"sudden deaths, causes not ascertained," and "ill-defined and not specified causes," are specially large, but have been greatly decreasing in recent years.

After a further examination of the figures from other points of view, Sir John Sibbald concluded by saying that it seemed clear that the alleged increase of suicide must be regarded as not proven; and he added a protest against the too frequent habit of accepting the conclusions suggested by what he designated as "crude statistics" without giving them the critical consideration that they ought to receive.

THE EYE SYMPTOMS OF LOCOMOTOR ATAXIA, WITH A CLINICAL RECORD OF 30 CASES.

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It is now generally recognised that the disease known as locomotor ataxia may include among its clinical manifestations symptoms other than those which depend on pathological changes in the spinal cord. A number of these are associated with the functions of the eyeballs. The Argyll-Robertson pupil is universally admitted as valuable confirmatory evidence of a diagnosis of locomotor ataxia; ocular paralyse, if less frequent, are certainly not less significant; and optic nerve atrophy is at least so well known in connection with the disease that its occurrence in any individual case would hardly call for comment.

A further step forward in our knowledge of the clinical possibilities of locomotor ataxia has been the recognition of the fact that ocular disturbances may precede the evidences of any spinal lesion. This advance necessarily means that the occurrence of any one of the ocular events above mentioned must, unless otherwise explained, generate the suspicion that the case may in its later events display the phenomena known to depend upon sclerosis of the posterior columns of the spinal cord.

It is very difficult to collect the evidence necessary to show in what proportion of cases this suspicion is justified by the event. For it is certain that ocular disturbances may long precede the manifestation of spinal symptoms. In the case of optic atrophy the interval may, according to Gowers, extend even to twenty years. Thus it can only be in very exceptional instances that one and the same physician will have the opportunity of observing at least a number of these cases through all the stages of their progress. Yet, if true, it is of manifest importance, for the sake both of exact knowledge and of accurate prognosis, that it should be clearly recognised that an optic nerve atrophy, an ocular paralysis, or a loss of the pupil light reflex, unless capable of other explanation, belongs in all probability to the order of events incident to locomotor ataxia, and that any one of these may well be the introduction to a more widely-spread manifestation of the disease.

For reasons stated above, the collection of complete histories necessary to afford actual demonstration of the truth of these propositions is difficult; and all the more so as there is reason to believe that in those cases in which the early stress of the disease falls upon the nervous apparatus of the eyeball the spinal symptoms are apt to be slight in degree as well as delayed in development. This is certainly the case when the ocular disturbance takes the form of optic nerve atrophy. "In a large number of such cases," says Gowers, "ataxy never comes on, the spinal malady becoming stationary when the nerve suffers."

Of course in a given case of optic nerve atrophy without spinal symptoms the question may fairly be raised whether it is right to place such a case in the locomotor ataxia group. All that can be said in reply is (1) that from cases of optic atrophy pure and simple one passes by an unbroken series of steps through cases with more and more distinct evidence of locomotor ataxia to, at the end of the series, optic atrophy in association with characteristic ataxic symptoms, and (2) that as already stated, a simple case of optic atrophy may remain unchanged for many years, and yet in the end display undoubted evidence of the development of a spinal lesion. But if optic nerve atrophy may be the primary symptom in the

