The migraine theories of Liveing and Latham: a reappraisal

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This article discusses the leading 19th century British contributions to theories of migraine pathogenesis: Edward Liveing’s theory of nerve storms and Peter Wallwork Latham’s vasomotor theory, providing a detailed accounts of their origin, and their rise and fall in the latter decades of the century, emphasizing the central role of William Gowers in their ultimate downfall. The article concludes by discussing the reasons behind the rising reputation of Liveing’s work, demonstrating how history may be made to serve contemporary ends.

Keywords: headache; history of neurology; migraine; University of Cambridge

Introduction

The major 19th century British contributions to theories of migraine pathogenesis were Edward Liveing’s theory of nerve storms and Peter Wallwork Latham’s vasomotor theory. Christopher Boes and Donald Dalessio, in their historical introduction to the most recent edition of Wolff’s Headache, dub them ‘the prototypical vascular and neurogenic theorists’ (Boes and Dalessio, 2007). This was a very active period in the history of migraine: many theories of head pain had been published over the preceding century, most treating migraine either as a neuralgia of the first division of the trigeminal nerve or as a consequence of cerebral vascular congestion; in addition, since the turn of the 19th century a number of prominent British scientists (including William Hyde Wollaston, David Brewster and George and Hubert Airy) had become interested in the optics of the visual aura of migraine (Schiller, 1975; Plant, 1986). Liveing and Latham were among the first, however, to articulate theories of migraine that attempted to explain the pathophysiological basis of all the phenomena of the migraine attack. Both studied mathematics and medicine at the University of Cambridge in the 1850s, developed their theories of migraine in the 1860s, and published their work—summarized in Liveing’s monograph On Megrim, Sick-Headache and Some Allied Disorders (Fig. 3) (Liveing, 1873) and Latham’s pamphlet On Nervous or Sick-Headache, Its Varieties and Treatment (Fig. 4) (Latham, 1873a)—in the early 1870s. Both theories were well known to contemporary neurologists, but had largely disappeared from view by the early years of the 20th century. Recently, however, Liveing in particular has re-emerged as a central figure in the history of headache. The first section of this article provides a detailed account of the origin of Liveing and Latham’s theories, concentrating on their different institutional backgrounds at Cambridge, and the consequent differences in the philosophical underpinning of their approaches to medical science. The middle section details the circumstances of the rise and fall of the two theories in the latter decades of the 19th century, emphasizing the central role of William Gowers in their ultimate downfall. The final section speculates on the reasons behind the rising reputation of On Megrim, demonstrating how history may be made to serve contemporary ends.
Background: the Cambridge connection

Liveing and Latham entered Cambridge at a time when the University was undergoing immense changes in its social and academic fabric. The medical faculty, saved from irrelevance by the efforts of John Haviland in the 1820s and 1830s, had faltered in the face of government inquiries into the medical profession in 1834 and 1848, and had yet to embrace wholeheartedly the new physiological and chemical sciences that would be its redemption in the 1870s and beyond (Rook, 1969). The social advantages of a Cambridge medical education had been under systematic attack in the radical medical press, epitomized by Thomas Wakley’s Lancet, a process aided by the increasing influence of the metropolitan medical universities and their alumni in the major London hospitals (Weatherall, 2000). Despite this, however, their careers demonstrate the advantages that still accrued to those who chose to spend a portion of their medical training in Cambridge.

Liveing was born in 1832 in Suffolk. He studied natural science and medicine at King’s College, London, before entering Caius College, Cambridge in 1854. His hot-tempered and red-haired brother George (‘the Red Precipitate’) was already regarded as the leading chemist in Cambridge, though he was not to succeed to the Professorship of Chemistry until 1861. Edward read mathematics, becoming 30th Senior Optime in the 1858 Tripos. He took the Cambridge MB the following year, moving back to London to become assistant physician at King’s College Hospital; much of the clinical material collated in On Megrim dates from this time. He sent his MD thesis on migraine to Cambridge in 1868, graduating 2 years later. He acted as an examiner in medicine at Cambridge in 1870–71, and was elected a fellow of Caius College in 1874.

