✓	0	✓	0	0	✓	0	✓	✓	✓
14. Euphoria.	✓	✓	✓	✓	0	0	0	✓	0	0	0	0	✓	0	0	✓
15. Emotional Upset.	0	0	0	0	0	0	0	0	✓	0	0	0	0	✓	0	✓
16. Mental Deterioration.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
17. Impairment of Smell.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
18. Diplopia.	0	0	0	✓	0	0	0	0	✓	✓	0	0	0	✓	0	✓
19. Dimness of Vision.	0	0	0	✓	0	0	0	✓	✓	0	0	0	0	✓	✓	✓
20. Restriction of Field.	0	0	0	0	0	0	0	✓	0	0	0	0	0	✓	0	✓
21. Altered Discs.	0	✓	✓	✓	0	0	✓	✓	✓	✓	0	0	0	✓	0	✓
22. Eye muscle paralysis.	0	0	0	0	0	0	0	0	✓	✓	0	0	0	✓	0	✓
23. Nystagmus.	0	✓	✓	✓	✓	0	✓	0	✓	✓	0	✓	✓	✓	✓	✓
24. Cervical Sympathetic Paralysis.	0	0	0	0	0	0	0	0	✓	0	0	0	0	0	0	0
25. Trigeminal Neuralgia.	0	0	0	0	0	0	0	0	✓	0	0	0	0	✓	0	0

	I5	I6	I7	I8	19	20	2I	22	23	24	25	26	27	28	29	30
26. Pupillary disturbances.	0	0	0	0	0	0	0	0	≠	0	0	0	0	0	0	0
27. Facial Paralysis.	0	0	≠	0	0	0	0	0	≠	0	0	0	0	≠	≠	≠
28. Deafness.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
29. Tinnitus.	0	0	0	0	0	0	0	0	0	≠	0	0	0	≠	0	≠
30. Vertigo.	0	0	0	≠	0	0	0	0	≠	0	0	0	0	≠	0	≠
3I. Ataxia.	≠	≠	≠	≠	0	0	≠	≠	≠	≠	0	≠	≠	≠	≠	≠
32. Difficulty in Swallowing.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
33. Constipation.	≠	0	≠	≠	0	0	≠	0	≠	≠	0	0	0	≠	≠	≠
34. Bladder upset.	0	0	0	≠	0	0	0	≠	0	0	0	≠	0	≠	≠	≠
35. Weakness lower limbs.	≠	≠	≠	≠	≠	≠	≠	≠	0	≠	≠	≠	≠	≠	≠	≠
36. Weakness upper limbs.	0	0	≠	≠	0	0	0	≠	≠	≠	0	0	≠	≠	≠	≠
37. Intention Tremor.	≠	≠	≠	≠	0	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠
38. Spasticity.	0	≠	≠	≠	≠	≠	≠	0	≠	≠	0	≠	≠	≠	≠	≠
39. Altered abdominal reflexes	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠
40. Babinski's sign.	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠
4I. Increased Knee-jerks.	≠	≠	≠	≠	≠	≠	≠	≠	0	≠	≠	≠	≠	≠	≠	≠
42. Ankle or Knee Clonus.	0	0	0	≠	0	0	0	0	≠	0	0	0	0	≠	≠	≠
43. Paraesthesiae.	0	0	0	≠	0	≠	≠	≠	≠	0	0	0	0	0	≠	≠
44. Lost Vibration sense.	0	≠	≠	≠	0	0	≠	≠	≠	≠	0	0	≠	≠	≠	≠
45. Hypertrichosis.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
46. Other Trophic Lesions.	0	0	0	≠	0	0	0	0	0	0	0	0	0	0	0	0
47. Remissions.	0	≠	0	≠	0	0	0	≠	≠	≠	0	≠	≠	≠	≠	≠

Cases I5 to 30 continued.

