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William Gowers: the never completed third edition of the ‘Bible of Neurology’

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William Gowers’ classic single-authored two-volume A manual of diseases of the nervous system appeared in a first edition in 1886 and 1888, and in a second edition in 1892 and 1893, with a third edition of Volume 1 in 1899. No third edition of Volume 2 ever appeared. However, in 1949 Critchley stated that he had seen part of a revision of this volume. Subsequent writers could not find this material, but it recently came to light at Gowers’ old hospital at Queen Square, London. The present paper describes the rediscovered material, containing Gowers’ handwritten alterations for a further edition of Volume 2, and substantial new material, at least in relation to nystagmus and myasthenia. Gowers’ declining health, or a conflict between his planned new text and his contributions to the neurology segments (1899) of Allbutt’s System of medicine, may explain why a third edition of Volume 2 of the Manual of diseases of the nervous system never appeared.

Keywords: Gowers; Manual of diseases of the nervous system; myasthenia; nystagmus; Queen Square

Introduction

In 1886 J & A Churchill, the London medical publishing house, brought out Volume 1 of William Richard Gowers’ A manual of diseases of the nervous system. Volume 2 followed in 1888. Volume 1 dealt with diseases of the spinal cord and peripheral nerves; Volume 2 with diseases of the brain, cranial nerves and general and functional diseases of the nervous system. Gowers (1845–1915) at the time was physician to the National Hospital for the Paralysed and Epileptic, Queen Square, and to University College Hospital, London, and Professor of Clinical Medicine at the latter. His intention in writing the Manual of diseases of the nervous system was to:

‘attempt to give an account of diseases of the nervous system, sufficiently concise to be within the compass of the time-pressed student or busy practitioner, and yet adequate in its outline of a subject which has become wide and deep beyond any other part of medicine.’

He succeeded well enough for the volumes to become known to several generations of British neurologists as the ‘Bible of...
Neurology’, though Spillane (1891) suggested that it should be regarded as neurology’s ‘New Testament’.

Enlarged second editions of the Manual of diseases of the nervous system’s two volumes appeared in 1892 and 1893, respectively, with a third edition of Volume 1, co-edited by Gowers and James Taylor, in 1899 (Fig. 1). No third edition of Volume 2 ever appeared, though Gowers’ obituary in the British Medical Journal erroneously stated that it had, in 1899 (Anonymous, 1915a). Critchley (1949) in his biography of Gowers, wrote that he had seen a draft revision of a third edition of Volume 2. Critchley’s Plate IX illustrated page 555 of Volume 2 of the second edition, dealing with insular sclerosis. On it, Gowers had made handwritten alterations. Years later, McDonald (1986), when preparing for his Gowers Memorial Lecture, could not find the draft revision, and regretted that it was ‘sadly now missing’.

The lost revision of Volume 2 of the third edition of the Manual of diseases of the nervous system was rediscovered at Queen Square in 2008. Ann Scott was researching the background for her biography of her grandfather Ernest Gowers (William Richard Gowers’ son), who was Chairman of the Board of Governors of Queen Square from 1946 to 1957 (Scott, 2009). The Queen Square Library had just taken over responsibility for the Hospital’s archives, then uncatalogued and stored in cupboards on one of the hospital corridors. A preliminary search by Scott and the librarian Louise Shepherd, revealed an album of William Richard Gowers’ holiday sketches. When the library catalogued the archives, more of Gowers’ papers were discovered, some in a bundle amid other old documents. The significance of this material was not immediately recognized. Later, handwriting on it was verified as that of Gowers by comparison with handwritten letters bearing his signature.

The present article discusses the rediscovered material, the changes Gowers intended for a third edition of Volume 2 of his Manual of diseases of the nervous system, and speculates on why this volume was never published.

The rediscovered material

Nearly all of the papers in the bundle were groups of consecutive single pages from the second edition of Volume 2 of the Manual of diseases of the nervous system. Each page was glued to the left-hand side of a larger sheet of blank paper, allowing written alterations made from the right-hand margin of the existing type to overflow onto the blank paper. There were also two sets of handwritten pages and a few fragments of pages, some containing handwriting, some shorthand and one a combination of both.

