THE TREATMENT OF LOCOMOTOR ATAXIA, WITH SPECIAL REFERENCE TO ATAXIA.*

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Locomotor Ataxia (tabes dorsalis) was first described by Todd in England in 1847, and by Romberg in Germany in 1841-51, although Schermer had described the peculiar gait in 1819, and Decker in 1838 called attention to the swaying and unsteadiness with closed eyes; but the first important recognition of the disease as a clinical entity was in a paper by Duchenne of Boulogne published in 1857-58, in which he described the symptomatology under the heading of "ataxie locomotrice progressive." It is interesting to observe that in this early account of the disease the progressive character of the ataxia is noted. The cause of the condition soon became a subject of much speculation and controversy. Shock, injury, heredity, fatigue, and various other causes were all in turn suggested as causal factors. Erb and Mœbius strongly believed that the disease was syphilitic in origin, and this view was even more strongly asserted by Fournier. Hitzig, on the other hand, taught that it was the result of some toxin liberated from some unknown source as the result of accident.

The discovery of the Spirochaeta pallida by Schaudinn, and its recognition as the cause of syphilis, renewed interest in the controversy. But the failure to find the spirochaete in the tabetic lesions, the lack of success of drug treatment directed against the spirochaete, and the fact that primary and secondary manifestations of syphilis are so often absent, all failed to lend support to the purely syphilitic origin of the disease, many influential authorities considered that tabes could occur quite apart from syphilis, and some even went so far as to assert that when tabes and syphilis were both present in the same individual, the association was accidental and of no causal significance. At the same time another group of observers were inclined to

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suspect that if the spirochaete was not the cause of the disease, a toxin resulting from the activity of the syphilitic spirochaete was responsible. The introduction of the Wassermann reaction and the fact that a positive result always denoted syphilis, although its absence did not preclude it, threw further light on this subject as it was discovered that the number of tabetics in whom this reaction was positive, varied from 90 to 100 per cent.

Finer and more modern histological methods have proved that the lesion involves the sensory neurons of the spinal cord. These appear to degenerate as the result of some process at work outside the cord itself. It is not due to chronic meningitis (thickening of the pia mater), because this is not found in all cases, and in the early stages of tabes the meninges often appear normal. Nageotte considers that tabes is the result of a localised lesion of the posterior roots between the dura mater and the posterior root ganglion, that the changes extend sometimes as far as the ganglion, and that the lesion is a transverse interstitial neuritis of the posterior root fibres. Orr and Rows regard tabes as “a system lesion, which begins as a parenchymatous degeneration of the sensory protoneurons starting at the point where the neurilemma (or external sheath) is lost.” This is just about the place where the posterior root fibres pass through the pia mater. Marie and Guillian consider that there is a syphilitic lymphangitis of the posterior system of the lymphatics of the spinal cord. This system of lymphatics does not communicate with the lymphatics of the lateral and anterior columns. The lymph streams ascend in the posterior column of the spinal cord; and there is a common lymph system for the posterior roots, posterior columns and the posterior pia mater. The lymph circulates in the perivascular lymphatic spaces, and ascends the posterior roots to reach the spinal cord. Hence infection more readily reaches the spinal cord by the posterior roots than by the anterior roots where the lymph flow is outwards. Noguchi has been able to demonstrate the spirochaete in these tabetic lesions and to make cultures from them. And as no organism, apart from the spirochaete has been isolated from inflammatory tabetic foci, the evidence is clear that as syphilis is always present, it has
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a causal relationship and that the spirochæte must be regarded as the exciting organism.

This brings us to the problem why it is that only a proportion of those who suffer from syphilis develop tabes. When an infection is general the tissues are equally accessible. The tissue attacked must thus be that which affords the most favourable soil for the growth of the infecting spirochæte. The spirochæte in a new host grows best in the same tissue as that from which it is derived, as the biochemical reactions of the same tissues in the two hosts must be identical. A spirochæte growing only in the nervous system of one host not uncommonly gives rise to an infection of a second host where only the central nervous system is attacked. The strain of spirochæte is thus somewhat specific for the tissue in which it grows and which it invades. McDonagh and Ross go so far as to state that these strains may be morphologically identified, but Noguchi and others deny this. A spirochæte which has adapted itself to the biochemical conditions in the nervous system as in tabes appears to lose its capacity to attack other tissues, such as the skin and mucous membranes and deeper tissues, as such lesions are the exception in tabes, or at least such strains only grow with difficulty in such situations where the biochemical conditions are different. A particular tissue in one host may thus acquire immunity to a particular strain, and this immunity may apply not only to the host where the successful defence was established but in any freshly infected host. Thus we get an explanation why syphilis of the central nervous system is so commonly considered to be non-infective. That special strains of spirochæte exist is proved by the example recorded by Pierre Marie and others of recurrences of tabes and general paralysis in the same family, or at least traceable to a common infection. It is also to be noted that there is a tendency for the cardio-vascular system to be attacked at the same time as the nervous system, for instance, aortic lesions not uncommonly accompany tabes; among ninety-nine tabetics Matsunaga found fifteen cases of cardiac disease. Cardio-vascular lesions are in fact one of the important causes of death in tabes. The longer the active period of the invasion lasts the less the primary tends to be the sole site.

