TABETIC OPTIC ATROPHY.

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Thesis for the degree of M.D.

presented by

Daniel Ferguson, M.B.,C.M.

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May 1906

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

This thesis contains the results of a study of the Clinical histories of cases of tabes Dorsalis in which optic atrophy was present. It is held by some that these cases run a course differing in some important points from those in which optic atrophy is absent. It is said for example by some authors that if optic atrophy developes early in a case of Tabes Dorsalis, ataxy never supervenes; by others, again, that if ataxy appears its advent is long delayed. It is obvious that if a contribution to the subject is to have any value, the first thing to do is to have a series of these two groups of cases - cases of Tabes Dorsalis with optic atrophy in the one group and cases of Tabes Dorsalis without optic atrophy in the other group - in which the facts are recorded with definiteness and then to analyse and compare the data furnished by the two groups.

Attention will also be directed to the condition of the pupil and the condition of the ocular muscles in Tabes Dorsalis.

The presence or absence of syphilitic infection will also be noted, and, when possible, the interval intervening between the inoculation of the syphilitic virus and the onset of Tabes Dorsalis will be indicated.

The clinical histories of the caseswill be first recorded and the results will then be examined and commented on.

Case 1. Male, aged 49, widower, a shoemaker (formerly for twelve years a soldier) by occupation, complains of severe pain in the

back and abdomen and incessant vomiting.

History. He states that he has suffered from recurring attacks of pain in the stomach and vomiting for one and a half years. His first attack occurred suddenly in the midst of perfect health and continued for a week during which time nothing would lie on his stomach. The pain and vomiting suddenly left and he felt quite well until three weeks afterwards when he was seized with similar attack. During the next nine months similar attacks occurred with increasing frequency. For the last two months he has had an attack almost every week; and on one occasion vomited half a cupful of dark blood. Between the attacks he is perfectly well. He contracted syphilis at the age of twenty-nine, eighteen years before the tabes developed. At the age of forty-seven he began to suffer with difficulty in micturition and shortly afterwards from incontinence, chiefly at night. He was also troubled with obstinate constipation. About a year ago he began to be troubled with lightening pains in his legs. He also had diplopia. For the last six months he has lost his sexual power and desire.

Present condition. Pupils unequal in size (3 mm and 2½ mm respectively) they do not contract to light but contract actively on convergence. There is well-marked thoracic and ulnar analgesia. He complains of cold feet. The calf-muscles are anæsthetic and there is some hypotonus. There is no ataxia and no Rombergism.

The knee-jerks and Achillis-jerks are brisk and equal on the two sides. The plantar reflex is normal in degree and the toe-movement on each side was flexion; the abdominal and cremasteric reflexes were not obtained. There was no optic atrophy; there was no girdle sensation and no numbness.

Case 2. Male, aged 39, married, warehouseman by occupation. He has no children. His wife had a miscarriage three years ago. Patient contracted syphilis fourteen years ago.

History. For four or five years he has suffered from pains in the legs. During the past year he had difficulty in starting micturition and the bladder was not completely emptied; has difficulty in walking in the dark and sways with his eyes shut. For some time past he has noticed that his legs would suddenly give way and had a feeling of constriction round the waist, shooting pains up the spine and down the legs, but never extending to the head; gastric crises, rectal crises, attacks of palpitation and laryngeal crises. His sight is not so good as it was.

Present condition. Deep reflexes lost, ataxic gait in first stage. Pupils unequal, Argyll Robertson. Abdominal and plantar reflexes exaggerated. No cutaneous anaesthesia; hypalgesia over thorax and in legs. Joint and bone sensibility unimpaired. Moderate hypotonus of hamstrings. No muscular wasting. No foot drop when

lying on back. Considerable static ataxia in the legs. Commencing grey atrophy of both discs.

Case 3. Male, aged 35, married, complains of a burning feeling in the legs, arms and trunk and of numbness of the hands and feet of several months duration.

History. Patient contracted syphilis at the age of 24, that is eleven years ago. For the past year he has suffered with shooting pains in the legs, body and sometimes the arms and difficulty in micturition. He has always been temperate. For several months he has been feeling out of sorts. Sometimes he suffers with sudden attacks of diarrhoea without any apparent cause.

Present condition. Patient is fat and well-nourished; tongue a little tremulous. Knee-jerks absent even on reinforcement. Achillis-jerk absent. Analgesia of calf muscles, which are firm and well-developed. Pupils are of medium size, the right a little smaller than the left. Both pupils react very sluggishly to light but quite actively on convergence. The ocular movements are natural, but he says that he frequently sees double. The optic discs are normal. There is a band of thoracie analgesia from the 2nd to the 6th ribs on both sides; marked analgesia over the left arm on ulnar and radial sides and slight analgesia on ulnar side of the right arm. There is absolutely no ataxia and no Romberism.

Case 4. Female, aged 37.

History. Patient was married at the age of 17 and eight months thereafter had a child which lived only two days. She found that her husband had venereal disease; she had a sore throat and her hair came out. She therefore left him. After his death she married again at the age of 23. She had three miscarriages and suffered with ulcerated legs. She noticed first a feeling of a cord round the waist; then rectal crises and a difficulty in holding her water two years ago. For twelve months she has suffered with pains in the legs and body and increasing difficulty in walking; frequent attacks of giddiness and flatulent eructatious with gastric distension.

Physical signs. Pupils equal, small, Argyll Robertson. Absence of deep reflexes; plantar reflexes lost; epigastric just present. Cutaneous anaesthesia from 4th to 8th segments inclusive; anaesthesia and paraesthesia of soles of feet; analgesia or hypalgesia to pricking of legs, and of thorax from 5th to 9th segments with some hyperaesthesia below and above. Marked inco-ordination and wasting of legs; no inco-ordination in hands. Marked hypotonus of Hamstring muscles.

Further note two years later. She is quite helpless; cutaneous distrubance has become more marked. Slight contraction of fields

of vision and pallor of the discs. Retching attacks without vomiting and epigastric pain. Foot drop from paralysis of the dorsal flexors; some inversion of the soles, especially of the right foot from peroneal paresis. She is now very depressed and has attempted suicide.

Case 5. Male, aged 34, married, house-painter by occupation, complains of failure of vision.

History. Patient contracted syphilis and gonorrhoea 15 years ago. He enjoyed good health until six years ago, when he began to lose the sight of his left eye; six months later the sight of his right eye became involved. Two years after his eyesight failed he began to suffer with sharp shooting pains in the legs. They have never been very frequent or severe and for the last three years have almost ceased to trouble him. About the same time (two years after the onset) he experienced some difficulty in making water, but this is now very slight. He used to be constipated but his bowels are now quite regular. The sexual function remained normal. During the past year he has had a sensation of formication in the soles of his feet, and sometimes numbness, tingling and coldness in the feet. He says that the ground feels soft under his feet, and in consequence of this want of feeling in his feet he has difficulty in going downstairs.

