

III The Clinical symptoms of Tabes.

Having now discussed briefly the region of the human frame in which the effects of tabes are most visible and having described the morbid changes which are visible in this region, and given a short summary of the various views put forward to account for the disease, it may be as well to run over the clinical symptoms visible in an ideal case of tabes.

The sufferer is commonly a man of between five and twenty and fifty. In many cases he is of that swarthy neurotic type which furnishes the world with an undue proportion of poets, musicians and madmen. In nine cases out of ten he has had syphilis, possibly a year ago, more probably four, eight, twelve or even twenty years before.

Eye symptoms are very commonly the first to drive the patient to seek medical advice. His wife calls his attention to the fact that he has developed a squint, or he finds a dimness come over his sight and the lines of his morning paper become blurred & blotted.

Very commonly one of his eyelids drop and he finds he cannot raise it. His medical adviser on examining his eye sight finds that the visual angle is contracted, that scotomata or blurred patches occur in the ^{circle} ~~center~~ of vision, and that the pupil which has perhaps been mydriatic is now myotic. Examination shows him also, ^{perhaps.} that this pupil while accomodating itself for distance, does not answer to the stimulus of light. Atropia ~~does~~ ^{may} not cause it to dilate. An ophthalmoscopic examination may show atrophy of the optic nerve, the arteries being reduced in size and the disc grey and diminished.

There are no more constant symptoms in tabes than the pupillary ones. Vincent found them in 47 cases out of 51.

The patient returns home after this examination and may notice little or no change in his condition for years. Various little symptoms show him however that the demon which has seized him has not relaxed its grip. He may have fleeting

attacks of facial neuralgia and even of facial paralysis. Strange flushes come over him and he perspires profusely without obvious cause. Numbness and pricklings alternate in different parts of his frame. His sexual desire which has possibly for some time been inordinate begins to wane. Vague pains which have been flitting about his lower limbs and which he has probably ascribed to rheumatism, become more intense and sudden in their character until he can only compare them to electric shocks.

The sufferer's appetite has been probably capricious for some time back and his digestion uncertain. Suddenly some day after a meal he is seized by an irresistible attack of nausea. He vomits for hours, throwing up not only all that he has eaten but also many pints of a clear mucoid fluid, occasionally stained with blood or mixed with bile. The attack continues until he is utterly exhausted. This is a gastric crisis. Or it may be a violent attack of diarrhoea with tenesmus and

innumerable watery stools. Or it may be a sudden cough with difficulty of breathing, simulating whooping cough and going to such a length that the sufferer becomes black in the face and may even expire of apnoea in the paroxysms. These latter are the intestinal and laryngeal crises.

For some time the friends of the patient have observed an uncertainty in his gait, which continues until walking becomes a matter of difficulty. He himself makes the discovery some night that without a light he is helpless, and falls to the ground. With this fresh budget of symptoms he seeks his medical man once more.

On examination the latter finds that the knee-jerk is gone, and possibly the cremasteric and gluteal reflexes as well. On being asked to shut his eyes the patient totters. There is no loss of muscular power, beyond that which is inevitable in a long illness.

After this, presuming the disease continues to run its course without check

the foregoing symptoms increase in severity while others gradually develop themselves. A feeling of constant constriction round the waist or under the armpits is a common phenomenon. Renal crises in which great quantities of pale urine of low specific gravity is passed are rarer. The victim seldom escapes vesical troubles however, with cystitis and a constant desire to micturate, or the bladder may be sluggish so that there is no desire to micturate, or there may be vesical crises causing intolerable agony during many hours.

Occasionally stranger symptoms may come upon the sufferer. A small raw spot upon the plantar aspect of his foot may deepen and enlarge until a perforating ulcer is established. Or certain of his joints may become flooded by a sudden copious effusion, which rapidly bursts the ligaments, destroys the joint and causes atrophy of the articular ends of the bones. Or there may be changes in the shafts of the bones themselves

by which they ~~they~~ are rendered brittle and liable to fracture.

The skin during this time has been pallid dry and cold, and subject to various eruptions, to herpes, pemphigus, erythema, and a condition resembling ichthyosis.

As the disease progresses the sufferer gets some relief from pain, the sudden shocks dying away and being replaced by analgesia and anaesthesia. Motor paralysis and acute muscular atrophy may supervene. Slowly the unfortunate victim sinks from one gradation of misery to another and can only look forward to the death which may reach him from pure exhaustion or may come from the involvement of the vital centres in the medulla. Here are the words of Heine the great German Jewish poet when after seven years of this torture he saw the shades of death gather round his couch. They are interesting as showing the thoughts evolved by a great brain when linked to what was practically a dead body.