The involvement of the posterior sensory fibres leads to
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disturbance of the reflex arc with loss of knee-jerks and other reflexes depending on the particular root involved. As a rule, before destruction of these fibres takes place, there is a period of irritation, so that exaggeration of reflex action usually precedes loss, but this period may be of very brief duration and is often overlooked. The so-called "lightning" pains develop at this time. Interference with the reflex arc result in hypotonia, the muscles tend less and less to resist stretching and allow unusual degrees of flexion and extension.

The posterior roots, however, not only carry sensory impulses centralwards, they also carry vaso-dilator impulses peripheralwards. The sensory nerve fibres at the periphery of the body bifurcate, one branch passing to the sensory end organ, the other branch ending in a blood-vessel. The hyperæmia which ushers in the whole process of the inflammatory reaction, result from an axon reflex limited to the two branches of this terminal bifurcation. Section of a sensory nerve does not interfere with this reaction, but if sufficient time elapses to allow the peripheral divided portion of the fibre to degenerate then this hyperæmic reaction cannot occur, and immediate death of the tissue occurs following injury. Thus perforating ulcers and other trophic phenomena are characteristic of tabes, and the swelling of joints, termed "Charcot's" joints, which are painless, is another example of the same condition. Although the afferent nerve roots carry sensory impulses centralwards only a small proportion of these enter consciousness as sensations of pain, touch, heat or cold. A large proportion of the afferent impulses are concerned with posture and remain on the physiological level. These arise from nerve fibres terminating in the tendons, muscles and joints.

But the main symptom with which we are concerned this evening is the ataxia. Ataxia, as a rule, develops gradually and insidiously. The tabetic begins to find he is not so sure of himself when he shuts his eyes, he tends to fall into the basin when he washes his face, he is no longer so certain where the ground is, and he becomes unsteady in his movements. Walking downstairs begins to present unusual difficulties and he becomes quickly fatigued. His uncertainty in movement necessitates great care in walking and he has to watch everything he does. These difficulties increase and he begins to

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find his movements becoming more and more awkward, jerky, and difficult. This inco-ordination increases until the whole process becomes chaotic. Later, the problem of movement becomes so difficult and complex that the tabetic surrenders to what he regards as the inevitable, and he leads an existence restricted at first to an arm-chair and later to bed. Hence the classification of tabes into the three divisions of (1) the preataxic, (2) the ataxic, and (3) the "paralytic" stages.

Ataxia, although usually of slow and gradual development, may develop suddenly. Dr James Taylor had a tabetic under his care at the National Hospital, London, who, while on duty as a policeman, captured a criminal after a severe and exhausting chase, but once he had captured the man he became suddenly so acutely ataxic that he had to be carried to hospital there and then. Maloney has pointed out that ataxia may develop acutely, not only following excessive fatigue but also from fear, and he records the case of an elderly man who, when visiting an underground cave lit by electricity, felt frightened at being so far under the earth alone with strangers. Suddenly the electric light failed and his worst fears seemed to be confirmed. He collapsed, had to be carried out of the cave to a local hospital where he was found to be a typical tabetic with an incapacitating degree of ataxia. Maloney has called this the ataxic moment, i.e. the moment when ataxia appears suddenly; at a definite moment before this movement is apparently normal, after this it is chaotic. The cause is obviously the state of fear or fatigue which immediately preceded it, without which it would have developed gradually and slowly in all probability. Clearly there is no question of a sudden increase in the extent of the lesion. It is an immediate effect and entirely mental.

The importance of this acute onset of ataxia is the fact that it is mental in origin, and thus it enables us to realise that possibly when the ataxia is slow and gradual in onset it may also simply be because the change in the mental outlook is slower in taking place. The onset and increase of ataxia has no relationship to any increase in the organic lesion in the posterior roots. A tabetic in the pre-ataxic stage may pass on to the ataxic stage and even to the "paralytic" stage without
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any increase in the content of the syphilitic processes involving his posterior roots. All that is happening is that he is changing his mental outlook in relation to his walking and a progressive mental deterioration is setting in.

The influence of the mental state on posture is well known. The difference in attitude of the tired from the vigorous, of the depressed from the excited, and of the frightened from the courageous is obvious. And in addition we are accustomed to use words to describe mental states from their resultant attitudes, such as “spine-less,” “weak-kneed,” “lacking in backbone,” “upright,” etc.

The effect of blindness on ataxic tabetics is remarkable, as a blind tabetic is usually not ataxic. Blindness is stated to accompany tabes in only about 8 per cent. of the cases, and thus the opportunities to observe the effect of blindness are not numerous, especially as the blindness may develop before the ataxia. A blind tabetic does not usually become ataxic, and an ataxic tabetic who becomes blind readily and frequently loses his ataxia. Benedikt of Vienna, in 1881, asserted that tabes associated with rapid optic atrophy shows benign spinal symptoms, and in 1887 he proclaimed a law that the specific motor symptoms of tabes would diminish in severity immediately the disease became complicated by optic atrophy, no matter how advanced these symptoms might be, and he also asserted that he knew of no exception to this law. That improvement and even disappearance of ataxia may occur following the onset of blindness has been confirmed by many observers. According to Mott 50 per cent. of cases with optic atrophy later develop general paralysis of the insane which takes them out of the category of tabes. The fact that ataxia may disappear with the onset of blindness is important in enabling us to understand the nature of the mental change which results in ataxia. Blindness simplifies the mental life as it cuts out visual images. The blind tabetic leads a life of enforced inaction. He only reacts to such simple reflex problems as are able to penetrate his restricted life. He thus worries less, and he is less fatigued.