On Megrim was published in 1873. Throughout this period (and indeed afterwards, as he rose in the hierarchy of the Royal College of Physicians), Liveing was following a path worn bare by generations of Cambridge-educated physicians. Caius College was—and had for several hundred years been—regarded as the premier medical college. This reputation rested on the shoulders of its famous alumni, and its social links with metropolitan medical society, not least the link between George Paget and his brother James, a steadily rising star of mid-century British surgery (Rolleston, 1932). There was no local impetus to carry through the exhortation of its most famous medical alumnus, William Harvey, ‘to seek out the secrets of Nature by means of experiment’, Paget having been unsuccessful in his proposal to have a laboratory built at the college in 1845, though one was constructed in the 1860s. The traditional early move to clinical studies in London with the intention of building a metropolitan public and private practice was facilitated by the close professional and personal links between the senior figures in the Cambridge medical faculty and their counterparts in the London medical schools. It is telling in this regard that On Megrim is dedicated to Henry Bond and George Paget, successive Regius Professors of Physic at Cambridge, ‘with the highest sentiments of respect and personal regard by their friend and former pupil’. Those who followed this path successfully, as Liveing did, could expect to
serve their college and the Cambridge faculty more generally in future as lecturers, examiners and external members of boards, committees and panels of electors, particularly if there were, as here, additional personal ties to the University. Liveing was a member of a class of Cambridge medical graduates who were highly influential in directing medical education in the University all the way up to the appointment of John Ryle as Regius Professor in the 1930s (Fig. 1) (Weatherall, 2000).

Latham’s origins were very different. He was born in Wigan, the son of a general practitioner. He worked as his father’s apprentice, before studying at Glasgow University. He too entered Caius College in 1854 to study mathematics, obtaining a higher place than Liveing (19th Wrangler). Unlike Liveing he continued his studies by taking the Natural Science Tripos, at that time still a recent—and to some minds dangerously innovative—development in the University’s curriculum. Latham holds the unique distinction of having achieved the First Class in all five sciences then examined. He was elected fellow of Downing College in 1860. Following a brief stint as Assistant Physician to the Westminster Hospital, he was elected unopposed in 1863 to the post of physician to Addenbrooke’s Hospital, Cambridge, a position that he held until 1899. During the 1860s, he acted as assistant to the Downing Professor of Medicine, William Webster Fisher, and ultimately succeeded Fisher to that post in 1874. Downing College was a relatively modern foundation, being only 60-years old when Latham became a fellow, and medicine had specifically been catered for in its endowment, creating the University’s first new medical professor since the Regius Professorship created by Henry VIII. This in itself would not have been enough to inculcate a spirit of investigation, without the early intellectual drive of Latham’s predecessor Fisher. Fisher has been regarded as an indolent figure, largely on the basis of an anecdote that described him lecturing in his bedroom, but in fact in the 1830s and 1840s he delivered and published well-regarded papers on ganglionic development, an interest that put him in the vanguard of the British clinical neuroanatomy movement (Geison, 1978; Jacyna, 1984). When Fisher applied for the Downing Professorship, he told the electors that its ‘permanent reputation’ could only be advanced ‘through original investigations made by its holder’. The demands of teaching, hospital and private practice, and college life ultimately frustrated him, but the spirit of science and medicine remained strong at Downing, as evidenced by Latham’s appointment to the fellowship in 1860, the award of two scholarships for proficiency in natural sciences in 1863, the first of their kind in Cambridge, and the creation of a college laboratory. Latham himself was an early—perhaps the first—member of a new class of local clinicians with an interest—but not a central interest—in the medical affairs of the
University. As the medical school expanded over succeeding decades, Latham was joined by an ever-increasing cadre of junior physicians and clinical lecturers, many of whom felt that they understood the interests of the ordinary medical student more clearly than their more senior colleagues. When a third interested party—the experimental physiologists led by Michael Foster—arrived on the scene in the 1870s and 1880s, it was Latham who was most vociferous in pointing out their deficiencies over succeeding decades, something that brought him the opprobrium of successive historians of Cambridge medicine (Fig. 2) (Rolleston, 1932; Langdon-Brown, 1947).