disease, if the occurrence of spinal symptoms may follow it after an interval of many years, and if again it may remain without at any time any existing ataxia, it is not unreasonable to presume that both the Argyll-Robertson pupil and an ocular paralysis may each have exactly corresponding relations to the development of the spinal evidences of locomotor ataxia. The collection of evidence to support this suggestion is even more difficult than in the case of optic nerve atrophy. The latter condition must ere long compel the patient to seek medical advice, and thus the opportunity for a complete investigation of the state of his nervous apparatus is afforded at a relatively early date. But an Argyll-Robertson pupil may exist, and presumably exist for years, without any inconvenience to the patient. Such a patient, therefore, will not consult his medical adviser until spinal or other symptoms display themselves, and thus the precedence of the pupillary condition cannot be determined. In the case of an ocular paralysis medical assistance is, no doubt, usually promptly invoked. But such an occurrence is open to a number of etiological explanations, for example, rheumatism, cold, etc., which it is difficult to exclude with confidence. Hence it is much less precise in its significance than either a double optic atrophy or the Argyll-Robertson pupil. It must be by the collection of observations extending over a long term of years that actual demonstration of the relationship of the ocular disturbances now in question to the occurrence of spinal disease can be established. But while falling short of the merit of actual demonstration, the presentation of the facts displayed by a number of cases which could only be observed over relatively brief periods is not without value. If no one case affords a complete history of all the stages of the disease the picture presented may none the less be fairly complete, provided the cases are sufficiently numerous, and they are seen at different points of development. It is believed that in the present series these conditions are fulfilled. The conclusions they afford, as far as the present purpose is concerned, are: (1) That an optic-nerve atrophy, an ocular paralysis, or an Argyll-Robertson pupil may exist as an isolated symptom for a considerable time, presumably for years; (2) that any two of these may be associated together, with a correspondingly increased presumption that the diseased process causing them is of the locomotor ataxia order; (3) that any one of the three or a combination of two or all of them may exist in conjunction with a greater or less degree of evidence of spinal disease; and (4) that occasionally a case which commences with purely ocular symptoms may be seen to develop with comparative rapidity characteristic symptoms of the spinal lesion of locomotor ataxia. The cases therefore may be held to justify the view that an optic-nerve atrophy, an ocular paralysis, or the Argyll-Robertson pupil (not capable of other explanation) must be regarded as affording a definite basis for suspicion in reference to a possible development of spinal disease. On the other hand, it must be admitted that the prognostic indication so far as spinal disease is concerned is not an absolute one, for the ocular defect may exist certainly for many years without any evidence whatever of the involvement of the spinal cord.

The cases here recorded have all been the subject of detailed and in most cases repeated examination, and unless the contrary is stated, it may be taken for granted that the thoracic and abdominal viscera are normal to physical and other methods of examination. In all cases, too, in which no specific statement is made, it is to be understood that the visual acuity, the visual fields (both for white and colours), and the fundus oculi have been proved to be normal. This last statement of course does not apply to cases in which optic atrophy exists. Particular care has been taken to be accurate in regard to the condition of the pupils and the knee-jerks. In nearly, if not absolutely in every instance where a departure from the normal is chronicled, the record has been confirmed by more than one observer, and in the case of a deficient knee-jerk the conclusion stated has never been formulated until the conditions insisted on by Gowers, Buzzard, and Jendrassik have been fulfilled. With a few exceptions in which only a single observation was possible, the patients have been watched for months, and in some instances for several years. The cases are arranged in series, with a view to show how, from a purely ocular condition, one may pass through gradually accumulating evidence to the

same ocular condition in association with characteristic signs of the spinal lesion of locomotor ataxia.

I.—CASES IN WHICH OPTIC NERVE ATROPHY¹ IS THE PRIMARY OR DOMINATING CONDITION.

(a) *Optic Atrophy without other Evidence of Disease.*

CASE I.—W. T., aged 25. Failure of vision extending over two years, with reduction of visual acuity to the power of counting fingers at 3 ft. Double optic atrophy; pupils medium, with distinct light response; knee-jerks distinct and no evidence of spinal disease, and no cerebral symptoms other than one or two attacks of giddiness. Urethritis but no syphilis.

CASE II.—F. R., aged 38. Double optic atrophy, with almost complete loss of vision, the defective sight having been observed for at least eighteen months; pupils dilated and immobile; no evidence of spinal disease, unless possibly some degree of failure of sexual power; no cerebral incidents; no history or evidence of syphilis.

