Gordon Holmes lecture: Gordon Holmes and the neurological heritage*

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Sir Gordon Holmes is one of the great figures in the history of twentieth century neurology. In him, there came together the traditions of German and British neurology which moulded a physiological approach to normal and disordered function of the nervous system with a commitment to interpreting clinical phenomena in the light of meticulous pathological examination. He, more than any other individual, was responsible for the form of the neurological examination as it is now performed; and his insights are fundamental to the way that, nowadays, we think about vision, somato-sensory function, the spinal cord and the cerebellum.

Keywords: cerebellum; recovery; shell-shock; spinal cord; visual cortex

Introduction

This article commemorates Gordon Holmes, one of the great figures in the history of our discipline and enables me to acknowledge two debts: one to Brain which, as the vehicle for publication of original work, has been central to the advancement of neurology since 1878 and of which Gordon Holmes was editor between 1922 and 1937; and the other to Gordon Holmes himself whose teachings influenced me indirectly through two of his New Zealand pupils.

J. E. ('Jock') Caughey (1904–2000) taught me how to examine the nervous system using the method (as Derek Denny-Brown used to point out) developed by Holmes. And Denny-Brown was a further conduit through which the personal teachings of Gordon Holmes have survived. Denny-Brown was profoundly influenced by two people—Holmes and Sherrington. In 1966, when he was professor of neurology at Harvard, the only photographs on the wall of Denny-Brown's study in Cambridge MA were of these two men. But as Holmes himself used to stress, there is a lineage in medicine as in all scholarship. Our heritage is a fabric woven of strands from many sources. In considering Holmes's contributions, I shall explore how the nineteenth century heritage had influenced him, and how, in turn, he contributes to ours in the twenty-first century.

The early years

Gordon Morgan Holmes was born in Dublin of protestant farming parents on February 22, 1876. After local schooling he went to Trinity College, Dublin. His neurological training began at the turn of the nineteenth century when, after a trip to New Zealand as a ship's surgeon, he took up a Stewart Scholarship from Trinity College, Dublin, to study neuroanatomy with Ludwig Edinger (1855–1918) in Frankfurt (see Fig. 1). Here he encountered the German neurological heritage which was in fact grounded in British scholarship. Thomas Willis (1661) had been the first to use the term 'neurology' but it was Sir Charles Bell of Edinburgh who provided the more direct stimulus when Romberg translated Bell's collection of papers, mostly published in Philosophical Transactions on 'The nervous system of the human body' into German. Romberg later wrote 'The researches of Sir Charles Bell fill me with enthusiasm, and, in 1831, I translated his great work and made known to my professional brethren in Germany his investigations which will ever serve as models of scientific enquiry.' In 1840, Romberg wrote the first textbook of neurology which was profoundly influential, not only in Germany, but also in Britain after it was translated by E. H. Sieveking (Romberg, 1853), one of the early physicians at Queen Square.

Holmes's time in Edinger's laboratory did not begin propitiously. Gerald Parsons-Smith (1982), who had long discussions with Kathleen Holmes, Sir Gordon's eldest daughter, records that Edinger told Holmes to draw sections of the spinal cord. He spent two days doing so. But when he brought them to the 'Herr Professor' they were torn up in...
front of him. He was finally successful, but only at the eighth attempt. It is noteworthy that Edinger was himself a fine artist. His daughter Tilly recounts how, when Lovis Corinth was painting his portrait, Edinger noticed that Corinth was having difficulty drawing the brain which he was dissecting; whereupon he got up and painted it in for him (Lewey, 1953). Holmes’s experience seems to have made a lasting impression, since he also was known to tear up case notes in the 1930s (Walshe, 1965), and his pupil Denny-Brown adopted this habit in Boston during the 1960s.

Soon however Edinger was so impressed by Holmes that he gave him the brain of Goltz’s famous dog without a forebrain for detailed histological study. This brain was important because it came from the contentious experiment performed in 1892 as a result of which Goltz concluded that normal walking, sleeping and response to visual and tactile stimuli remain in the absence of the cerebral hemispheres even though ‘higher psychical faculties were completely absent’ and there was no evidence of memory. He concluded that ‘all the actions of the animal can be explained as reflex responses to immediate excitation’ (Schaefer, 1900). By this time Holmes’s drawing skills were impressive as can be seen from his paper in the Journal of Physiology (Holmes, 1901: see Fig. 2). Parsons-Smith (1982) states that the drawings were by Holmes; and Walshe (1966) refers to the beauty of the engravings which were done in Frankfurt. Thus, from Edinger, Holmes acquired the techniques which provided the basis for his anatomical and pathological studies of the next decade. Denny-Brown’s view of the development of German neurology was that it was impeded in the late nineteenth century because of a ‘preoccupation with pathological detail almost wholly divorced from clinical significance’ (Denny-Brown, 1965). Edinger was different from most of his contemporaries in emphasizing the functional significance of anatomical investigations, and this he communicated to Holmes (Kreft, 1997).