"Do I really exist" he writes "My

body is so shrunken that I am hardly anything but a voice. In my mattress grave in the great city I hear early and late nothing but the noise of vehicles, hammering, quarrelling and piano-strumming. A grave without repose, death without the privileges of the dead who at least need not spend any money nor write letters or books — that is indeed a pitiful condition. Long ago the measure has been taken for my coffin and my obituary; but I die so slowly that the process is tedious for myself as well as my friends. What avails me that youths and maidens crown my marble bust with laurel when the withered hands of an aged hag are putting blisters behind my ears what avails me the incense of the roses of Shiraz when in the wearisome loneliness of my sickroom I get no perfume but the smell of hot towels? But patience, everything has an end! You will one day find the booth closed where the puppet show of my humour has so often delighted you"

IV Vascular and Vasomotor peculiarities in tabes.

In a previous portion of this thesis the fact has been pointed out that given a general derangement of the whole vascular system by which its nourishing qualities should be impaired all over the body, the point at which this morbid condition would first show itself would probably be in the lower dorsal and lumbar portion of the cord. The reason for this is, as has been shown, that the vascular supply of that region is carried on under exceptional disadvantages, the vessels from above being longer and more slender than any others in the body, while from below the reinforcing branches which accompany the nerve roots are much longer and therefore weaker than those of the superior segments of the cord.

Not only might it be predicted that a vascular derangement would show itself in that region, but the posterior portion of the cord might be foretold to be the area in which such changes would first appear. The reason of this

is that ~~that~~ the vascular supply of the front of the cord is furnished from above by a single larger artery, while the posterior portion is nourished by two smaller ones, the total supply of which is not greater than that conveyed through the single anterior vessel. Now presuming that a vasomotor influence was at work (as I hope to show there is, through another chain of reasoning) it would tell more upon the part which is nourished by the two small arteries than that which depends upon the single larger one, for the smaller vessels are more susceptible to vasomotor influence and their contraction would therefore reduce the blood supply in the posterior portion of the cord, more than it would be reduced in the anterior part, even though the artery there had responded also to the vasomotor stimulus.

Now looking at the fact that the vascular arrangement of the cord is such that its weakest portion is in the posterior part of the ^{lower} dorsal and lumbar regions, and that this is the very spot which is most

frequently and most obviously the seat of the morbid changes in *tabes dorsalis*, then it is no very wild flight of fancy to surmise that there may be an intimate connection between vascular derangement and the disease in question.

If however we can prove that by the use of a drug the main action of which is to contract small arteries both clinical symptoms and pathological appearances can be produced by no means discernible from *tabes dorsalis* then I think that our surmise becomes almost a certainty. On the one hand we have anatomical reasoning to show that this portion of the cord is the one which is most susceptible to vascular changes. On the other we should have proved that a substance producing vascular changes does affect that portion of the cord, and affect it in an exactly similar manner to the disease with which we have to deal. If the second proposition be established the double chain of reasoning which links *tabes* and vascular derangement

together appears to me to be irresistible.

The peculiar influence exercised by ergot upon the spinal cord has been long recognized. When bread contaminated with ergot of rye is taken for some time either gangrene of the extremities may set in, or a peculiar disease identical with tabes. Tuzcek has had opportunities as recently as 1879 of studying an outbreak of this disease in Frankenberg in Hesse when as much as ten per cent of the flour was found to consist of ergot of rye.

In the majority of these cases the clinical symptoms were numbness, pins and needles, lightning pains, girdle pain, analgesia, staggering on closing the eyes (Bauch-Romberg's symptom) and loss of the patellar reflex (Winkler's or Westphal's symptom). The pupils were usually dilated. Thus the symptoms were identical with those of ordinary tabes dorsalis.

A histological examination of the cord proved that the pathological lesion was what might have been expected. Bardach's

columns were found to be sclerosed and in some cases Goll's were also slightly affected. The lateral and anterior columns were normal. There were numerous corpora amylacea. The principal changes in the cord appeared to be atrophy of the nerve fibres and overgrowth of the neuroglia.

Dr. R. Robert of Strasburg has within the last few months published in the "Archiv für experimentelle Pathologie und Pharmakologie" some researches of his upon the active principles of ergot which throw some light upon which of them it is which has this remarkable effect upon the human spinal cord. He has obtained three bodies from ergot, ergotinic acid, cornutin and sphacelinic acid.

Ergotinic acid appears to be a powerful depressant, causing narcosis, paralysis of the cord, and stoppage of respiration. Being a glucoside however it is split up into sugar and an inert base in the alimentary canal, so that its action

unless it is injected is of no practical importance.

Sphacelinic acid is the constituent of ergot which gives rise to the characteristic gangrene. Cocks when dosed with it rapidly lost their combs which turned black and dropped off. Intestinal derangement and profuse watery diarrhoea was a frequent result of a dose.

Sphacelinic acid, according to W^R Robert by its action on the vasomotor centre causes a spasmodic contraction of the arterioles and raises the blood pressure. He found that continuous small doses of the acid caused changes in the spinal cord with the result that the animal showed an ataxic gait and lost all power of co-ordination.

Thus we arrive at the fact that a substance which causes excitement of the sympathetic system and consequent constriction of vessels will also have the effect of causing changes in the spinal cord leading to ataxic symptoms.

Cornutrin is a new alkaloid which was discovered by Dr Robert in the course of his researches. It is a poison causing tonic and clonic spasms and epileptiform convulsions. Its presence explains the epileptic attacks which have been constantly observed as concomitants of every epidemic of ergotism.