Present condition. V.A.R. counts fingers at 12 inches. V.A.L.

Light and darkness. He has no headache, no pain in the back and no girdle sensation. There is absolutely no ataxia and no Rombergism. - he can stand on either foot with the eyes closed with perfect steadiness. The finger-nose test is perfectly performed. There is marked analgesia of the calf muscles. There is ptosis, more marked on the left side than on the right, ocular movements normal; no nystagmus. Pupils widely dilated, the right more so than the left. Right pupil measures 10½ mm. left 8½ mm; they do not contract to light, but contract very actively on convergence. The optic discs are in an advanced state of white primary atrophy. The knee-jerks and Achillis jerks are completely absent even on reinforcement. The plantar, cremasterie and abdominal reflexes are active. A distinct band of analgesia is present on both sides of the thorax between the 3rd and 6th ribs; muscular analgesia in the calves; considerable hyperaesthesia of the soles. No crises; intelligence, memory and intellectual faculties unaffected.

He sleeps badly.

Further notes thirteen months later.

Lightening pains in the legs have returned and are now worse than they ever were. He is now markedly ataxic.

Further notes seven months later than the last note.

Ataxia is much more marked in the lower extremities. When supported on each side and told to walk, patient throws out his legs in a

wildly inco-ordinate way. He has entirely lost the sense of position and movement in the lower extremities. He has frequent attacks of severe lightening pains and urination has again become difficult.

Case 6. Male, aged 50, single, mill-band worker by occupation.

History. He contracted syphilis thirty years ago. The illness began with cold feet and pains in the legs, followed by difficulty in walking, double vision and inability to distinguish colours. Sight gradually became worse and six or seven years ago he became quite blind. About this time he also had difficulty with his water. He is now unable to stand, walk or feed himself, partly because of the loss of sense of position and partly through muscular wasting and weakness. He is much emaciated and the feet are very cold and in a position of talipes equino-varus. They are cyanosed and the skin is smooth and glossy. He is unable to turn himself over in bed and is quite helpless. All the deep and superficial reflexes are lost in the limbs and there is marked hypotonus. He has no joint sensations and he cannot tell whether there is anything in his hands or not when it is put there, and the only way he knows is to put the object up to his lips and feel with this sensitive part of the body. He is absolutely blind. The right pupil measures 6 mm, the left 5 mm. There is a cataract in the right eye. Both pupils are inactive to light and pain

and there is hardly any movement on convergence. He is unable to converge the eyeballs, although he made the effort to do so and thought he had succeeded.

Case 7. Male, aged 40, labourer by occupation.

History. Patient complains that his illness commenced seven or eight years ago with what he terms "weakness in the abdomen" and costiveness. This "weakness" gradually increased, his walking became difficult and he suffered with sharp shooting pains in the lower extremities. On two or three occasions at the commencement of the illness he saw double but is unable to remember the exact dates. He had syphilis 16 years ago.

Present condition. He is quite unable to stand or walk and only seems willing or able to move the lower extremities when he sees them. When strongly urged to try to make a step or two, one of his legs is thrown forwards with great force, the motor discharge and resulting muscular contraction being out of all proportion to the movement which the patient desires to accomplish. There is no paralysis of the muscles of the lower extremities; the muscular development (especially of the lower extremities) is poor. The power of co-ordinating the muscles of the lower extremities, when the eyes are closed, and the muscular sense in the lower extremities, seem quite lost. He is unable to appreciate differences in weight with the lower extremities. There is no loss of muscular

sense in the upper extremities.

Sensory functions. The shooting pains in the lower extremities are much less severe than they were a year ago. He often suffers with sharp shooting pains in the abdomen but these are not accompanied by gastric disturbance. The tactile sensibility of the skin of the lower extremities is practically unimpaired.

Reflexes. The knee-jerks, plantar, cremasterie and abdominal reflexes are absent, urination is precipitate and requiries straining to empty the bladder. The bowels are obstinately constipated.

Pupils. Small, the right measures 2 mm. the left 2½ mm. It is doubtful if the pupils contract to light and if they do so it is certainly very feeble; contraction on accommodation is active.

Sight both for white and colours seems good. The other special senses are normal.

Case 8. Male, aged 37, Tobacco-packer by occupation.

Patient contracted syphilis 16 years ago.

Present condition. There is paralysis of the right external rectus Argyll-Robertson pupils; pains in the legs first felt three years ago; history of several gastric crises; knee-jerks present and exaggerated; no Romberg symptom; no ataxia; no girdle sensation; no difficulty with the bladder.

Further note eighteen months later.

He is suffering with pains in the legs;- no change in condition except that there is no squint now. Knee-jerks still brisk. There is no trace of ataxic and no hypotonus.

Case 9. Male, aged 39, married, painter by occupation.

History. Twenty years ago when a soldier he contracted gonorrhoea but denies chancre or secondary syphilitic symptoms. Symptoms began nine years ago with diplopia which lasted for six months. Soon afterwards he had some numbness and loss of feeling in the legs and difficulty in walking. Seven years ago he began to suffer with occasional incontinence of ~~mine~~ urine. For the past seven years he had suffered with lightening pains in the legs, arms and trunk but not the head. He has had occasional attacks of pain in the stomach and vomiting, and pain in the bowels and diarrhoea. The difficulty in walking and ataxia which have been present since the early stage of his illness, have during the past two years become much worse and for the last two years he has been quite unable to stand and walk unless supported on each side.

Present Condition. Patient is unable to stand and walk unless assisted on each side. There is no muscular paralysis. Knee-jerks and Achillis-jerks are absent. The pupils are unequal in size, the right measures 2½ mm. the left 3½ mm. Argyll Robertson. Girdle sensation is present. There is marked anaesthesia and analgesia round the thorax and upper part of the abdomen, down

inner side of upper arm, on front of thighs and over soles of feet. He has difficulty in urination and at times incontinence. Bowels are very costive. There is loss of sexual power and desire; no ataxia in the arms.

Case 10. Male, aged 57, married, shoe-finisher by occupation. History. Patient contracted a chancre six years ago. His symtoms began five years ago when he suffered with ptosis and squint in the right eye. Under treatment the squint went away in seven weeks but left the pupil large. Two years ago the sight in both eyes became dim and got gradually worse. He experienced numbness over the breasts, arm pits and flanks. He knew that there was impaired sensation there. He had cramp in the calves but no numbness in the feet. For the last few months he has had difficulty in passing water, and has been constipated for several years. There have been no gastric crises but there is tenderness over the epigastricun. There has been no difficulty in walking. He has had loss of smell since paralysis of the right eye set in, but has a very disagreeable sensation of smell like a drain.