(b) *Optic Atrophy with other Ocular Evidence suggestive of Locomotor Ataxia.*

CASE III.—(By permission of Mr. Ernest Clarke, F.R.C.S.) R. C., aged 39. Double optic atrophy, reducing right visual acuity to the power to count fingers at 4 ft., and left to mere perception of light; right pupil dilated and three times the size of the left; neither any light response, but free movement on convergence; entire absence of symptoms and objective signs of spinal disease; "gleet" twenty years before, no syphilis.

CASE IV.—A. S., aged 25. Double optic atrophy, with observed failure of vision for twelve months. V. A. right-hand movements only, left $\frac{1}{2}$ part; pupils 2 mm., no light response, but contract on convergence; knee-jerks difficult to obtain, but movement, though possibly wanting in promptness, is normal in extent; no ataxia or other evidence of spinal disease; mother of three healthy children, no miscarriages.

(c) *Optic Atrophy with some Evidence of Spinal Disease.*

CASE V.—W. A., aged 37. Double optic atrophy with reduction of visual acuteness to "hand movements"; pupils dilated and immobile; knee-jerks absent, but no other evidence of spinal disease; venereal sore when aged 20; no recognised secondaries, and father of four healthy children.

CASE VI.—J. G., aged 35. Failure of sight (six months); optic atrophy gradually increasing whilst under observation for twelve months; pupils not definitely abnormal; knee-jerks absent throughout, but no further appearance of spinal disturbance; urethritis, but no history of syphilis; father of two healthy children, wife no miscarriages.

CASE VII.—F. L., aged 39. Double optic atrophy, reducing visual acuteness to $\frac{1}{2}$; pupils very small, and with Argyll-Robertson phenomenon; subsequent to failure of sight (twelve months) has had shooting pains in thighs, and failure in retention power of bladder; knee-jerks distinct; no ataxia or sensory defect in lower limbs; venereal sore twenty years before; no recognised secondary syphilis; wife healthy, seven pregnancies five miscarriages.

(d) *Optic Atrophy with distinctive Evidence of Spinal Disease.*

CASE VIII.—C. H., aged 38. Failure of sight (two years) from double optic atrophy; pupils medium, with Argyll-Robertson phenomenon; moderate double ptosis, but no ocular paralysis; shooting pains in lower limbs (eight years); knee-jerks absent; considerable ataxia and failure of control over bladder; syphilis at 19 years.²

CASE IX.—G. S., aged 40. Pallor of discs and peripheral contraction of visual fields; four months later loss of knee-jerks and gradual development of ataxia; pupils normal throughout; death at the end of twelve months with symptoms of meningitis; syphilis at 25 years.

II.—CASES WITH ARGYLL-ROBERTSON PHENOMENON.

(a) *Argyll-Robertson Phenomenon without other Evidence of Disease.*

CASE X.—A. L., aged 33, the subject of slight hypermetropic astigmatism. Pupils small, not quite circular, with Argyll-Robertson phenomenon; no other ocular defect, and no evidence of a spinal lesion. No history of syphilis.

CASE XI.—K. S., aged 43. Pupils rather small, unequal, quite destitute of light response, though moving freely in convergence; no other ocular defect except some presbyopia; no evidence of spinal disease, though left knee-jerk not easily obtained. Unmarried; syphilis seems highly improbable.

CASE XII.—G. G., aged 60. Pupils small, with distinct Argyll-Robertson phenomenon. Knee-jerks not easily obtained, but not definitely abnormal, and no other evidence of spinal disease. Patient suffers from defective vision, probably from tobacco poisoning (central scotoma for red); no history or evidence of syphilis.

(b) *Argyll-Robertson Phenomenon with other Ocular Disturbance suggestive of Locomotor Ataxia.*

CASE XIII.—D. T., aged 42. Pupils below medium size, destitute of light response, with free movement in convergence; had for seven days suffered from diplopia, and under observation gradual development of complete paralysis of right external rectus; no other ocular defect. Knee-jerks distinct, and no suggestion of spinal disease; chancre of lip and secondary syphilis nine years before.