Inferences in the clinic: Liveing’s theory of nerve storms

Liveing and Latham derived their theories of migraine during the 1860s and 1870s, a period during which there were extensive debates and arguments about how medical knowledge could best be advanced, and more specifically about how medicine should be made scientific. Liveing states in his introductory remarks that his views on migraine were derived ‘in the way of inference from a collation and comparison of cases’ (Liveing, 1873, p. 3). He presents ‘in a tabular form, for easy reference, the results of an analysis of sixty cases from my own notes and other sources’, his own cases dating for the most part from 1863–65. The published version of Liveing’s work began life as a Cambridge MD thesis, read in April 1870, and the Thruston address at Caius College, delivered in May 1871. A synopsis of the thesis was published in the British Medical Journal in 1872, followed in 1873 by an extended version of the whole, published as a monograph by the London publishers J and A Churchill.

Liveing attempts to demonstrate that ‘nervous afflictions’ including ‘hemicrania, migraine, sick-headache, biliousness, blind-headache, suffusion dimidians, hemiopia, neuralgie ophthalmique and so forth’ are all members of the same ‘pathological family’, and further that they are part of the ‘whole family of functional nervous disorders’ that ‘exhibit that kind of accession, culmination, and subsidence which essentially belong to our notion of a paroxysm or fit of any kind’, attacks which ‘may be not inaptly characterized as a “nerve-storm”’ (Liveing, 1873, pp. 32–3). The technique by which this is done is that of argument from analogy: Liveing sites migraine within the ‘whole family of nervous disorders of which Epilepsy is the type, including Epileptic Vertigo, Spasmatic Grouph, Spasmodic Asthma, Angina Pectoris, Gastralgia, Tic-douloureux, Intermittent and Paroxysmal Insanity, with many others…’. He does this by exhibiting ‘the common characters of some leading members of the group…with a view to show how closely megrim conforms in these respects to the family type’ in ‘a similar order to that which I have observed in tracing the general features of megrim… and leave the reader to complete the parallel, which will thus be rendered sufficiently obvious’ (Liveing, 1873, p. 151). In the subsequent section on the pathology of migraine, Liveing extends the analogy to healthy actions such as sneezing, yawning, hiccupping, vomiting, laughter, ‘the natural development and gratification of our appetites’ (including sexual appetites), sudden fits of anger, and so on. ‘The form of the neurosis or the particular character of the seizure’, Liveing concludes, ‘will be determined for the most part by the concentration of this explosive tendency about particular foci, or its limitation to particular territories, of the encephalitic system; and in accordance with this view a certain correspondence may be traced between the characters of various neuroses and the principal physiological divisions of that system’ (Liveing, 1873, p. 391). By inference from the observed phenomena, Liveing concludes that ‘the “Storm” has its point of departure or principal focus in the optic thalamus, and that its normal course from above downwards, or from before backwards, in the sensory tract’ (Liveing, 1873, p. 396).

This approach to the scientific enterprise derived its legitimacy from appeals to the primacy of Baconian inductive reasoning. Inductive clinical science was supposed to create general laws from analogies drawn from wide clinical experience. The drawing of empirical laws from clinical observations was a matter of judgement, which, according to elite physicians who affected a fashionable adherence to Baconian principles, only came from proper education and long experience. Liveing’s connection with the elite physicians based at Caius College and his subsequent successful metropolitan practice in London put him firmly within this tradition. ‘Experience’ included a just appreciation of the classics of ancient medical literature; it is unlikely to be a coincidence, for example, that most of the members of Liveing’s analogous ‘family of neurosal disorders’ are those which the pre-Galenic physician Aretaeus discussed in his works on the Diagnosis and Treatment of Acute and Chronic Diseases, works which were set texts in the Cambridge MB examination well into the 1850s. Though Bacon’s work had been criticized, qualified and superseded by the middle of the 19th century, an attack on induction having been launched by philosophers such as the Cambridge polymath William Whewell, appeals to Baconian philosophy continued to be a central rhetorical resource for the processes of validation and legitimation by which elite scientific and medical communities established themselves within society as a whole (Barclay, 1864; Yeo, 1985). Liveing was clearly aware of some of the potential limitations of inductive reasoning: when he discusses the paroxysmal character of neurological disorders, a character ‘implied in the name ἐπιληψία, a seizure, by which one of the most typical members of this group is known’, for example, he comments that the terms ‘epileptoid’ and ‘epileptiform’ have been ‘somewhat too loosely and widely applied’ in recent years, though he concludes that ‘their ready adoption is a distinct recognition of a real pathological affinity, and of the need of some such term to express it’ (Liveing, 1873, p. 165). Ironically, as we shall see, it was the growing realization that Liveing himself had ‘too loosely and widely applied’ the nerve-storm concept in his theory of migraine, that ultimately led to his work being forgotten.