Physical signs. Knee-jerks present on left side, absent on the right side; hypotonus on both sides; no loss of sense of position of legs or toes, a little loss in hands; no loss of sexual power. He used to lurch to the right when he had paralysis of the right eye, the superficial reflexes are present in the trunk and lower

limbs. There is anaesthesia to light tactile impressions in 4th and 5th thoracic segments on the left side and over a region corresponding to the sacral roots. There is no thermo-anaesthesia either to cold or heat. Optic atrophy exists in both eyes and there is marked curving of the vessels which are of normal size.

Case 11. Male, aged 36, married, barman by occupation.

History. Patient says he has always been temperate and is most of the day on his feet. He denies syphilis and gonorrhoea. His wife has not had any miscarriages.

Six months ago difficulty in walking and ataxia developed; he began to suffer with sharp shooting pains in his legs, chiefly the calves, and sharp shooting pains in the back.

Three months ago he began to suffer with difficulty in passing water and dimination in sexual power and desire.

Two weeks ago he felt his legs stiff and weak, but had not noticed any difficulty in walking in the dark.

A week ago he took a along walk (3½ miles); this was followed by the development of acute ataxia and complete inability to stand and walk.

Present condition. He is completely unable to stand and walk, the result of ataxia in his lower limbs. There is absolutely no paralysis and no rigidity. There is marked hypotonus of knee and hip muscles. The muscles of the calves and thighs are firm and

less sensitive than normal to pressure. The movements of the lower limbs when lying in bed were widely inco-ordinate; the patient could not tell the direction of passive movements or the position of the limbs after passive movement; there was great impairment of the muscular sense.

The knee-jerks and Achillis-jerks were completely absent even on reinforcement; the deep reflexes in the upper limbs were all present and normal in degree. There was no ataxia of the arms. The cremasterie and abdominal reflexes were absent, the plantar reflex was absent, the toe movement being flexion. There was some difficulty in micturition owing to loss of expulsive power; the bowels were constipated. Sexual desire and power are said to be present.

The pupils are unequal and widely dilated, the right measures 8 mm. the left 10 mm. They do not contract to light and only very imperfectly on convergence. There is well marked analgesia on the chest (between the 3rd and 8th ribs), the inner sides of the arms and the greater part of the lower extremities. Sight and other special senses are normal. The optic discs are normal.

Case 12. Female, aged 48, married, sewing machinest by occupation. History. One child was born eighteen months after marriage; there had been no pregnancies since. The illness began with lightening pains and numbness in the hands, followed by ataxy.

Present condition. There is marked ataxy; Romberg's symptom is present. There is loss of joint sensation in feet and hands. The deep reflexes are lost and there is loss of sense of position in both upper and lower limbs. Superficial reflexes plantar absent, epigastric present.

Pupils unequal, the right is irregular and measures 3½ mm, the left measures 5 mm. inactive to light and accommodation. Thoracic anaesthesia is present in the area of distribution of 7th and 8th cervical, upper dorsal, 3rd 4th and 5th lumbar and 1st sacral roots and there is hyperaesthesia between. There is imperfect localisation over the lower part of the abdomen and upper part of the legs. Pricking with a pin is described as a touch all over the body. There are papery scars all over the body undoubtedly syphilitic.

Case 13. Male aged 48; married, metal polisher by occupation. History. Patient married at the age of 21 and his wife had a son twelve months after marriage and a miscarriage two years afterwards, but no children simce. At the age of 28 the patient began gradually to loss his sight and he became completely blind in about twelve months. The doctor told him that he had "white atrophy". At that time he was mentally unstable. During the last two years patient's legs have seemed to give way under him a little; he became strange

in his manner and squandered his means. Two years ago he had a fit. Four months ago he had mental symptoms, hallucinations and delusions of persecution.

Present condition. Knee-jerks are absent; sensation is apparently normal. He stands and walks very well for a blind man. The pupils are irregular and immobile, measure 3 mm, the left being slightly the larger. There is slight nystagmus, white atrophy of both discs. Memory very good. Speech, tongue & lips and facial muscles tremulous. He is quite blind.

He died two months afterwards in a state of general paralysis.

Case 14. Male, aged 45 mason by occupation.

History. Patient's wife has had sixteen children, six of whom are alive, one miscarriage, and three born dead in succession. Patient denies syphilis. Four years ago he had double vision and gastric crises.

Present condition. He has no ataxy, no pains in the limbs, no paraesthesia, no difficulty of walking in the dark. Knee-jerks are present on both sides on reinforcement; triceps and wrist-tap both obtained; no loss of sense of position; joint sensation and localisation perfect. The only symptoms are double vision and severe gastric crises. The pupils present the Argyll-Robertson phenomenon There is paralysis of the left external rectus.

Further note nine months later.

Patient still suffers with gastric crises and is very depressed. He has shooting pains down the spine, paralysis of the left external rectus, and Argyll-Robertson pupils which measure 3-5 mm on both sides. The knee-jerks are present on the right side and present on reinforcement on the left side. He suffers from retention of ~~mind~~ urine.

Case 15. Male, aged 29, married, carman by occupation. History. Patient enjoyed good health up till three years ago when he began to complain of pains in the legs; two years five months ago dimness of vision of left eye; 2 years 3 months ago dimness of vision of right eye and vision of the left eye almost gone. For two years vision of the right eye has been almost lost and for 1 year 9 months he has had unsteadiness in walking. For 9 months he has had shooting pains in the back of the head on both sides, and for one month shooting pains across front of chest. He had syphilis before marriage.

Present condition. Gait slightly ataxic, also movements of arms. Shooting pains are present as above, tingling in hands and feet. Sensibility to touch, pain and temperature good. Knee-jerks absent, also the other tendon reflexes. Sphincters unaffected. Vison - hand movements. Optic discs atrophic. Pupils unequal, loss of light reflex and nystagmus are present. Upward movement of both eyes and outward of the left is defective.

Further notes. Two years afterwards he had a fit followed by auditory hallucinations.

One year after the onset of the fits his state was as follows. Gait ataxic; he can stand well with his heels together and his eyes closed. He is quite blind. The pupils are unequal, the right being dilated and the left contracted; neither react to light. He suffers with pains in his arms and cannot touch the tip of his nose with his right forefinger or make the two forefingers meet. He is deaf in both ears but can hear loud shouting. There is nystagmus when told to look towards his right hand. There is no tremor of tongue or face but a little hesitancy and slurring of speech. He says he lost power of speech for some time seven months ago. There is white atrophy of both discs; the vessels are normal in size.