A similar emphasis was pervasive in the next stage of Holmes’s training when he came to London in 1903 and, on the casting vote of the chairman, became an assistant house physician at Queen Square. There, he worked for Hughlings Jackson who emphasized strongly the need for meticulous pathological study to complement clinical investigation. Moreover, in his view (expressed in 1882), the physician should do the post-mortem examination himself (Jackson, 1932): ‘A practitioner must not be pathologist only, although unless he be a pathologist, he cannot be a good
Some of us work chiefly at physiology, some at pathology, and some of us at clinical medicine. But to be a good practitioner, a man must know much of all three, and most of us nowadays carry on the three lines of investigation.

Jackson’s method, as Holmes described it, ‘was simple—owing nothing to apparatus or instrumental techniques; it may be summed up as that of the unceasing contemplation of facts of observation, scrupulously and untiringly acquired’ (Holmes, 1954). This emphasis on anatomy (including pathology) and physiology reinforced the teachings of Edinger. The additional element was meticulous clinical observation. Fifty years later, contemplating Jackson’s contribution, Holmes wrote: ‘He left a rich legacy of thought to his successors; one not yet exhausted either in its content nor in its stimulating quality. Hidden in his writings ... are penetrating generalizations and hints for further research awaiting the alert student’ (Holmes, 1954). He goes on to say ‘It is easy to lapse into a kind of ancestor worship of Jackson, but those who know his thought best know that it has a life and a pertinence to modern problems in neurology that justify the admiration of the most critical...’ Admiration of Jackson by his contemporaries—often the harshest of critics—did indeed approach idolatry as an amusing document in the Sherrington Archive at the University of British Columbia in Vancouver shows. In a letter to Sir Charles Sherrington dated October 8, 1925, Henry Head refers to a ‘remarkable’ ‘jeux d’esprit’ put together by ‘certain members of the Neurological Society about the year 1880’. It is a parody of the Athanasian Creed (‘Whosoever will be saved, before all things it is necessary that he hold the Catholic faith...’) containing the essentials of Jackson’s doctrine. Head commented: ‘It certainly shows how closely Jackson had arrived at the sort of position we hold today.’

‘Quicunque vult ... whosoever will be saved, before all things it is necessary that he hold the Neurological faith, which faith except everyone do keep whole and undefiled, without doubt he shall perish everlastingly. And the Neurological faith is this: that Unity in the Nervous System, and the Nervous System in Unity, is to be recognized. Neither confounding the Function, nor dividing the Structure. For there is one portion of the Brain, another of the Cord; and another of the Sympathetic. But the structure of the Brain, and of the Cord, and of the Sympathetic is all one; the cells similar, and the fibre[s] identical... Furthermore it is necessary to Everlasting Salvation; that he also believe rightly the doctrine of Dissolution. The right faith is, that we believe and confess; that the Special fails before the general, and the Voluntary before the Automatic... This is the neurological faith; which unless a man believe faithfully he cannot be saved. Glory be to the Brain, and to the Cord, and to the...”

Fig. 2 Section of the cerebellum and brainstem of the dog without a forebrain (Holmes 1901). (Courtesy of The Journal of Physiology).
Sympathetic. As they were discharging, are now, and ever will be, Reflex without end. Amen.

Let us return to Holmes. We have seen that he was an anatomist. But he thought physiologically and, by 1915, was recognized as a physiologist by physiologists: he was invited to edit the volume on the nervous system in the English translation of Luciani’s *Human Physiology* (Luciani, 1911–1921). He became, moreover, one of the supreme clinicians of his day, and was recognized as such by his peers such as Walshe (1966) and his juniors (Critchley, 1979). The general view is reflected in an anecdote from the 1930s. When the residents (one of whom told me the story) would sit round in the evenings and discuss whom they would consult themselves if they developed neurological symptoms the consensus was that they would ask C. P. Symonds to take the history [he was widely acknowledged as having refined the art of history taking (Denny-Brown, 1965)], Gordon Holmes to carry out the physical examination and W. J. Adie to interview the relatives because he was such a nice man.