It is still an open question whether ergot acts directly upon the vascular walls or whether it acts through the nervous system. The balance of evidence is rather in favour of its acting through the vasomotor system, when the power of the drug over involuntary muscular fibres is considered. In any case the undeniable fact remains that however produced its action is to cause spasm and of the arterioles and to raise pressure, and that this physiological action brings about among other changes, a morbid condition of the posterior columns of the spinal cord.

Looking at the matter from two entirely different points of view then, an

anatomical and a therapeutic we find ourselves led to the same conclusion, namely that an exciting influence in the system which shall act upon the vasomotor centre and produce constriction of the small arterioles, will produce all the symptoms of tabes dorsalis. Considering that no other influence as far as we know will have this effect, and that there is no other feasible explanation for the particular seat of this lesion, we may take the converse of the foregoing statement and formulate the opinion that tabes dorsalis is produced by some influence in the human frame which excites the sympathetic system and so causes constriction of the small arterioles.

Looking at the symptoms of tabes dorsalis from this new standpoint two very distinct stages can be made out, corresponding roughly with the first and second stages of Duchenne. The first is the stage of sympathetic excitement, the second the stage of sympathetic paralysis.

The first might be called the stage of the dilated pupil and the second the stage of the contracted pupil.

Let us take this theory of the disease and follow the sequence of events implied by it and see how it tallies with the actual facts of the disease.

Presuming the existence of an exciting influence in the human frame, this influence being probably the syphilitic poison in the case of tabes, it may be very long before it shows its influence upon the arterioles. In the case of ergot it was not uncommon for the ergotinic tabes to become developed long after the diseased rye had been eaten. During all that time no doubt nutrition had been to some extent impaired but so gradually that it took months or years for the system to break down under it.

This malnutrition would show itself first as has been already explained in the lower part of the spinal cord, that being the weakest point of the vascular system. It

would not confine itself to that point however but being a general process would show itself more or less in every part, the most susceptible suffering first. Thus the whole of the spinal cord would in time show traces of the morbid action, the process travelling from below upwards as fresh segments found the effect of a curtailed blood supply. Why the posterior column should be most sensitive to vascular changes it is impossible in the present state of our knowledge to say. The action of ergot indicates that it is so. Possibly the reason already put forward that its nutrition depends upon two small vessels instead of the one larger one in front, may have something to do with it.

While these changes are going onⁱⁿ the cord we should expect to have symptoms of vasomotor derangement in other parts of the body. More particularly we should imagine that the nerve centres would be highly sensitive to any interference with their blood supply, and would either show signs of deranged action or become

entirely paralyzed. Accordingly in the early stages of tabes we find many signs of derangement of the nervous centres. The first is more rarely affected than some others, but anosmia is not a very uncommon symptom. In the optic nerve various conditions have been observed some pointing to central and some to peripheral changes. These peripheral changes when they take the shape of amblyopia, or atrophy of the optic nerve are most important to our argument. Here through the ophthalmoscope we can actually see those changes going on the existence of which we have been endeavouring to establish by reasoning and deduction. What could throw more light upon the general condition which leads to tabes than the examination of the disc of a tabid patient? Here the very process is going on which is occurring in his spinal cord. There are no traces of inflammatory changes. The edges of the disc are clean-cut & well defined with none of that blurred appearance which characterizes optic neuritis. The arterioles however are notably lessened in calibre, with

the result of giving the disc a whitish greyish appearance. On account of the diminished blood supply the nerve fibrils degenerate and break down, while the neuroglia being a less sensitive element than the actual nerve tissue shows the impairment of its nutrition by changing its character and becoming coarser & more fibrous. At the same time the attenuated arteries of the retina show that this contraction of vessels is not confined to the trunk of the nerve but is a general condition.

The third nerve is frequently involved early in the disease, as is shown by ptosis resulting from paralysis of its branch which supplies the levator palpebrae superioris. Recent experiments of Bechterew have shown that the reflectory centres for the nerve fibres which contract the pupils are situated in the nucleus of the third nerve. It is probable therefore that the myosis so common in the later stages of tabes depends upon excitement of this nucleus as well as upon sympathetic paralysis.

Signs of changes in the nuclei of the

fourth and sixth nerves are not uncommon, as shown by paresis or paralysis of the recti externi or interni and the appearance of a convergent squint.

The fifth nerve shows signs of derangement early in the course of the disease. Lightning pains frequently occur along its course. Anaesthesia of the head & face is also a common symptom.

The auditory centre is frequently affected, tinnitus aurium and complete deafness being not uncommon.

The *partes dura* and the glossopharyngeal are not involved so frequently as the others, nor is the spinal accessory. The pneumogastric however shows signs of excitement along all its branches -- which signs may however be due to other causes.

Now in the case of all these derangements of nervous mechanism, many of which are temporary, passing away and then recurring again, what possible explanation can account for them.