There was progressive physical and mental enfeeblement and he died nine months later.

Case 16. Male, Married, sailor by occupation.

History. Patient contracted syphilis 22 years ago. His present trouble began 19 years ago with pains in the back extending both transversly and vertically all over the back. At first the pains only lasted about a day. As soon as they became acute vomiting commenced and relieved them. There were no pains in the region of the epigastrium. At first these only came once in six months but

later they became more frequent and more severe. Five years ago the attacks of pain occurred every three months and would last three or four days. The attack of vomiting would last two or three hours but now did not relieve the pains. He now began to notice that on washing his face he had a tendency to fall down and he began to have very sharp laminating pains shooting down the legs, His sexual powers also began to decline. For the last three or four years he has had a sensation of wool under the soles of his feet. He has also had occasional difficulty in passing his water. His bowels act regularly. For the last five or six years he has had a sense of constriction round the body and synchronous with the pain. He has noticed no change in his gait and powers of walking. His chief trouble is the increase in frequency and severity of the pains and sickness. These attacks now come on every two or three weeks, and last for a few days a week. He has lost flesh during the last few years.

Present condition. Muscular power in both upper and lower extremities is good; the muscles of the legs are flabby but show no signs of atrophy. There is no inco-ordination of movement; there is slight hypotonus of muscles of right leg. Cutaneous hypaesthesia is present over the thorax but there is no definite anaesthesia. The muscular sense is unimpaired. Plantar reflex is well marked on left side, but absent on the right side; cremasteric

reflex present on right side but very sluggish, brisker on left side but less marked than normal. Epigastric reflex brisk on both sides. Knee-jerks absent on both sides. The gait is normal. He stands pretty well on one leg and stands erect with his eyes shut. The pupils are slightly contracted and unequal, the left being smaller than the right. They present the Argyll-Robertson phenomenon. Gastric crises still continue and there is diplopia owing to paralysis of the left external rectus.

Case 17. Male, aged 37, agent by occupation, complains of failing sight.

Present condition. There is concentric limitation of the fields of vision. The pupils are small, unequal and show the Argyll-Robertson phenomenon. There is no ataxy; Romberg's sign is not obtained. Shooting pains are present in the legs. The knee-jerks are absent. There are no signs of mental affection. The optic discs are in a state of atrophy. The patient has had syphilis.

Further notes five years afterwards.

He has had business worries and six months ago became very depressed and threatened to cut his throat.

Physical condition. Knee-jerks absent; walks with a shuffling gait; tremor of lips tongue; speech slurred and syllabic; exalted expression.

Mental condition. Memory impaired; no idea of time or place; indifferent to personal appearance and surroundings; delusions of wealth.

Patient died five months afterwards in a congestive seizure.

Case 18. Male, aged 48, married, ship's steward by occupation.

History. He had a hard chancre 25 years ago followed by sores on the body and arms. He was under treatment for three months. His wife had three miscarriages and one still-born child. Ten years ago (i.e. 16 years after infection) he noticed that he saw double for ten or eleven days. Twelve months ago sight began to fail in the right eye of which he has now completely lost the sight. There is also dimness of vision of the left eye. He has complained of no shooting pains numbness formication or girdle sensation or inability to walk in the dark, but for sometime he has noticed a frequent desire to make water and occasional incontinence.

Physical Condition. There is optic atrophy on both sides; pupils are unequal and somewhat irregular; the right measures 5½ mm the left 4¾ mm. they show the Argyll-Robertson phenomenon. There is limitation of the field of vision on the left side ~~that~~ but he recognises colours. Knee-jerks, right brisk, left present. There is paraesthesia, no Romberg symptom, no ataxic gait, no anaesthesia or analgesia anywhere.

Case 19. Male, aged 36, porter by occupation.

History. Four years ago patient began to suffer with giddiness, bad sight and squint. One year ago he began to suffer with staggering gait and also began to drop things. He denies syphilis; but had rheumatic fever several times, and jaundice 20 years ago.

Present condition. The gait is staggering but Romberg's symptom is absent. He says he has difficulty in walking in the dark and cutting pains in the legs, most severe in the left and suffers with severe abdominal pain at times. There is an external squint of the left eye and the movement outwards is incomplete. The pupils show no reaction either to light or on accommodation. Thereis white atrophy of both discs. The knee-jerks are present and ankle clonus is obtained. There is tenderness over the spine in the 6th dorsal, 8th dorsal and lumber regions. Muscular nutrition is good and there is no wasting. Sensation is impaired all over the face and chest and both arms except the outer side of the left arm. Sensation to cold is also slightly impaired, to heat good. Leg sensation to touch is almost absent with the exception of the soles of the feet; sensation to heat impaired. Both feet and hands are very cold. At times he cannot pass water. Colour vision is very imperfect.

Further note. Two years afterwards the patient died of general paralysis and septic meningitis.

Case 20. Male, single, labourer by occupation.

History. Patient contracted syphilis 14 years ago and has been a free drinker for the last two years. For the last eight

weeks he has had difficulty in passing water and pain when he endeavours to do so. Sometimes he has difficulty in passing motions. For the last two weeks he has had difficulty in walking. For the past four or five weeks he has had a peculiar sensation in the abdomen, such as a sense of constriction like a tight cord and at times sensation of cramp. Shooting pains have occurred in the right leg only. His vision has been failing for the last two years and now he cannot see to read.

Present condition. His present illness began with violent pain in the abdomen and vomiting so that he was thought to be suffering from acute intestinal obstruction but it was noticed that the pupils did not dilate during the paroxysms of pain and on further examination he was found to be suffering with locomotor ataxy. He is a well-developed and well-nourished man. There is no wasting of muscles and power in arms & legs is good. Hypotonus of the muscles is well marked. The buccal mucous membrane on the left side and the right side of the tongue are anaesthetic and analgesie, as well as the whole cutaneous surface of the right half of the body, but the cornea is very sensitive to the slightest touch. There is slight inco-ordination of arms and legs. Argyll-Robertson pupils are present. There is no paralysis of the external ocular muscles. There is great diminution of the field of colour vision on both sides and there is optic atrophy on both sides.

The muscular sense is much impaired on the right side of the body. The gait is ataxie. Romberg's symptom is well marked. The knee-jerks are absent on both sides. The plantar, cremasterie, and epigastric reflexes are considerably diminished.