Deductions in the laboratory: Latham’s theory

Latham’s monograph On Nervous or Sick-Headache was a summary of two lectures on the subject given at Addenbrooke’s
Hospital. It was published as a pamphlet in 1873, much of its contents having previously appeared in the *British Medical Journal* in March 1872; it was in this pamphlet that Charles Dodgson, the author of *Alice in Wonderland*, first found a recognizable description of his own migrainous aura (Podoll and Robinson, 2009). Further expositions of Latham’s views are contained in two further articles in the *British Medical Journal* in January and February 1873, the main purpose of which is to defend and define his theory that migraine is due to defective tone in the cerebrospinal system, causing excitement of vasomotor sympathetic nerves, producing the ‘prodromata’ (which we would call aura), followed by ‘suspension of the function of this nerve’, producing vasodilatation and headache. Liveing dates the origins of this theory to the early 1860s, recalling that Latham told him of his vasomotor theory ‘in the course of conversation…while discussing some of the more remarkable phenomena’ (Liveing, 1873, p. 319) and is at pains to point out that Latham had formulated his theory without being aware of the views of Du Bois-Reymond (which had been published in German in 1860, then translated into French and criticized by Charles Edouard Brown-Séquard the following year), and before the views of other continental authorities such as Möllendorf had appeared (Koehler, 1995).

Latham’s clinical lectures start with accounts of cases of migraine, the purpose of which is to illustrate a general truth that attacks of migraine are precipitated by ‘exhausting the powers and, therefore, lowering the tone of the system; putting it out of tune, disturbing the harmony in the functions, and, at the same time, exalting the susceptibility of the nervous system’. This, Latham believed, caused the regulating power of the brain over the sympathetic ganglia to be lost and, ‘instead of tranquil, even, harmonious action in the various organs, as in perfect health, we have convulsive, excited, and painful movements’. Latham outlines the actions of sympathetic ‘vasi-motor’ nerves, showing that overactivity of these nerves causes constriction of the blood vessels, while underactivity causes vasodilatation. Proceeding to ‘apply this to the disorder that we have been considering’, Latham outlines a theory (‘advanced not as certain and proved, but only as possibly true’) of the phenomena of migraine that explains them in vascular terms: the observed aura phenomena of migraine are caused by a contraction of the blood vessels of the brain, followed by headache brought on by dilatation of those vessels (Latham, 1872).

Latham’s interest in, and knowledge about the sympathetic nervous system derives as much from his exposure to Fisher and upon physiological observations made in the Downing College laboratory, as from his readings in contemporary physiology. In his clinical lectures, for example, he grounds his vascular theory in observations made of cutting the sympathetic supply to the neck of a rabbit, and then stimulating the distal portion of that nerve by ‘galvanism’ (Latham, 1872). In his subsequent article on ‘The Pathology of Sick-Headache’, Latham sets out to prove his theories, basing his proof on three propositions: first, that the cerebrospinal system contains fibres that inhibit the sympathetic system, supported by a quotation from John Marshall’s 1867 *Outline of Physiology, Human and Comparative*; secondly, that violent emotions can cause the same effects as stimulation of the sympathetic or section of the cerebrospinal nerves, supported by a quotation from William Benjamin Carpenter’s 1869 *Principles of Human Physiology*; and thirdly, that medications that cause ateriolar contraction and stimulate the sympathetic ganglia (he chooses digitalis and gives supporting quotations from Thomas Lauder Brunton and Jan Purkinje), can cause visual symptoms identical to those described by Hubert Airy in the *Philosophical Transactions* in 1870. ‘The deductions which I draw from these propositions’, Latham concludes, ‘are the following: 1. If violent mental emotion stimulate the sympathetic, it does so by withdrawing the inhibitory influence of the cerebro-spinal system, and what violent emotion produces rapidly, long-continued mental effort or fatigue will accomplish more slowly, but as surely. 2. The prodromata of sick-headache are due to excitement of the sympathetic; and this is proved to be the case, by the same phenomena appearing when vision is affected, as are produced by the action of digitalis. 3. It is a physiological axiom that excitement is followed by depression; and consequently excitement of the sympathetic is the cause of the “function of the nerve being temporarily in abeyance”, and the headache associated with this condition’ (Latham, 1873b).

