On 10 May 1893, William Gowers began a series of weekly clinical demonstrations at the National Hospital for the Relief and Cure of the Paralysed and Epileptic at Queen Square, London. The contents of some of these demonstrations were published as ‘Post-graduate Clinical Lectures’ in the Clinical Journal, and in other learned periodicals. Some were also later included in his book Clinical Lectures on Diseases of the Nervous System. Recently, the manuscripts of what appear to be verbatim transcripts of two further but unpublished demonstrations from Gowers’ course in 1895 came to light, one containing alterations made in Gowers’ handwriting. The first concerned a case of disseminated sclerosis and its differentiation from hysterical paraplegia, the second transverse myelitis and its consequences for bladder function. Why these lectures were never published remains uncertain, but their relatively unedited contents reveal something of the neurological knowledge, diagnostic reasoning, clinical examination and teaching methods employed by one of the great pioneers of clinical neurology.

Keywords: bladder; disseminated sclerosis; Gowers; hysteria; myelitis

Introduction

On 10 May 1893, William Gowers, Physician to the Hospital for the Paralysed and Epileptic at Queen Square, London, and already author of the classic two volume work, A Manual of the Diseases of the Nervous System, explained to a group of his post-graduate students that he would institute a series of Wednesday afternoon clinical case demonstrations to replace his outpatient teaching. He then proceeded to give the first of these, on syringomyelia (Gowers, 1893). During the next 4 years, 18 of these demonstrations appeared as ‘post-graduate lectures’ in the Clinical Journal. Another six were published elsewhere (Table 1). The first 19 of the 24 were reprinted in his book Clinical Lectures on Diseases of the Nervous System (Gowers, 1895a), though not in chronological order. The latest of these, on severe optic atrophy, had been given on 16 January 1895 (Gowers, 1895b). Gowers gave the earliest of the remaining five lectures, on adult anterior poliomyelitis, on 22 May 1895, but it was not published until 1896. Two of the published lectures, both on locomotor ataxia, were given in consecutive weeks (14 and 21 June 1893). Two of the unpublished 1895 demonstrations have been recently unearthed in the Archives at Queen Square (NHNN/RES/2/1 and NHNN/RES/2/2). These mainly unedited manuscripts reveal something of the state of specialized neurological knowledge at the end of the
19th century. They also provide an insight into the approach used by a leading Victorian neurological authority to the neurological examination, clinical reasoning, bedside teaching and the preparation of material for subsequent publication.

The unpublished lectures

The demonstration on Disseminated Sclerosis, took place on 10 June 1895; that on Acute Myelitis, on 27 November 1895. Both manuscripts appear to be transcripts of verbatim shorthand records taken down during the demonstrations. The manuscripts are handwritten in ink. The writing is well-formed, written on ruled paper, in double-spaced lines.

The first paragraph of the Acute Myelitis lecture deals mainly with ‘house-keeping’ matters unrelated to the lecture topic and are unlikely to have appeared in any published version. This, and a number of transcription errors, make it very likely that these are verbatim transcripts from the shorthand record. Whoever prepared the manuscripts appeared conversant with medical and neurological terminology, though defeated by occasional specialized words because of the dependence of Pitman’s shorthand on phonetics. Thus space was left in the Acute Myelitis lecture to accommodate a word which probably was ‘kymograph’, a term probably unknown to someone who had not worked in experimental physiology but familiar to Gowers through his earlier research (Gowers, 1878). The transcriber also had trouble with the latter part of the word ‘neuroglia’. Furthermore, the word ‘expected’ has been mis-transcribed as ‘accepted’ in the first paragraph. This is the type of error that occurs when transcribing from a phonic system.

The manuscripts were in the same handwriting, but in parts of the Disseminated Sclerosis transcript changes of variable legibility, some in a darker ink and later ones in pencil, were inserted in a different handwriting, almost certainly that of Gowers himself (Fig. 1). The amending script was compared to documents in the Queen Square archives known to be in his handwriting, and with writing inserted in pages from a copy of Volume 2 of the second edition of his Manual of the Diseases of the Nervous System (1893), probably in preparation for a third edition that was never published. Gowers made no alterations on the final three handwritten pages of the Disseminated Sclerosis lecture, and made none on the Acute Myelitis manuscript.

Table 1: Titles of post-graduate lectures based on Gowers’ Wednesday afternoon clinical demonstrations, with their dates of publication

<table>
<thead>
<tr>
<th>Publication date</th>
<th>Title</th>
</tr>
</thead>
<tbody>
<tr>
<td>The Clinical Journal</td>
<td>A case of syringomyelia</td>
</tr>
<tr>
<td>31 May 1893</td>
<td>Locomotor atax—part 1</td>
</tr>
<tr>
<td>27 September 1893</td>
<td>Locomotor atax—part 2</td>
</tr>
<tr>
<td>October 1893</td>
<td>Acute ascending myelitis</td>
</tr>
<tr>
<td>20 December 1893</td>
<td>Facial paralysis</td>
</tr>
<tr>
<td>14 February 1894</td>
<td>Bulbar paralysis</td>
</tr>
<tr>
<td>2 May 1894</td>
<td>The infantile causes of epilepsy—part 1</td>
</tr>
<tr>
<td>5 September 1894</td>
<td>The infantile causes of epilepsy—part 2</td>
</tr>
<tr>
<td>12 September 1894</td>
<td>Facial contraction after palsy</td>
</tr>
<tr>
<td>19 September 1894</td>
<td>Optic neuritis</td>
</tr>
<tr>
<td>5 December 1894</td>
<td>The treatment of muscular contraction</td>
</tr>
<tr>
<td>6 March 1895</td>
<td>Lead palsy</td>
</tr>
<tr>
<td>1 May 1895</td>
<td>Severe optic neuritis</td>
</tr>
<tr>
<td>5 June 1895</td>
<td>Caries of the spine (3 July 1895)</td>
</tr>
<tr>
<td>6 November 1895</td>
<td>Caries of the spine (3 July 1895)</td>
</tr>
<tr>
<td>13 November 1895</td>
<td>Adult anterior poliomyelitis (22 May 1895)</td>
</tr>
<tr>
<td>12 February 1896</td>
<td>Adult anterior poliomyelitis (22 May 1895)</td>
</tr>
<tr>
<td>18 March 1896</td>
<td>Slight multiple neuritis (26 February 1896)</td>
</tr>
<tr>
<td>6 May 1896</td>
<td>International Medical Magazine</td>
</tr>
<tr>
<td>October 1893</td>
<td>Syphilitic hemiplegia</td>
</tr>
<tr>
<td>March 1895</td>
<td>Neuralgia</td>
</tr>
<tr>
<td>British Medical Journal</td>
<td>Mistaken diagnosis</td>
</tr>
<tr>
<td>7 July 1894</td>
<td>Argyria and syphilis</td>
</tr>
<tr>
<td>1 December 1894</td>
<td>Medical Magazine</td>
</tr>
<tr>
<td>April 1894</td>
<td>Saturnine tabes</td>
</tr>
<tr>
<td>May 1895</td>
<td>The foot clonus and its meaning</td>
</tr>
</tbody>
</table>