Signs and Symptoms.	3I	32	33	34	35	36	37	38	39	40	4I	42	43	44	45	46	47
I. Age.	28	5I	45	I9	32	34	30	32	33	28	45	36	26	22	36	52	29
2. Sex.	M	F	F	M	F	F	M	M	M	M	M	F	M	M	M	M	F
3. Type of Work.	H	L	L	O	L	L	L	L	H	S	H	S	S	L	H	L	S
4. Duration of Illness	4	9	5	½	I0	I	4	3	3	5	7	6	¾	I	2	I3	2
5. First Symptom.	35	35	36	35	43	35	I9	I8	36	30	35	38	37	35	30	35	35
6. Chief Symptom now.	35	35	35	35	36	35	35	I8	36	35	34	37	35	I8	30	34	30
7. Previous Infections	O	O	O	O	O	O	O	O	O	O	O	O	✓	O	O	O	O
8. Hereditary Disease.	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
9. Venereal Disease.	O	O	O	O	O	O	O	O	O	O	O	O	O	O	✓G	O	O
I0. Wassermann.	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
II. History of Injury.	✓	O	O	✓	O	O	O	O	O	✓	O	O	O	O	O	✓	O
I2. Blood Counts etc.	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
I3. Speech Disturbed.	✓	O	O	O	✓	O	✓	O	O	✓	✓	✓	O	O	O	✓	O
I4. Euphoria.	✓	✓	O	O	O	O	✓	O	✓	✓	✓	✓	✓	O	✓	✓	✓
I5. Emotional Upset.	O	✓	O	O	O	O	O	O	O	✓	✓	✓	O	O	O	✓	O
I6. Mental Affection.	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	✓	O
I7. Impaired Smell.	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O	O
I8. Diplopia.	✓	O	O	✓	✓	O	✓	✓✓	O	O	✓	O	O	✓	O	O	O
I9. Dimness of Vision.	✓	O	O	O	✓	O	✓	✓	O	O	O	O	O	O	O	✓	O
20. Restricted Field.	O	O	O	O	O	O	✓	O	O	O	O	O	O	O	O	✓	O
2I. Altered Discs.	✓	O	✓	O	✓	✓	✓	✓	O	✓	✓	✓	O	✓	✓	✓	✓
22. Ocular Paralyses.	O	O	O	O	O	O	✓	✓	O	O	✓	O	O	✓	O	O	O
23. Nystagmus.	O	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	O
24. Sympathetic Lesion.	O	O	O	O	O	O	✓	O	O	O	✓	O	O	O	O	O	O
25. Abnormal Pupils.	O	O	O	O	O	O	✓	O	O	O	✓	O	O	O	O	O	O
26. Trigeminal Lesion.	O	O	O	O	✓	O	✓	O	O	O	O	O	O	O	O	✓	O

Cases 3I to 47 continued.

Signs and Symptoms.	3I	32	33	34	35	36	37	38	39	40	4I	42	43	44	45	46	47
27. Facial Palsy.	0	0	0	0	≠	≠	≠	0	0	0	0	≠	0	0	0	≠	≠
28. Deafness.	0	0	0	0	0	0	0	0	0	0	≠	0	0	0	0	≠	0
29. Tinnitus.	0	0	0	0	≠	0	0	0	0	0	≠	≠	0	0	≠	≠	0
30. Vertigo.	≠	≠	0	0	≠	≠	≠	≠	≠	≠	≠	≠	0	≠	≠	≠	≠
3I. Ataxia.	≠	≠	0	0	≠	≠	≠	≠	≠	≠	≠	≠	0	≠	≠	≠	≠
32. Swallowing.	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
33. Constipation.	0	≠	0	0	≠	≠	0	≠	0	0	≠	≠	0	0	0	0	≠
34. Bladder Upset.	≠	0	0	0	0	0	≠	≠	0	≠	≠	≠	0	0	0	≠	0
35. Leg Weakness.	≠	≠	≠	≠	≠	≠	≠	≠	0	≠	≠	≠	≠	≠	0	≠	≠
36. Arm Weakness.	≠	≠	≠	0	≠	0	0	0	≠	0	≠	≠	0	0	0	≠	0
37. Intention Tremor.	≠	≠	≠	≠	≠	≠	0	0	≠	≠	≠	≠	≠	≠	≠	≠	≠
38. Spasticity.	0	≠	≠	≠	≠	≠	0	0	0	≠	≠	≠	0	0	≠	≠	≠
39. Abdom. Reflexes altered.	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠
40. Babinski's sign.	≠	≠	≠	≠	≠	≠	≠	≠	0	≠	≠	≠	0	≠	0	≠	≠
4I. Increased Knee-jerk	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	≠	0	0	≠	≠	≠
42. Clonus.	0	≠	0	0	≠	0	0	0	0	0	≠	≠	0	0	0	≠	0
43. Paraesthesiae.	0	0	≠	0	≠	0	0	0	0	≠	≠	≠	0	0	0	≠	≠
44. Lost Vibration S.	0	≠	≠	≠	≠	0	≠	0	≠	≠	≠	≠	≠	0	≠	≠	≠
45. Hypertrichosis.	0	0	0	0	0	0	0	0	0	0	≠	0	0	0	0	0	0
46. Other Trophic Les.	0	0	0	0	≠	0	0	0	0	0	0	0	0	0	0	≠	0
47. Remissions.	≠	≠	≠	0	≠	0	≠	≠	≠	≠	≠	≠	0	0	0	≠	0