One set of handwritten pages described the initial part of the history of a patient with epilepsy. Its content was almost identical to the account of a subject (James S) described in Gowers’ Hughlings Jackson lecture (1909). These pages contain internal evidence of being written in 1904, or later, and are not considered further. All of the remaining material, except perhaps for a few of the paper fragments, appear relevant to a third edition of Gowers’ Manual of diseases of the nervous system. At least four copies of Volume 2 of the second edition of the manual must have been taken apart in preparation for a new edition, since there were two sets of pages 421, 422, 423, 424 and 543. At some time, consecutive pages from Volume 2 must have been separated into sections that largely corresponded to the book’s chapters, or major parts of chapters. Nearly always, whole sections either had survived intact, or were absent. Including the title page and the index, pages for nearly half the total text of Volume 2 of the second edition were present in the bundle. Table 1 lists the full set of chapters and section headings of this volume and their corresponding page numbers. The surviving pages and sections are highlighted in Table 1. Some of the surviving sections were not annotated; others contained handwritten alterations in ink or rarely in ink superimposed on handwritten pencil amendments. The revised groups of pages are indicated in bold type in Table 1.

The groups of sheets of backing paper to which printed pages had been glued all had one, two or occasionally three perforations, presumably for binding devices, towards their upper left hand corners. This suggests that the bundle of pages had been taken apart and reassembled, probably more than once, at various stages of its existence.
Gowers’ intended revisions

The revisions fall into three classes as follows: (i) removal of the more speculative interpretations and redundant text; (ii) insertion of new material; and (iii) general text shortening and simplification. Gowers deleted a considerable proportion of the section on the motor nerve supply of the eyeballs, its accompanying line drawings and the associated references in the footnotes (Fig. 2). He removed details of the anatomy of the relevant nerves, indicating that this was described earlier in the volume, but on page 168 added details of the innervation of the eyelids. He crossed out detailed material on patterns of abnormal eyeball position and movement, stating that these matters were now well covered in textbooks of ophthalmology. Interestingly, this particular section had been the only one singled out for detailed criticism by the British Medical Journal’s reviewer of the second edition of Volume 2 of the Manual of Diseases of the Nervous System (Anonymous, 1894). On page 180, he replaced the section on isolated palsies of single external eye muscles with the following account of congenital abnormalities that was not present in the second edition.

‘Congenital anomalies are not rare, and sometimes entail diagnostic difficulty. One eyeball & ocular fissure may be a little higher than the other, & it is very common for the upward movement to be unequal in degree. Sometimes some movement is oblique instead of straight in one eye; the left eye for instance in looking horizontally to the right, moves upwards as well as inwards. In these cases there is often also a difference in level. The fact that in congenital cases double vision can never be found is an important aid to their recognition.’

Gowers also deleted statements that by then were probably considered inaccurate, for example, that partial paralysis of an external eye muscle could cause nystagmus (p. 171). He also deleted certain case histories of his own patients, and certain interpretations of altered physiology that seem to have depended on reasonable inference rather than established facts. The entire section on nystagmus was to be replaced with a handwritten account inserted at the appropriate position in the papers.

The earlier part of the second edition’s section on cerebral haemorrhage was missing. In the remaining part, Gowers’ deletions were sporadic and minor, or involved removing accounts of his own illustrative cases. Similarly, the deletions were trivial in the sections on brain degeneration and disseminated sclerosis. The overall tone of what remained seemed slightly more conservative than in the second edition. In the annotated parts dealing with narcolepsy, hypochondriasis and neurasthenia, Gowers’ deletions were all minor.

Throughout his revision Gowers made relatively short alterations, mainly elisions, shortening the text. Thus the sentence in the second edition (p. 400; Fig. 3)

‘the enduring symptoms, which persist after the initial stage is over, are due to local interference with the functions of the damaged part of the brain, and are determined by the situation of the lesion.’

became

‘the enduring symptoms, due to destruction of tissue, depend on the situation of the lesion.’