The dates of delivery of the lectures not contained in Gowers’ book of Clinical Lectures (1895a) are shown in parentheses. The bibliography of Gowers’ publications provided by Critchley (1949) lists an additional lecture on anterior poliomyelitis, but this is the result of a duplication.

a Not included in Gowers’ book of Clinical Lectures (1895a).
Disseminated sclerosis

The Disseminated Sclerosis lecture (see Appendix A for the version as Gowers modified it) concerned a case of fairly advanced disease of the cerebrospinal type, then considered the most frequent of Charcot’s (1877) three varieties of the disorder, namely the cephalic, the cerebrospinal and the spinal. Gowers did not describe the evolution of the disorder in his patient, simply presenting the findings as they existed at the time. He demonstrated what he considered were the major features of the condition, paraparesis with hyper-reflexia, severe intention tremor of the upper limbs, impairment of vision due to optic atrophy with loss of the pupil reaction to light, and nystagmus. He considered this combination of findings diagnostic. He analysed the pattern of the visual field defects, as revealed by confrontation and charting, and reasoned that the right optic tract as well as the optic nerves must have been affected. He was particularly concerned with distinguishing whether the young female’s paraplegia was due to hysteria or to organic disease and pointed out features of her pattern of attempted leg movement which he considered made hysteria unlikely.

He emphasized that the presence of other features such as exaggerated knee jerks and ankle clonus, the pattern of tremor, an ‘intention’ one that was absent at rest, and the eye signs, provided unequivocal evidence of physical disease. He did not seek psychological triggers for the malady or discuss the possibility of recent emotional trauma which, in his understanding of the basis of hysteria, might have predisposed towards that disorder.

Gowers did not record the outcomes of some clinical neurological tests that today’s reader might have expected. He did not mention the ankle jerks, though in his Manual he had described a technique, not used today, for eliciting them. Instead, he relied on the presence or absence of ankle clonus. He tapped downwards on one of his fingers already pressing distally on the upper border of the patella, with the knee extended, to determine whether a knee jerk was exaggerated. If so, the quadriceps was felt to jerk. The extensor plantar reflex was not described till the following year (Babinski, 1896). Gowers did not use the tendon reflexes in the upper limbs, probably because the distribution of the dermatomes was not well defined before the work of Head and Campbell (1900). For the same reason he did...
not use sensory changes on the trunk in his case with acute myelitis to determine the level of the spinal cord lesion. However, he knew that the ensiform cartilage corresponded to about the fifth and sixth dorsal cord segment. Spinal fluid examination was not yet available as an ancillary examination.

During the demonstration Gowers discussed what was known about the pathology of disseminated (or insular) sclerosis. He described the apparently random distribution of the patches of sclerosis throughout the CNS, and their macroscopic and microscopic features with overgrowth of neuroglia and wasting of ‘nerve elements’. Gowers made no mention of the relative preservation of axis cylinders despite loss of myelin, though he was aware of this phenomenon (Gowers, 1893), originally recognized by Charcot and emphasized by his disciple Pierre Marie (Marie, 1895, originally 1892). Gowers considered that the sclerosis was unlike that which occurred in a ‘system degeneration’ like tabes, and believed that the damage to the neural elements resulted from a primary developmental glial disorder.

Acute myelitis

This demonstration (Appendix B) involved an instance of clinically diagnosed acute transverse myelitis, probably at thoracic level, in a 24-year-old male. It had developed a year previously, a few hours after surgical drainage of an abscess in the left side of the neck. Gowers appears not to have had access to detailed records of these earlier events. He did not try to deduce the exact level of the spinal cord pathology from the clinical findings, and was seemingly content to locate the pathology above the lumbar enlargement of the spinal cord. He argued that the pathological process had not extended into the enlargement because there was increased reflex activity in the legs, and because of the pattern of disturbed sphincter function that had occurred. The latter was his main focus in the demonstration. He stated that three possible patterns of altered bladder function might result from spinal cord pathology: first, there could be retention of urine and subsequent overflow incontinence, with secondary renal damage from back pressure and infection (Gowers did not explain the neural mechanism that produced this state); second, if the lumbar enlargement of the cord was destroyed the bladder was paralysed and urine flowed from the bladder as it entered it; and third, if spinal cord disease isolated the intact lumbar enlargement from higher centres there was intermittent involuntary reflex bladder emptying whenever the bladder became distended enough. Gowers thought the third pattern applied in the patient he demonstrated. As the editing was not complete, Gowers might eventually have recognized that he had not explained the mechanism of his first type of bladder dysfunction, if or when he finished editing the lecture for publication.

He gave little attention to the nature of the neuropathology in his patient, confessing that the contemporary understanding of myelitis was unsatisfactory. At the demonstration he had arranged for the audience to be able to view histological material showing examples of transverse myelitis. Figure 2 shows Gowers’ own drawing of such material (Gowers and Taylor, 1899), though one cannot know whether the sections illustrated were shown on 27 November 1895.

Discussion

In 1895, the clinical manifestations of disseminated sclerosis had not long been widely known in British medicine. Charcot’s influential description of the disorder and of its differentiation from paralysis agitans, first published in 1868, was not translated into English until 1877 (Charcot, 1877). The first British clinical case of disseminated sclerosis confirmed by autopsy dated from 1873 (Moxon, 1873). The accounts of disseminated sclerosis in Wilks’ Lectures (1878), Ross’s Treatise on the diseases of the nervous system (1882) and Gowers’ Manual (1893) appear to depend heavily on Charcot’s Lectures (Charcot, 1877). Despite Gowers’ full account of the disorder in his Manual he did not include an
illustration of the pathology of the disorder. Even if he had none of his own, such illustrations should have been available to him from British sources, e.g. Bramwell's (1882) illustrations of the spinal cord (Figs 3 and 4) making this a surprising omission. Gowers' only other publication on disseminated sclerosis, more than a decade later, was in shorthand (not yet transcribed) in the *Phonographic Record* (Gowers, 1909).