Signs and Symptoms etc.	48	49	50	51	52	53	54
1. Age.	38	35	23	22	23	22	38
2. Sex.	F	F	M	M	F	F	F
3. Type of work.	S	L	O	L	L	L	S
4. Duration of Illness.	18	8	6	3/12	4	2	4
5. First Symptom.	43	18	35	35	35	35	36
6. Chief Symptom Now.	18	36	37	34	30	38	31
7. History of Infections.	0	0	/	/	0	0	0
8. Hereditary Disease.	0	0	0	0	0	0	0
9. Venereal Disease.	0	0	0	0	0	0	0
10. Wasserman Reaction.	-	-	-	-	-	-	-
11. History of Injury.	0	/	0	0	0	/	0
12. Blood Counts and Films.	0	0	0	0	0	0	0
13. Speech Alteration.	0	/	/	0	/	/	/
14. Euphoria.	/	/	0	/	/	/	//
15. Emotional Upset.	/	0	0	0	/	0	0
16. Mental Deterioration.	0	0	0	0	0	0	0
17. Impaired Sense of Smell.	0	0	0	0	0	0	0
18. Diplopia.	//	/	0	/	0	0	0
19. Dimness of Vision.	/	/	0	0	0	0	/
20. Restricted field of vision.	//	0	0	0	0	0	/
21. Altered Optic Discs.	/	/	/	/	0	/	/
22. Eye Muscle Paralysis.	/	/	0	/	0	0	0
23. Nystagmus.	/	/	0	/	/	/	/
24. Cervical Sympathetic paral.	/	0	0	0	0	0	/
25. Abnormal Pupils.	/	0	0	0	0	0	/
26. Trigeminal Neuralgia.	0	0	0	0	0	/	0

Cases 48 to 54 continued.

Signs and Symptoms etc.,	48	49	50	5I	52	53	54
27. Facial Paralysis.	✓	0	0	0	✓	0	✓
28. Deafness.	0	0	0	0	0	0	0
29. Tinnitus.	0	0	0	0	✓	0	0
30. Vertigo.	0	✓	0	0	✓	✓	✓
3I. Ataxia.	✓	✓	0	0	✓	✓	✓
32. Difficulty in Swallowing.	0	0	0	0	0	0	0
33. Constipation.	✓	✓	0	0	✓	0	✓
34. Bladder Disturbance.	0	0	0	✓	0	0	✓
35. Weakness in Lower Limbs.	✓	0	✓	✓	✓	✓	✓
36. Weakness in Upper Limbs.	✓	✓	0	0	✓	0	✓✓
37. Intention Tremor.	✓	0	✓	✓	✓	✓	✓
38. Muscular Rigidity.	✓	0	✓	✓	✓	✓	✓
39. Altered Abdominal Feflexes.	✓	✓	✓	✓	✓	✓	✓
40. Babinski's Sign.	✓	0	✓	✓/	✓	0	✓
4I. Increased Knee-jerks.	✓	✓	✓	0	✓	✓	✓
42. Ankle or Patellar Clonus.	0	0	0	0	0	✓	0
43. Sensory Disturbances.	✓	✓	0	✓	0	✓	0
44. History of Remissions.	✓	✓	✓	0	✓	0	✓
45. Loss of Vibration Sense.	✓	✓	0	✓/	✓	✓	0
46. Hypertrichosis.	0	0	0	0	0	0	0
47. Other Trophic Lesions.	✓	0	0	0	0	0	✓

In the following table, I have localised the chief symptoms occuring in this series.