When the seeing tabetic begins to find that his postural images are beginning to mislead him, and that he is beginning to fail to localise exactly where his limbs are in space, he
substitutes for these postural images visual images, and in this way is able to get the information he requires. But the substitution of a visual image for a postural image throws the whole spinal reflex integration out of gear, and is the substitution of an inefficient for an efficient method, of a complex integrative action for a simpler one, and the ataxia is the symptom of the resultant muscular incoordination.

When a seeing tabetic recognises that his postural images are misleading, and commences to rely upon visual images in their place, his next tendency is to repress these misleading postural images as far as he can. Just as a case of diplopia finds two visual images misleading, and finally adjusts himself by repressing the false image, so the tabetic attempts to make a similar adjustment, but in this case the final result is not so successful, as unfortunately it is the true or postural image which is repressed.

Movements are originally learned by watching the earlier attempts and controlling and directing them by vision. If the movement is not exactly what is intended, the error is seen, recognised and corrected. By concentrating attention on these various postures we gradually learn to associate a postural image with each position, and with repetition and practice we learn to know from postural information alone the position of any part of the body in space. As these movements become more and more established, visual control is unnecessary and superfluous, and is finally dispensed with and remains in reserve, only to be called into service if required. The more visual direction is utilised, the more the mental effort required, and the greater the resultant fatigue.

Postural reflex control of movement is more efficient than visual control because postural control is effected at the physiological level, and is not subject to the same degree of fatigue as visual or conscious control which is effected at the psychological level. It is a simpler reflex integration and is therefore more efficient. A substitution of visual for postural control is thus a retrogression and a return to a more primitive and cruder arrangement. It is a conscious process substituted for an unconscious.

Visual stimuli far outnumber all the other sensory stimuli
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from the sensory fields. They thus excite far more associations than any other tactile, auditory or sensory image. Effortless attention is largely characteristic of vision. This external distraction and competition of visual images for attention makes it necessary to minimise their influence. Hence teatasters and wine connoisseurs close their eyes, when they wish to appreciate a flavour, as they smell or taste. An expert pianist keeps his eyes fixed on his music and allows his fingers to wander up and down the keyboard of the piano controlled only by postural information. A good shot keeps his eyes on the bird and brings his gun into line entirely by posture. In golf, the reason we are told to keep our eye on the ball is so that there may be fixation of the visual image. The swing may then be carried out entirely by postural information. Removal of the eye from the object on which it is fixed in the above movements immediately throws the automatic co-ordinated muscular action out of gear and a disintegration of the reflex pathways occurs with resulting chaos. Thus in the tabetic, the substitution of visual images for postural images in walking results in inco-ordination and chaos. Once this substitution is made, attempts to repress the postural images lead to an aggravation of the condition until the arrangement become unworkable and the tabetic takes to bed.

The reason why blindness in tabes is rarely accompanied by ataxia is because a tabetic who is blind is unable to make a substitution of postural images by visual images, and thus is forced to rely solely upon his postural images. If he becomes blind in the pre-ataxic stage the ataxic stage will not develop, and if he becomes blind after the ataxic stage is present he is forced to re-learn his postural images and thus his ataxia disappears.

Postural images may be developed if special attention be directed to this particular end. Jugglers and acrobats require to do this, and are obviously successful. But probably no better example or illustration could be suggested than that of Blondin who was able to walk across the Falls of Niagara on a tight-rope.

The line of treatment for ataxia is thus evident. The ataxic tabetic has found his postural information diminished
and delayed by the lesion in the posterior root. The sensory impulses are fewer, feeble and misleading. He becomes conscious that his information of posture is erroneous, that his gait is becoming clumsy and awkward in consequence, and that his equilibrium is affected. He attempts to substitute visual information for the misleading postural information, and finally tends to repress this latter altogether. He throws out of gear his whole integrative pathways, substitutes a more complex one in its place, changes an unconscious physiological process into a conscious psychological one, with resulting disintegration of nervous function. But all this time no further structural change is taking place necessarily in his posterior roots. The organic lesion may be just the same in the pre-ataxic stage as in the atactic or paralytic stages. All that is happening is a progressive mental deterioration which is running its course independently of the organic lesion which was responsible for originating the condition and which itself may be very largely stationary.

Treatment obviously requires (1) removing the dependence on visual images, and (2) re-learning the postural images. To a large extent treatment has thus to be directed towards improving the mental state. Visual images may be cut out by blindfolding the patient, but this is not always necessary. He must not however be allowed to watch his movements. The presence of Romberg's sign and the commencement of ataxia are to be regarded as signs that inadequate mental control over movement has set in. If this mental factor is clearly understood and the necessary mental treatment given in the pre-ataxic stage, ataxia need never develop, except as the result of some sudden calamity. The treatment in the pre-ataxic stage consists of an endeavour to prevent the patient from having to undergo any acute mental stress, to limit his capacity of action to what his mental energy will permit, to prevent fatigue, and to establish complete and proper mental control of all his doings and movements.