A century ago, the clinical recognition of disseminated sclerosis depended heavily on the presence of a particular constellation of physical signs, namely lower limb weakness, intention tremor of the upper limbs, scanning speech, nystagmus and vision disturbance. There was little emphasis on the relapsing–remitting course of the disorder that later took on increased diagnostic importance, though Gowers (1893) mentioned that the disease might remain static for appreciable periods of time. Charcot (1877) had described the anatomical pathology of the disease and the relative sparing of axons, but Gowers did not comment on this sparing during his demonstration. Gowers, and his contemporaries like Charcot, did not seem to perceive that demyelination might be the primary event in disseminated sclerosis and thought that the disease had its roots in the neuroglia. McDonald in the ninth Gowers Memorial Lecture later reported that Gowers’ intended correction for the unpublished third edition of Volume 2 of his *Manual* stated ‘It is essentially a process of morbid growth but may sometimes commence as an interstitial inflammation’. He also noted that ‘the tendency of residual embryonic tissue to overgrow in adult life’ and speculated that the islets of sclerosis may arise from points of developmental origin (McDonald, 1986). Many of the mysteries about multiple sclerosis that baffled Gowers remain unsolved today and astroglial proliferation is again considered an important aspect of pathogenesis.

Charcot held the view that hysteria was a neurological disorder with an, as yet, unrecognized structural pathological basis, a ‘neur-osis’ in Cullen’s (1789) sense of that word. Untreated, it had a reasonably stereotyped set of patterns of clinical expression. Freud’s theories on hysteria were beginning to appear in print,

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**Figure 3** Carmine staining of a series of transverse sections of the spinal cord from the upped cervical region (a) to the filum terminale (i), showing areas of gliosis in a case of disseminated sclerosis. The dye is taken up by neurons and glia, but not by myelin. From Bramwell (1882).

**Figure 4** Osmic acid stained section of spinal cord at the level of ‘C’ in Fig. 2. The demyelinated areas are unstained while the intact myelin stains darkly with the acid. From Bramwell (1882).
but in 1895 had not yet achieved any widespread notice, let alone acceptance. Gowers interpreted the disorder and its range of possible manifestations somewhat differently to Charcot. He thought that predisposing personality and emotional traits might have a role in causing a loss of the governance that the cerebrum normally exerted over lower level neural mechanisms. He also attached much less importance to gynaecological origins for the disorder (Gowers, 1893). However, in his demonstration he was not concerned with the mechanisms of hysteria but with differentiating hysterical paraplegia from leg weakness due to structural spinal cord disease. In doing this he described a pattern of ankle movement during attempted dorsi-flexion of the foot that he considered indicative of organic paresis. This pattern was not described in his Manual. The differentiation between the two disorders was important at a time when disseminated sclerosis was almost certainly under-diagnosed at Queen Square. A total of 5% of Gowers’ in-patients carried a diagnostic label of paraplegia or paraparesis, of whom one-third were labelled as hysterical paraplegia or hysteria. Insular sclerosis was mentioned only very occasionally in his case notes as a cause for leg weakness.

In discussing acute myelitis, Gowers admitted that the basis of myelitis was not adequately understood, and did not suggest its likely cause in his patient. Perhaps disseminated sclerosis was the cause here too, but the diagnostic criteria then accepted for that disorder (see above) would have made its recognition unlikely. Denial of a history of syphilis in a 24-year-old soldier who had developed his illness in India might not suffice to exclude that aetiology, but for some reason Gowers chose to bypass this. A spinal septic thrombophlebitis appears less likely and a pyogenic intraspinal process even more unlikely, though there had been a neck abscess. Sufficient details are not available for any reasonably soundly based retrospective diagnosis to be suggested. Gowers’ main concern in his demonstration was the disordered sphincter function, and his interpretation of its mechanisms left an important aspect unexplained, namely the basis of urine retention with overflow incontinence. His audience at the time may not have noticed this, unlike readers of a printed version. It is interesting that Gowers regarded palpable changes in the tone of the anal sphincter as indicating the probable simultaneous behaviour of the bladder sphincter, and that following research at University College London he had previously published on the human anal sphincter in neurological disease (Gowers, 1878).

Taken together, these two manuscripts show something of Gowers’ modus operandi that is not obvious from his more polished published writings. The unedited Acute Myelitis lecture reveals the fluency of Gowers’ speech at his clinical demonstrations, something that might have been lost in final published material after Gowers had corrected it and perhaps changed its style.

Gowers started teaching informally at the National Hospital for the Paralysed and Epileptic in 1873, after he was appointed an assistant physician at Queen Square, and it was not long before the outpatient room was ‘thronged with physicians from all over the world standing in the gangways and straining the capacity of the accommodation’ (Critchley, 1949). He also gave formal lectures at University College Hospital and later at Queen Square, and many of these were published in the British Medical Journal and the Lancet.

Together with his other, extensive publications, often republished in the USA and Europe, his ideas on neurology reached far beyond the fortunate audience who could attend his demonstrations at Queen Square. His formal lectures were always meticulously prepared (Holmes, 1954), but in the informal and less predictable situation of the clinical demonstrations it seems likely that he was more spontaneous, thinking on his feet as the details of the case were revealed.

When writing a commentary on his use of shorthand, he described the way he prepared his formal lectures by writing what he wanted to say in shorthand and then obtaining ‘a type-written copy made by a shorthand reader and type writer’ (typist), but bemoaned the cost of having a manuscript typed (Gowers, 1909). Gowers’ Queen Square case records were handwritten until 1905, when typewritten records were introduced. He bought a typewriter of his own in 1885, which he probably used in his consultancy and which he used for his ‘Children’s Diary’. All his children were taught shorthand as soon as they could read, and they were sometimes conscripted to help with copying his work before publication (Scott et al., in press).

Gowers was a firm advocate of shorthand for taking lecture notes and case notes. ‘In a previous generation, when neurology was more feared than studied in England, if a man knew this subject well, and especially if he carried an ophthalmoscope ready for use in his pockets, he was probably an old student at Queen Square, and if, in addition, he could write shorthand, almost certainly one of Gowers’ house physicians there’ (Anonymous, 1915). Shorthand had ‘an abiding fascination and usefulness for him, and was a royal road for his house-physicians and students to his heart’ (Anonymous, 1915). It was not an unusual skill for doctors in those days, and it was fundamental to Gowers’ scientific method in an era without electronic recording devices or computers.