Essai sur les symptômes céphaliques du Tabes.
Perrin. Paris. 1876

except that of vaso-motor changes in the nuclei. From what we know of the vaso-motor centre and system we know that it acts sporadically rather than continuously. The variations of the blood supply in the nerve nuclei brought about by these sympathetic influences would fully account for these sudden fleeting paralysis of motor nerves, and shocks of pain in sensory ones. This, the only explanation which appears to cover the facts, will tally exactly with the view which we have arrived at as to the nature of the changes going on in the spinal cord and also with the appearances which we actually observed in the ophthalmoscopic examination of the optic disc.

The brain is so vascular an organ that a general contraction of arterioles would not affect it to any great extent, beyond rendering it sluggish and impairing its general efficacy. Nevertheless in many cases temporary aphasia, monoplegia

and hemiplegia which can only be due to vaso motor changes show that it participates in the general condition. Lecog dwells upon peculiar apoplectic attacks which he has observed in the course of the disease and which are due no doubt to the same cause.

Thus the condition of the brain and of all the cranial nerves support the view of tabes which is founded upon the study of the anatomy of the cord and upon the action of ergot as a poison. There is another test we may apply. In addition to malnutrition of the nervous centres as a consequence of the constriction of arterioles, we should expect to find signs of impaired vitality in the body itself. Such changes would naturally be looked for in the foot as being the portion furthest removed from the circulatory centre.

This link in the chain of evidence is supplied by the perforating ulcer of the foot, a symptom which often appears early in the disease and in one

Revue de Médecine. Paris. 1882.

case quoted by Althaus was the first thing to draw attention to the patient's condition. It is a symptom analagous to those gangrenes of the extremities which appear in ergot poisoning. The constriction of the arterioles upon which we have founded our argument is sufficient to cause functional and eventually organic change in sensitive nerve centres, but is not as a rule enough to cause the coarser tissues of the body to undergo any marked change. In the case of the aged however or in those whose circulation is naturally weak the condition of affairs is different and the extremities of the body may lose their vitality to such an extent as to become gangrenous. It is in the aged usually that the symptom of perforating ulcer is seen and it is associated often with a tendency to acute bedsores which shows that it depends upon a general rather than a local condition.

There is a case which was read by Mr. Pepper before the clinical Society of London which illustrates this view of

British Medical Journal. Oct 25 1884

perforating ulcer. The woman complained of 'stinging sensations' all over her body. There was anaesthesia of both feet and loss of the patellar reflex, with partial ptosis of both eyes. A sinus in one of her feet led through a mass of heaped-up epithelium down towards the second metatarsal bone. Shortly after her entrance into hospital there was gangrene and sloughing of the skin upon the dorsum of the foot, which went so far that the foot had to be amputated. For four days after that all went well, then the heel flap sloughed away, bedsores formed rapidly over the sacrum, the lower part of the stump became black and the patient died exhausted. After death extensive degeneration of the posterior columns was found, proving the case to have been a genuine one of tabes dorsalis. The patient was sixty years of age.

In this case it was evident that the perforating ulcer and the subsequent gangrene did not depend upon any lesion of the local trophic nerves, because similar

changes were going on in the back, producing acute bed sores. The result can only be attributed to impaired circulation, and the sequence of events in the foot was exactly what would have been expected in a case of senile gangrene with atheromatous arteries.

In the cord, the nerve nuclei, the brain, the peripheral end of the optic nerve and in the foot we have symptoms all of which are consistent with the idea of excitement of the sympathetic nerve and consequent contraction of arterioles. Apart however from the vasomotor action we have other indications of sympathetic irritation.

One of the chief of these is the dilation of the pupil which almost invariably ushers in the disease. The radiating fibres of the iris are supplied by sympathetic branches from the lenticular ganglion. By their irritation the pupil is widely dilated, an effect which is increased if there is any accompanying paresis of the third nerve which supplies the circular fibres. Later on in the second stage the

sympathetic nerve becomes paralyzed and the third nerve acting unopposed produces the myosis which is characteristic. The same effect is to be observed in ergotinic tabes the pupil being largely dilated at first and afterwards myotic. The condition of the pupil may be taken as an index of the state of the sympathetic nervous system.

It has frequently been remarked that in tabes the pulse rate is frequently permanently increased, averaging from a hundred to a hundred and twenty per minute. This has been ascribed to a paralysis of the inhibitory branch of the vagus. It is quite as probable however and more in consonance with the train of symptoms cited already that the increase in the pulse rate is due to the stimulation of the accelerans branch from the cervical sympathetic ganglia. This seems the more probable as it has been recorded that ^{at} the period of gastric and other crises the pulse rate slows down, sometimes to the extent of

thirty a minute. Now the phenomena of gastric crises can only be due, according to the theory of the sympathetic origin of tabes, from a sudden paralysis of the vaso motor nerves supervening upon their excited condition. Thus the membrane of the alimentary ^{tract} canal which had been anæmic from the contraction of its arterioles, becomes in a moment extremely vascular and congested, gastric juice is poured out in great quantities, the organ becomes irritable owing to the sudden change, and there is persistent reaching, during which great quantities of the acid juice ^{are} ~~was~~ thrown up. What we may see upon the surface in some of those rare cases in which there are crises of perspiration bears out this view of the cause of these phenomena. The skin in some cases of Pithers' which I hope to refer to afterwards was intensely hyperæmic in the perspiring region, showing every symptom of the ~~a~~ condition being caused by vascular change. What better

confirmation could there be of this view than the sudden change from an abnormally high to an abnormally low pulse rate. The former testified to the accelerating influence of the excited sympathetic system upon the heart, and just at the moment when other symptoms lead us to suppose that stimulating influence to be paralyzed we find the pulse change its character completely exactly as we should expect to do were the heart under the unopposed inhibitory influence of the vagus.