A tabetic who, however, has progressed to the atactic stage is merely a tabetic whose mental stage has deteriorated below the level for effective action, who has begun to lose confidence in his postural images and is trying to substitute for them visual images. This complication in his reflex
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paths, however, completely deranges all his old landmarks, and every movement merely tends to decompose further his mental state. As the more he walks the worse his ataxia becomes, he becomes dull and depressed, as it is difficult to see how a symptom which is constantly being aggravated can be cured.

When standing, the average tabetic stands with his feet wide apart and with his heels closer together than his toes. His weight is largely placed on his heels and the toes tend to cling to the ground. The antero-posterior and transverse arches give way and the hypotonic muscles elongate. Such a foot affords a most inadequate basis for support. The only stable element in it is the os calcis, and the rest of the foot may move as a flail attached to the ankle and be little better than a flabby conglomeration of bones and muscles. The ligaments stretch beyond functional utility. This unusual distribution of the weight of the body on the heel and clutching of the ground with the toes, leads to blisters and perforating ulcers if the ordinary healthy trophic functions of the tissues are impaired. Needless to say, the lack of support and stability which such a stance causes in addition to his tabetic swaying results in uncertainty and fear. Every step is accompanied by a lavish expenditure of muscular and nervous energy out of all proportion to what the situation demands and rapidly produces fatigue. Thus his troubles multiply and he goes from bad to worse.

The normal position for the feet in standing is when each external malleolus is in the same vertical plane as the great trochanter, and the long axis of each foot directed forward with the feet more or less parallel. In this position the weight of the body is distributed across the longitudinal and transverse arches and is directly transmitted to the ground by a tripod, consisting of (the internal tubercle of) the os calcis, the sesamoid bones and head of the first metatarsal and the base of the fifth metatarsal. The tabetic foot must be so supported that the weight is distributed in the above manner on this tripod, and for this purpose a cast of the foot is taken first at rest and second with the full weight of the body on it, so that the exact points upon which the weight is falling, the degree the natural arches have given way and the amount of spread in all
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directions permitted by the overstretched muscles and ligaments may be understood. From these a special boot is made which corrects these faults and supplies the support properly on to the normal pressure points. The patient now can use his whole foot and not merely his heel as a support and immediate relief is experienced. He feels he has a secure and rigid base. He is more stable, and feels it. He re-learns his postural images for his feet. He fears less, and as fear begets ataxia and ataxia begets fear, his increased reassurance improves his gait. This artificial support to the foot is merely a temporary expediency until his own muscles and ligaments recover their tone by appropriate exercises.

A record of the gait is best taken on paper, the feet being dipped in potassium permanganate and his walking-stick, if used, has some wool at the point also so dipped. This will give us an indication and measurement of the degree of ataxia present.

Next we must consider the fact that most of the muscles of the limbs and body are also in a hypotonic state and need support until they also are strengthened. Abnormal mobility of joints must therefore be prevented. The restricting apparatus must be light, and a simple belt or bandage is all that is required. It is simply asked to perform the duty of a temporary artificial muscle. The extent and mode of application varies with each case. For abduction and outward rotation of the leg a bandage attached to a loop at the back of the boot is carried several times spirally round the leg and attached to a belt at the waist. A corset may be considered in some cases.

A series of graduated postural exercises are now required to enable the tabetic to re-learn his postural images. A mental state as free from external and internal distractions as possible must be created. Blindfolding may be necessary to achieve the first object, although this can often be omitted. As regards internal distraction, the most important factors to eliminate are fear and fatigue. The confidence of the patient must be established, and at first he will tend to rely largely upon his teacher. Exercises must be carefully controlled and must be followed by rest and recuperation. The first necessity is to discover a position in which the relationship of the body
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to its environment is stable. This can almost always be found in the sitting attitude. Various movements and exercises can be performed under these conditions without fear on the part of the patient and can be repeated until practice has made them perfect. He now proceeds to the creeping or "all-fours" position. The amount of support required is usually not very great, because there are four points of support and in a very short time movement along the floor soon becomes easy. The head should be held well up so that movements are carried out without watching each action.

After stability and movement in the creeping position is learned, kneeling is the next advance. The head must be held erect and usually safety in the kneeling position is soon established. The walking on the knee is practised (if necessary with suitable knee-caps). This stage is usually not of long duration as it presents no great risk to the performer since he can assume the all-fours position if his balance is lost. Next he tries standing in a corner with the support of both walls. He then has to practise a series of movements of his limbs until he feels some return of confidence and has repeated every posture until it has become effortless. The series of postural images which accompany each movement must be completely re-learned. Then he can stand facing the wall, and, touching it with his hands, he moves sideways along the room. Then he can turn his back to the wall and make the same movement, both with his eyes shut and with them open. He then can make attempts to cross from one wall to the other at the corner of the room. Any return of loss of balance indicates some postural image has not been satisfactorily learned, and he must practise this over and over again until this is again learned. He can then be taught to walk about the room, first with help, second without it, to kneel down and rise up in the middle of the room away from support, to go upstairs and then downstairs, to lengthen his distance, to discard his walking-stick and finally to move about with confidence unaided in public.

No matter how advanced the degree of ataxia, improvement is quick along these lines, provided the tabetic is free from organic mental disease. The importance and significance of the
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mental factor is very strikingly thrust upon anyone attempting this method of cure. As a rule improvement is rapid, and it is specially to be observed how pain and other symptoms may diminish and disappear as freedom of movement returns. The mental relief is enormous.