It seems likely that Gowers arranged for a student to record his clinical demonstrations in Pitman’s shorthand. This student (or perhaps another) would then be expected to transcribe the shorthand into longhand. Gowers had been instrumental in the on-going development of a booklet ‘Phonographic Outlines of Medical Terms’, which had been a project he first envisaged when as a 16-year-old apprentice he had written and asked Sir Isaac Pitman when one might be produced (a project that had to wait 40 years for fruition in 1902) (Scott et al., in press). Shorthand and shorthand transcription are skills that can only be learned through constant practice. It seems likely that Gowers could have deliberately imposed the routine of shorthand practice on his students and his house physicians.

Although his demonstrations were published as ‘lectures’, they really were instructive analyses of actual clinical situations and because they were verbatim records no references were provided. Gowers was sometimes sparing in citing references in his writings but there is evidence that he was up to date with the current literature in the amendments he was adding to his unpublished third edition of Volume 2 of his Manual of Diseases of the Nervous System. By present day standards, the manuscripts have obvious deficiencies. Some may have been accidental; many are consequences of advances in knowledge and changes in attitude over the intervening century. Most of the facts recounted in the
demonstrations may be found scattered through Gowers’ previous writings. However, in the manuscripts they are brought together to help him interpret the clinical findings and arrive at an accurate diagnosis, e.g. whether paraplegia was due to hysteria or to nervous system disease.

Why did Gowers not publish these particular 1895 lectures, and cease working on the Disseminated Sclerosis text? Five of the lecture demonstrations he gave after that on Disseminated Sclerosis were published, and one that was later than the Acute Myelitis demonstration. He had suffered a complete breakdown in health in the 1890s, from which he never fully recovered. In June 1894 he had added to his workload when he launched the shorthand journal, the Phonographic Record of Clinical Teaching and Medical Research, which he edited for a number of years, while continuing his usual professional activities. He completed the preface to his Clinical Lectures on the Diseases of the Nervous System in June 1895. He may have intended to add the Disseminated Sclerosis lecture to this book, but either found himself too busy to meet his publisher’s deadline, or was dissatisfied with the quality of this manuscript. Throughout his career Gowers remained perplexed about the nature of Disseminated Sclerosis and he might have been reluctant to rush into print at this stage.

In 1898, Gowers went on a cruise to South Africa on doctor’s orders. At the time, he was working on the final proofs of the third edition of Volume 1 of his Manual. By its later editions, the two-volume Manual contained approximately 810,000 words and 374 illustrations, most drawn by Gowers himself. Exhaustion and competing priorities may both have contributed to him stopping work on the manuscripts and the third edition of Volume 2 of the Manual also never reached completion (Queen Square Archives NHNN/RES/1/1).

Probably the majority of Gowers’ weekly clinical demonstrations never appeared in print, even though their contents were likely to have been recorded and then transcribed. Gowers may never have intended to publish either lecture, and the manuscripts could have survived by pure chance.

These unpublished lectures reveal something of Gowers’ teaching style and methods and illustrate the neurological diagnostic practice and clinical reasoning at the end of the 19th century. However, it would be unfair to judge them as if they were at a stage that satisfied Gowers himself, when he might either have rejected them as not good enough for publication, or at least had not yet finished editing them to his own exacting standards. But they provide a glimpse into the early days of what were to become the famous neurological clinical demonstrations and lectures at Queen Square, later described by Holmes (1951). It is possible from the verbatim record of these manuscripts to picture the way in which a great Victorian clinical neurologist engaged in the dynamic process of teaching the art of diagnosis.

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Appendix A
A post-graduate lecture on Disseminated Sclerosis delivered at the National Hospital for the paralysed and epileptic, Queen Square, London, 10th June 1895 by WR Gowers MD, FRS, Physician to the Hospital and Consulting Physician, University College Hospital Gentlemen,

Professional knowledge grows apace. By professional knowledge I meant the general knowledge of medicine possessed by the profession as a whole, as distinguished from that possessed by those occupied in the advancement of medical science.

The patient before you is a girl suffering from loss of power in the legs. If she had been seen by a dozen doctors 15 or perhaps even 10 years ago, I believe that more than half of them would have diagnosed the case as hysterical paraplegia. At the present time I think the proportion who would make that diagnosis is not more than a quarter. The diagnosis would have been made by the
process of reasoning. Paraplegia, functional in nature, is an occasional symptom of hysteria. Most cases of hysteria occur in girls: therefore the probability is great that it is functional paraplegia; when the patient was examined it would be found that there was irregularity in the movement of the arms, and it would be remembered that tremor and unsteadiness of movement are occasional symptoms of hysteria. Then it would be found that her sight was bad, and she would have said that the sight of one eye was affected; and amblyopia, chiefly unilateral, is a common symptom of this disease; the diagnosis would have been confirmed; the diagnosis made at the outset would have been supported by a further study of the case. That sequence of reasoning is the usual one. Whatever conclusion is reached at the outset dominates all further perception, carries with it in memory all the other indications presented by the patient unless they are absolutely opposed to it. Preconceived ideas are an immense help to facile diagnosis. But the ‘royal road’ is used now by fewer than formerly employed it, because the knowledge of the profession has become, on the whole, vastly better, especially as regards those subjects in which knowledge is a modern product. The efforts of the teachers during the last 25 years are now producing an enormous effect throughout the whole profession. And among the principles generally recognized is this: that any symptom which is an absolute proof of organic disease, however slight, proves it and neutralizes the significance of any general possibility or probability of a purely functional nature. When this patient is examined the power in the legs is to be first observed. The right leg she raises, the left she cannot; when she tries to flex the right ankle she can do it, but not the left. That at once should raise a grave suspicion, and more than a suspicion. Hysterical paralysis of the leg is chiefly one-sided in only very rare cases. It is generally bilateral. A marked difference in the power of the two legs would at once, or should at once, make mere functional paralysis unlikely. Note further that one foot inverts as the ankle is flexed. This means that there is unequal loss of power in the flexors of the ankle, the peroneal muscles and the tibialis anticus. Such unequal palsy in muscles which combine to produce a single movement is never met with in functional diseases. That slight deviation of the foot as she flexes the ankle should alone exclude hysterical paraplegia. But the real point is to ascertain whether or not there is foot clonus and excessive knee jerk. The knee-jerk is excited by sudden tension of the rectus, the muscle having previously been quickly extended, those conditions can be readily produced in the recumbent posture, though then the mechanism adopted is insufficient to produce contraction unless the knee jerk is in excess. In this patient, the muscle being relaxed, the patella is pressed down towards the foot by the finger being placed above it; the finger is then tapped in the direction of the pressure and instantly the muscle contracts. In the right leg the same thing is conspicuous. That means that the knee-jerk is present, and, moreover, that it is in excess. I now push up the foot, as you see, and there is a regular characteristic foot clonus; the same in each leg. In functional disease there may be some excess of these muscle reflex actions. There may be enough to enable you to obtain the reactive contraction in the way I have obtained it; there is not enough to give rise to a foot clonus. In functional disease you may indeed have a clonus when the foot is pressed up, but it is always preceded by a pressure downwards of the foot, from a voluntary contraction in the calf muscles. Indeed, slight increases of this muscle reflex action that is met with in many cases of functional disease may be enough to enable you to get the contraction in this way, and to enable a voluntary tonic contraction of the calf muscles to clonus. Its significance has been disputed. I will not affirm that such a clonus as this is never met with in what is called ‘functional’ disease, but I am certain that such clonus, if constant, never can depend on a more functional affection. If it is not due to what we call organic disease, it is always due to definite nutritional changes in the nerve channels. If you adopt the principle, as a practical rule, that it is not met with in functional disease—that it always indicates definite changes—I am sure that not more than once in the remainder of your lives will you find that that rule does not guide you to a correct conclusion. If you regard it, on the other hand, as of small significance you will many a time find yourselves in error. Remember especially that this increase in muscle irritability in moderate degree in so-called functional disease, can not depend on that which alone we are justified in calling ‘hysterical’. It does not depend on the cerebral condition, but on secondary or associated changes in the nutrition of the spinal cord. It is an evidence of disordered function of the cord itself, and we have cannot include the cord under the domain of true and pure hysteria. The patient moves her legs with fair steadiness, but not her arms. The unsteadiness of the arms I can show you best by getting her to hold this rod. If, afterwards, you ask her to favour you with a friendly grasp, you will perceive still better the irregularity of the muscular contraction. A short time ago, this irregular, jerky incoordination was even more marked, but its degree has lessened with lessened degrees of muscular power. This sudden jerky incoordination is itself a characteristic of organic disease and of one organic disease in practice in particular.