This approach to scientific medicine—based in the rhetoric of rationality and deduction—required pre-existing scientific laws from which the causes of diseases could be derived. Such projects involved, as their most vocal advocate, the Edinburgh physician–physiologist John Hughes Bennett, who in the late 1860s, ‘the skill of the anatomical operator, the analytical power of the chemist, and the varied knowledge, theoretical and practical, of the histologist, physiologist, physicist, pathologist, therapist, as well as of the physician whose knowledge of diagnosis is unimpeachable’ (Weatherall, 1996). Firmly established laws were few and far between in the experimental neuroscience of the 1870s, however; in addition, serious doubts existed that laws derived in healthy animals were applicable to sick people; and to cap it all, in the 1870s a powerful anti-vivisection movement, which threatened to make experimental physiology, and the science derived from it, socially unacceptable. Of these potential problems, it was the first—the relentless advance of clinical neuroscience—that was ultimately to prove the undoing of Latham’s theory.

Reception, criticism and disappearance

Liveing’s monograph was well received. The review of *On Megrim* in the *British Medical Journal* praised it as ‘a work, which, for elegance of style, deep research into previous and contemporary literature, lucid expression, and a scientific interpretation of clinical facts, has had no equal during the last decade’, although the reviewer confessed to disappointment that Liveing had not provided more guidance on the treatment of migraine (Anon, 1873). Contemporary British neurologists were well aware of *On Megrim*, and of Latham’s lectures: John Hughlings Jackson, for example, delivering an oration in 1877 to the Medical Society of London on
ophthalmology in its relation to general medicine, summarized the theories of both authors. Hughlings Jackson regarded the visual and sensory disturbances of migraine as ‘the sensory analogue of spasmodic turning of the two eyes to the side in unilateral epileptic form seizure’. ‘Both are, I think,’ he wrote, ‘epilepsies in the sense that there are “discharging lesions” of some part of the cortex cerebri’. Hughlings Jackson localized the discharges of migraine to the optic thalamus, though he noted that this theory was not yet supported by the kind of clinico-anatomical correlative research into cerebral localization being pioneered at that time by David Ferrier in London and Jean-Marie Charcot in Paris (Hughlings Jackson, 1877). Ferrier himself regarded migraine as a ‘neurosis of the cerebral membranes’ (Ferrier, 1879a, b). When Hughlings Jackson returned to the topic 2 years later, on this occasion in lectures on epilepsy given to the Harveian Society, he restated his opinion that migraine was scientifically to be classified with the epilepsies, though he noted that this theory was not yet supported by the kind of clinico-anatomical correlative research into cerebral localization being pioneered at that time by David Ferrier in London and Jean-Marie Charcot in Paris (Hughlings Jackson, 1877). Ferrier himself regarded migraine as a ‘neurosis of the cerebral membranes’ (Ferrier, 1879a, b). When Hughlings Jackson returned to the topic 2 years later, on this occasion in lectures on epilepsy given to the Harveian Society, he restated his opinion that migraine was scientifically to be classified with the epilepsies, though he noted that this theory was not yet supported by the kind of clinico-anatomical correlative research into cerebral localization being pioneered at that time by David Ferrier in London and Jean-Marie Charcot in Paris (Hughlings Jackson, 1877).