The insertions Gowers proposed for the revised sections mainly comprised short statements of new facts or new interpretations. For example, that there was increasing evidence that particular
eye movements were represented in particular cell groups within the oculomotor nuclei in the brainstem, and that the optic neuropathy of disseminated sclerosis progressed less rapidly than that of tabes. Such changes modernized the text though, individually, none was of any great moment. However, there were two more substantial insertions, one lengthy, the other brief and both of interest.

More major changes

Pages 207 to 210 of Volume 2 of the second edition were absent from the material that survived, but were obviously to be replaced by a new handwritten account of nystagmus (Fig. 4), transcribed in Appendix I. The revision is better organized and lacks the personal case material of the earlier version. It may not have been at
those that are the direct effect of the destruction; those persist for months, and, unless the loss is such as to render that it can be supplemented by the action of the other hemisphere, they continue for the rest of life. The symptoms may be mainly motor in the homolateral side, and the symptoms they produce last longer than do those that result from the slighter pressure on distant parts. The symptoms produced by these two mechanisms have been distinguished as "direct" and "indirect" symptoms. In those cases the homolateral hemisphere, and can only be distinguished in practice by the gradual disappearance of the one and the persistence of the other.

In some cases there is a slight increase of the local symptoms during the period of "inflammatory reaction," due, not to the damage to adjacent structures by the inflammation around the lesion. Chronic Stage.—The enduring symptoms, which persist after the initial stage in part due to the local interference with the functions of the damaged part of the brain and are determined by the situation of the lesion. Persistent general cerebral symptoms, such as, for instance, as are complications in cases of tumour, are for the most part about in hemiplegia. Headache is trifling; optic neuritis is commonly seen. Consciousness is rare. Some mental change may occur, slight or considerable, and evidence objects by defective memory, probability, and emotional instability, but is less than in other conditions. Local symptoms persistent in most cases are about when the lesion is too placed to spare the structures concerned directly in mechanisms.

The most common symptoms is hemiplegia, because hemihemorrhage is most frequent in the region of the corpus striatum and internal capsule, and the anterior part of the thalamus, lesions containing the parts of the motor path rarely escapes laceration or compression. When damage is due to the motor centre of the hemisphere, the hemiplegia is complete, hemiplegia frequent near the posterior part of the capsule, in which the sensory tract is contained. But this is an uncommon occurrence, there being some blunting of sensibility, especially at the extremities of the limbs, and especially in hemispheres transfixed by a cranial wound, and may co-exist with tactile anesthesia, and be accompanied by spontaneous pains, but these are less common in cerebral softening. Trophic disturbances in the hemiplegic side vary in frequency and degree; there may be persistent sensitiveness, blunted or even abolished sensibility.

A not infrequent after-effect results from the pressure effects as much as the direct effects of the hemorrhage as to the localisation. Only the local effects are, strictly speaking, indirect, but these cannot be separated practically from those that are due to pressure.

Figure 3  Gowers’ annotations on page 401, dealing with cerebral haemorrhage, with two perforation holes in the upper left hand corner. Image courtesy of the Queen Square Library, Archive and Museum. Copyright National Hospital for Neurology & Neurosurgery, London.