Symptoms.	Localisation.
Loss of power in upper or lower limbs.	Pyramidal tracts in cord.
Muscular hypertonus	Upper motor neurone lesion, as above.
Speech disturbances	Speech centres: brain-stem lesion.
Involuntary laughing and crying, and other emotional and mental upsets.	Lesions of cerebral cortex.
Euphoria.	Brain disturbance.
Nystagmus.	Vestibulo-ocular mechanism.
Paraesthesiae and dysaesthesiae in upper and lower limbs.	Cutaneous sensory paths in the cord or brain.
Numbness or anaesthesiae of fingers.	Sensory fibres in lateral columns.
Vertigo.	Vestibular apparatus.
Loss of vibration sense.	Paths of tactile sensation in spinal cord or brain.

Positive Romberg.	Spino-cerebellar tracts.
Loss of abdominal reflexes.	Lesion of spinal or cortical arc for these reflexes.
Girdle pains.	Posterior root involvement.
Bladder and bowel upset.	Injury to nerve supply of sphincter of bladder, and paresis of bowel wall.
Parosmia.	Olfactory nerve lesion.
Alterations in acuity and field of vision.	Optic nerve lesion.
Ptosis, with dilated pupils and loss of accommodation.	Third nerve lesion.
Diplopia below horizontal plane.	Fourth nerve lesion.
External ophthalmoplegia with diplopia.	Sixth nerve lesion.
Loss of tone in facial muscles.	Motor root of fifth nerve.
Facial paralysis.	Seventh nerve lesion.
Tinnitus and Vertigo.	Vestibular nerve.
Deviation of the uvula.	Vagus nerve lesion.

Deviation of the tongue, without wasting.	Supranuclear lesion of the twelfth nerve.
Enophthalmos, with ptosis and contracted pupil.	Cervical sympathetic paralysis.
Hypertrichosis.	Trophic disturbance.

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Scotland. Deaths from Disseminated Sclerosis by Sexes, 1935-1937.

Year		All ages	Ages																			
			-1	1-	5-	10-	15-	20-	25-	30-	35-	40-	45-	50-	55-	60-	65-	70-	75-	80-	85-	90-	95-
1935	m.	61	-	-	-	-	1	1	1	3	6	7	7	9	7	6	6	4	2	1	-	-	-
	f.	86	-	-	-	-	-	3	5	5	9	4	14	10	15	6	6	5	2	2	-	-	-
1936	m.	87	-	-	-	-	2	2	-	6	6	6	8	13	9	12	11	8	2	2	-	-	-
	f.	71	-	-	-	-	1	2	1	3	6	12	3	18	2	6	4	4	7	-	2	-	-
1937	m.	88	-	-	-	-	-	3	1	4	9	12	10	8	15	10	8	2	4	2	-	-	-
	f.	85	-	-	-	-	1	1	4	9	5	8	11	12	3	10	8	8	3	1	1	-	-

STATISTICS 19 DEC 1938 BRANCH SCOTLAND

Deaths from Disseminated Sclerosis in Scotland for the years I935, I936, and I937.

For the further interest and information of my reader, I have interleaved opposite this page, the Registrar's statistics for deaths from Disseminated Sclerosis in Scotland, during the past three years.

From this information it may be observed, that the total number of deaths from this disease is 478 for the period of three years, giving an average annual number of deaths of almost I60. Included in this three years total, are 236 or 49% males, and 242 or 5I% females. For the two decades, 40 to 60 years of age, 262 deaths were registered: 5 were for individuals under twenty years of age, and eleven for people over eighty years old. The maximum figure for five year age periods, is 70 deaths between the ages of 50 and 55 years.

Taking the average annual number of deaths from Disseminated Sclerosis as I60, and the population of Scotland as approximately 4,800,000, I find that the death rate is 0.033̇ per thousand.

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The Domestic and Economic Management of Disseminated Sclerosis.

Permanent incapacity for remunerative employment, from whatever cause it may arise, is necessarily accompanied by sacrifices, denials, and hardships, affecting both the sufferer and his dependents. The true circumstances surrounding chronic invalidism can only be appreciated fully by personal contact with the patient in his own home. The family doctor, because of frequent domiciliary visitation, is afforded ample opportunity for the study of these circumstances, and disseminated sclerosis, because of its usually long course, forms an excellent subject for discussion from this angle.