(c) *Argyll-Robertson Pupils with more or less Evidence of Spinal Disease.*

CASE XIV.—T. F., aged 55. Right pupil 2 mm., left 3 mm., each with Argyll-Robertson phenomenon; no other ocular defect except presbyopia. Ten years ago had difficulty in passing urine, and since then occasionally voids it involuntarily, and for eighteen years has been liable to seizures of pain in calves, insteps and heels; knee-jerks normal, and no objective sign of spinal disease. Venereal sore when aged 20 but no secondary symptoms, and father of six healthy children.

CASE XV.—H. W., aged 38, is the subject of hypermetropia, 4.5 D. Pupils very small, especially left; neither moves under light but distinct contraction during convergence. Admits recent difficulty in descending stairs, saying he "frequently misses the bottom step," and has suffered from "sciatica" for two years. No objective evidence of spinal disease,

and urinary and sexual functions undisturbed. Admits gonorrhoea but denies syphilis. Wife miscarried eight months after marriage; no further pregnancies.

CASE XVI.—R. S., aged 43. Hypermetropic and presbyopic. Pupils small, unequal, not quite circular, and with definite Argyll-Robertson phenomenon. Knee-jerks cannot be obtained (confirmed on three different dates), but no other sign or symptom of tabes dorsalis unless "rheumatic pains" in lower limbs for several years. Unmarried; no history of syphilis. Four years ago had, after "catching cold," to have urine withdrawn by a catheter, but no subsequent disturbance of bladder function.

CASE XVII.—E. W., aged 48. Myosis with Argyll-Robertson phenomenon; right ptosis and crossed diplopia (one month) without obvious ocular paresis; absence of right knee-jerk (confirmed on two occasions), and failure in retention power of bladder (six months), but no other evidence of spinal disease; vulval sores and skin eruption six years before.

III.—CASES IN WHICH AN OCULAR PARALYSIS IS THE EARLIEST OR DOMINATING SYMPTOM.

(a) *Ocular Paralysis without other Evidence of Disease.*

CASE XVIII.—(By permission of Mr. J. T. James, F.R.C.S.) F. D., aged 37. Dilated and immobile pupils, without any other ocular defect. No evidence of spinal disease; syphilis nine years ago; no change while under observation for three years.

CASE XIX.—K. K., aged 28—Iridoplegia, double, followed by paralysis of left external rectus, the condition being under observation for nearly a year, but without the discovery of any satisfactory explanation. No evidence of spinal disease. Married, four healthy children, no miscarriage; during one pregnancy very free loss of hair (now grown again), but no other occurrence to suggest syphilis. No family or personal history of gout or rheumatism.

CASE XX.—G. H., aged 33. Dilated and immobile pupils, with incomplete ptosis and divergence of eyeball on each side. Present condition of four years' duration, and separated by an interval of three years from venereal sore and skin eruption. No other ocular defect; no evidence of spinal disease and general health good throughout. No change while under observation for three months.

(b) *Ocular Paralysis with other Ocular Evidence suggestive of Locomotor Ataxia.*

CASE XXI.—E. S. (aged 43 years) diplopia from paralysis right external rectus, pupils small, each with Argyll-Robertson phenomenon; visual left acuity only $\frac{1}{2}$, and small but distinct central scotoma, with some contraction of the peripheral field; knee-jerks distinct and no ataxia or other evidence of spinal disease. Three early miscarriages, no full-time child. No change while under observation for nine months, but on two occasions severe attack of vomiting and abdominal pain, extending over several days and without recognised cause (? gastric crises).

CASE XXII.—A. M., aged 55. Left ptosis and paralysis of external ocular muscles supplied by third nerve in 1887, the pupils being normal, followed by incomplete recovery. In 1897 development of identical conditions on right side, and pupils found to be small and with Argyll-Robertson phenomenon; knee-jerks very slight and with great difficulty, but no other evidence of spinal disease. No history of syphilis.