final draft stage for it seems less polished than Gowers’ usual accounts. There is more on Gowers’ proposed pathophysiology of nystagmus, centred on the hypothesis that the phenomenon results from disturbed reciprocal inhibition, the mechanism whereby an opposing muscle relaxes when its corresponding prime mover contracts. Sherrington had investigated this phenomenon since 1893 and published a series of papers on it including his 1897 Croonian lectures (Sherrington, 1897) whose full text became available in 1898 (Sherrington, 1898). By 1899 Gowers knew of the role of the muscle spindles (Beevor wrote a short section on the spindles at the end of the third edition of Volume 1 of the Manual of diseases of the nervous system), and
that they did ‘not present the same aspect in the ocular muscles as in others’. Gowers suggested that nerve impulses from the tendons of the external eye muscles travelled via the fifth cranial nerve to the brainstem to act on neurons in the external eye muscle nuclei, thus providing the anatomical background for his interpretation of the mechanism of nystagmus. In the second edition of the Manual of diseases of the nervous system Gowers had suggested that nystagmus arose from various sites in the CNS, including the spinal cord. For the third edition he limited the neural sites of origin of nystagmus to the brainstem and cerebellum, and the labyrinth. Gowers’ new account provided a major revision of the topic. It showed that he had kept abreast of advances in physiology, and could utilize these advances to develop new explanations for disease phenomena.

The second significant alteration proposed for Volume 2 appeared at two separate places in the surviving material. On
Discussion

There is little doubt that, though others failed to find it in the interval, a substantial part of the revised text for the third edition of Volume 2 of Gowers’ Manual of Diseases of the Nervous System, with proposed revisions made in Gowers’ handwriting, has been rediscovered some six decades after Critchley (1949) last recorded having seen it. The identity of page 555 (Fig. 5) of the rediscovered material and the page that Critchley (1949) illustrated makes it virtually certain that the set of pages recently found at Queen Square is that which Critchley saw. Unfortunately, a little over half of the original text of Volume 2 is missing.

How did the material come into Critchley’s hands?

James Taylor (1859–1946), the disciple and friend of Hughlings Jackson and Gowers, was responsible for compiling Hughlings Jackson’s Neurological fragments (1925), editing his Selected writings (1931) and co-editing Volume 1 of the third edition of Gowers’ Manual of diseases of the nervous system. It seems likely that he was also to have been joint editor of Volume 2 of the third edition of the manual, though Gowers would have played the leading role in the writing. Possibly Taylor and Gowers each received a set of second edition pages prepared for revision and Gowers’ annotated set came into Taylor’s hands later and at some stage found its way to Queen Square. In his preface to Gowers’ biography, Critchley (1949) acknowledged the assistance he received from Mrs James Taylor, and an etching ‘The mouth of the lyn’ by Gowers, reproduced in the biography (Plate X, facing p. 88), was loaned to him by Taylor’s daughter. The revised sheets may have been given to Critchley by Mrs Taylor. It would explain the duplicates of a few pages of the manual in the surviving material.

Why was the revision never completed and published?

The existence of a significant part of the revised text of Volume 2 of the Manual of diseases of the nervous system raises the question as to why the work was never published. When Volume 1 of the first edition appeared its readers were told that Volume 2 was in press. When Volume 1 of the second edition appeared, readers were reassured that Volume 2 would soon be ready. No assurances were given regarding to the appearance of Volume 2 of the third edition when Volume 1 was published.

Work on Volume 2 of a third edition appears to have begun with the cooperation of J & A Churchill. Gowers’ revisions were written partly on the larger sheets of paper to which pages from the second edition were glued. Such sheets were the sort of materials that Churchill’s successor, the firm of Churchill-Livingstone, provided to its authors for revising a book in the days before word processing. Gowers’ written changes show that he had made a serious attempt to modernize and shorten the text of Volume 2. Why he chose to shorten it and omit case histories is unclear.
Gowers and Taylor had expanded the text of Volume 1 of the third edition from 616 to 692 pages. Comparison of some random sections of the second and third editions of Volume 1 (those on sciatica, acute ascending paralysis, ataxic paraplegia and Thomsen’s disease and paramyotonia) show no attempt to carry out the editorial style shortenings of text that Gowers apparently intended for Volume 2 of the third edition.

Critchley (1949) wrote that Gowers suffered a breakdown in health in 1894, suffering severe back pain which Gowers later hinted was ‘lumbago’ (Gowers, 1904). He went on a voyage to South Africa and back to recuperate, returning ‘with the second edition of his Manual re-written, corrected and ready for the press’ (Critchley, 1949; p. 93). We now know that he went to South Africa in 1898, not 1894 (Scott et al., 2012). It must therefore have been the third edition of Volume 1 that Gowers worked on during the voyage. Possibly when unwell, Gowers may have allowed what he considered reasonably satisfactory material from the earlier edition of Volume 1 to remain unaltered. Later, perhaps in better health, he was more radical in revising Volume 2.

