Neuralgia: The history of a meaning

Chris N Alam, H Merskey DM FRCP FRCPC FRCPsych

A review is presented of the meaning of neuralgia – from its introduction in 1801, to indicate an affection of one or more nerves causing pain, to its present day use. There was early dispute whether neuralgia required a lesion to be present in the nerve, and whether peripheral branches, nerve roots or the neuraxis was involved. Affections of organs were seen as a cause and the concept was extended to include links with psychological illness. In the last decades of the 19th century and throughout the 20th century, opinion on neuralgia gradually moved closer to the original meaning.

Key Words: History, Meaning, Neuralgia

La névralgie : Histoire d’une définition

RÉSUMÉ : Une revue de la définition de la névralgie – de son introduction en 1801 pour décrire une affection d’un ou plusieurs nerfs causant des douleurs jusqu’à son utilisation actuelle – est présentée. Dès le début, une discussion s’est engagée pour savoir si la définition de la névralgie exigeait la présence d’une lésion dans un nerf, et si les branches périphériques, les racines nerveuses et l’axe cérébro-spinal étaient concernés. Les affections des organes étaient vues comme une cause et le concept a été élargi pour inclure les liens avec une affection d’ordre psychologique. Dans les dernières décennies du 19ème siècle et pendant le 20ème siècle, l’appréciation de la névralgie s’est graduellement rapprochée du sens originel.

University of Western Ontario, London, Ontario
Correspondence and reprints: Dr H Merskey, London Psychiatric Hospital, Room C204, 850 Highbury Avenue, PO Box 2532, London, Ontario N6A 4H1. Telephone 519-455-5110, fax 519-455-5090
Received for publication September 7, 1995. Accepted November 16, 1995

PAIN RES MANAGE VOL 1 NO 3 AUTUMN 1996 165
The broad formulation of the symptoms of neuralgia is similar to that encountered today— for example in the description of trigeminal neuralgia by Elliot (6). The pain usually involves the second or third division of the nerve, or both, and it may spread to all three, but it is seldom confined to the first division. Pain occurs in recurrent bouts which last days, weeks, or months, with intervals of complete freedom. During each bout the pain is intermittent, but as time goes on it tends to become persistent and more severe, with fewer and shorter intermissions.

The pain is variously described as knife-like, red-hot needles under the skin, or painful electric shocks. The face may suddenly screw up with pain— hence the term tic.

As we shall show, the pristine versions of neuralgia were subject to changes that occurred for a variety of reasons. Some authors wished to identify a particular place as the cause of neuralgia. This could be anywhere from the spinal cord (7) through the nerve roots, trunks, branches, or terminal portions of nerves and even the visceral organs (Rowland, page 147; Graham [8], page 257). At a later stage, neuralgia was made to subsume almost any undefined pain anywhere in the body. General influences, such as chronic inflammation, syphilis, rheumatism, malaria and anemia, were also incriminated in the production of the disorder (Rowland, pages 18-29, 37-42).

Not surprisingly, age, sex, previous constitution, predisposition and psychological factors were all blamed for the production of neuralgia as time went by. In this article we will describe the initial view of neuralgia, its subsequent attribution to certain organs and to general factors, the extension of the term to include neuralgic headaches, the further extension to include indeterminate pains, the presentation of psychological notions, and, finally, the return to a more conservative and specific definition of neuralgia.

**THE INITIAL VIEWPOINT**

Warren (9) favoured a local cause and treated patients with severe facial pain by removing portions of the infra-orbital and supra-orbital nerves, or the inferior alveolar nerve within the mandible, or, on at least one occasion, a portion of the facial nerve. Graham (pages 256-7) also favoured locating the cause of neuralgia in nerve trunks and branches close to the site of the pain. Graham thought that local irritation of a nerve might cause neuralgia, but also observed (pages 256-7) that efforts to treat neuralgia as a localized affliction had not proved altogether successful and that “a great disorder of the general health” was more likely to trigger tic doloreux than a localized cause of neuralgia. The idea of a focal cause became more established after Brodie (10) described lesions in different parts of nerves that produced pain at a distance. Another type of local cause was favoured by Teale (7) and Anstie (11 [page 141]), although the latter also accepted that lesions of the trunks of nerves and affections of the viscera could cause the problem (pages 20,69). Teale’s idea about the spinal cord was stated as follows:

The symptoms of this affection [irritation of the spinal cord] consist in an infinite variety of morbid function of the nerves of sensation and volition which have their origin in the spinal marrow, and the parts in which these morbid functions are exhibited, of course, bear reference to the distribution of the spinal nerves.