Her sight is affected; this might not cause you to look at the eyes of a patient, because almost the first thing you should do in such a case is to carefully observe them and especially their movement. If you do, many will be the cases in which the aspect of the eyes—the size of the pupil or the position of the globe will put you on the right track. When this patient looks to one side or the other the eyeballs are not steady; there is the oscillation known as ‘nystagmus’. The practical value of nystagmus is that it indicates disease that is more than functional. It is never present in mere hysteria. The significance of nystagmus is of high practical importance. It is the more important because in a large number of the cases in which it is met with, other symptoms are equivocal. We would fain know more of what the symptom means; at present the secret of the special meaning has baffled us.

The loss of the light reflex of the pupil, which is present, is another symptom that may always be treated as a sign of structural disease. This loss, with a nystagmus, irregularity of the movements of the arms, weakness of the legs with the clonus which shows degeneration of the lateral columns constitutes a sign of one strange malady alone—of disseminated sclerosis. We do not know why the combination has this conclusion. That which is specially significant of the disease is the peculiar jerky state of the arms. We do not know how this symptom is produced. In its special form it is almost, but not quite, constant to the disease. I need not tell you what the nature of the malady is. You know
that spots of sclerosis are scattered through the brain, pons, medulla and spinal cord; they are quite irregular in their position just as tumours are but they differ from morbid growths in that the new tissue simply replaces the normal structures. There is no increase in the size of the substance in which the tissue is developed: these strange roundish grey areas are due to an overgrowth of neuroglial elements and a wasting of the nerve structures; their random distribution compels us to regard the overgrowth as the primary change and yet it has none of the effects of a new growth in causing enlargement of the part, i.e. it is not a tumour. The situation of the mass has no correlation to function or to that liability to disease which so many parts of the nervous system present. You know how certain structures in the nervous system—nerve endings and nerve cells, undergo degeneration, the distribution of which is limited to those that have a certain function. Of two strands close together one may be affected and the other remains normal, solely, as far as we know, on account of the difference in function. Even in one centre, as in the centre on which on which the action of the pupil depends, we may have an affection of some structures leading to the loss of light reflex of the eyes, while other adjacent structures, subserving accommodation and its associated contraction of the pupil, are normal, in absolute contrast to system disease, in which the distribution of the lesion depends upon the function, is this random insolated sclerosis. The significance of the difference is that in the ‘system diseases’ the change begins by failure of nutrition of the nerve endings, and the sclerosis, which we can so easily see, is secondary. In this affliction of random distribution, the change begins in the connective tissue elements, and the wasting of the nerve elements is secondary. Of course, if there is damage to the nerve elements in a tract which is liable to undergo secondary degeneration, there is, combined with the effects of the sclerosis, a secondary degeneration of the fibres which pass through the sclerotic area. In the dorsal region of the spinal cord, the lateral columns of the cord may be the seat of these irregular, scattered spots of sclerosis, and there such spots cannot fail, at one place or another, to damage the fibres of the pyramidal tracts. These fibres of necessity degenerate below the point at which they are damaged. Hence we have true secondary ‘lateral sclerosis’ and in the legs the symptoms are identical with those produced by primary lateral sclerosis. This is a point of considerable practical importance. We have this condition here. There is weakness of the legs with the increase of the muscle reflex action, which indicates lateral sclerosis, and might be due to the primary form. We know from the other symptoms that there is more than that. We have evidence of disseminated spots of sclerosis elsewhere; and so we conclude that the state of the legs is not one of simple primary lateral sclerosis. We might have more difficulty in discussing the nature of the process in the spinal cord.

In some cases of insular sclerosis the islets occur only in the dorsal cord or at least are absent even in situations in which they cause no local symptom. Then the case is indistinguishable from one of primary lateral sclerosis. But the case presents other points that deserve notice.