Gowers probably worked on the revision of Volume 2 in the 1898–1900 period. On page 1040 of the second edition, despite making alterations, he left unchanged a statement to the effect that hypochondriasis had been recognized only in the early years of the present century. This suggests that he was writing before 1901. Furthermore, in his new section on nystagmus he made substantial use of Sherrington’s concept of reciprocal inhibition, an idea he probably would have become aware of around 1898, or slightly earlier.

Could poor health have prevented Gowers from completing the revision of Volume 2, in which he would almost certainly have been the dominant partner? On medical advice to reduce his workload he gave up editing his pet project, the Phonographic Record of Clinical Teaching and Medical Science, in 1899. However, he published several major papers between 1899 and
In 1900, he produced a second edition of his monograph *Epilepsy and other chronic convulsive diseases*, which included his analysis of his records of 3000 patients. Although Foster Kennedy wrote in 1908 that Gowers was ‘breaking up badly’ (Butterfield, 1981), his book *The borderland of epilepsy* appeared in 1909. On the whole, declining health does not seem a sufficient explanation for Gowers’ failure to complete the third edition, although it may have contributed.

Gowers’ *Lancet* obituary (Anonymous, 1915b) mentioned that he had contemplated producing a single volume shortened version of the *Manual of diseases of the nervous system*, omitting anatomical and physiological material. This possibility may have diverted him from his revision for the third edition, but if so, neither project ever came to fruition, whereas other subsequent major publications from his pen did. Also, there had been an American single volume 1357—page version of the first edition of the *Manual of diseases of the nervous system*, and its format had been criticized for being ‘exceedingly clumsy’ (BS, 1888).

Critchley (1949) suggested that sales of the third edition of Volume 1 of the *Manual of diseases of the nervous system*, published in 1899, may have been adversely affected because Macmillan brought out Volumes 7 and 8 of Allbutt’s *System of medicine* in the same year. These volumes contained the full multi-authored neurological content of the *System of medicine*. Perhaps this is the explanation, though it seems unlikely that, as early as 1900, poor sales of Volume 1, which had been published only in the previous year, would have been sufficiently evident to cause further work on Volume 2 to be abandoned. Also, three reviews of Volume 1 (Anonymous, 1899a, b, c) in the *British Medical Journal, Lancet and Journal of the American Medical Association*, were highly favourable.

Volume 7 of Allbutt’s *System of medicine* contained Gowers’ account of epilepsy (Gowers 1899a), and Taylor’s chapters on ‘The cerebral palsies of children’ (Taylor, 1899a), and ‘Occlusion of cerebral vessels’ (Taylor, 1899b). Gowers had annotated the text relating to Taylor’s latter topic for the third edition of the *Manual of diseases of the nervous system* before his revising appeared to cease. Allbutt’s Volume 8 contained Gowers’ account of paralysis agitans in which he developed his ideas relating to stress as a triggering factor (Gowers, 1899b). These recently available accounts in a work produced by one publisher and written by the authors of Gowers’ *Manual of diseases of the nervous system*, dealt with substantial topics that would have had to be considered in Volume 2 of a new edition of the manual (with its different publisher). This situation may have produced copyright or other commercial issues. Whether such matters explain why a third edition of Volume 2 of the *Manual of diseases of the nervous system* never appeared can now be only a matter for conjecture. However, the timing, and Gowers apparent abandoning his revision part of the way through the task, are consistent with this possibility. Whatever happened then, relations between Gowers, Taylor and J & A Churchill seem to have remained amicable. As well as the second edition of Gowers’ *Epilepsy and other chronic convulsive diseases* in 1901, Churchill brought out his *The borderland of epilepsy* in 1909 and Taylor’s *Paralysis and other diseases of the nervous system in childhood and early life* in 1905.