In accordance with this theory of the disease the first stage of tabes is marked by a contraction of all the systemic arterioles and a consequent impairment of nutrition. In the second stage the vasomotor system is paralyzed and the vessels are dilated. There have been however such morbid changes by that time in the vascular coats, owing to the vasa vasorum being involved in the general attenuation of vessels,

that the passage of nutrient serum is interfered with and the parts around are no better off than when the vessels were constricted.

Taking the cord as being the part which is most affected by vascular change we may trace the sequence of events in it. For a long time the vessels become gradually narrower and smaller, with perhaps occasional short returns to their original calibre, for nervous influences seldom act continuously. This prolonged malnutrition tells first upon the highly-organised nervous elements in the posterior column - that being, as has been shown by studying the action of ergot, the part of the cord which is most sensitive to vascular influences. The nerve fibres begin to atrophy, to break up and to disintegrate as they do in Wallerian degeneration. In the mean time the delicate neuroglia also feels the effect of impaired nutrition. It becomes coarse and fibrous, and

takes the place to some extent of the atrophied nerve tissues. The vessels in the mean time become thickened in their muscular coats owing to its permanent ^{contraction} ~~action~~, while all the coats lose in vitality and the perivascular lymphatics become choked with ^{granular} cells and leucocytes, the products of irritation. This contracted condition of vessels would of course never be seen in the post mortem room for the reason that the fact of death would remove the nervous influence and restore the calibre.

After the paralysis of the vasomotor system which according to this ^{theory} ~~system~~ marks the second stage of the disease the vessels become irregularly enlarged. Their coats however are thickened and altered, the adventitia being studded with oil globules and granular corpuscles. These latter are seen also in the lymphatic spaces between the outer and middle coats, and even in the smallest capillaries. Along the course of the vessels numerous amyloid bodies are to

be found. The nerve fibres have wasted away to such an extent as to have almost disappeared, while their place has been taken by the altered neuroglia.

Ordoñez and Brian Lewis have as already mentioned (p. 27) laid special stress upon the evidences of vascular change. He has described what he himself ^{designated} as an 'initial lesion' of the arterioles of the posterior columns, the coats of which he found blocked up with oil globules and granular corpuscles — an appearance which he did not explain, but which according to the view worked out here would be caused by the degeneration of the coats owing to the impaired nutrition received through their vasa vasorum. Such a condition, he remarked, would conduce to imperfect exchange of nutriment between the vessels and the tissues, and hence to atrophy and sclerosis of the nervous tissues. These views of Ordoñez are borne out by the already quoted case of Buzzard's which was investigated by Brian Lewis who found periaarteritis and

will marked vascular changes. The vessels were much dilated, but irregularly so and their coats were thickened and diseased.

These cases and observations do not make it certain that the vascular changes in the cord are the primary ones. It makes it certain however that changes go on in the vessels, and in deciding the question of cause and effect it certainly seems far more probable that diseased vessels would cause changes in the regions supplied by those vessels, than that disease of those regions would cause extensive morbid changes in the supplying arteries.

As regards the theory put forward in these pages the observations of Oedonez and of Brian Lewis have a most important bearing upon it.

In studying the anatomy of the cord we made out that the lower portion of it was extremely sensitive to vascular changes.

We then proved that a drug which produced vascular changes, will also

produce, clinically and histologically the symptoms of disease in the lower part of the cord.

We then endeavoured to show that the idea of general vascular changes was consistent with the other symptoms of the disease.

We have now come to the fact that most competent observers have seen in the cord that which we should have expected to find there by our inductive method of reasoning — viz changes in the walls of the vessels.

The four fold corroboration of the view which makes tabes dorsalis a disease dependant upon vascular disturbance arising from a morbid state of the sympathetic system appears to me to be as conclusive as scientific circumstantial evidence can ever be.

There is only one other view which might be advanced to cover all these

facts, and that is that perhaps there is in the blood in such cases an irritant matter which produces its effect by a local action upon the walls of the blood vessel and not through the medium of the vaso motor system. This view becomes untenable however when one considers the evidence of sympathetic disturbance which exists apart from the state of the vessels and also takes into account such symptoms as temporary cerebral troubles, crises in various organs and sudden effusions into joints, all of which indicate abrupt alterations in the vascular supply only to be explained by nervous influences.