Latham’s most trenchant early critic was in fact Liveing. On Megrim contains a long discussion of previous theories of migraine, culminating in the vascular theories of Du Bois-Reymond (that migraine is caused by constriction of the cerebral blood vessels), Möllendorf (that migraine is caused by arterial vasodilation and venous congestion) and Latham. Liveing’s criticism is based on ‘the inconsistency of the phenomena on which they are based’, asking why—if ‘the contraction of the vessels and the cerebral anæmia are the necessary antecedents of the subsequent hyperæmia and headache’—do many attacks simply consist of pain, and in others the visual disturbance of aura continues into the headache phase? (Liveing, 1873, p. 323). For Liveing the observed vascular disturbances are epiphenomena, and he cites Möllendorf’s view that the origin of vasomotor fibres of the sympathetic lie in the optic thalamus as a possible anatomical basis for this (Liveing, 1873, pp. 397–8) William Gowers, in his discussion of the pathology of migraine in his Manual of Diseases of the Nervous System, published in 1886, summarizes the
problem with the vasomotor theory succinctly: ‘we must assume, first, and initial spasm of the arteries in a small region of the brain; secondly, that the contraction always begins at the same place; and, thirdly, that it can give rise to a definite, uniform, and very peculiar disturbance of function. There is no evidence of the truth of any one of these assumptions’ (Gowers, 1886, p. 789). Latham himself moved on to other interests, concentrating in his 1886 Croonian lectures at the Royal College of Physicians on the organic chemistry of rheumatism, gout and diabetes, and in his 1888 Harveian Oration on the ‘blood-changes’ in disease brought about by the presence of micro-organisms (Latham, 1886, 1888). In Cambridge, the work of John Langley and Walter Gaskell advanced the understanding of the sympathetic, placing it within the context of a more ubiquitous autonomic nervous system, interest in which came increasingly to lie in the description and definition of the role of its various neurotransmitters (Langley, 1921; Langdon Brown, 1939).

Oliver Sacks, in his essay on Liveing that precedes the 1997 reprint of On Megrim, notes that Gowers ‘often paid homage to his colleague’s great book’ (Sacks, 1997). While this is true—he mentioned Liveing’s work specifically in the Manual, and in his lecture on migraine in the series on The Border-land of Epilepsy he calls it a ‘classical work…which, as a medical monograph, has few rivals on any subject’—it was nonetheless Gowers’ critique of Liveing’s theory that did most to ensure its descent into obscurity. In the Manual Gowers echoes—surely deliberately—Liveing’s own comment on his use of the term ‘nerve-storms’, calling it ‘a somewhat inapt metaphor’ (Gowers, 1886, p. 789). He also criticizes Liveing’s localization of the aura processes to the optic thalamus, a ‘suggestion…based on theories…that we now know to be erroneous’; specifically Gowers states that while a visual disturbance might arise from this structure, it could equally well relate to the ‘half-vision structure in the occipital cortex’; he also notes that sensory information is now thought to bypass the optic thalamus, ascending to the cortex via the internal capsule; and finally points out that aphasia is now known to be due to left hemisphere dysfunction, placing the neural origin of migrainous aura in ‘a peculiar kind of functional disturbance in some of the nerve cells of the cerebral cortex’ (Gowers, 1886, p. 791). In this publication, Gowers stressed the analogies between migraine and epilepsy, drawing attention to the ‘common features’ shared by epilepsy and migraine, specifically the combination of ‘arrest of action and of over-action in the nerve-cells concerned’ (Gowers, 1886, pp. 787 and 789) and over the following decade several further cases were published in support of this viewpoint (summarized, with additional cases, by Wilfred Harris in an 1897 Cambridge MD thesis, subsequently published in Brain) (Harris, 1897). The contrary view, put forward most cogently by Samuel Wilks, physician to Guy’s Hospital, that there was no link between migraine and epilepsy, was rarely expressed (Wilks, 1888).

In the section on migraine in the Manual, Gowers mentions 12 cases in which epilepsy and migraine co-existed in the same patient, and puts much emphasis on this relationship. Twenty years later, lecturing on migraine in his series on The Border-land of Epilepsy, his views had clearly changed. ‘It is,’ he says, ‘indeed, somewhat curious that the most frequent relation of migraine to epilepsy that we have encountered in our survey of the facts is as a source of error. The danger of this is greater than I had anticipated when I began to consider the cases that brought the two maladies together, and to lessen this has become of greater importance than I had expected.’ The ‘actual evidence’ of a link between epilepsy and migraine is, he concludes, ‘very slight’, and that while ‘in each malady a state of nerve tension seems slowly to develop, which is at last relieved by a violent functional disturbance…this disturbance differs so much in character that we should, perhaps, not be surprised to find that there is so little evidence of a direct relation between them’. The differences between the two conditions were, he concluded, ‘definite and distinctive’ (Gowers, 1906). In two stages, therefore, Gowers completely undermined Liveing’s theory: in 1886, he had criticized Liveing’s terminology and dismissed his anatomical localization of migraine; in 1906 he swept away the most important supporting analogy. Little remained thereafter, allowing Liveing’s obituarist in the British Medical Journal to dismiss On Megrim as a work that had simply been ‘much esteemed in its day’ (Anon, 1919).