Over a period of ten or more years, almost every conceivable social problem may be encountered by the sufferer, and obstacles, which, in good health would be taken in his stride, may, in his weakness, prove well-nigh unsurmountable. As may readily be understood, this applies particularly in the case of an insured worker, with a wife and family to provide for. W.N.P. Barbellion, who was himself a victim of this disease, wrote the following exclamatory sentence in his 'Journal of a Disappointed Man'; ''Paralysed, with a wife, and a child, and no money --- ugh". What words of mine could more adequately or succintly describe the social outlook of a man in such a predicament, than this terminal declaration

of self-pity, and self-disgust?

To the physician in consulting practice, a patient with early disseminated sclerosis, is more or less a pathological package, sent to him by the family doctor to be correctly labelled. To resident hospital doctors and undergraduates, the sufferer is apt to become just another case -- a curiosity, for neurological experiment. To the general practitioner, however, such a patient becomes a constant charge, requiring advice and guidance over a period of many years. Throughout the whole illness, almost daily explanations are requested by the patient and by his relatives regarding the course of the illness, and the significance of individual signs and symptoms. Added to these, the practitioner is required to carry out the usual regulations of the Health Insurance Act, and to give due consideration to all matters affecting the family welfare. Explanations and answers to pertinent questions can not always be truthfully given, but in the circumstances, such inexactitudes are both permissible and excusable. At all costs, the patient's fears have to be allayed, and his hope of ultimate recovery kept alive. From the practitioner's point of view, the occurence of remissions in this disease, particularly in the earlier stages, is often a saving grace. After many weeks of reassuring and optimistic advice to such a patient, I have frequently found myself almost defeated in my effort to explain

away the arrival of a new symptom, or the exaggeration of an old one. I have found myself telling a patient that I thought he was keeping better, when he was not; that he was walking better to-day, when his paresis and ataxia were more pronounced than previously; or that his hands were not so shaky, when the intention tremor was obviously more marked. I adopt these measures entirely in good faith, for the disillusionment of the patient regarding the gravity of his condition. Treatment by suggestion is always attended by a measure of success in Disseminated Sclerosis, because of the euphorism invariably present. This mental state allows of greater proclivity on the part of the patient, towards such moral support and suggestion. Should the sufferer be mentally stable, or have a propensity to mental depression,however, I have found a greater disinclination for, and disbelief of, such advice.

This form of encouragement may be continued successfully for about two years, but breaking-point is reached sooner or later in every case. The patient must be at least partly informed of the seriousness of his state of health, otherwise he will lose faith in his doctor, and is likely to go elsewhere for advice. I have reached this stage with one or two patients during the past few years, but in one case in particular, I was saved from further explanations by the occurence of a marked remission of symptoms, which renewed the

former feeling of mutual trust and goodwill, for a further period.

Unfortunately, the breaking-point between practitioner and patient, when such exists, does not always synchronise with the onset of a remission. In these circumstances, it is either a matter of telling the patient the whole truth, or risking the loss of a patient. It is well that I should warn the young practitioner at this point, that the general public is an extremely fickle quantity which has to be handled with much adroitness and finesse. This inconstancy is peculiar to all classes in times of sickness, and is chiefly a manifestation of fear for the future. To a great extent, it is a natural reaction to illness.

The ease with which an insured patient can change to the panel of another doctor, is at least partly to blame for loss of faith. I have always advocated the eradication of this weed from the field of State Medicine, and this could easily be accomplished by transforming the local panel committee into a sort of court of inquiry. This court, made up by all the doctors in the district, could meet every three months to consider applications by insured patients for transfer to other panels. Such transfers could be granted or refused, according to the voting of the members.

At the present time, a disgruntled patient, who may be a source of useful information to a doctor, may, by a mere stroke of a pen, change over to another

panel list where the capitation fee is the only incentive to accept him. Such a transfer would be refused by a local court of inquiry, as I have suggested, and the patient would be told that his treatment had been adequate. Let us consider for a moment, the other side of the story. If an insured patient becomes permanently incapacitated, mainly in his own opinion, through ailments like chronic muscular rheumatism, or nervous debility etc., he will continue to draw sickness benefits until he becomes sixty-five years of age, and prove himself, over a number of years, to be a nuisance to the panel doctor: but can the already overworked practitioner rid himself of such a patient by requesting the removal of his name from the list? No, he can not. I have a score or more of such patients in my practice, patients whose certificates should be signed as suffering from Ergophobia.