CASE XXIII.—H. F., aged 37. Right ptosis with diplopia (seven days) and defective inward excursion of right eyeball; pupils very small, not quite equal, and with Argyll-Robertson phenomenon; optic discs pale and marked contraction of visual fields, but normal central vision; knee-jerks scarcely to be obtained, but no other evidence of spinal disease. No history of syphilis. Father of four healthy children. Seen after a month's interval, paralysis of all external right ocular muscles supplied by third nerve, and knee-jerks absent.

(c) *Ocular Paralysis with more or less Evidence of Spinal Disease.*

CASE XXIV.—G. S., aged 41. Ptosis and complete ophthalmoplegia externa on left side, with dilated and immobile pupils and some degree of right ptosis, these conditions or some of them having been present for five years. Knee-jerks distinct, and no ataxic phenomena, but imperfect control over bladder and failure of sexual power during last six months. No admitted syphilis.

CASE XXV.—J. L., aged 42. Diplopia and drooping left upper eyelid for four years. Ptosis left side, and marked defect of ocular movements in each eye; left pupil dilated and immobile, right small contracts during convergence, but no light response; no other ocular defect. Knee-jerk scarcely obtained on either side; no ataxia, but attacks of "twitching pains" in lower limbs, and for some time difficulty in starting the flow of urine. Venereal sore in 1882, and subsequent loss of hair, but no other secondary symptoms. Patient watched for twelve months without appreciable change.

CASE XXVI.—L. D., aged 44. Crossed diplopia (one month) without obvious ocular paralysis and pupils small with Argyll-Robertson phenomenon. Knee-jerks absent; no ataxia to usual tests, but has noticed tendency to stagger in the dark; is troubled with pains in the knees, has difficulty in commencing the act of micturition, and recent marked failure of sexual power; venereal sore at 20 years, and subsequent sore throat, but no skin eruption or loss of hair. Father of three healthy children.

CASE XXVII.—J. H., aged 47. Double vision of two months' duration; similar attack three years ago, with complete recovery. Paralysis of right external rectus; pupils, visual acuity, and visual fields normal. Knee-jerks absent, but no other evidence of spinal disease. Patient the subject of albuminuria, and presents physical evidence of an aneurysm of the ascending aortic arch. Youngest child has marked evidence of inherited syphilis.

CASE XXVIII.—W. M., aged 35. Paralysis of left third nerve, without iridoplegia or cycloplegia; pupils normal. Knee-jerks absent, but no ataxia or other evidence of spinal disease. Several venereal sores ten years ago, but no recognised secondary syphilis. There is, however, evidence of a former iritis.

CASE XXIX.—M. C., aged 54. Paralysis of left external rectus, with history of two previous attacks of diplopia during last four years; no other ocular defect unless some imperfect light response in left pupil; knee-jerks absent, and complaint of "sciatica" for two years, but no other evi-

dence of spinal disease. Albuminuria distinct, and physical signs of hypertrophy of left ventricle. No history of syphilis.

CASE XXX.—(By permission of Mr. N. M. MacLehose, M.B.) H. Y., aged 30. Homonymous diplopia observed over a period of six months without appreciable ocular paralysis; pupils of medium size, with definite Argyll-Robertson phenomenon; knee-jerks absent, and in later months decided ataxia and sensory defects in lower limb; visual acuity unaffected to ordinary tests, but gradual contraction of visual fields, especially on right side; chancre and secondary syphilis four years before.