A good deal of attention has been directed of late, especially by French observers, to the vaso motor and secretory troubles in tabes dorsalis, and many instructive cases have been chronicled which illustrate what we may call the primary symptoms of the disease in distinction to those secondary ones which are

due to the affected cord and which have hitherto received more attention than is their due. Such symptoms as inco-ordination, anaesthesia of legs, static ataxia, loss of reflexes &c may according to this view be classed as secondary, while salivary, gastric, intestinal &c crises, perforating ulcer, joint and bone lesions of Charcot, optic atrophy and pupil symptoms, sudden flushes, perspirations, vertigo, ecchymoses may all be referred directly to the vasomotor or sympathetic system and as such are primary.

Strauss, Lépine, Parrot, Petres Putnam and Olivier are all recent French writers who have given attention to the less obvious but possibly more suggestive and therefore important symptoms of locomotor ataxy. Strauss has drawn attention to the ecchymoses of the tabic - a symptom pointing to severe spasm of the arterioles or else to deficient vitality in their walls which permit serum and colouring

'Des ecchymoses labiales à la suite de crises de douleurs
fulgurantes.' Archives de Neurologie. 1881.

matter to escape into the tissues. Strauss has seen after intense lightning pains, irregular patches of bruising to come out in the area of the pain, from the size of a sixpence to that of a five shilling piece. They are painless in themselves and become rapidly absorbed. They do not correspond with the course of any of the cutaneous nerves.

Professor Pitres of Bordeaux gives two cases in which he has observed these ecchymoses after lightning pains. Upon hearing casually of my contemplated thesis the Professor most kindly forwarded me a copy of a brochure of his published last year (*Sur quelques troubles vaso-moteurs et sécrétoires de l'Ataxie locomotrice*) from which I extract a brief epitome of these two cases, which are interesting as illustrating this rare symptom.

L—, 38 years of age, had had a chancre at 27. At 35 he had lightning pains, at 36 changes in his eye-sight, at 37 want of steadiness in his walk. He had

patches of anaesthesia in the front of his thorax. His reflexes were absent. Upon being admitted to hospital after a severe bout of lightning pains a large patch of ecchymosis was observed on the inside of his right thigh. On having his attention called to this the patient showed no surprise. He remarked that he had received no blow there and that the discolouration was due to the violent pains which he had had. He said that this appearance was no unusual thing, and that after lightning pains he had often observed bruises like the one upon his thigh. Four days afterwards he left the hospital. At that time the marks upon his leg had already begun to disappear.

M - X - had for many years been suffering for many years from severe pains coming on once or twice a month. His reflexes were absent. After an exceptionally severe bout of pain, principally in the right fore arm, he came to consult Professor Pitres. At an examination a large ecchymosis was discovered covering the seat of the pain,

which could not be ascribed to any cause except the pains. It rapidly faded away and became absorbed.

In neither of these cases was there any tenderness in the bruised parts and in both the patches had appeared in the region which had just been the seat of lightning pains.

Such cases as those of Strauss and the above-quoted ones of Pitres throw a new light upon the cause of lightning pains. If Charcot's supposition that the pains are caused by central irritation of the nerve roots were correct then it would be impossible to account for their having so powerful a peripheral effect as actually to cause ecchymosis. The inference rather appears to be that lightning pains are intimately connected with local vaso-motor spasms of such violence that the blood is forced through the walls of the blood vessels. Such an explanation would account for the fact that lightning pains are occasionally a prominent symptom in cases in which there is no

corresponding amount of spinal sclerosis.

In connection with abnormal activity of the sweat glands - another curious symptom which is probably vaso-motor in its nature, - Mons. A. Ollivier has recently made a number of observations.

In one case quoted by him, in which all the typical symptoms of tabes were present, the soles of the feet and the palms of the hands were continually covered with sweat, which was increased by any exercise or emotion. This sweat had a slightly acid reaction, and was perfectly limpid. It was inodorous on the hands but somewhat offensive on the feet. In the parts which ^{were} ~~are~~ subject to these sweats the skin was of a rosy tinge, which varied with the amount of sweating. The last point is of importance as showing that the increase of the secretion depended upon vascular changes rather than on any special excitement of sudoral nerves. In this case of Ollivier's the same affection

attached the patient's scalp, his attention being called to it by the fact of his hair becoming greasy and staining his coat-collars.

In a case quoted by Henrot a continual sweating of the feet terminated in a perforating ulcer.

I have notes of another instructive case of the same nature in the wards of Professor Pitres. In this instance the patient was troubled in addition to the ordinary symptoms of the disease, with a troublesome and persistent itching of the lower extremities. After a time these turned to a bright pink colour and were constantly bathed in a profuse perspiration. The colour of the legs was sometimes uniform and sometimes in patches. Occasionally it vanished altogether. It could be driven away by pressure and was not accompanied by any swelling or pain. There was no abnormal heat and the sweat after being wiped away could be seen reappearing in little drops. The nails of the great toes were badly nourished. There was excessive reflex excitability of the region involved

the slightest touch on the foot causing violent motion of the whole limb. The only other unusual symptom in the case was permanent flexion of the three outer fingers of the left hand. There was incoordination, Brauch Romberg's symptom, optic troubles, lightning pains and all the other symptoms of tabes. The sweating symptoms did not improve under treatment.