Her sight is greatly impaired; the acuteness of vision has failed considerably during the last 2 years, and rapidly of late, so that she can now only recognize fingers. If you presently look into the eyes, you will see characteristic white atrophy of the optic discs. They are devoid of the entire normal tint; there is a sharp-cut deep and a hollow physiological cup, large, without any of the tissue which indicates previous inflammation. They are indistinguishable from the discs which we meet in locomotor ataxy. Such simple atrophy of the optic nerves is not uncommon in disseminated sclerosis and, alike in its aspect and in the affection of vision to which it gives rise, it is the same as the atrophy of locomotor ataxy. That is startling; more startling than you may at first perceive. Locomotor ataxy is a ‘system disease’; it consists in primary degeneration of the nerve elements and a secondary overgrowth of the connective tissue, the neuroglia, which increases, whereas the proper structures waste. In the degeneration of the optic nerves we have however, much greater overgrowth of the connective tissue than in the spinal cord; yet we are compelled by the facts to regard it as primary degeneration of the nerve fibres. Indeed, it seems to be essentially a peripheral degenerative neuritis, like that which occurs also in the same disease in the sensory nerves of the limbs. This great production of interstitial tissue in the optic nerves compared with that which is seen elsewhere is remarkable. It deserves note, for two reasons. First, in spite of it, the character of the failure of sight shows that the affection is primarily a wasting of the nerve fibres and the overgrowth of interstitial tissue, great as it is, is secondary and the amount of tissue present is no proof therefore that its overgrowth is primary. Secondly, in the course of the degeneration of tabs there is occasionally evidence that the process takes on a more acute character. This seems sometimes to be the case in the spinal cord, and also in the optic nerves, especially and the chiasma. It assumes the character of interstitial inflammation, acute failure of vision may occur in the course of a few days. It may take the form of bi-temporal hemianopia, in consequence of the secondary affection of the interstitial tissue taking on such independent inflammatory character at the middle of the chiasma, so as to damage the decussating fibres. That is perhaps a stepping stone to the fact which this case illustrates. Here we have evidence not of the system degeneration of tabs, but of a secondary overgrowth of connective tissue, and yet, in the optic nerves, there is degeneration just like that of tabs. It may be a ‘stepping stone’ because, even in tabs, there is evidently a tendency in the optic nerves atrophy although it begins by degeneration, for the secondary interstitial overgrowth to have a more independent character than elsewhere. The fact which probably underlies all these phenomena is that the neuroglia and nerve structures develop from the same embryonal tissue. You know how in the course of development of the cord, in the various forms of syringomyelia, there are found remains of tissue identical with microglia. This persists in inverse proportion to the nerve elements, illustrating the fact that the nerve tissue develops from the same elements, and therefore we are not surprised to find that in some cases we find an excess of the persistent neuroglia and deficiency of the nerve elements. So we need not wonder if that inversion in development is reproduced in disease, as we see it is, by the arrangement of tissue when the nerve elements waste, and by the association of primary atrophy of the optic nerve fibres with this local excessive overgrowth of the neuroglia.
Time does not permit me even to enumerate the leading features of insular sclerosis but there are some features of which I should like you to take away a distinct conception. One of those is the half-blindingness from which the patient suffers.

When you wish to ascertain the presence or absence of lateral hemianopia, that is loss of the half field in one side, but on the same side in each eye, the readiest method, also the simplest, is to make the patient look at your face while you hold up both your hands (one on each side) within the limits of your own field, and ascertain if the patient can see both, or only one. On whichever side the half loss exists, one temporal half field must be lost, one hand will only be seen. Bitemporal hemianopia in which both outer half fields are lost, is also revealed by this but it is comparatively rare. A less rough test, which, with ease, yields remarkably accurate result, sufficiently so to enable you to construct an appropriately correct chart, is to attach a small piece of white paper to a black pen holder, making your own eye the fixing point and your own field of vision the standard by which you estimate that of the patient. Testing the patient thus you perceive that there is a loss of the left half of the field and reduction of the remaining half; she does not see an object in the periphery of the side on which vision remains. You note also that it is similar in the two eyes; in each there is half-sided loss, or if you prefer, a formidable lesion, there is homonymous hemianopia, same-named half blindness. I show you some charts which have been obtained. Such hemianopia may be due to disease anywhere between the chiasma and the occipital lobe; in the optic tract, possibly the extremity of the optic thalamus, the white substance of the occipital lobe or the cortex at the hinder part of occipital lobe. You perceive that, on each side, the seeing half includes a small region around the fixation point. This is invariable. It is due to the fact that some of the fibres just around the fixation point pass by both optic tracts to both hemispheres. Disease of one optic tract leaves vision in an area around the fixation point, which varies in extent in different persons. But this involves also, as you might expect, a diminution in the acuity of central vision in both eyes. In disease of the optic tract, therefore, the patient loses one half completely, but there is no restriction of the field of vision in the other half. Yet in many cases of hemianopia there is such a peripheral restriction of the field of vision as we have here with absolutely normal ophthalmoscopic appearances. In those cases the disease is in the cerebral hemisphere. The lesion involves the fibres going to the angular region, as the grey matter of the angular region in which the higher visual centre is situated. Is that the case here? Is the cause of the hemianopia an area of sclerosis in the hemisphere? It is possible, but I think it is not the case. We have here an atrophy of the optic nerves in addition to the hemianopia which alone will account for the restriction of the field of vision. If she had no hemianopia I conceive that her field of vision in that eye would be reduced to the area I show you, in consequence of the degeneration of the optic nerves. So that the case is potentially instructive in that respect, in so much as it presents a condition of hemianopia associated with the contraction of the half-fields, which would show cerebral disease—disease of the cerebral hemisphere, in which the contraction of the half fields is probably only of local origin, from degeneration of the optic nerves. I conceive that the hemianopia probably depends on an islet of sclerosis in the opposite optic tract.

I said I do not propose to discuss this disease in general, nor is it practicable at these lectures to discuss any disease in general. I always like, if I can, to have some practical consideration. Alas, this malady is one which baffles practice. There is no disease more resistant to every effort which can be brought to bear upon it than insular sclerosis, developing, as it has in this patient, to this degree in the course of 2 years or so, and its progress is such that it seldom fails to continue with unhindered rapidity, until the end. Yet all manifestations of disease manifest vagaries, which influence the prognosis, alike for good and for evil. I have only once or twice said a patient would surely die, and in each of them the patient got better, and I naturally hesitate to repeat such a statement. I would not mind being wrong if I could cure the patient, but in this disease the course is often a mysterious one. Sometimes it is arrested, but very seldom, but what is most distressing is that we cannot discern on what the arrest depends. Though I know of no affection which so completely baffles effort as this, yet it is always right to try. It is possible we sometimes do good when we cannot discern it, as certainly we are often supposed to do harm when we are innocent of being the cause of the evil which follows our treatment. Arsenic, small doses of mercury, counter-irritation by the actual cautery especially, are the most important neurine influences, together with nerve tonics. Of especial importance is attention to the general health and the maintenance of an equable temperament. The last is the easier; curiously enough, patients suffering from disseminated sclerosis are singularly happy, not quite in the degree met with in general paralysis, in which the patient will say ‘Oh! if this is to be bad I wish everyone were bad’—so happy are they. Their enjoyment, the way in which they are unaffected by disability and undeterred by progressive weakness, is a feature, however, which this patient does not possess, but is yet one of the many mysteries of disease.