The morbid states of sensation include every variety, from the slightest deviation from the healthy sensibility of any part to the most neuralgic affections....

Teale perceived two ‘seats’ in neuralgia: the first was the immediate location of the neuralgic pain; the second was the remote cause of the pain, usually in the central nervous system. If treatment was to be successful, then it would have to be applied to this second seat. To some extent one may perceive an overlap between the theories held by Graham as well as Hall (12), and those held by Teale, Brodie and Rowland. While all recognized a contribution from a general disorder of a patient’s health and the possibility of a localized cause, the latter group related neuralgia more to the spinal cord and nerve roots.

Hall (page 121) held that ‘inflammation’ of the affected nerves was the cause of neuralgia, but argued that those suffering from neuralgia also suffered from a general disorder. He noted that the neuralgic patient cannot bear the slightest touch near the affected nerve. This symptom is rather like the trigger points that Valleix (page 2) identified. It is worth noting that the trigger points of Valleix lay along the course of nerves at locations, such as the supra-orbital and infra-orbital foramina, rather than in muscles to which current theory allocates trigger points.

Numerous 19th century authors also believed that neuralgia originated in the nerve roots or in an affection of the spinal cord, which manifested itself as pain at a distant point on a nerve trunk. Such authors include the aforementioned Teale, Brodie, Rowland and Anstie, along with Peters (13), Rigal (14), Chapman (15), Tuke (16) and Behan (17). Granville (18 [pages 99-100]) emphasized the importance of the local disorder rather than general health, which is not surprising considering his interest in counter-irritation.

Granville’s focal interest stands in marked contrast to the view of Rowland (page 1) that the causes for neuralgia could be overstretching of nerves, pressure on nerves, dental cavities, affections of the urinary organs, uterus, heart, liver and large vessels, spinal irritation, organic diseases of the brain, malignant diseases, chronic inflammation, syphilis, rheumatism, malaria and anemia.

The sufferings which frequently attend this dreadful malady cannot be exceeded, debarring the unfortunate patient from the pursuit of either business or of pleasure. When he seeks relief by mixing in society, he is constantly exposed to the action of causes which excite the return of his torture, and he is therefore doomed to a life of seclusion, generally when at an age of which hope and enterprise are the natural characteristics (Rowland, page 1).

Valleix was the most notable student of local effects. His discussion of trigger points in this context was not unique because Hall had observed something similar, but it was more extensive than those of others, and Valleix also offered reasons for their existence. He is worth quoting:

According to the results of numerous observations I have made, neuralgia must be defined: a more or less violent
pain, having its seat along the distribution of a nerve, scattered with limited points: veritable painful foci from which depart, in varying intervals, lancinating or other analogous pains, and at which pressure, conventionally exercised, is more or less painful (page 2).

Laycock (19 [page 327]) believed that neuralgia could arise from lesions on a nerve such as “a small cicatrix, or a tubercle”. He followed Brodie in describing the existence of points that were unbearable to touch and superficially resembled the tender points of Valleix’s trigger points. However, Brodie’s points are described as always being located on the median line and thus differ from the trigger points of Valleix, which occur at such places as the supra-orbital and infra-orbital nerves.

**NEURALGIA FROM ORGANS**

Both Graham (page 257) and Teale (pages 46-7) described neuralgias affecting the organs. Graham observed “although the influence of severe derangement in the digestive organs, in producing excessive nervous irritation and pain is so evident, yet I have been frequently greatly surprised at the slight consideration given to this fact”. Teale delineated neuralgias of the heart, lungs and stomach. Eight years later, Rowland (page 147) claimed that neuralgia could attack “every organ with sensibility”. Rowland accepted both local and central causes. He regarded the overstretching of nerves and the placement of pressure on nerves and cavities in the teeth as localized causes of neuralgia. He also held that a neuralgia of the foot had resulted from an affection of the urinary organs, that facial neuralgias could be caused by uterine disorders and that pains in the shoulder resulted from a disease of the liver, as well as neuralgias caused by the heart and “large vessels” (pages 18-23). Both Teale and Rowland placed a limit on visceral neuralgias because the seat of the affliction remained the nerves, and Rowland (page 131) noted that the organ’s functions were not impaired. Rowland also cited neuralgias of the heart and lung, angina pectoris, gastralgia, hepatalgia, nephralgia and hysteralgia (pages 125,132,137,140,147).