In Airdrie and district, like other industrial areas, men over fifty years of age are considered too old for heavy work. If a man of that age takes ill from any cause, and is laid aside for even a few weeks, employers are quick to seize the opportunity of procuring the services of a younger man in his stead. Thus the fifty year old is thrown out of work, and has very little chance of securing other employment. How is he to secure a living? He may have sons and daughters bringing home their weekly earnings, but they have little to spare to keep him. He is refused unemployment benefit through the administration of

the Means Test, and is also considered ineligible for Public Assistance relief. Then he remembers that for the past twenty odd years, he has paid Health Insurance contributions, and feels entitled to don the mantle of illness, in order to draw sick benefit. That is the process of evolution of most of my chronics in this area, and who could blame them?

It is a general opinion among insurance practitioners, that the patient has the upper hand all the time: that he has toomuch power and authority, and that too little consideration is afforded the doctor.

With regard to the changing of doctors under Insurance Act regulations, the above considerations are prompted by a personal experience within recent years. A male patient of mine, who suffered from Disseminated Sclerosis in its earlier stages, was a prolific source of information for the subject of this thesis. Quite suddenly and unexpectedly, he left my panel. As I afterwards discovered, he had become dissatisfied with his progress, and had gone to another doctor in the district. He had done so, because a friend with a similar complaint, had been cured by the other doctor. Further inquiry on my part, revealed that the friend actually suffered from Disseminated Sclerosis, but that in his case, a marked remission of symptoms had occured. It may thus be realised, that in this disease, a remission may be a godsend, or it may result in the loss of a useful patient.

To return more closely to my subject, I wish, in this concluding chapter of my thesis, to describe the outstanding points of interest in the home management of cases of this disease, both from the medical and social aspect.

It has been my experience, that Disseminated Sclerosis is seldom wholly incapacitating during the first four years. Short periods of absence from work are often necessary, when acute exacerbations occur, or when a course of treatment has to be administered. Usually, when the disease has lasted over four years, the periods of unfitness become more prolonged until employment ultimately ceases. Certain patients are able to continue at work for much longer periods than others, even up to fifteen years from the onset of symptoms, but such cases are more the exception than the rule. This period of capacity for work is modified firstly by the rate of progress of the disease process: secondly, by the vocation or type of work performed by the sufferer: and thirdly, by the anatomical localisation of the disease, in relation to the parts of the body essential for the patient's employment.

I have found that those patients whose work demands great accuracy and neatness, such as bank clerks, accountants, book-keepers and typists etc., find themselves wholly unable to carry on their work much sooner than men and women doing coarse manual labour, such as in certain mill and factory jobs, coal mining, or building. Shop assistants

are often able for work, for many years after the onset of the disease, as I have already indicated in my descriptive cases. The chief determining factor in most cases, is whether or not the anatomical parts affected by the disease, are essential and indispensable for the patient's work. For example, a clergyman, school-teacher, or a politician, may continue in employment for many years, if the speech mechanism remains unaffected. In this instance, the control of the speaking voice is the essential wage earning factor, and is independent of other manifestations of the disease. Many other examples might be made, but this one is sufficient to illustrate the point.

Following the cessation of work, the mode of obtaining a livelihood, depends on several factors. It first of all depends on the financial position of the patient, and his immediate relations. Should the sufferer be of independent means, or be born of wealthy parents, there is obviously no lack of medical and nursing attention: the patient will have good food, clothing, and excellent home surroundings: he will also be well provided for, in the way of outings and holidays.

Consideration of the more common type of case, as it occurs in middle and working class practice, presents to us, a different picture. A disabled worker finds himself forced to draw sickness benefit from his Approved Society, at the rate of I5/- weekly if he has made I04 or more weekly contributions, or

9/- weekly, if only between 24 and I04 contributions have been made. We may thus appreciate the difficulties encountered by a young man, say of 25 to 30 years of age, who may have been earning any sum up to £5 per week. If the loss can be made up in other ways, the outlook is not so gloomy, but when, for example, a widow is depending on a son's weekly wage for the upkeep of their home, such a drop in the family income can not be tolerated, and so Public Assistance relief money has to be requested. Likewise, a married man with a family is forced to become a pauper, for the maintenance of his home.