Tabes is a disease which does not as a rule shorten life. In the absence of complications the life of a tabetic may almost be regarded as of average duration. The great incapacitating factor is the progressive development of the ataxia. If we are now able to prevent the development of this symptom, and to cure it if it does appear, as we now are able to do, then we practically restore the tabetic to a more or less normal existence. The treatment of the ataxic tabetic differs in no way from that of the pre-ataxic tabetic, and as the greater part of this treatment consists of the prevention of a progressive mental deterioration and demoralisation, such treatment is more or less comparable to the treatment of the syphilitic neurasthenic.

In conclusion, attention has to be drawn to the failure of metallic injections to kill the spirochæte in the central nervous system, and at best such methods of treatment can only be regarded as an attempt to reinforce the natural defence reactions of the tissues. There is too great a tendency at the present moment to direct attention to the spirochæte and too little to the host. Antisyphilitic remedies are pushed in an attempt to replace natural human defence reactions rather than to assist them. What is wanted is rather to strengthen and develop the natural defensive reactions of the tissues. But the important point is that although tabes is recognised universally as a syphilitic disease; even if antisyphilitic treatment could be successful in killing the spirochæte, such a result would have no benefit in curing tabetic ataxia because this is not primarily a direct effect of the syphilitic lesion. It is an indirect and secondary reaction due to a disturbance of the normal integrative action of the nervous system which has no proportionate relationship to the underlying organic syphilitic condition. It is a mental condition and requires mental treatment.

It has also to be noted that the understanding of the nature of ataxia and its essential prevention and curability, makes the
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classification of tabes into the pre-ataxic, the ataxic, and the paralytic stages no longer necessary. This is a classification based on symptoms and was useful in defining the usual progress of the disease. It has served its day and generation, until the advance of knowledge of the integrative action of the nervous system allows us to dispense with it. The true organic lesion in tabes appears to be a syphilitic interstitial neuritis of the posterior roots.

Tabes is an excellent example of the fact that recent investigations on the physiology of the central nervous system make it necessary for us to orientate ourselves anew in relation to nervous diseases and their symptoms. These symptoms must be interpreted with the aid of this new knowledge and treated in accordance with this new interpretation. In tabes we have a definite organic lesion producing certain structural defects and leading to certain physiological and psychological consequences. These physiological consequences bear no relation to the extent of the organic lesion and progress while the organic lesion may remain stationary. There is thus a definite divorce between the extent of the symptoms and the extent of the lesion. These symptoms may react to proper physiological or psychological treatment, and the results may appear marvellous. A neglect to realise and appreciate this relationship between symptoms and mental states, and a consequent failure to give relief, may result in such patients finding their way into the hands of the unauthorised who, ignorant of the source of symptoms, but able to influence the mental state, may be able to bring relief where misguided medical efforts have proved unavailing. What would appear to be required is that the patient must be taught through adequate mental training to defend himself against the tyranny of symptoms and to exert efficient mental control over his mental processes.

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Discussion.

Professor Bramwell said—Tabes was one of the diseases of the moment when I was studying in Paris nearly thirty years ago. Indeed the mention of tabes in the Neurological Society of Paris brought half the members to their feet. The controversies on various points were very keen and it is even reported that one member of the Society challenged another to a duel since he believed that he had not received credit for priority in relation to a certain observation. I have listened with much interest to Dr Bruce's remarks with reference to the axonal reflexes and to the part postural images play in relation to the ataxia.

Cases of tabes with pronounced ataxia, which were comparatively frequent twenty or thirty years ago, are, I venture to think, but seldom seen nowadays either in the medical wards or in those set apart for venereal disease. This is certainly my impression and I think it must be a very general experience. Assuming this to be a fact, it is difficult to avoid the conclusion that the explanation is to be found in the introduction of the arsenical preparations and the more thorough treatment of syphilis in its early stages. Dr Bruce has referred to the ataxia as the most disabling symptom of the disease. While I agree that pronounced ataxia is a very disabling symptom, there are other symptoms, such as the gastric crises, the arthropathies and severe pains, which are at least as disabling and at the same time less amenable to treatment than is the ataxia.

Dr Bruce has given us a very lucid exposition of the mechanism by which the ataxia is produced, but I would protest against the statement in the epitome of his paper as to the "failure of Frenkel's exercises to cure ataxia." In 1900 I had the opportunity of seeing something of Frenkel and his treatment of ataxia by co-ordinated exercises, and I believe that I was one of the first, if not the first, to introduce the method into this country. Frenkel's observation, that when a tabetic repeats a movement several times his co-ordination improves, was the origin of the treatment, the preferred explanation being that with the repetition of a movement the patient comes to learn the position of his limb by means of the nerve impulses carried by the afferent fibres which are still intact. I have treated many cases by this method and there can be no question that great improvement in the ataxia, corresponding very closely with the trouble taken by the physician and the patient's perseverance, may be predicted in every case, provided that the treatment is not interfered with by severe pains or crises, by a disabling arthropathy, or by mental deterioration.
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I am glad that Dr Bruce has emphasised the mental factor in relation to the ataxia. There can be no doubt that it is very important. When twenty-five years ago I wrote a paper on the treatment of tabes by Frenkel's exercises, I was under the impression that the ataxia was to be explained by defective impressions received from the periphery, but further experience has convinced me that there is often a large mental element which varies greatly in different cases. Perhaps the best illustration of this fact is to be found in instances such as Dr Bruce has mentioned, in which the patient suddenly becomes ataxic. It is inconceivable that in these cases the suddenly developed ataxia is a result of an acute organic lesion. There is often a history of a fall or of an emotional factor immediately preceding the aggravation of the ataxia and one is forced to the conclusion that the sudden increase in the ataxia is a direct consequence of loss of confidence. A fall, for instance, disturbs the patient's confidence, just as when one is learning to skate and has a fall one's confidence is shaken. The explanation must be found, as Dr Bruce has suggested, in a physiological disturbance of the postural mechanism.