In Holmes, the German and the British neurological traditions came together, thus enabling his own contribution, and in turn creating his own legacy to subsequent generations. As a perusal of his bibliography shows (see Phillips, 1979) Holmes’s achievements were as remarkable as they were diverse; in anatomy (see Brodal, 1979), pathology, physiology (especially in relation to the cerebellum and the cortical representation of cutaneous sensation and vision); and, more generally, in relation to disorders of the visual system at every level from the lowest to the highest, from the pathology of papilloedema (Paton and Holmes, 1911) to visual agnosia (Holmes, 1918a, b). Three examples from many which could have been chosen illustrate these credentials: the spinal cord and vision, and the response to intolerable stress, again of interest with respect to the concept of ‘shell shock’ developed in the First World War, and of its present day analogues.

**The spinal cord**

With his baptism of fire in Frankfurt, Holmes acquired the techniques of Weigert (Edinger’s colleague) and Bielschowsky for staining myelin and silver impregnation for axons, respectively. These he exploited in an early paper (Holmes, 1906) following appointment as Pathologist and Director of the Research Department at Queen Square in 1904. Its subject is the relationship between loss of function and structural change in focal lesions of the central nervous system. His starting point is the difficulty of correlating the severity of clinical impairments with the extent of axonal degeneration at post-mortem. He chooses to explore the problem by comparing the clinical features with pathology in four cases of compression, three of the cord and one a brainstem glioma. He shows that paraplegia can occur in the absence of secondary (axonal) degeneration. From this he concludes that ‘...the presence of myeline [sic] sheaths is necessary for the functions of the tract fibres of the cord as conducting strands,

and that when these are lost there may be physiological block in the fibres. This condition, from the point of view of function, is equivalent to a structural break in the fibres but differs in the fact that the anatomical integrity of the axis cylinder and its trophic cell remain unaffected ...’

This conclusion became part of the stock-in-trade of Holmes’s colleagues at Queen Square and was articulated with characteristic flair by Walshe 50 years later: ‘... It should not be necessary at this date to pile Pelion on Ossa to establish that the secondary degeneration of spinal cord tracts, pyramidal or other, subject to focal lesions, is not an essential condition of physiological block, for demyelination alone may achieve this’ (Walshe, 1956).

This view, however, did not at once become incorporated into physiological and neurological thinking. Denny-Brown (1944a, b) reached the same conclusion for the peripheral nervous system when studying experimentally the effects of peripheral nerve injury. A. K. McIntyre, professor of physiology at Otago, knew these papers which provided one of three options for an experimental model in which to study the physiological effects of demyelination. Direct electrical proof that conduction block commences precisely where demyelination starts came in the peripheral nervous system (though with a different model) in the early 1960s (McDonald, 1963). Proof in the spinal cord soon followed (McDonald and Sears, 1970). Both these sets of experiments provided new data on the properties of demyelinated fibres, in particular that conduction was slowed, a finding that was important in interpreting clinical nerve conduction studies and later led to the introduction of evoked potentials as a diagnostic tool (Halliday et al., 1972, 1973).

Having said that in the presence of demyelination ‘the anatomical integrity of the axis cylinder and its trophic cell remain unaffected’ Holmes goes on in his 1906 paper: ‘reparative processes would consequently be possible. That such return of function may occur has been frequently observed in the rapid recovery of power and sensation that follow the removal of tumours and the draining of tubercular abscesses which have produced symptoms of compressive paraplegia.’ The implication that the axon must somehow have acquired the ability to conduct again is not spelled out by Holmes. Nor was it made explicit 60 years and more later in work that struggled to explain slowing by the effects of thinning of myelin and widening of nodes (McDonald, 1963, McDonald and Sears, 1970, Rasminsky and Sears, 1972). Not until Bostock and Sears (1978) demonstrated by single fibre recording in demyelinated ventral roots that demyelinated fibres could conduct, did the explanation become obvious. Conduction in these circumstances was very slow indeed, and subsequent calculations showed that delays in the visual evoked potential could be accounted for on this basis, though synaptic factors might also contribute (Bostock and McDonald, 1982). The latest addition to this story is the convergence of evidence from experimental investigations and human pathology (in the Holmes tradition) that such conduction is mediated by
sodium channels inserted into the demyelinated axon (see review in Compston et al., 2005).