Outside the rarefied atmosphere of the National Hospital, old views of migraine proved persistent. In 1883, Clifford Allbutt, later to succeed George Paget as Regius Professor at Cambridge, but at that stage a physician in Leeds, contributed a case of ‘epileptiform migraine’ to Brain, in the conclusion of which he comments that ‘however strong the evidence may be that migraine is a primary neurosis…yet it is impossible to ignore the testimony of many sufferers who attribute its recurrence…to “bilious” dyspepsia’ (Allbutt, 1883). Thomas Lauder Brunton, one of the leading turn-of-the-century authorities on pharmacology and therapeutics, who in the 1880s had published observations on the calibre of the arterial walls in migraine that might have indicated an attachment to the vasomotor theory, stressed in his lectures to medical students the ‘bilious’ nature of the attacks, commenting that ‘the liver probably has a considerable part to play in the production of headaches’, possibly due to the accumulation of bile, and advising his listeners to give patients emetics, which might bring about a ‘natural cure’ (Brunton, 1897; Schiller, 1975). In contrast to this view Henry Head, then working at University College Hospital, disputed that ‘true megrim’ was in any sense due to pain referred from elsewhere, a view based on his extensive studies of the localization of pain referred from head and neck structures, as well as from the thoracic and abdominal viscera (Head, 1894).

By the time Harold Wolff penned the first edition of Headache and Other Head Pain in 1948, most of the more vibrant contemporary theories of head pain were based in psychology—migraine as a neurosis—or neurosurgery—migraine as a manifestation of processes affecting intracranial pain-sensitive structures. Wolff’s views on migraine derive from both these prevailing strands of thought, but not at all from the late 19th century debates theories above. Liveing’s work, for example, served him solely as a convenient English language summary of the reports of Parry and Möllendorf that migraine headache could be diminished by compression of the ipsilateral common carotid artery, and Latham’s work was not mentioned at all. Wolff had no interest in, or need to, position his researches within a long historical tradition,
an approach that served his claims for the novelty and importance of his experimental observations. He concluded that the ‘preheadache phenomena’ were due to cranial vasodilatation, a hypothesis supported by two reports of amyl nitrite (presumed to act as a cerebral vasodilator) temporarily abolishing visual aura; and that the pain of migraine was due to distension to the cranial arteries, particularly the extracranial, based on his observations of the effect of ergotamine taretate. There was no mention whatsoever of the autonomic nervous system, and just a brief paragraph in which he dismissed any link between migraine and epilepsy as not ‘important from the clinical point of view’ (Wolff, 1948).

Conclusion: the re-emergence of On Megrim

Many recent accounts of the history of migraine accord a prominent place to Liveing, rather less so to Latham. This resurgence in interest in Liveing may be traced to the advocacy of two contemporary neurologists: Oliver Sacks and J M S Pearce. In the introduction to his monograph on migraine, published in 1970, Sacks describes On Megrim as a ‘remarkable Victorian masterpiece’ that in its historical depth and generality of approach ‘has never been equalled’ (Sacks, 1970). Sacks was far more effusive in his praise, for example, than Macdonald Critchley had been in 1966, when in his widely cited historical lecture ‘Migraine: From Cappodocia to Queen Square’, he had merely called On Megrim a ‘minor classic’ (Critchley, 1966). Sacks returned to the fray nearly three decades later, penning an introduction to a reprint of On Megrim published on the occasion of the 8th Congress of the International Headache Society, held in Amsterdam in June 1997, calling On Megrim ‘a treasure of clinical observations; such a mingling, at once, of intellectual passion and human feeling; so riveting, so genial, so easily and naturally written’, recalling that the reading and re-reading of it ‘was crucial for the generation of my own thoughts’ (Sacks, 1997). Pearce calls On Megrim ‘[t]he best text . . . without doubt’, commenting in 1986 that the world was little wiser then about the pathogenesis of migraine than it had been in 1873 (Pearce, 1986). Elsewhere Pearce lauds Liveing’s ‘remarkable and prophetic scholarship’ (Pearce, 1987).