Appendix B

A post-graduate lecture on Acute Myelitis delivered at the National Hospital for the paralysed and epileptic, Queen Square, London on 27th November 1895 by WR Gowers MD, FRS, Physician to the Hospital and Consulting Physician, University College Hospital

Gentlemen,

Before I begin my lecture today I should like to say that I propose to finish earlier than usual, not only that you may look at the patients, but because Dr Colman, the Pathologist, is good enough to say that he will put under the microscope some sections of the disease which we are to consider. Moreover, an autopsy which he had just completed shows some specimens well worthy of consideration which he will have for your inspection in the adjacent museum. Although that was not as accepted, a case of an affection of the nervous system, it is an instructive example of malignant pelvic growth, which has compressed the nerves to give rise to leg symptoms, and of narrowing of the pulmonary artery in consequence of secondary growths within the lungs. It is not my case; therefore I have really no right to show you the specimens.
But it is the case of one of the most benevolent and noble-minded, as well as one of the most able physicians of the day—Dr Hughlings Jackson—so that I have no hesitation whatever in inviting you to see the specimens.

The subject which I have to ask you to consider now is an illustration of the fact that the knowledge that we need and the knowledge that we have are often in inverse proportion. There is no disease of which we know, as a disease, less than of acute myelitis; and there is no disease of which we need to know more. Moreover, it is a disease the early treatment of which is of paramount importance. I am not sure that with all the increase in our knowledge of its pathology, we have a proportionate increase in the practical knowledge that we need, of the treatment which alone can be effective—that is the early treatment. I propose to show you the case and remark upon it then in a discursive fashion.

The man before you is a soldier, 24 years of age. Twelve months ago he had a swelling on the left side of his neck. He denies having had either gonorrhoea or syphilis (the notes do not say whether they can be excluded but the swelling on the left side of the neck occurred when he was in India; it was apparently an acute swelling, with suppuration. He went into hospital, and in 2 days time the abscess was opened. Eight hours after the operation, he was unable to pass urine, and presently found his legs weak. A catheter had to be passed the same evening, and on the following day he was unable to move his legs. I should like, here, to impress upon you that inability to pass urine is frequently the first symptom of acute myelitis. It is a most important fact; it is strange how well known the fact is, and yet how frequently its significance is not perceived. Its importance arises from this; that it is at the very onset of such myelitis the chief scope for treatment exists. Complete loss of power over the bladder and over the rectum continued. He had not a continual flow of urine; the bladder emptied itself spontaneously 8 to 10 times a day without his knowledge. That fact is also of significance. There are three chief conditions of the bladder in spinal disease; one is, when there is inability to empty the bladder and gradually increasing retention of urine, so that at the last, one or two pints or more of urine may be always in the bladder. Then, the urine, being still secreted by the kidney, comes down into the full bladder, and the elastic strength of the wall of the bladder is sufficient to cause the urine which comes down into it to flow away, or rather, to make this added quantity of urine cause a continual overflow. It is that condition which has attracted much notice by everyone, and has led to Sir Henry Thompson’s aphorism that incontinence means retention, an aphorism which contains a considerable and imperfect truth, and yet only represents a reality in a very partial manner. But the important fact regarding this retention and incontinence is that the increased pressure within the bladder which always exists, involved resistance to the passage of urine from the ureters; in spite of the valvular opening of the ureters there is of that fact no doubt. It is by this mechanism that organic disease of the kidneys is produced—the surgical kidney; it is the increased pressure within the pelvis of the kidney which is the first and chief agent in leading to that formidable disease. Afterwards, cystitis, an organisinal infection, may lead to propagation of inflammation to the kidney; but I am sure the chief mechanism is the increased pressure, and that state may develop, insidiously. You hear that in this patient, although there was no loss of sensation in the limbs, he was unaware of the involuntary evacuation of the bladder. That is an illustration of the way in which a patient may not have the sensory notice of that which goes on. In locomotor ataxy especially, and in spastic paraplegia and any other disease of the spinal cord, this over-distension of the ureter, this pressure on the kidneys, and this organic change in the kidney may bring a patient to the verge of disaster without the slightest consciousness on his part that there is anything wrong; because in these cases there is always the constant overflow, and there may be some involuntary evacuation in spite of the persistent over distension. This has been impressed on me by many cases, and its importance cannot be exaggerated.