There are in these series of cases many facts which might reasonably be made the subject of remark, and several of the cases are certainly of great individual interest. But they are here displayed in the above grouping for the purpose of illustrating the clinical order and sequence in which, as a matter of actual experience, the ocular disturbances of locomotor ataxia may manifest themselves in relation to the spinal evidences of that disease. Of course, in those cases in which there exists only a single ocular symptom unaccompanied by any sign of spinal disease, it may be objected that it has yet to be demonstrated that such cases are of the nature of locomotor ataxia. It is doubtless to be desired that such cases should be under exact observation as long as the opportunity for further developments exists—that is, for the entire life of the patient. But to insist upon such a condition is a mere counsel of perfection. One must make reasonable use of such evidence as the brevity of life and the exigencies of practice permit. And the evidence here set forth affords at least a very strong presumption, to say the least of it, of the truth of the doctrines stated in the earlier paragraphs of this paper. Probably the particular proposition which is most likely to be contested is the one which places the Argyll-Robertson pupil equally with optic nerve atrophy, and an ocular paralysis, as a possible first event in the eruption of the phenomena of locomotor ataxia. But on turning to the records it will be found that the facts support this suggestion almost as strongly as they support the corresponding suggestion in reference to optic nerve atrophy and ocular paralysis. Attention in this respect may be particularly given to Case XIII. The man complains of a quite recent diplopia, and he has undoubtedly had syphilis; the pupils show the Argyll-Robertson phenomenon. It is in the highest degree probable that had the patient been under observation a week or two earlier the condition of the pupils would have been the sole existing ocular abnormality. Yet in the light of the development of an ocular paralysis, it can scarcely be doubted that whether he develop spinal symptoms or not, his nervous system is the site of diseased processes of the locomotor ataxia order. When to these facts there are added, as in Cases XIV to XVII, illustrations of the various forms and degrees of evidence of spinal disease that may be associated with the Argyll-Robertson pupil, it seems impossible to resist the conclusions that the condition of the pupil so named may be the first evidence of locomotor ataxia; that it may precede by varying intervals other evidences of the disease; and that at least very probably, in a certain number of cases, the symptomatology of the disease may be permanently restricted to this one event. In some examples of its spinal form locomotor ataxia is undoubtedly an extremely chronic disease, with few and imperfectly developed symptoms; and it is thus not unnatural to expect that similar limitations may obtain in the ocular manifestations of the disease. That evidences of grave nervous disease may be limited to the pupil is well seen in Case XVIII, where a syphilitic patient was under observation for three years without the discovery of any abnormality other than paresis of each sphincter iridis. There is certainly no obvious reason why a similar restriction should not determine the Argyll-Robertson pupil as a purely isolated phenomenon with, it must be added, the same unfortunate possibilities that are undoubtedly attached to the patient whose case has just been quoted. The conclusions above adopted in reference to the Argyll-Robertson pupil are applicable *mutatis mutandis* to optic nerve atrophy and to ocular paralysis, as is abundantly demonstrated in the corresponding series of the cases recorded with this paper.

NOTES.

¹ In all the cases the ophthalmoscopic appearances were those of primary optic atrophy. ² This statement, both here and in other cases, must be taken as implying that there is a quite undoubted history of a primary sore followed by secondary symptoms; when the evidence is less conclusive the record carries a mere statement of the facts.

A SUM of 13,981 roubles has been collected in Russia for a monument to Pasteur.

CASE OF TABES DORSALIS WITH BULBAR SYMPTOMS.

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MANY observers have noted the affection of the larynx in tabes dorsalis, varying in degree from a slight alteration in the voice (depending, possibly, on ataxy of the intrinsic muscles of the larynx) to severe abductor paralysis, but I believe the following case to be exceptional from the severity of the laryngeal paralysis and the involvement of the other bulbar centres:

J. L., aged 50, married, and formerly a warder in a gaol, first came under my care as a out-patient at Prince Alfred Hospital some six years ago. He was subsequently admitted to the hospital as an in-patient on two or three occasions, and died while under my care in the early part of the year 1897. He gave the following history: About four years before he came to the hospital he suddenly lost power in the legs, and fell down as if he had been struck. He had no antecedent pains, and the weakness had been getting gradually worse. Up to a few months previously he had control over his bladder, but since then he had occasional incontinence of urine. His bowels have been very constipated. About two months before admission he noticed a difficulty in swallowing his food, and about a week later difficulty in breathing, especially at night; at the same time he began to make a great noise during inspiration, and to have some trouble in speaking. These symptoms have been getting gradually worse.