As regards the question of antisyphilitic treatment. Every case must be considered as an individual problem. Cases of tabes are met with in which the condition appears to be absolutely stationary. Under such circumstances I do not think that it is necessary to emphasise the fact, if the patient does not already know it, that the disease is the result of syphilis and to advocate antisyphilitic treatment for one may thereby produce a state of misery which more than counterbalances any benefit which may be expected to result from the therapeutic procedure. In other words, the personality and mental attitude of the patient must be taken into account in every instance.

Dr Robert A. Fleming said—I agree that one rarely sees the marked cases of tabes with ataxia which one remembers in days gone by. It is a gratifying fact if it means that such cases are less common because the disease is more efficiently treated than it used to be. I noted that Dr Bruce refers to the pre-tabetic stage of the disease in his synopsis, but did not state what he meant by it in the paper. I presume he implies the earliest stages of the disease before any of the marked phenomena develop? To my mind this is a most important stage because it is then that one has the best chance of arresting the disease by vigorous treatment. I have noted in certain cases the altered zones of sensation to pain described by Gordon Holmes which may be found at the upper part of the chest. In my wards I had recently just such an early case, and these zones were helpful in enabling the patient to
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be energetically treated. The colloidal gold reaction gave in that instance a definite luetic response. I think one would be more than justified in prompt treatment with one of arsenical preparations in every early case, and certainly without any fear if there were no evidence of any optic atrophy being present. I agree with Dr Bruce that the terms pre-ataxic and ataxic do not help in describing the so-called stages of locomotor ataxia. I should like to say something in support of the value of Frenkel’s exercises which Dr Bruce criticises. I have carried out this method of treatment in many cases of ataxia both in tabes and disseminated sclerosis. In all of these I use sight greatly and I was somewhat disturbed to hear Dr Bruce’s denunciation of a method I had so long practised. I always insist on the patient slowly and carefully carrying out certain movements of legs or arms using his eyes in the process. In the case of the legs I then make him try to walk with the go-cart or some supporting apparatus, using his eyes to acquire again the postural movements he has lost. Once he has learnt to walk I make him look up to the ceiling so that he learns to dispense with his visual help. This method has yielded such excellent results in my hands that I do not feel convinced that sight is a mistake in the re-education of ataxic patients. I admit that the often-stated fact that tabetic patients who lose their sight from optic atrophy do not become ataxic. This made me listen with interest to Dr Bruce’s paper, because it made me question whether sight is as desirable a factor in the treatment of ataxia as I have always in the past considered it.

I note Dr Bruce refers to patients who have become blind in cases of tabes becoming ataxic if they regain their sight. I do not believe that any tabetic who develops primary optic atrophy ever can recover his sight although it may be possible for a tabetic who has temporarily lost his sight as a purely functional condition to regain it. Such cases are and must be very rare.

Mr Lees said—There are several statements which Dr Bruce has made in the course of his paper with which I am not in complete agreement. He spoke of the aetiology of tabes and expressed the view that it was due to a neurotropic type of Spirocheta pallida as opposed to a dermotropic type. He quoted in support of this view the fact that in many cases of established tabes there was no history of either a primary sore or a skin rash. This argument is a rather flimsy one as the history is often unreliable in such cases and not infrequently we find on careful clinical examination definite evidence of the scar of a primary lesion, while in other cases, a history of gonococcal infection is admitted and this condition in such cases may often have masked a
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Meatal or a urethral chancre. Few bacteriologists accept the view that there are different types of *Spirocheta pallida*. The question of there being a neurotropic virus was fully debated some years ago and the protagonists of this theory failed completely to establish a case for it. Speaking from actual clinical cases, there are numerous cases met with in which it is impossible to explain why one patient should have nerve syphilis and another somatic syphilis.

At the present moment I have under my care a family of six children suffering from inherited syphilis. The father died of G.P.I.; the mother suffers from somatic syphilis; one of the children is a juvenile G.P.I., the other five all suffer from varying forms of somatic syphilis such as interstitial keratitis, osteomyelitis and paroxysmal hemoglobinuria. There are many other cases one could quote to the same effect against there being a neurotropic strain of *Spirocheta pallida*.