What lies behind these encomia? For Sacks, Liveing quickly becomes ‘an old friend . . . both an ancestor and a colleague’. He lauds the ‘sheer descriptive richness’ of On Megrim, the quality of the writing, the freshness and breadth of vision, and ultimately the ‘sheer beauty of the book’. On Megrim is therefore the epitome of an era in which there was no division between scholarly, technical writing, and popular or general literature. It is precisely this blurring of boundaries that characterizes Sacks’ own literary contributions, which have created (or perhaps recreated) an extraordinarily successful contemporary literary genre—the clinical vignette. Pearce, on the other hand, positions Liveing as a direct ancestor in his quest to develop a neural theory of migraine, an approach which Pearce finds in the works of Liveing and Gowers, before it disappears, waiting for Pearce to ‘resuscitate’ it in the late 1960s (Pearce, 1987, 1999). Pearce’s early advocacy, and the subsequent ascendency of the neural theories of migraine pathogenesis in the 1980s and 1990s, has created an environment in which more recent reviewers find it natural to accord Liveing a central place in the history of migraine: Giorgio Zanchin, for example, credits Liveing with ‘an original concept, that of migraine as a cerebral disorder’ (Zanchin, 2010), and Russell Lane and Paul Davies, in the historical chapter of their recent monograph on migraine, review Liveing’s theory at some length, concluding that ‘his views were more in keeping with our current understanding of migraine pathogenesis than those of his peers’ (Lane and Davies, 2006). Latham, in contrast, is mentioned only in passing in many reviews; Pearce has always been even-handed in his treatment of his work, though ultimately relegates his ideas to an ‘important but secondary role in migraine’ (Pearce, 2006); Francis Schiller discusses his views at some length in his idiosyncratic but often insightful historical review of ‘The Migraine Tradition’ (Schiller, 1975).

Of contemporary historians of migraine perhaps only Hansruedi Isler is less laudatory; in some reviews he barely mentions Liveing (Isler, 1987), and where he does, he damns with faint praise: in his historical introduction to the first edition of The Headaches (toned down somewhat by the most recent third edition), for example, he highlights a ‘British preoccupation with epilepsy’ in which tradition he places Liveing, Hughlings Jackson and Gowers, and suggests that the enduring reputation of On Megrim rests as much on the fact that it was written in English as on its clinical observations and insights (Isler, 1993; Isler and Koehler, 2006). Isler is correct to identify this tradition, but like others before him he misses the point that it was Gowers himself who effectively killed it off. Isler is similarly dismissive of Latham, suggesting that the contrasting views of Du Bois-Reymond and Möllendorf were first synthesized by Eulenburg in his 1871 Lehrbuch der funktionellen Nervenkrankheiten auf physiologischer Basis, and that Latham’s theory was merely a ‘similar synthesis’.

Where, then, are we to place Liveing and Latham in the history of migraine? Latham should be credited with being one of the first people to attempt to articulate a vascular theory of migraine that explained the phenomena both of aura and of headache. This theory did not stand up to the steely gaze of great clinical neurologists of Latham’s era; the longevity of Wolff’s vascular theory, superficially comparable to Latham’s and potentially susceptible to exactly the same critique, may be attributed to the fact that one limb of the theory (the role of extracranial vasodilation in headache genesis) was supported by the acknowledged therapeutic successes of ergotamine.

To see Liveing’s theory as in any sense a precursor to modern neural concepts of migraine is simply not tenable. As we have seen in this article, Gowers effectively demolished the theory of nerve storms in its contemporary sense; and to reach back to claim Liveing as an ancestor to modern neural concepts, as Pearce did in the 1980s, requires us to strip his theory of all the aspects that do not accord with modern understanding of the paroxysmal disorders that Liveing believed were analogous to migraine; to do so is to impoverish the theory by divesting it of all its contemporary richness and depth. Sacks’ view—that the value of On Megrim lies primarily in its beauty, range and depth—is to my mind closer to the mark. On Megrim is the culmination and
summation of 300 years of medical speculation, observation and theorizing on migraine, and remains the best monograph ever written on the subject.

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