Case 21. Male, aged 26, married, lahourer by occupation. History. Patient married five years ago and had three children alive and one dead which lived one month. Twelve months ago patient saw double and since then has suffered with shooting pains in the head, arms and legs and pains across the back. Eight months ago he suffered with weakness in the bladder and could not hold his water. Six months ago he had difficulty in walking, especially in the dark. Patient had a chancre eight years ago.

Present condition. Patient can stand with his eyes shut, his heels together, but cannot stand on his toes; he cannot stand on his left foot but can on his right. The knee-jerks are absent. The pupils are Argyll-Robertson; the left measures 5 mm. the right 4½ mm; both are irregular in outline. Sexual desire was lost four months ago but was not excessive before that time. The tongue and lips are tremulous. He has a depressed anxious expression on his face and suffers with frontal headaches. Articulation is fairly good. He has shooting pains in the back of the ear; has not slept well but has had no dreams. There is an old scar on the dorsum of the penis; the glands in the groins and neck are shotty.

The muscular strength is good; there is marked hypotonus. The plantar, cremasteric and epigastric reflexes are present. There is no loss of sense of position of the hand. Tactile sensation is good. In the left foot and lower part of leg some pricks are felt as touch or give the sensation of contact with a cold body. Pins and needles or shooting pains are present in arms legs. There is no muscular wasting, but there is slight loss of sense of position of the lower limbs. There is some slight thermo-anaesthesia in the legs and both feet. He is nearly blind in both eyes but the left is more affected than the right. There is primary optic atrophy on both sides.

Further notes nine months afterwards.

He has still pains in the legs and is quite blind. Expression is emotionless. There is marked tremor in upper lip and tongue and slight hesitancy in speech but no slurring. He has no fits but suffers with headaches occasionally. He still complains of weakness of the bladder; he can stand with his heels together but cannot stand on one foot. There is no ataxia on walking. The right pupil measures 5 mm. the left 4¾ mm; both are irregular in outline and inactive to light and pain, but react to accommodation and on convergence. On the outer peroneal surface of the right leg there is a patch of light tactile anaesthesia and a few spots scattered about on the feet and legs where he does not appreciate a prick from a touch. There are scars of heeps on the right side over the area

of the 12th dorsal. The superficial reflexes are rather exaggerated. There is no thermo-anaesthesia and little change in the sensory condition. Although blind he imagines he sees people walking in at the door and tries to get out of the way for fear of colliding with them.

Case 22. Male aged 47 years.

History. He lost his sight between 19 and 20 years of age. He accounted for this by a blow on the head when 11 years of age. Twenty-five years ago he was told that he had locomot or ataxy. He is very positive in saying that he had nothing to do with women until he went blind. He says he does not see how it can be locomotor ataxy for up to a few weeks ago he was able to walk eight or ten miles a day. Four years ago he was treated for severe gastric crises. He has now gastric crises which last two or three days. He has the feeling of a tight cord round the waist, cutting pains in the legs and a somewhat ataxic gait. Cutaneous sensation is nowhere lost in the legs, but the light tactile sensibility is lost over the thorax and back between the 4th and 10th segment inclusive. In this region there is also some hypalgesia and considerable delay. The joint sensation is good everywhere except in the great toe. The knee-jerks and triceps-jerks are lost. There is moderate hypotonus in the legs. Pupils are under atropine so that they cannot be examined. There is optic atrophy of old standing.

Syphilitic infection.

It will be noted that out of the 22 cases there was evidence that the patient had been infected with syphilis in 16 cases - a percentage of 72·7; in one case the patient was probably the subject of congenital syphilis (case 22); in one case there was no history or positive evidence of syphilis; and four patients absolutely denied that they had syphilis. Of these latter one had had gonorrhoea and the wife of another a miscarriage and 3 still-born children. In 13 of the cases the exact date of syphilitic infection could be definitely fixed and the following table is drawn up to show the interval of time that had elapsed between the syphilitic infection and the onset of tabes. The onset of the disease can, of course, only be fixed by the appearance of the first sign or symptom of the disease.

Table 1.

Table showing the interval of time intervening between the contraction of the chancre and the onset of Tabes in 13 cases.

Case.	Interval between the chancre and onset of Tabes.
1.	18 years
2.	9 "
3.	10 "
4.	18 "
5.	9 "
6.	23 "
7.	8 "
8.	13 "
10.	1 "
16.	3 "
18.	16 "
20.	12 "
21.	7 "

This gives an average interval of 11·3 years between the chancre and the onset of Tabes; the longest interval being 23 years and the shortest 1 year.

Of these 13 cases of Tabes in which the exact interval between the contraction of the chancre and the onset of the disease could be calculated, there are 8 in which optic atrophy was present and 5 in which optic atrophy was absent. If these two groups of cases are compared - in the one group the cases that had optic atrophy and in the other the cases that had not optic atrophy it will be seen that there is not much difference, in the average interval between the contraction of the chancre and the onset of Tabes, between the two groups of cases. This is shown in the two following tables.

Table. 2.

Table showing the interval of time between the chancre and the onset of Tabes in 8 cases in which Optic Atrophy was present.

Case.	Interval of time between chancre and onset of Tabes.
2.	9 years.
4.	18 "
5	9 "
6	23 "
10.	1 "
18.	16 "
20	12 "
21	8 "

This gives an average of 12 years; the longest interval being 23 years, the shortest 1.

Table. 3.

Table showing the interval of time between the chancre and onset of Tabes in 5 cases in which optic atrophy was not present.

Case.	Interval of time between the chancre and onset of Tabes
1	18 years
3	10 "
7	8 "
8	13 "
16	3 "

This gives an average interval of 10·4 years between the contraction of the chancre and the onset of Tabes; the longest interval being 18 years, the shortest 3 years.

It thus appears that the average intervals of time in the two groups of cases do not differ in any marked degree.

It is of interest to note the percentage of cases of Tabes Dorsalis in which a syphilitic history or evidences of syphilis was obtained by various authors. This is shown in the following table.

Statistics relating to syphilitic antecedents by various Authors, after Redlich.

Buzzard	45 per cent	Sequin	72 per cent.
Frankel	50·7 per cent	Collins	75 " "
Gerhardt	51 " "	Friedherin	75 " "
Bernhardt	60 " "	Voigt	76 " "
Eisenlohr	60 " "	Rumpf	85 " "
Mager	60 " "	Althaus	90 " "
Borgherini	61 " "	Raymond	90 " "
Remak	63·5 " "	Strumpell	90 " "
Gowers	70 " "	Dejerine	97 " "
Mendel	70 " "	Quinquadel	100 " "
Senator	70 " "		

The incidence of ataxy in Tabes dorsalis.