In other cases, parents may be able to keep a young man or woman so afflicted, in fairly comfortable circumstances, without Public Assistance money. In a few cases, I have seen these young paralysed people making baskets and rugs etc. in order to earn a few shillings. Further, when the patient is a married man, I have found that his wife often takes casual work, such as scrubbing out schools, office premises or cinemas. The money obtained from such work, is very welcome, and creates a greater working margin for the patient's needs, and for the running of the house. As often as not, no official return is made for casual earnings of this type, so that the invalid head of the house is usually quite comfortable in every way.

It may seem surprising that so many individuals fail to make returns for casual labour, but it is

is a very common practice in Industrial Lanarkshire, where unemployment is rife. At the present time I know of many able-bodied unemployed, who are doing casual jobs such as carrying coals in Glasgow, assisting in licensed premises on Saturdays, or acting as caddies at the local golf clubs. Even the man who comes to wash my car in the mornings, admits rather confidentially, that he makes no return at the Labour Exchange of money received for such work. This man was employed by a local firm of undertakers for thirty years, but when the firm changed from horse drawn vehicles to motor cars, his services were no longer required. This practice has become a fine art with many of these men, and they have continued unmolested for years.

A female insured worker, stricken by an incapacitating malady like Disseminated Sclerosis, finds, on relinquishing her employment, that her Approved Society pays her the magnificient sum of I2/- per week, if I04 or more contributions have been made, and 7/6 weekly, if 26 to I04 only have been made. Again, Public Assistance relief has to be applied for, and the ignominy of becoming a pauper is her lot, through no fault of her own.

Approved Society payments are thus seen to be ridiculously low, with the result that many decent working people are forced to use up their life savings in times of illness, and many do so, rather than live on the ratepayers. When the illness is prolonged, as in Disseminated Sclerosis,

self-support and pride are overcome by poverty in the end, and relief has to be sought.

It is thus easily realised, that prolonged sickness and poverty go hand and hand, especially among the great working classes of the people. In my opinion, individuals, such as those suffering from Disseminated Sclerosis, should be looked after by the nation, in a much more adequate manner than at the present time. If we consider the fact that sufferers from this disease are mostly young people in the prime of life, some just at the beginning of promising careers, then we must realise how unfortunate they are to be so afflicted, and to be gradually reduced to poverty, with prospects of nothing beyond a Poor Law Hospital.

What could be done to better their lot in life? I would venture to suggest that the founding of a colony, maintained by the nation, for the reception and care of these patients, would be a great advance in this direction. One hospital of such a kind would be sufficient for the whole of Great Britain, and once established, intensive research work could be carried out, with a view to finding the cause of the disease, and a successful therapeutic measure. The money would be well spent on such a venture as this, and the required amount would be infinitely less than the Government is at present spending on other extensive health schemes of less importance.

I am of the opinion that substantial findings would accrue from such concentrated research, and that until such measures are taken, the etiology of this disease will remain a mystery.

In the later stages of Disseminated Sclerosis, when the patient is more or less confined to bed, it is generally the case that all his private means have been absorbed, and that his dependents are in receipt of Public Assistance money, necessary for their upkeep. They may also receive small sums of money from local charitable institutions, and benevolent societies. Sometimes the benefit derived from such organisations may be in kind, in the form of food, coals, or clothing.

In Airdrie and district, the chronic invalid is very well looked after. Public Assistance payments are made in accordance with a uniform scale of aliment, and this scale is applied in all county areas and burghs in Lanarkshire. It is not my intention, in a work of this kind, to discuss the application of this scale in detail, but for the interest of my reader, I have set out below, a list of the payments made under this scale, insofar as they would be related to a chronic invalid.

There is, firstly, an allowance for subsistence. In the case of an invalid son or daughter over eighteen years of age, the parents are allowed II/- per week. A single person over 2I

years of age, receives I5/- per week. A married couple living with a relative, are allowed 22/6 weekly, or if living in a rented house, 2I/- weekly. An additional 2/6 weekly is paid for each child, but the maximum payment is restricted to 35/- per week.