Secondly, I should like to join issue with Dr Bruce on the statement he made that there was evidence to show that salvarsan treatment had increased the incidence of nerve syphilis. The reverse is more true; this loose statement was made a few years ago by a prominent neurologist and was completely refuted by the evidence collected by Colonel Harrison. The statement made was that the treatment given in the army, an average of 4 to 5 grammes of “914”, had given rise to an increase of neurosyphilis in those particular cases. Every clinic carrying out antisyphilitic treatment was circularised and all cases of ex-soldiers were investigated with the result that the incidence of neurosyphilis was found in these special cases to be almost a negligible figure. When we consider that the average amount of treatment then given in the army was necessarily dependent on the exigencies of the campaign and was not considered complete, the true facts brought out by Harrison are remarkable and completely contradict such statements regarding salvarsan which are made without any valid proof.

There is definite evidence, however, that lack of salvarsan and bismuth or mercury treatment will increase the incidence of neurosyphilis. The amount of treatment given to patients suffering from syphilis by medical practitioners throughout England is quite inadequate. On an average it is less than 2 grammes of the drug “914” per patient and yet the most experienced syphilologists and neurologists are of opinion that any case of syphilis, however early seen, requires a minimum of 8 to 9 grammes of “914” and with it at least 30 grammes of bismuth or the equivalent of 60 grammes of metallic mercury.

If neurosyphilis develops lack of salvarsan should be blamed and
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not the drug itself. Dr Bruce laid emphasis on the failure of salvarsan drugs to cure tabes dorsalis. Tabes is only one of the end-results of syphilis and that the true treatment of it should be the preventive treatment. If all cases of syphilis are diagnosed and treated in the seronegative stage of syphilis, before the development of a positive Wassermann and if treatment is continued for a sufficiently long period, I have no doubt whatever that tabes will become a very rare condition, much more rare than even Professor Bramwell and Dr Fleming find it now.

In addition, nerve syphilis can be diagnosed early when it is still an inflammatory lesion and curable, if every case of syphilis, while under treatment, is watched both clinically and serologically and if it is remembered that the time of infection of the nervous tissues is during the generalisation stage of the syphilis when 20 to 30 per cent. of all cases of syphilis show some pathological change in their spinal fluid. In this early inflammatory stage much can be done for them. I was glad to hear Professor Bramwell emphasise the importance of observing clinical signs and symptoms in cases of syphilis and of not relying only on highly scientific tests such as the Wassermann test. Every patient who is under treatment for syphilis requires periodic clinical observation even if he feels well and his blood gives a negative Wassermann reaction. If there is even the slightest clinical sign of nerve involvement the spinal fluid should be examined at once. In addition, it is advisable in every case of syphilis to examine the cerebrospinal fluid at the end of the first year and also before the patient is discharged as cured. When this is carried out and cases of syphilis are kept under clinical observation there are few cases of tabes that will not be diagnosed in the early pre-tabetic stage when they are curable. The question raised by Professor Bramwell as to whether we should treat all cases and especially quiescent cases of tabes is a very difficult one. It is rare in syphilis to find only one part of the body, such as the posterior nerve roots, to be involved. There may be and often is an aortitis, myocarditis or some other lesion. We must consider every case on its merits and decide whether the lesions elsewhere are not progressive and in need of treatment even if the tabes is quiescent. I agree with what Dr Bruce and others have said about the importance of treating the mental state in all cases of tabes. There is a big mental strain in every case of syphilis and syphilophobia is often much more difficult to deal with than syphilis. Everyone of these patients require encouragement and as favourable a prognosis as it is possible to give.

Dr Bruce, in speaking of the failure of drugs in the treatment of cases of tabes, mentioned that salvarsan possibly damaged the natural
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defensive mechanism of the patient against syphilis. Over-treatment may do so but careful dosage in any case of syphilis tends to do the opposite, as seen in the feeling of well-being, the increase of weight, and the physical improvement that is apparent in patients having salvarsan treatment. In addition, we have now at our disposal in cases of nerve syphilis arsenical drugs such as tryparsamide which do not act as strong treponemacidal agents and exercise only a slight influence on the Wassermann test, but which undoubtedly increase the defensive mechanism of the body and stimulate the patient's resistance. If a case of nerve syphilis is diagnosed in the early inflammatory stages and this drug is exhibited before gross damage has been done to the nervous tissues then a great deal can be done to retard the progress of the disease and assist the patient. In any case in which ataxia has developed, antisyphilitic treatment alone is of limited value unless in conjunction with it we give the patient the benefit of courses of Frankel's exercises. I have seen marked benefit from combining the two methods, re-education of the muscles and antisyphilitic treatment.

The lucid exposition of the methods advocated by Dr Bruce for dealing with ataxia interested me very much and I shall be very glad to apply the methods he has outlined and watch their effect in some of the cases which come under my care.

Dr Traquair asked whether recovery or improvement ever took place in optic atrophy in cases of tabes. He questioned the view that signs of optic atrophy or other changes in the fundus oculi contraindicated treatment by tryparsamide and suggested that, so far from this being the case, such evidence constituted an added indication for this treatment.