A point on which various, and sometimes contradictory, opinions have been expressed, is the relationship that exists between the optic atrophy and the motor ataxy in Tabes Dorsalis. Some of the writers on this subject will be cited further on, but meanwhile the twenty-two cases, whose histories have been recorded, will be examined from this point of view. We will divide the twenty-two cases into two groups, including in the first group those cases in which optic atrophy was present and in the second group those

cases in which optic atrophy was not present, and note in each group whether ataxy was present or absent, and when present, its degree.

There were twelve cases with optic atrophy as a prodromal symptom, and these will be first examined. In one case (Case 4) the optic atrophy was a late symptom; this case will be considered separately.

In the remaining nine cases optic atrophy was absent.

Table. 4.

Table of twelve cases of tabes Dorsalis, with optic atrophy as a prodromal symptom, showing whether ataxy was present or absent; and, when present, its degree.

Case	Ataxy	Degree of Ataxy.
2.	Present	Slight.
5	Present	Marked.
6	Present	Marked
10	Absent	
13	Absent	
15	Present	Slight.
17	Absent	
18	Absent	
19	Present	Slight
20	Present	Slight
21	Absent	
22	Present	Slight.

It thus appears that out of twelve cases of tabes Dorsalis with optic atrophy as a prodromal symptom there were -

5 cases in which there was no ataxy = 41·6 per cent

5 cases in which there was slight ataxy = 41·6 per cent.

and 2 cases in which there was marked ataxy = 16·6 per cent.

Table. 5.

Table of nine cases of Tabes Dorsalis without optic atrophy showing whether ataxy was present or absent, and when present, its degree.

Case	Ataxy	Degree of Ataxy.
1	Absent	
3	Absent	
7	Present	Marked.
8	Absent	
9	Present	Marked
11	Present	Marked.
12	Present	Marked.
14	Absent	
16	Absent	

Thus out of nine cases of Tabes Dorsalis without optic atrophy there were -

5 cases in which there was no ataxy = 55·5 per cent

4 cases in which there was marked ataxy = 44· 4 " "

Table. 6.

Table showing the incidence of ataxy in both groups of cases.

	Cases with Optic Atrophy.	Cases without Optic Atrophy.
Ataxy Absent in	41·6 per cent	55·5 per cent.
Ataxy slight in	41·6 per cent	
Ataxy marked in	16·6 per cent	44·4 per cent.

It appears, therefore, that in this series of twenty-one cases of Tabes Dorsalis ataxy developed oftener in the cases with optic atrophy than in the cases in which optic atrophy was absent in the ratio of 1·31 to 1.

It will be observed, however, that all the cases without optic atrophy, that developed ataxy, did so to a marked degree and that marked ataxy developed much less often in the cases with optic atrophy than in those without optic atrophy in the ratio of 1 to 2·6.

In case 4 the optic atrophy developed three years after the ataxy and four years after the beginning of the illness and the ataxy advanced to such a degree that the patient became quite helpless.

It has been mentioned above that it is the opinion of some observers that when ataxy manifests itself in Tabes Dorsalis in which optic atrophy is an early symptom, it does not come on so

early in the disease as in cases without optic atrophy. It is necessary therefore, to examine the two groups of cases in respect of the time incidence of ataxy.

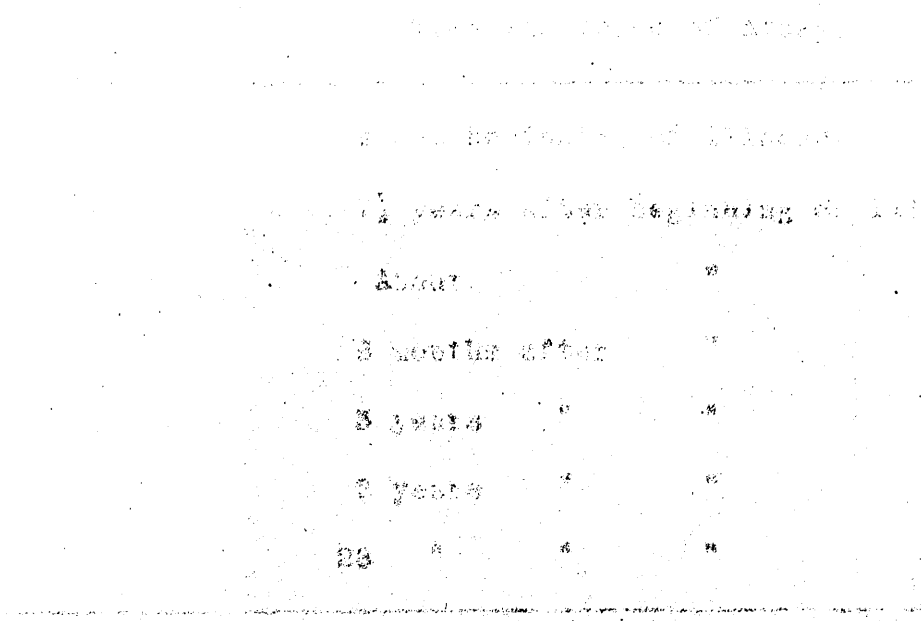

Table. 7.

Table showing the time incidence of ataxy in seven cases of Tabes Dorsalis with optic atrophy as a prodromal symptom.

Case.	Time incidence of Ataxy.
2	About beginning of illness.
5	7½ years after beginning of illness.
6	About " "
15	8 months after " "
19	3 years " " "
20	2 years " " "
22	28 " " " "

Table. 8.

Table showing time incidence of ataxy in four cases of tabes Dorsalis without optic atrophy.

Case	Time incidence of ataxy.
7	Soon after beginning of illness.
9	" " " " "
11	At " " "
12	Soon " " " "

It will be seen that, of the seven cases without optic atrophy as a prodromal symptom that developed ataxy, two became ataxic about the beginning of the illness, one 8 months after the beginning of the illness, one two years, one three years, one 7½ years and one 28 years after the illness commenced.

In the four cases without optic atrophy that developed ataxy, the ataxy came on at or soon after the beginning of the illness. With regard to the cases that have not developed ataxy we may note the time that has elapsed since the beginning of the illness up to the present time.

Table. 9.

Table showing the duration of the illness in five cases of Tabes Dorsalis with optic atrophy as a prodromal symptom, that did not develope ataxy, from the beginning of the illness up to the present time.

Case.	Duration of illness up to present time.	
10	5 years	
13	20 "	dead
17	5 "	dead
18	10 "	
21	1¾ "	

Table. 10.