The real reason for Volume 2 of the third edition of Gowers’ masterpiece never appearing may forever remain uncertain, but evidence is again available in the archives at the National Hospital for Neurology and Neurosurgery at Queen Square that he had gone some distance towards completing what was possibly his only unfinished major project.

References


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Nystagmus

Nystagmus is an oscillation of the eyeballs, rarely of one only, due to an alternate contraction of the opposing muscles. They should act together, the opponent supporting yet yielding to the acting muscle. The alternation causes a to and fro movement, usually of both eyes and synchronous. It occurs on voluntary movement, but sometimes goes on in the mid position. In the direction in which the will acts the movement is more rapid than in the return. It may recur on movement in any direction or only in one. In the horizontal movements, it is usually greater in the outward moving eye, which is the more important. Sometimes the motion is rotatory, & then is slight and often unilateral—the degree of movement varies much; a slight jerking may attend defective power after it has rested for some time and has been thought to be different in nature from the pronounced oscillation, but this is uncertain. The motion sometimes varies in degree, even under observation. Usually uniform, a considerable movement sometimes separates two or three smaller jerks. Its time varies, and is the more rapid the smaller the range of movement: roughly speaking, & then is slight and often unilateral—the degree of movement varies much; a slight jerking may attend defective power after it has rested for some time and has been thought to be different in nature from the pronounced oscillation, but this is uncertain. The motion sometimes varies in degree, even under observation. Usually uniform, a considerable movement sometimes separates two or three smaller jerks. Its time varies, and is the more rapid the smaller the range of movement: roughly speaking, the double movement occurs from 80 to 180 per minute Other features will be considered presently.

From the common form, the acquired nystagmus of central disease, two other varieties may be distinguished, which are important in connection with its origin. An infantile form begins in the first month of life. Sometimes it is associated with a similar alternate contraction of the muscles of the head and neck causing nodding movements. More commonly it develops when there is a condition lessening the amount of light which enters the eye, such as a superficial disease of any kind. It also occurs in albinism, when the amount of light is in excess. Thus, there is an abnormal amount of light during the time when the child acquires the power of fixing a light, a process in which a reflex action on the muscles takes an important share. This form consists of an alternate action of the same rate, there is not the quicker motion in the direction of volition.

Miner’s nystagmus develops in those who have worked for years in getting coal, lying in a constrained position with the head inclined. It occurs especially in badly lighted mines and is disposed to by defects of general health and alcoholism. It is often rotatory.

The common form results from organic disease or degeneration, near or in the mid-brain, pons, or cerebellum. It is not met with in disease of the cerebral hemisphere, above the central ganglia; rarely from disease in these, chiefly the optic thalamus. It may be caused by affections of the labyrinth of the ear that induce vertigo, an important fact in connection with the influence of cerebellar disease in causing it.

Pathology. The slightest degrees of nystagmus may develop into those that are considerable, and in these there is a deliberate alternation of the opposing muscles. This is apparently the result of an excessive activity of the muscle-reflex action between reciprocals. We cannot doubt that there are structures that subserve this action in the nuclei of the ocular nerves, analogous to those that exist in the spinal cord. Sherrington has obtained, in the latter, a similar insubordination by cutting off the voluntary impulse. The same alternate movement occurs, and he has proved that the arrest of action in one muscle, followed by a contraction in the opponent, is due to an afferent nerve impulse produced by the extension of the latter which inhibits the spinal centre for the former and is followed by activity of its own centre. Thus, the same effect is produced and the alternation goes on. The resemblance to nystagmus is perfect. In this the acting muscles, say those moving the eyes to the right, contract under the will, but just before they reach the limit of movement, they suddenly relax from central inhibition; their opponents, the left-sided muscles, contract, and the eyes move back, rather more slowly, but just before reaching the mid-position, they are in turn arrested and a quicker action of the right sided muscles again moves the eyes to the right.