Putnam records a case in which the position of the sudoral disturbance was exactly the converse of the last. In it the upper portion of the patient's body was continually bathed in sweat while the lower part was perfectly dry. The administration of pilocarpine served only to make the contrast more striking.

While dwelling upon those symptoms which appear to be primarily due to vaso-motor changes, one cannot omit those salivary, gastric and intestinal troubles in which great quantities of fluid are voided.

Gastric crises, first described by Volamare in 1868 and afterwards by Charcot in 1873 are principally characterised by violent pain in the stomach, in the course of which there occurs vomiting of food and glairy fluid frequently stained with bile or blood. In some patients the paroxysms of vomiting come on without any pain. The amount of fluid thrown up is sometimes very great. In a case recorded by Putnam as much as from ten to twelve pints a day would be vomited, the attacks being followed by overpowering thirst.

It has been already remarked in these pages that the gastric attacks are accompanied by a remarkable slowing of the heart's action. According to the theory of tabes put forward here this arises from a paralysis of the sympathetic fibres which regulate the blood supply of the one, and counteract the inhibitory action of the vagus in the other.

Copious discharges of fluid from

Leçons sur les maladies du système nerveux.
Charcot.

the intestine is a not uncommon symptom in the first stage of tabes. In these cases there is no abdominal pain or rectal tenesmus. It may come on suddenly and remain in spite of all treatment for many consecutive weeks or months. This symptom is often accompanied with bladder trouble. Althaus suggests that the symptom is due to sudden loss of power in the inferior mesenteric plexus of the sympathetic.

Salivary crises are less common than the others. It occurs without any pain in the salivary gland or redness of the mucous membrane. It is not connected with the use of mercury. Putnam remarks on the suddenness of its appearance. Patients are occasionally attacked during their sleep and awake to find themselves bathed in saliva. The attack may cease as suddenly as it began, leaving no signs of irritation behind. From sixteen to twenty ounces of saliva have been known to escape in one seizure from a single patient.

Pitres describes a case where the salivary crisis came on every morning at the same hour. The patient first experienced a warmth of the dorsum of the tongue and then the saliva began to run out with such rapidity that he could only sit with a vessel upon his knees to catch it, until it stopped as suddenly as it began.

Buzzard has remarked that these crises are very often seen in those patients who are subject to Charcot's Joint lesions. This is not unnatural since the latter have also every appearance of being due to sudden vasomotor changes. Indeed the sudden effusion of a large quantity of fluid into a joint might be well called an arthrodial crisis.

This short sketch of some recent observations on different obscure symptoms of tabes, is added not so much as affording support to the view advanced here as to the origin of the disease, as to illustrate the class of symptoms which are primary in their nature, in contradistinction to those more

obvious ones which depend upon the weakened spine and nervous centres.

Looking back at the ground over which we have gone it appears to me that every successive fact points to the one unavoidable conclusion. When one considers the grey skin of the tabid patient, his flushings and perspirations, his pupil changes, the signs of impaired nutrition in his feet and other regions remote from his heart, the effusions in his joints, the atrophy in his bones, the acute bedsores, the copious discharges of fluid poured out in his salivary, gastric or intestinal crises, the transient cerebral symptoms — one cannot avoid the conclusion that a disturbance of that system which regulates blood-supply and blood-pressure is a prominent if not the most prominent symptom of the disease.

But when in addition to this one learns that there are anatomical reasons why the cord should be specially sensitive to any such influence. And when also it is shown

that a drug the action of which is to diminish blood supply produces the identical clinical symptoms and pathological changes found in the disease then I think probability deepens into certainty.

It may seem a small matter to mention and yet it shows how reliable an inductive method of reasoning such as is used here may be, when I say that having worked out the rest of the evidence I remarked to a friend that I was sure that ergot in continual small doses would produce tabes - although at the time I had never heard the fact mentioned or seen it in print. He called my attention to Tuzek's account of the outbreak of ergotinic tabes, which showed that my surmise was correct.

As to the question why the posterior columns should be specially affected it is a difficult thing to explain in our present state of knowledge. They are not the only parts affected. There is often posterior spinal meningitis, wasting of the posterior roots and

cornua of grey matter and atrophy of the anterior ganglia, Clarke's vesicular column and of the cranial nerves. If the posterior columns are most affected we may safely assume that it is on account of some such anatomical reason as is suggested on page 39.

What is the exciting influence which acts upon the vaso motor system? That again is a question which can only be answered when we know more of the chemistry of those morbid products which may vitiate the human blood. It is probable that in many cases the syphilitic poison is closely connected with it. Fournier has found syphilis in 91 per cent of his cases - Erb in 90, Althaus in 86, Ross in 95, and Vulpius in 75.