Previous History.

He had been a healthy man except for a slight touch of fever and ague about twenty years ago, and gonorrhoea seventeen years ago. This was not followed by any symptoms of secondary syphilis. He had been moderate in the use of alcohol. Family history showed nothing of importance.

Condition in February, 1895.

He is a tall thin man, but well built. Intelligence and memory good. No headache or pains anywhere; no convulsions, but has attacks of giddiness. Sleep is disturbed by his noisy respiration. His speech is thick and hesitating, and at times he appears to stammer.

Special Senses.—Smell is unaffected. Sight is fairly good, though he is presbyopic. Taste is impaired over the right side of the tongue. Hearing normal.

Cranial Nerves.—3, 4, 6. Pupils are unequal, left is slightly larger than the right. In the right eye the pupil does not react to light or on convergence, the left one reacts to light very feebly, and not at all on convergence. There is no ptosis or external ophthalmoplegia. 5. Tactile and painful sensation is impaired over right side of face; movements of jaws are normal. 7. No facial paralysis. 9, 10, 11. There is difficulty in swallowing, especially solid food. There is also complete paralysis of the abductors of the vocal cords, and during inspiration the cords do not separate, but tend to close together. The noise accompanying inspiration is so loud that he can be heard all over the ward. 12. Movements of the tongue are restricted; it is distinctly wasted on the right side, and it cannot be protruded very far. On protrusion the tip deviates to the right side.

Motion.—Muscular power is impaired in both legs, more so in the right than in the left. His arms are also weaker than normal, but there is no localised atrophy of muscle. He walks with difficulty, and cannot easily turn round.

Sensation.—Tactile and painful sensation is much impaired on both legs and arms. There is loss of muscular sense in the legs and arms, but more marked in the right than in the left arm. When standing on a hard floor he says he feels something soft under his feet.

Reflexes.—Abdominal and epigastric are feeble; cremasteric absent; plantar present on both sides. Left knee-jerk is very feeble, and right one is absent. No ankle clonus.

Sphincters.—Slight incontinence of urine, and bowels are constipated. There is complete loss of sexual desire and power. Romberg's symptom is well marked.

There are no vasomotor or trophic lesions. No gastric crises. He suffered somewhat from indigestion, but his other systems did not show any thing unnatural. His urine is normal.

Progress of Case.

The abductor paralysis seemed to progress, so that he became very cyanosed at times, and had great dyspnoea. This became so bad that tracheotomy had to be performed soon after his admission to hospital, and he was obliged to wear the tube up to the time of his death.

On his readmission to the hospital in February, 1897, the following notes were made: He is now very weak, and cannot walk in consequence of his inco-ordination. His eyesight has been failing much during the last few months; the pupils do not react to light, or on convergence. The tongue still deviates to the right on protrusion. He has lightning pains in the legs at times, but no girdle sensation. He has no control over his bladder, and the bowels are very constipated, so that he requires an enema always to secure relief. The knee-jerks are quite absent on both sides. His appetite is fair, but on attempting to swallow he almost chokes, so that feeding by the nasal tube is necessary. He subsequently developed signs of lobar pneumonia at the left base, and died after a few days' illness.

Necropsy.—There was some delay in getting permission to perform a necropsy, and the weather was very hot at the time. The whole brain and cord was removed and put into Müller's fluid, but as it was not in very good condition at the time, it did not harden properly, so that practically only the spinal cord could be examined microscopically. The cord showed sclerosis of the posterior columns, more marked in the upper dorsal and cervical regions than in the lumbar region, though some degeneration was found in those columns throughout the whole length of