There is one other aspect of Holmes's spinal cord studies that needs to be mentioned: his investigation of the acute spinal injuries of warfare, that formed the basis for his Goulstonian lectures to the Royal College of Physicians in 1915. His careful observations made under appalling conditions resolved a number of controversies about pathways in the spinal cord (Holmes, 1915). What is missing from this description is an account of the evolution of bladder disturbance in paraplegia. It did not appear until 1933, in the same issue of Brain as the second of the classical papers by Denny-Brown and Robertson (1933a, b) on the physiology of the normal human bladder (based in part on experiments on themselves) and the chronic neurological bladder. Holmes was prompted to write up his World War I experience because of the rarity with which acute loss of bladder function from spinal trauma was seen at that time in civilian neurological practice (Holmes, 1933). The paper is a masterpiece of clear clinical description, contrasting in this respect (and in this respect only) with the no less important but opaque papers of Denny-Brown and Robertson, of which it is said that F. M. R. Walshe, on encountering Denny-Brown in the corridor at Queen Square, told him that he was waiting for the English translation before reading them.

At the same time as Holmes was making observations on the spinal cord, he was engaged in another endeavour that led to one of his greatest achievements, the elucidation of details of visual representation in the cerebral cortex.

The visual cortex

At the outbreak of war in August 1914, Holmes applied for a commission in the Royal Army Medical Corps. He was rejected because of myopia. But keen to play his part, he joined Percy Sargent, one of the surgeons at Queen Square, and together they went to a Red Cross Hospital just behind the front line (Fig. 3). Their success in dealing with brain and spinal trauma led to a reconsideration of Holmes's fitness, and he was appointed consultant neurologist to the British Armies in France (Parsons-Smith, 1982). Just where work was done in the Red Cross Hospital, close behind the lines.

Readers of Pat Barker’s First World War Regeneration Trilogy (Barker, 1992) or John Garfield’s The Fallen (Garfield, 1990) will know something of the horrific conditions so well captured by the official war artists, in particular C. R. W. Nevinson (Ingleby et al., 1999). There was a constant stream of casualties of all kinds. Harvey Cushing (1936) described his visit to the Unit in 1915 and was amazed by what he saw, up to 900 acutely ill soldiers with head and spinal injuries, convoys of up to 300 wounded men arriving daily, and managed by only 10–16 doctors (Cushing, 1936). Holmes’s energy was seemingly boundless (Walshe, 1965). After a long day at work with the casualties, Holmes would sit up on cold nights in his ‘British Warm’ writing up the day’s research observations. (Phillips, 1979; Parsons-Smith, 1982). The material fell into three main categories: spinal, cerebellar and cortical.

As Holmes states in the introduction to the first paper on vision (Holmes and Lister, 1916) it had been established in the opening years of the twentieth century that the visual area coincides with the striate cortex; the upper part of each retina is represented in the upper part of this area, and the lower in the lower walls and lips of the calcarine fissure. However ‘nothing [was] definitely known on the correspondence between various concentric zones of the retina and the different segments of the area striata’ and the representation of the macula was still controversial.

It is relevant, here, to consider a confusion that developed in the 1990s, and to set the record straight about Holmes' contribution. It has been asserted that Holmes was neglectful, perhaps wilfully, in failing to acknowledge the work of the Japanese ophthalmologist Inouye based on his experience of gunshot wounds of the head in the Russo-Japanese war, published in German in 1909 and recently translated (Inouye, 2000). But this is wrong. In the introduction to his 1916 paper Holmes says ‘... Inouye, in his excellent monograph on the visual disturbances observed after gunshot injuries of the occipital lobes inflicted in the Russo-Japanese War, brings evidence showing that the macula centre must lie towards to the pole of occipital lobe...’ Others thought it lay anteriorly. Holmes makes clear that one of the principal aims in his first study was to clarify this issue. The point is made, not to criticize modern commentators, but to indicate an aspect of Holmes's work bequeathed to his successors: the scrupulous setting of his own observations in the context of observations and interpretations of others—with which, of course, he might or might not agree. He was also careful to discuss the limitations of his own material. In this paper, there were obvious constraints dictated by the variable time elapsing between injury and examination, difficulty of follow-up and the technical limitations of the visual field plots which had often to be performed with the patient lying in bed, although some were later confirmed by means of an ingenious modification, the Bjerrum (tangent) screen, described in the paper. A further difficulty was that localization of the foreign body and its track depended on primitive X-rays of the skull, the results being mapped on to a set of co-ordinates, or at surgical exploration. He is careful not to imply a precision greater than was reasonable.
As a result of his observations on over 2000 cases of head injury and the detailed analysis of 23 cases, he concluded that Inouye was correct in understanding that the centre for macula vision is located rather posteriorly and, more specifically, that it lies in the posterior extremities of the visual areas. He adds further details and says that the centre for vision subserved by the periphery of the retina is probably situated at the anterior end of the visual area.