Whatever the morbid influence may be however the object of this thesis has been to maintain that tabes dorsalis depends^{primarily} upon the presence in the human system of some agent which acts upon the sympathetic~~sympathetic~~ system in such a way as to derange the vascular mechanism and thereby

impair the vitality of the different organs of the body.

In conclusion a short synopsis of the various arguments adduced in favour of this position may not be superfluous. The theory then that *tabes dorsalis* has its origin in the sympathetic and vasomotor system is founded

1. On the peculiarities of the vascular supply of the cord, which make the lower part of it very sensitive to vascular change — a step towards proving that a disease which is usually associated with the lower end of the cord is dependant upon vascular change.

2. On the action of sphacelinic acid, one of the active principles of ergot, as investigated by Robert. Here a substance which produces constriction of arterioles produces also symptoms and post mortem appearances which are identical with those of *tabes*.

3. On the changes of the peripheral end of the optic nerve which may be actually seen in many cases by means of the ophthalmoscope.

Here one can see the narrowing of the vessels, the atrophy of the nerve fibres and the deterioration of the connective tissue just as we should expect to find it in the cord.

4. On those fleeting varied changes in different nerves pointing to vasomotor influences in their nuclei.

5. On temporary monoplegia, aphasia, and other cerebral troubles

6. On perforating ulcers of the foot, malnutrition of the toenails, and other symptoms which are consistent with the theory of diminished arterial and impaired nutrition.

7. On the pupil changes, the initial mydriasis and the subsequent myosis, the first being coincident with sympathetic excitement, the second with sympathetic paralysis.

8. On The increased pulse rate frequently observed in the first stage of tabes, which accords with the idea of excitement of the cardiac branch of the sympathetic. When however gastric crisis points to temporary

paralysis of the sympathetic, the pulse slows down.

9. On the various crises, including artbrodial crises, in all of which great quantities of fluid are poured out which must indicate violent vasomotor changes.

10. On the sudoral troubles occasionally met with which are accompanied with vascular congestion of the region affected, pointing to ^{local} vasomotor paralysis.

11. On the occurrence of patches of ecchymosis occurring in connection with lightning pains, which tends to show that this symptom is connected also with spasm of the arterioles.

12. On the general protean nature of the symptoms of tabes, which can only be accounted for by its dependance upon a morbid condition of a power such as the vasomotor system, pervading the whole frame but varying in its influence upon different parts in different individuals.

13. On the actual condition of the spinal

arterioles which according to Ordoñez and Brian Lewis show clear evidences of the changes which have occurred in them.

It is no part of the object of this small thesis to go into the large and important subject of treatment. There is one note however which I should like to make. Presuming that the theory of the disease here advanced has any truth in it, the earliest condition of it is a general contraction of arterioles. Under these circumstances a drug which will dilate those vessels again ought to exert a favourable influence upon the disease. Of such drugs there are two which have a powerful action, nitrite of amyl and nitroglycerine. The former is a somewhat awkward drug to exhibit and its effect appears to be more transient than the other. Nitroglycerine however in a one per cent solution is a most handy and convenient preparation. The dose, beginning

with one drop may be safely increased to fifteen or twenty, a congestive head ache being the first sign of an overdose. I have myself taken as many as forty minimis of Murrell's solution without inconvenience.

Only on one occasion have I been able to try this drug upon a tabid patient. Empiric experiments of the sort should only be tried with the knowledge and consent of the patient, and this makes it a delicate matter for a young practitioner. He is liable to fall a victim to the 'post hoc propter hoc' fallacy, and all subsequent developments of the malady be laid to the door of that unfortunate innovation in the treatment. In this one case however the sufferer being an intelligent man I proposed to him that he should try this remedy to which he readily consented. He had loss of reflexes, Brauch Romberg's symptom, amblyopia and every other sign of the malady. For two or three weeks he appeared to improve considerably

both in his general health and in his particular symptoms. Though confined to a bath chair he pursued his avocation as a commercial traveller, and following this he passed on to another town, taking some of the drug with him. I have never heard from him since nor can I find out his address. It is such cases which tend to make medical men cynical.

Looking at the disease from this point of view we can understand how it is that occasionally very aggravated symptoms are produced by the use of ergot. Both Grassett and Charcot have recorded such cases. On the other hand in the later stage where there is vasomotor paralysis ergot would be of use in restoring the tone of the vessels.

In concluding this thesis, of the imperfect nature of which I am deeply conscious, I must acknowledge thankfully the assistance which I have received from various publications upon the subject.

Progrès Medical. Paris. 1883.

In the opening description of the cerebro-spinal tract I borrowed largely from Professor Grainger Stewart's "Introduction to the study of nervous diseases" and from Byron Bramwell's "Spinal Cord", as well as from D^r Moxon's Croonian lecture. Subsequently in epitomising the clinical symptoms of the disease and the various opinions put forward as to its genesis, I fell back upon Althaus' "Spinal Cord" and Ross' "Nervous Diseases". I subsequently quoted from Robert in the "Archiv für experimentelle pharmakologie" and from Pitzis' and Ollivier's recent brochures upon vasomotor changes. A list is appended of the other works consulted or referred to in the course of the thesis.

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