Secondly, there is an allowance for rent and rates, with a minimum weekly payment of 3/6, and a maximum of I0/- . National Health Insurance payments are, of course taken into consideration. The accepted regulation throughout the country is that the first 7/6 derived from this source is not counted in the assessment, for the purpose of applying the scale. That is to say, that a married man, with a wife and six children, who is in receipt of sick benefit at the rate of I5/- per week, will receive 27/6 weekly from Public Assistance, instead of 35/-. His total income for subsistence is therefore 42/6, and with rent and rates allowance added, the total becomes £2.I2/6. per week.

This total may become still further inflated by means of casual work done by the wife, which I have already discussed. In Airdrie, we are particularly well supplied with private trusts and bequests, the annual interest from which amounts to about £300. This money is spent on food, clothes, coals, and monetary gifts to the sick and poor. Further to these sources of income, there are privately-run sick-funds, yearly societies, and aid-funds in connection with certain public works: there is also the

Female Benevolent Society, and other religious societies; and lastly, but by no means leastly, there are three Masonic Lodges in the burgh, each in a sound financial state, which give liberally to a brother in need.

Thus we see, that in a small provincial burgh like Airdrie, the chronic invalid is well catered for, and need want for nothing. I have known several families, where the children were all of school age or under, and the father a chronic invalid, to have weekly incomes in the region of four pounds per week.

No effort of mine, to describe the lot of the paralysed patient at home, would be complete, without reference to the magnificient work carried on so willingly and efficiently, by the District Nurses. They are indeed, the general practitioner's best friend, and I should not ever wish to work in an area, where their services were not available. Fortunately, this is not likely to happen nowadays, since district nursing has been organised in nearly every town and village throughout the country. They have come into being, as an organisation, so that the sick poor may be satisfactorily nursed in their own homes. Such district nursing was first organised in Liverpool, in 1859, and it was there first shown, that not only could valuable help be given in the nursing of the sick, but that with this help could be associated, even more important assistance, in lifting a family towards independence

and moral health. This is the outstanding feature of their work in this district. In a case of Disseminated Sclerosis, the nurse is undoubtedly of great help in keeping the patient clean and comfortable, attending to the state of the skin, performing a bladder lavage, or even passing a catheter when requested, but the moral uplift resulting from her daily visit to the patient can scarcely be fully realised.

Liverpool set this wonderful machine in motion, and a few years later, other towns followed her example. In I887, Queen Victoria gave £70,000, the Womens' Jubilee Offering, in memory of her fifty years reign, to promote and encourage District Nursing, and to establish a training centre in London, where nurses could be fully equipped for this type of work. Since that time, central training schools have also been established in Edinburgh, Cardiff, and Dublin. Thus was established, the Queen's Institute of District Nursing. In I897, the occasion of Queen Victoria's Diamond Jubilee, a further sum of £48,000 was handed over to the Queen's Institute, and on the death of the Queen, a further Memorial Fund of £84,000 was contributed to the Institute.

Our Airdrie and District Nursing Association, was founded in I897, the year of the Diamond Jubilee, and became affiliated with the Scottish Branch of the Institute. Seven years later, the gift of a home for the accommodation of the three nurses, was made by a native of the

district, at a cost of £I,500. The Home was officially opened by the Countess of Eglinton and Winton in December I9o4. Besides their ordinary duties, our nurses in Airdrie now carry on several auxiliary agencies, including the care of cripple children.

Affliction's sons are brothers in distress,
A brother to relieve, how exquisite the bliss.

Burns.

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SUMMARY AND CONCLUSIONS.

I have made an intensive study of Disseminated Sclerosis, in its medical and social aspects. I have failed to enlighten the medical profession as to the cause of the disease, or to prove the efficacy of any particular line of treatment; but I have made certain suggestions for the elucidation of these mysteries, which may have far reaching effects in the hands of those more competent, and in better surroundings for research work, than the general practitioner. From my own experience among these cases, and from careful criticism of the literature, I have formed the following summarised opinions:-

1. Little or no progress has been made towards discovering the cause of this disease.
2. The attitude of the medical profession as a whole, is apathetic towards this disease, and is lacking in urge towards finding its cause.
3. No solution will be forthcoming, until colonisation is procured, for the purpose of concentrated research.
4. State grants should be awarded to sufferers, so that they might be spared the ignominy of being branded as paupers.

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