Table showing the duration of the illness in five cases of Tabes Dorsalis without optic atrophy, that did not develope ataxy, from the beginning of the illness up to the present time.

Case	Duration of illness up to present time.
1	2 years
3	1 "
8	4½ "
14	4¾ "
16	19 "

We will likewise note the duration of the illness from the beginning up to the present time in those patients who became ataxic.

Table. 11.

Table showing duration of the illness from the beginning up to the present time in seven cases of Tabes Dorsalis with optic atrophy as a prodromal symptom, that developed ataxy.

Case	Duration of illness up to present time.	
2	4 years	
5	7 2/3rd "	
6	7 "	
15	6¾ "	Dead
19	6 "	Dead
20	2 "	
22	28 "	

Table 12.

Table showing duration of the illness from the beginning up to the present time in four case of Tabes Dorsalis without Optic Atrophy, that developed ataxy.

Case	Duration of illness up to present time.
7	7 years
9	9 "
11	6 months.
12	Uncertain.

It will be noticed in the history of case 15 that the slight ataxy that was present had disappeared when the patient was examined three years afterwards.

If we examine Table 9 we see that cases of Tabes Dorsalis with optic atrophy as a prodromal symptom may live for along time without becoming ataxic; the disease may even run its course up to the death of the patient without ataxy appearing.

If we look at Table 10 we learn a truth that is apparently forgotten or ignored by certain writers, namely that cases of Tabes Dorsalis without Optic Atrophy may likewise live for along time in the pre-ataxic stage.

Conclusions.

From an examination of the foregoing facts the following conclusions are warranted.

1. When optic atrophy developes in Tabes Dorsalis it does so, in much the larger proportion of cases, early in the disease; optic atrophy coming on as a late symptom is comparatively rare.

2. A patient suffering with Tabes Dorsalis, who has developed optic atrophy early in the course of the disease, may become ataxic, and may do so at any stage in the course of the disease.

3. When a patient, the subject of Tabes Dorsalis and who is ataxic, developes optic atrophy, the optic atrophy has no influence on the ataxy.

4. When a tabetic without optic atrophy becomes ataxic, the ataxy tends to come on early in the illness, and tends to be more marked in degree than in one with optic atrophy.

5. A patient suffering with tabes dorsalis and optic atrophy may live for a long period of time without becoming ataxic, and the same statement is true of a patient suffering with tabes dorsalis without optic atrophy.

6. The probability of ataxy never appearing is not greater in a tabetic with optic atrophy than in one without optic atrophy.

7. Tabes dorsalis, in a subject with optic atrophy as a prodromal symptom, may run its entire course up to death without the patient becoming ataxic.

We may now examine what has been written on the subject by various authors.

Benedikt of Vienna in 1881 (Wiener medizinische Presse, 1881. p 102) stated that the abortive cases of tabes are the cases in which optic atrophy is a prodromal symptom.

This statement is quite erroneous in both particulars. The abortive cases of tabes are not the ones in which optic atrophy is a prodromal symptom. That abortive cases of tabes sometimes have optic atrophy as a prodromal symptom is quite true, but it is quite a different statement. If we look at table 4 we see that out of 12 cases of tabes in which optic atrophy was a prodromal symptom, seven became ataxic and two of these did so to a marked degree.

In the second place there was quite a number of cases, (see Table 5) without optic atrophy which did not develope ataxy and of these (see Table 10) one had been in the pre-ataxic stage for 1 year, one for 2 years, one for 4½ years, one for 4¾ years and one for 19 years.

Benedikt in another communication in 1887 (Wiener medizinische Presse 1887, p. 11 30) stated that it is a law, from which he knew no exceptions, that the tabetic motor symptoms, no matter what

development they may have reached, vanish as soon as optic atrophy appears.

It is only necessary to refer to case 4, in which ataxy was present before the optic atrophy showed itself, but did not vanish on its appearance, to see that, whether it is a law or not, it is certainly not a law without exceptions.

On this point Raymond speaks the truth when he says - (Leçon sur les maladies du systeme nerveux second series, p 576) "I ought to add that, in the cases where - an exceptional contingency - the ambly opia appears in the ataxic period of tabes, it is without influence on the progressive evolution of the disease."

Ormerod (Allbutt's System of Medicine Vol. VII. p. 120) devotes very little space to the subject and merely makes the following remark- "Upon the general prognosticimportance of atrophy, involving as it does the prospect of utter blindness, it is unnecessary to insist. The gloomy outlook is somewhat relieved by a belief, which is commonly, entertained, that when tabes begins with optic atrophy the ataxic symptoms are likely to be less marked and less progressive than usual."

The following is an abstract of Martin's work - De l'atrophie du nerf optique et de la valeur pronostique dans la sclérose des cordons posterieurs de la moelle 1890. Thèse de Berne - which appeared in Brain XIV, p 413.

The fact that where there is atrophy of the optic nerve in Tabes Dorsalis the other symptoms of the disease remain stationary at an early stage, or attain only a slight degree of intensity has been remarked by many observers since it was first pointed out by Benedikt of Vienna, and the object of this memoir is to give more exact information on the antagonism which exists between atrophy of the optic discs and disorder of co-ordinated movement. Dr. Martin gives a summary of the writings of various authors on this subject, and states that his own observations confirm the statements of Benedikt, as to the influence of atrophy of the optic nerve on the development of Tabes in the pre-ataxic period. It is very rare for a tabetic patient, the subject of amaurosis at an early stage of the disease to become ataxic, while in many cases lightening and other pains become less severe after amaurosis is established. On the other hand he has not found that disorders of movement, if once well marked, improve with the subsequent development of optic atrophy. As a matter of fact amaurosis is rare in patients who have reached the ataxic stage, but in cases where it has occurred, no improvement in disorders of locomotion has be observed.

Out of 106 cases of tabes Dorsalis he found 21 who were amaurotic and who inaddition to optic atrophy suffered with lightening pains, loss of knee-jerks, and other unequivocal symptoms of the disease.

A full account is given of the clinical history and present state of each of the cases and the most important points are brought out in the following analysis.

The cases fall into two groups; the first group comprising Nos 1-15 inclusive, are in the pre-ataxic stage, or in other words during a greater or less number of years have developed no ataxy of movement. The duration of this pre-ataxic period has been from 3 to 33 years in different cases, and in 10 instances varies from 10 to 30 years. In the second group of 6 patients, optic atrophy came on as a secondary phenomenon to pre-existing ataxy.