In the next paper on this topic (Holmes, 1918a) Holmes begins with a résumé of the conclusions of the previous article stating that they 'agree with those that were previously arrived at by Inouye' and 'they conform with Marie and Chatelin's valuable observations on men with wounds similar to those of [his] patients' (Marie and Chatelin, 1914–1915). Here, he confirms the previous observations and extends them to a more detailed description of representation of the concentric zones of the retina and of the vertical and horizontal axes. He concludes that cortical lesions result in congruous defects, that in less severe lesions objects may be detected only when moving and thus, as he says, confirming Riddoch. Visual inattention, analogous to the cutaneous inattention which he and Head had described a few years earlier (Head and Holmes, 1911) is described. In further papers in 1918 and 1919, he reports in detail on other aspects of higher visual function and provides convincing evidence (including from post-mortem examination in two cases) that loss of visual orientation and depth perception, together with disturbances of fixation, convergence and blinking are due to bilateral lesions of the angular and supramarginal gyri and their contiguous areas (Holmes, 1918b; Holmes and Horrax, 1919). In both papers Holmes explicitly acknowledges that his observations conform to those of Inouye and Riddoch. Informative cases were rare. His procedure was to build up an argument in which the cumulative weight of his own evidence and that of others is persuasive. Holmes’s conclusions from this work remain central to the clinical evaluation of vision. The point could not be better put than Holmes did himself in the Ferrier lecture on the organization of the visual cortex in man (drawing on his interest in cathedral architecture for his metaphor): 'My own work on the visual cortex... has required the collection of a large number of observations, for while the physiologist can rely on experiments which he can select and control... the clinician must depend upon the analysis of observations which are rarely so simple or clear cut... The physiologist may be compared with the builder in ashlar or hewn stones which can easily be fitted together, the physician resembles the mason who has to use irregular rubble and therefore requires more time and labour to obtain his end. But in some branches of neurology the
‘rubble’ collected and put together by the clinician is essential …’ (Holmes, 1945).

Subsequent experience has not confirmed all of Holmes’s interpretations. He argued, for example, that in patients with normal acuity following occipital lesions perception of motion could not be dissociated from perception of objects, in contrast to what Riddoch had said (and to what in our own time has been confirmed by Zihl et al. (1983) and Plant and Nakayama (1993), and that loss of colour vision does not occur with purely cortical lesions, whereas this phenomenon was clearly demonstrated much later by Meadows (1974).

The fact that some interpretations based on data obtained with extreme difficulty have not stood the test of time does not diminish one’s admiration for the extraordinary range of Holmes’ definitive contributions to our knowledge of vision. What he said of Jackson is equally true of himself: ‘… we can salute his genius without believing that all he said is final and forever adequate to generalize the great additions to knowledge that have accrued since his day.’ (Holmes, 1954).

Let us return to the first of the 1918 papers already discussed (Holmes, 1918a) and consider one other set of observations contained therein because they relate to recovery of function. Holmes poses the problem as follows: ‘It is not uncommon, however, in work in France, where the earlier stages of gunshot injuries of the head can be observed, to find a complete hemianopia or a large area of total blindness disappear during the course of a few days or weeks. … To what extent can these defects which tend to recover more or less quickly be due?’

Kinnier Wilson (Wilson, 1917) had, as Holmes put it, invoked ‘temporary organic changes, as capillary haemorrhages, minute lacerations and disintegrations of the myelin’ as the basis for reversible visual loss following cerebral ‘concussion’. How he envisaged this working is not clear. Holmes took the view, based on his experience of the spinal cord, that oedema is the principal cause of these reversible changes. How that interfered with function was not clear then, as it is not now (Smith and McDonald, 1999). It is however worth considering further the issue of ‘disintegrations of the myelin’.