Dr Webster said—I think that the very pronounced cases of locomotor ataxia that we used to see were always alcoholic, and that seems to let us see into the cause of the nervous symptoms. The whole system seems to be upset, by some inflammatory process attacking the fibrous tissue, for which the spirochaete has a predilection. I believe the nervous symptoms are due to slight affection of the covering of the nerves which upsets the arc and root of the reflex, and the whole system seems to be disturbed, more so when the tissues are lowered by alcohol. If tabes develop, I have found it useful to teach the patients themselves to massage their extensor muscles—not the flexor muscles, they are strong enough to look after themselves—but the extensor muscles have lost their tone. The reflex massage of the extensor muscles will correct those symptoms which we often find in paralytic cases.
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Professor Russell said—Personally, I am very grateful to Dr Ninian Bruce for coming here to-night. I am responsible for prompting him to come, and I thank him. I am sure you all agree that the way in which he expressed his view of the pathology and development of locomotor ataxia was both lucid and beautiful. I was particularly charmed with his dealing with visual images and with postural and other images and travelling by the sensory tract and his picture of cerebral reflex disintegration, and of the recovery of the postural impression and the re-establishment in that way of physiological function, and loss of ataxia.

I was interested in the remarks made by subsequent speakers, in regard to the decrease in the number of cases of locomotor ataxia that we see, and the idea I got from the speakers was that this was the result of modern treatment. I may, of course, have misunderstood, but as it is not so very long since the spirochaete was discovered, and modern treatment followed upon that discovery, then the modern treatment can hardly be the explanation. Still I am quite sure we don't see so many. In an institution I have to deal with, I used almost always to have two or three chronic cases, and I have not had one for years. That is quite in keeping with the observations that have been made to-night, but I don't myself quite understand the cause of it. It does not seem to me that the explanation lies entirely in the newer treatment, because the newer treatment has been too recent.

With regard to treatment, I was interested in the somewhat cross opinions which have been expressed. I am not in a position to judge between these expressions of opinion on the treatment that should be adopted in these cases and therefore I leave it alone, although I am, of course, interested in the observations which Mr Lees has made in his own special department, and I think we would all have been glad if the speakers who have taken part to-night could have laid down more definitely the value of the modern specific treatment of tabes, I am not in a position to judge and I think it has not been in use long enough for anyone to be certain of its value. It is an extraordinarily difficult subject in connection with life insurance—as to how you are to regard a proposer who has had syphilis and who has been treated by modern antisypophilic methods and a positive Wassermann has become negative. How exactly are we, who have to do with insurance cases, to deal with cases of this kind? Are they to be taken as cases that are as good as cases that have not had syphilis? Then we have the unpleasant suggestion made by Dr Bruce that this modern treatment of syphilis may be, in the long run, a factor leading to the development later of nervous phenomena, including general paralysis and tabes, and
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therefore, while I appreciate the points that have been made to-night, I still want more information. Perhaps Dr Lees will, at some not distant date, give the Society a paper on this important subject.

Dr Ninian Bruce in reply said—When I started the paper I defined the main object as the question of the treatment of ataxia, and my object in doing so was partly because the treatment of ataxia along the lines I have elaborated to-night, is an interesting example of a functional nervous disturbance which is taking place, on the physiological, rather than on the psychological, level. At a time when psychological disorders of function are being discussed at such length, I thought it was important that we should remember there are physiological disorders of function which may also complicate organic nervous disease, and thus it is of considerable help in understanding many processes which are taking place in the central nervous system, if we should have a clear idea of the integration of reflex pathways, of what happens if anything should cause a disintegration of reflex action, and lastly of how the whole thing may be reintegrated; and locomotor ataxia is singularly helpful in explaining and illustrating these disturbances.

The remarks of mine to which Professor Bramwell referred were largely on subjects which are controversial, and any criticism which Professor Bramwell made upon these remarks would rather be a contribution to our knowledge than an attempt on my part to lay down a definite statement to-night. The particular point I mentioned about ataxia was the mechanical disturbance and its readjustment, and that the physiological disorder of function is not directly proportionate to the organic lesion.

Mr Lees mentioned that he had a case of syphilis with a neurotropic strain, which he used as an argument rather against the neurotropic possibility, but that seemed to me to be an argument in favour rather than against it, because general paralysis and nerve deafness are what a neurotropic strain would tend to produce.

In regard to Dr Fleming's remarks about the satisfactory results he had got from the use of Frenkel's exercises, what struck me was that he noticed great benefit when the patient did not watch every footstep. Dr Fleming said that he made the patients always look up. I think Dr Fleming is right and that actual blindfolding is not so necessary, so long as we see that the patient does not watch every movement he makes.

When I spoke of a patient who was blind and on recovering his sight became ataxic, I did not mean a patient with complete optic atrophy. As a matter of fact, I entirely agree with both Dr Traquair
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and Dr Fleming, that optic atrophy, once established, is permanent, but what I actually said was a tabetic who was "blind," and you have to remember the particular influence of what I have described as the mental chaos. If a patient is becoming blind, he often develops a large number of fears and phobias, so that the treatment of tabes is very similar to that of syphilitic neurasthenia. When I spoke of a tabetic recovering his sight, I meant recovering from the functional blindness which may be present and complicate tabes—not actually from the optic atrophy. Part of the blindness may be associated with some particular fear, and when that fear is dispersed, the sight partially recovers.

The object which I had in mind was that we might perhaps manage to acquire a certain amount of insight into these disturbances found in cases of organic nervous disease, if we learned how the integrative action of the nervous system may be altered and how much benefit can be derived by re-establishing the previous physiological pathways. I also wished to draw attention to the fact that many of the symptoms present in organic nervous disease are functional disturbances, not directly related to the organic lesion, and thus often curable if their nature is understood.