Taking the first group, and leaving on one side the loss of the knee-jerks on account of the impossibility of determining the date of its disappearance, the first sign of the onset of the disease in 9 cases was the occurrence of lightening pains with or without concomitant amaurosis; in 6 patients it was ambly opia with or without oculo-motor troubles, and in three of the latter lightening pains only came on 17,18 and 19 years respectively after the development of complete amaurosis. Of 11 patients who suffered from lightening pain at the time when amaurosis was fully established, in 5 the pains have been ameliorated since the loss of sight, in 6 they have remained stationery, and in no case has an exacerbation of the pains occurred.

In two cases who showed ataxy of movement subsequent loss of

sight has been followed by relief of pain.

In the second group, where amamosis supervened on ataxic symptoms, the steady progress of the disease has been unchanged. In one case only ataxy came on 6 years after the total loss of sight, and in this patient as in the others blindness was accompanied by amelioration of pain. The degree of ataxy was slight, and another exceptional feature was the presence of Romberg's symptom, which is not generally found in blind tabetics".

I agree with Martin when he says that amaurosis supervening on ataxic symptoms has no influence on the progress of the disease. With regard to the other statements in this thesis I am convinced that ataxy supervenes as often in tabetics with optic atrophy as a primary symptom as in tabetics without optic atrophy, but there is this difference, that when optic atrophy exists the ataxy tends to be slight and tends to come on later than in tabetics without optic atrophy. But, as I have already pointed out, ataxy may come on at any period in atabetic with optic atrophy and, moreover, may be so well marked as to render the patient unable to stand or walk without support.

With regard to the pain being lessened or remaining stationary after blindness comes on, a perusal of the history of Case 5. will show that the pain may become much worse after blindness is established.

The conclusions of Gowers are in entire agreement with my

own. He says (Gowers' Manual and Atlas of Medical Ophthalmology, 4th Edition, p.p. 180 et seq) Atrophy of the optic nerve is, as is well known, frequent in locomotor ataxy. In what proportion of cases it occurs is difficulty to say. Those who observe cases from the ophthalmoscopic side are naturally impressed with its frequency, since a large proportion of cases of simple atrophy has this association. The proportion has much increase since the loss of the knee-jerks has been recognised as a sign of the early stage of tabes. But we must not infer from this the converse proposition that a large proportion of cases of tabes present optic nerve atrophy. It has been said that about 1 ataxic in 14 suffers from optic atrophy. Of the last 400 cases of tabes that I have seen optic nerve atrophy existed in 26 or 6·5 per cent. This is probably nearer the truth. When it occurs it is more often an early than a late symptom, occurring before, rather than after, difficulty in walking, and in many cases ataxy does not develope. But the frequency with which tabes does not pass beyond the first stage is great, and it is doubtful whether the frequency with which optic nerve atrophy is associated with stationary pre-ataxic tabes is greater than corresponds to the proportion of tabetics that do not advance beyond the first stage.

But optic nerve atrophy often supervenes after ataxy has developed and has become great; the loss of visual guidance

necessarily increases the inco-ordination. Further, the disorder of movement may, come on after the atrophy and at any interval. I have known atrophic blindness to be complete for 20 years before ataxy of the legs developed, which speedily became such as to prevent standing. In another case the interval was 16 years between blindness and the inco-ordination."

In a foot-note Gowers makes the following ~~Opposite~~ Opposite remark. "The fact of the frequency with which ataxy does not come on when optic nerve atrophy developes, was pointed out in the first edition of this work (1879). A few years later the fact was emphasised by Benedikt who disregarded the great frequency of stationary early tabes and enunciated a law that the development of optic atrophy tends to prevent the occurrence of ataxy. Any so called "law" easily obtained recognition, however doubtful are the facts on which it rests. Those which seem to support it, attract much more attention than those which do not."

The Ocular Muscles in Tabes Dorsalis.

It is remarkable the frequency with which diplopia is complained of in the early stage of tabes. Sometimes it is the first symptom to attract the patient's attention. It may be permanent, persisting during the patient's life, but in much the larger proportion of cases it is a transitory symptom. It may be present only for a few days or weeks and disappear altogether or

it may appear and disappear several times before finally leaving the patient.

Of the series of twenty-two cases of Tabes, 12 suffer with diplopia, a proportion of 55·5 per cent. In nine of the 12 cases the diplopia was transient (75 per cent), in the remaining three it was permanent (25 per cent).

In one case there was bilateral ptosis and one of the cases with diplopia was associated with ptosis on the same side as the ocular paralysis.

In one case there was nystagmus and in another nystagmus with defective upward movement of both eyes and outwards in one. Both of the patients with nystagmus were quite blind.

In only five of the cases of diplopia could the muscle at fault be diagnosed since the diplopia in the other cases had vanished before the patient came under observation. In four of these cases it was the External rectus (two on the right side and two on the left side) and on one the internal rectus.

The Pupils in Tabes.

Inequality in the size of the pupils on the two sides was the most constant pupillary phenomenon. It was present in 14 cases out of 17 in which the size of the pupils could be determined, equal to 82·3 per cent.

Reflex iridoplegia (the Argyll-Robertson pupil)- loss of contraction of the iris to light whilst still active on convergence- was present in 15 of 20 cases where it could be determined equal to a percentage of 75.

In one case the pupils acted sluggishly to light but were quite active on convergence; in one case the reaction to light was doubtful but on convergence the pupils were quite active; and in three cases the pupils were quite immobile.

The size of the pupils could be measured in 17 cases and the following gives the results. Out of the 17 cases the pupils were -

Of small size in 8 equal to 47·0 per cent.
" medium " " 5 " " 29·4 "
" Large " " 3 " " 17·6 "
Large on one side and contracted on the other
in one case equal to 5·8 per cent.

In four of the cases the pupils were irregular in outline; and in three of these four optic atrophy was present.

Mental Symptoms.

In conclusion I would direct attention to the remarkable fact that 6 of the 13 patients with optic atrophy developed mental symptoms. Four of these six became general paralytics and died

(Cases 13,15,17, and 19), one developed dementia (case 21) and one attempted suicide (Case 4).

It is possible that the proportion developing mental symptoms may increase for it must be remembered that they have not all finished their course.

In this connection I would quote the following remark from Mott's article on Tabes Dorsalis in the Encyclopaedia Medica Vol.12, p. 57. "It is usual for neurologists to give comfort to their patients by telling them that although they are blind, they will probably remain free from locomotor troubles for a long time to come.

There is another side of the picture which is seen especially by the alienist, and it is my experience that a large number of these cases become tabo-paralytics"

The following is the duration of the illness in the four fatal cases.

Case 13	20	years	11	months.
15	6	"	9	"
17	5	"	5	"
19	6	"	0	"

Average duration of life in 4 fatal cases 9 years 9½ months.