The structures (muscle-spindles), which are believed to be sensitive to tension, do not present the same aspect in the ocular muscles as in others, but their tendons present sensitive neural structures, and the fact that a twig from each muscle nerve passes to the fifth nerve shows that afferent impulses must arise in them; they doubtless pass to the highest cells of the fifth nucleus, adjacent to the motor nuclei. But the muscle-reflex alternation is not produced by a defect in the volitional impulse, as in the spinal centre. Its causes are such as may disturb the balance of the mid-brain structures, which subserve all ocular reflex action, and also the association of the two eyes. The structural arrangement for the alternation of opposing ocular muscles must be rendered insubordinate with readiness, as is shown by the readiness by which it becomes excessive in infantile nystagmus from a mere disproportion in the amount of light. This must act on the same centre, as far as concerns its influence on the ocular muscles, and enables us to understand that dim lighting is an element in causing the miner’s form.

Nystagmus, when in moderate degree, only occurs on voluntary movement of the eyes, it is absent at rest, in the midposition. The reflex alternation is present only when the centre is energised by.
the volitional impulse. But the latter causes the movement to be quicker in the direction of volition. In time, the nystagmus in a certain direction may attain such a degree that it continues when the will is not acting, and even in opposition to the will; e.g. the quick motion to the right may persist in the mid-position, on upward and downward movements, and even during half the movement of the eyes to the left. These features show clearly that the symptom depends on the lower mid-brain structures. We can understand that the derangement may be slight, so as to cause the nystagmus to have only the semblance of a slight jerking, to which it is reasonable to ascribe to the same mechanism until there is evidence to the contrary. Nystagmus accompanies partial paralysis only when this has lasted for a time. It must be remembered that the impressions from the weakened muscle are lessened and that they exert an influence on the centre. Moreover, the tendency to alternate action seems sometimes to be so partial as to involve individual muscles and, in the oblique movements, in which two muscles take part, the movements may correspond to only one of them, with its opponent. The two eyes act alike, showing that the disorder is of structures above those for the binocular connection. We can conceive the complexity of the arrangement, but anatomy has not yet enabled us to localize or unravel it.

Among the influences that take part in balancing the lower centres are those exerted by the labyrinth, doubtless the semicircular canals and their relation to equilibrium. Increased pressure in an exposed tympanic cavity and therefore the labyrinth causes nystagmus, the quicker movement being towards the other side; diminished pressure also causes it, quick to the same side. Brief nystagmus follows rotation of the body, and the injection of hot or cold water or probing. Disease on one side of the pons, abolishing conjugate movement the side diseased, causes energetic nystagmus on movement to the other side, no doubt by the loss of balance of the reflex structures.

The subjective symptoms are often absent. The oscillation of the eyeballs must cause the image to move to and fro on the retina, but an apparent movement is confined to cases in which the oscillation is wide and then only in the direction of the quicker motion. It is indeed hardly more than a tendency to move, in the same direction as the eyes, as may be perceived by a voluntary quick movement of the eyes, to and fro. Perhaps the tendency to move is only the result of an after image, which disappears in the direction of motion. There is no reason why there should be apparent movement. A movement of the eye, with an opposite and equal movement of the image on the retina, gives the impression that the object is still. Enduring infantile nystagmus is never attended by any sense of movement. On the other hand, it is frequent in the nystagmus of miners and is not easy to explain. The “dancing” of objects renders this form peculiarly disabling.

The practical importance of definite nystagmus is the evidence it affords of organic disease, visible or degenerative. In such a malady as disseminated sclerosis, for instance, with symptoms easily misunderstood, its indication is often of great service. But it is of small localising value except in showing disease in the cerebellum or medial mesial structures, and a lesion outside the pons is probably on the side towards which the chief nystagmic movement takes place. Slight nystagmus may be left by an old paralysis of an ocular nerve which has quite passed away, doubtless from an enduring partial disturbance of balance in the reflex centre. A trifling nystagmus, on certain trained movements, may be congenital but too rarely to be important.