We have already seen how, in 1906, Holmes concluded that demyelination produces conduction block, that this is an important factor in compression paraplegia and that the survival of the axons is probably the key to recovery after decompression. Later, others developed an interest in this phenomenon and found that recovery of conduction occurs over the course of a couple of weeks after the induction of a pure demyelinating lesion in the cord, a time course similar to that observed by Holmes in cases of traumatic visual loss (Smith et al., 1981). Both remyelination and the development of new sodium channels in the exposed axon membrane are probably involved (see review in Compston et al., 2005). A similar time course is seen in the natural history of optic neuritis. It is now clear that the situation is complex because, although conduction block is reversed, the responses as judged by the visual evoked potential remain substantially delayed in most—but not all—cases for months or years. Holmes does not address the importance of timing in nervous function, although one of the house physicians at Queen Square when Holmes was first on the consultant staff, E. D. Adrian, later did in his analysis of the neural mechanisms underlying sensation (Adrian, 1932). The importance of timing in synaptic function was recognized by the Sherringtonian school, its mechanisms at the membrane level being established in the 1950s by Eccles and his colleagues ( Creed et al., 1932; Eccles, 1953). How then does it come about that substantially normal vision can return despite the persistence of gross slowing which, because of the patchy distribution of demyelination, will be unequal in different fibres? It seems clear that the central nervous system can adapt to make good use of the distorted input. What is involved remained wholly obscure until the beginning of the twenty-first century and the exploitation of functional MRI.

David Werring and Ahmed Toosy working with Alan Thompson and David Miller in the NMR unit at Queen square took a step towards elucidating the mechanism of compensation by showing that after recovery from optic neuritis a simple visual stimulus activates a much larger volume of cerebral structures subserving vision than normal and that this was an integral part of the recovery process. (Werring et al., 2000; Toosy et al., 2005). The fact that there is comparable extension of cortical activation in association with recovery of motor function after stroke (Weiller et al., 1993) and in multiple sclerosis (Pantano et al., 2002) suggests that recruitment of additional areas for central processing may reflect a rather general recovery strategy. What the synaptic mechanisms are that underlie these changes is not yet known. For the present, we must take the position that Holmes adopted in 1918 in relation to his studies on visual agnosia: ‘I have not … at present sufficient evidence to discuss this interesting question’.

Holmes and mental function
The last aspect of Holmes’s place in the neurological heritage to be discussed is an aspect less certain than those relating to the spinal cord and visual function, and yet more troubling: his approach to mental function and its disorders.

Let us begin with the facts. In Holmes’s classic work, the Introduction to Clinical Neurology of 1946 (Holmes, 1946), which represents the embodiment of his contribution to examination of the nervous system, his account of how to examine the mental state is clear and succinct and, even by modern standards, provides a useful framework. His emphasis is on the way in which changes in mental state can provide clues to the existence of organic cerebral disease. There is a section on suggestibility. As he says: ‘even normal persons are suggestible; any one can walk along a plank lying on firm ground, but may be unable to do so when it spans an abyss’. And he makes the point that suggestion is
particularly important in clinical neurology: first, because it may be the origin of symptoms; and secondly since it is the most effective agent in their removal, a point he made in relation to shell shock in World War I (Army, 1922). If that failed other methods were used which are described in unpleasant detail by Pat Barker (1992), often quoting verbatim from Yealland (1918). Holmes was unsympathetic towards soldiers with shell shock. As Critchley remarked to one Postgraduate, ‘He never liked those people’ (T. J. Murray, personal communication). Nevertheless, a case can be made that he supported and perhaps facilitated the official adoption of changes in practice in dealing with acute psychiatric casualties which had probably been initiated by doctors in the field; the policy from 1917 focused on ‘temporary respite from battle, sleep, food and (relative) comfort followed by return to active duty’, an essentially modern approach (Macleod, 2004). It was clearly effective since the number of cases of shell shock declined markedly.