You know that when a catheter is passed in health occasionally there is an irritation of the kidney, a reflex effect; therefore how easy it is to understand that when a kidney has been diseased in this way, the unfamiliar irritation aroused by the first passage of the catheter may have such a reflex effect on the vessels of the kidney as to suddenly derange its functions still further, and pitch the patient over the precipice on the brink of which he unconsciously was. That is what the late Sir Andrew Clark termed ‘catheter fever’; which by the early use of the catheter might have been entirely obviated; so that ‘a catheter fever would be the correct designation. The second condition which is met with in spinal disease is when there is destruction of the centre for the bladder in the lumbar enlargement; then there is no action of the bladder or the sphincter. The urine passes from the bladder as fast as it enters it, and the same with the bowel. The lumbar centres for the bladder and the bowel are near together, and commonly suffer together. You can recognize the condition by finding that, with continual incontinence there is no dullness over the pubes, and no urine is to be found, or at least no considerable amount if you pass a catheter into the bladder. But you can also ascertain it by a rectal examination in the majority of cases, because, as I said, the rectum is governed by the centre near that for the bladder. In health, if the finger is passed into the rectum, in a moment it is felt to be gripped firmly by the sphincter, whereas when there is this disease of the centre, no such grip occurs. [SMALL BLANK SPACE] The third condition is when there is disease cutting off the lumbar centre absolutely from the cerebral control and influence; then the bladder is left in perfect independent relaxation. When enough urine is within to excite, by the stimulation of the nerves at the neck of the bladder, the reflex action, the wall contracts and the bladder is emptied. I have no doubt that that was the case in this man, in whom there was the involuntary passage of urine 8 or 10 times a day. The finger being introduced into the rectum, at first the bowel seems flaccid, but in a moment the sphincter is felt to firmly contract. If then, the point of the finger be removed, so as to stimulate the mucous membrane of the rectum just above the sphincter, the sphincter at once relaxes, and then again contracts. It is possible to take a tracing of that which happens, by means of a [?] kymograph] You find then that if you stimulate the mucous membrane of the rectum just above the sphincter, there is a relaxation, and the pressure falls, but is followed by a firm contraction. If you complete that by a secondary line, you have that which probably perfectly represents the peristaltic action of the bowel.
Now there can be no doubt that this man has acute inflammation of the cord; we have now evidence of this from his present state. He bears evidence of disease above the lumbar enlargement. The story after the onset is that he remained stationary for two months, then began to improve and became able to walk, but, after a cold bath injudiciously given, renewed weakness came on; he was again unable to stand; it was 3 months more before he could stand, and 7 months from the time of the bath before he could walk again. During the last 4 months there has been some improvement. He has now considerable weakness of the legs, which differs a little from that which is ordinarily met with, because it is greatest towards the extremity of the legs. You know that weakness greatest towards the extremity of the legs is common in peripheral neuritis, but rare in central affections. There can be no doubt that this man has a central affection, and yet you have this unusual distribution. He can extend his knees, but with his heel on the ground he is unable to draw his toes up. In cerebro disease, the loss of power is greater in the foot than in the hip; in spinal disease, as a rule, in the dorsal region, the loss of power is greater in the upper part of the leg and the hip than in the foot. In multiple neuritis it is greater in the foot. Here, however, we find loss of power is greatest towards the extremity of both limbs, and yet it is certainly dorsal myelitis. He has extreme foot clonus in each leg, and you saw the curious tremor of the legs when I first exposed them. That was the same phenomenon as the foot clonus; it was the tonic contraction of the muscles under the reflex influence, broken into clonic spasm, such as I doubt not can be easily shown you in the rectus. You see, I only have to press the patella down and you perceive how much the rectus is agitated, so extreme is the excess of myotatic irritability. That proves that the path from the muscles to the lumbar grey matter is intact; the grey matter is intact in the lumbar enlargement, and the path from the grey matter to the enlargement is intact. It excludes any disease of the lumbar enlargement. I say ‘any disease’, but it does not include that secondary degeneration of the pyramidal fibres passing into the lumbar enlargement which, as you know, always occurs from disease higher up. But we cannot call that ‘disease’; we can only call disease that which is the primary morbid process. The other is the secondary—I was going to say the physiological—result of the primary affection. These symptoms conclusively show that the disease was in the lumbar enlargement. An acute onset of disease in the lumbar enlargement involving both sides of the cord shows that the disease is inflammation—myelitis. A sudden onset would suggest that it is haemorrhage, but sudden onset due to haemorrhage is usually accompanied by pain. There is occasionally an almost sudden onset of the symptoms in myelitis, that is to say a considerable degree of weakness may be reached in 10 or 15 min. In such cases it is probable that the inflammation is itself haemorrhagic, but it is not primary haemorrhage.

First, let me remind you that the symptoms of what is called transverse myelitis are: interruption of the conducting functions of the cord at a certain level. For instance, you will find total palsy of the legs, paralysis of the abdominal muscles, and probably loss of sensation up to the level of the ensiform cartilage; that means simple interruption to conduction at the level of about the fifth or sixth dorsal nerves. But remember that that tells you nothing whatever of the state of the cord below. There may be a transverse myelitis only a half an inch in extent, or the whole of the cord below may be the seat of inflammation; and so far as motion or sensation are concerned, the symptom is the same. It is only by the central function of the cord and reflex functions that you can estimate the state of the cord below. When you find transverse myelitis limited in its extent at a certain level in the dorsal region, the functions of the cord below are intact, and the muscle reflex action gradually becomes augmented by a sort of functional hypertrophy—if the expression may be used,—in consequence of the loss of the restraining influence from above until you find the state of spasm, tonic and clonic, which is present in this patient. A simple transverse myelitis does not permanently abolish reflex action in the lumbar enlargement; it has been said to do so, but the statement is an error. It is due to misapprehension of other cases in which after or with a primary myelitis of the dorsal region there has been a descending myelitis into the lumbar region. I remember one case, for instance, in which a patient had a transverse dorsal myelitis, with complete paraplegia and excess of the muscle reflex action, excess of the knee jerk and foot clonus developed in the course of a week. About a fortnight after the onset, slight returning power suddenly disappeared, with that the knee jerk disappeared also, the foot clonus could no longer be obtained, the muscles began to waste, and presented the degenerative reaction. The myelitis had spread down the cord and involved the lumbar grey matter. Cases such as that have, after having been seen only at a later stage, been suggested as evidence that a transverse myelitis may cause loss of reflex action in the lumbar enlargement, a conclusion which this case and cases which you will see almost every day absolutely disprove.

Two other points I would mention. First, as I have said, we know little of the real pathology of myelitis; we know very little of its causes. The little that we do know suggests that it is due, in some mysterious way, to a morbid blood state, and that is due of transverse myelitis, as well as of other forms. I hope, at the next lecture, to consider the other forms in somewhat more detail. But we have distinct evidence of a blood state in poliomyelitis in which we have constitutional symptoms—pyrexia, etc, conspicuous in the majority of cases, but varying without any proportion to the spinal lesion. We have similar facts regarding some other forms of myelitis.

I described to you once here, in a lecture which has been published in the volume just issued, a case of myelitis of gonorrhoea, in which apparently some post-gonorrhoeal chemical poison or chemical influence (we could find no organisms) led to severe, intense, fatal ascending myelitis. And when cold produces myelitis, we are still unable to escape from the compulsion of regarding the mechanism as upwards, as must be the mechanism through cold caused such a malady as acute rheumatism, or cold pneumonia, the disease which perhaps has a closer relation to acute myelitis than any other. Hence it is most significant that in this patient the attack should have come on immediately after severe suppuration in the neck; the abscess must itself have been due to some morbid blood state, and perhaps capable of augmenting it. It is a point on which I cannot urge you too strongly to keep your attention open, because I believe that many are the facts regarding acute myelitis which come under the notice of practitioners, which, grouped with
others would be of great significance, but which to them are isolated, or look so, and therefore they are too apt to think unimportant. We sadly need an organization for enabling men to communicate facts which come under their notice, but which seem to them not worth the trouble and not worth the labour of getting from their scanty leisure enough time to put in order and send to be published to rank with the all too-numerous facts which crowd our papers. But we do need an organization by which facts might be sent, perhaps processed and classified and compared with other facts which may come in the future, in order that the great mass of knowledge and information now lost may be saved.