Let us turn now to Holmes and hysteria in civilian life. His reputation in the 1930s, as handed down by my teachers who were house physicians at Queen Square, was that, to say the least, he lacked sympathy. Critchley (1979) records that house physicians would hide patients thought to have an hysterical illness during Holmes’ ward round (partly, at least, to save their own skins, Holmes having ordered that the patient be discharged). Denny-Brown recalled that Holmes was rough with such patients (S. Gilman, personal communication), an approach he shared with his colleagues. The American neurologist Charles Aring, who had been a postgraduate student at Queen Square in the 1930s, observed that the ‘great men from the National Hospital in those decades’ freely ‘showed their impatience with psychological

Fig. 4 Sir Gordon Holmes (Courtesy of the Institute of Neurology).
problems’ and showed an ‘ineptness [in handling psychiatric patients] which was never exhibited towards sufferers from somatic neurologic disease’ (Aring, 1965). He suggested that the Queen Square position derived from the austerity of a Victorian moral stance hedged by tradition. What remains surprising is that the staff at Queen Square, who had developed a very high degree of sophistication in analysing the symptomatology of organic neurological disease based on precise observation, did not consider that, in the mental sphere, there was a need for equal precision and penetrating analysis. This is perhaps particularly true of Holmes with his dual inheritance from Jackson who viewed psychiatric disorder as a consequence of dissolution of the highest level of function of the brain, and Edinger, whose institute was predicated on the assumption that with departments of comparative neuroanatomy, neuropathology, clinical neurology and psychology, bridges would be built from structure to an ‘objective psychology’ (Kreft, 1997). We must conclude that Holmes’s legacy in relation to mental problems is problematic.

**Conclusion**

Perhaps this confusing chapter is best left without looking back lest—one lot’s wife—we are turned to pillars of salt. It should not shroud Holmes’ overall contribution. In concentrating on his intellectual legacy, little has been said about the man. The formidable side is plain enough. But this perception must be tempered by the evidence, written and oral, of the affection in which he was held by colleagues, contemporaries abroad (such as Wilder Penfield), and pupils such as Aring, Denny-Brown and N. S. (‘Barney’) Alcock (1909–2006), who considered that Holmes was supportive and agreeable to work for (N. S. Alcock, personal communication). Others, however, record that his intolerance of error and ignorance could lead to tough reproval. He was given to shaking his students by the lapels to reinforce a point, and to grasping a passing house physician by the arm while asking ‘What are you doing, my boy?’ (in research, being implied; Walshe, 1966; Critchley, 1979) Critchley regarded this as ‘his form of blustering, benevolent, humorous encouragement.’ It must nevertheless have been somewhat intimidating since in his middle years Holmes was a massive figure. As editor of *Brain*, he was helpful to the young Critchley, inviting him to dinner and afterwards taking a blue pencil to the manuscript he had been invited to bring along, deleting unnecessary adjectives in pursuit of the principle of the unadorned clarity which characterizes his own prose. He avoided committee work which he disliked intensely (Walshe, 1966). Beyond neurology he had serious interests in literature and ecclesiastical gothic architecture. In his younger years he was a keen walker and later an
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oarsman. He and his wife played golf together (often midweek) throughout his professional lifetime and, in retirement, he gardened passionately, especially enjoying making bonfires of the rubbish, a scene spectacularly captured on an amateur cine film in the Archives at Queen Square. Though immensely distinguished and acknowledged internationally as being so, he was very modest and reticent (Walshe, 1965; Parsons-Smith, 1982). Of his knighthood, he wrote to the Harveyan Librarian at the Royal College of Physicians that he had ‘never been keen in (sic) honours, but my friends have overwhelmed me with congratulations and my family seem pleased.’ Much persuasion was needed to secure his agreement to having his portrait painted at the time of his eightieth birthday (see Fig. 4); this captures his austerity and contrasts with the more benign image taken from the cine film (see Fig. 5).

What is Holmes’s place in our neurological heritage? We have seen how his rigorous approach to observation and documentation, clinical and pathological, derived from both the German tradition through Edinger and the British through Jackson. From both too came his physiological mode of thinking. His contribution is embedded in the way we think about somatosensory and cerebellar function, the spinal cord and vision. It is his examination of the nervous system that we—and countless others—perform daily. Its power derives from the union of a detailed knowledge of anatomy with precise clinical methods for analysing function, grounded in a profound understanding of normal physiology. Holmes (1954) referred to Jackson in the context of Isaac Newton’s comment (borrowed from earlier writers—Merton, 1985) in a letter to Robert Hooke: ‘If I have seen farther, it is by standing on the shoulders of giants.’ Holmes believed that Jackson was a giant as, we conclude, was Holmes himself.

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