Besides supporting the notion of localized neuralgias related to the nerve, Laycock also suggested that there were localized visceral neuralgias, including tenderness of the breast, intermittent uterine pains from inflammation or hysterical colic (pages 215,244,246). Laycock surpassed the limits that Graham, Teale and Rowland had observed. Laycock held that neuralgic organs would become inflamed and he did not insist that such neuralgias would have a seat limited to their nerves (page 224). Overall, he described neuralgia of the breast, heart, spinal column, hip, uterus, intestines, joints, ribs, rectum, head, face and teeth, as well as ‘neuralgic convulsions’ (pages 215,217,224,225,246,327,328,333-5) described as a modification of epilepsy (page 327).

By 1851 this expansion of the concept of neuralgia was well under way. Sandras (20) listed the heart, stomach, gastrointestinal tract, kidney, bladder, eye, ear and uterus as sites for neuralgia, as well as delineating temporal, infra-orbital, supra-orbital, upper dental, inferior maxillary, anterior cervical, occipital, brachial, intercostal, ilioscrotal, anal-genito-urinary, crural, plantar, rachidian types of neuralgia, and ‘angine de poitrine’ (260ff,281ff). Angine de poitrine or ‘angine névralgique’ could be caused by ossification of arteries, damage to the circulatory system, or other nervous illness (pages 290-2). Other visceral neuralgias could arise from various types of abuse to the affected organ (page 325). Angine névralgique could result in asphyxiation due to a paralysis of the diaphragm (pages 290-2). Neuralgia had become something of a catch-all diagnosis.

The ideas on visceral neuralgia by Pagès (21) broke away definitively from the generally accepted theory of neuralgia; he noted that while some visceral neuralgias could be traced to nerve lesions, these were completely absent in others, and he noted functional troubles associated with neuralgia of organs. Romberg (22) is particularly notable because he wrote the first general textbook of neurology. He held that there were respiratory, gastric, gastodynamic, hypogastric and spermatic neuralgias, and explained that they could be caused by bronchitis, gout, hypochondriasis, pregnancy, menstruation and venereal excess (pages 103,104,142,144).

Laboulbène (23) did away completely with the idea of lesions of nerves as a cause of neuralgia. “Pain, the essential element of neuralgias, can not suffice to characterize them, except in its condition of being unaccompanied by any appreciable material lesion” he affirmed (page 1). Laboulbène sorted neuralgias into three categories – cranial, thoracic and abdominal – and 17 types of neuralgia, including headaches and various visceral neuralgias (pages 15,16). Although this was more than most authors listed, Laboulbène was not far out of line because Dowse (24 [page 132ff]) listed nine visceral neuralgias; Axenfeld (25 [pages 244-306], Anstie (pages 73ff,82,107ff) and Chapman (pages 63-104) each listed seven; and Rosenthal (26) listed four.

This expansion of the concept of neuralgia was short-lived. It is difficult to explain why this was so, other than to suggest that the ideas of Valleix, who did not list visceral neuralgias, were more widely accepted than others and were thus more enduring and more often propagated. Also, squabbling within the group promoting the idea of visceral neuralgias may have damaged the credibility of their theories. Chapman (page 20) and Lawson (27) (who accepted the idea of visceral neuralgias) disagreed with Anstie’s theories and attacked them directly and vigorously. As well, attacks came from Hammond (28), who did not accept visceral neuralgias and described neuralgia as an abused term.

**NEURALGIC HEADACHES**

Links between trigeminal neuralgia in particular and migraine, may have been seen by a number of clinicians, and the tendency to call certain headaches neuralgic has been very persistent. The description of headaches as neuralgic seems to have been at its peak in the second half of the 19th century, declining by the early 20th century, but the term ‘periodic migrainous neuralgia’ was still in use as late as 1979 (29).

Valleix was the first author in our series to describe both a peripheral cause for neuralgia and neuralgic headaches. However, his discussion of headaches focused on whether migraines could be neuralgias – a question he did not answer definitively, although he leaned towards the opinion that migraine could indeed be a form of neuralgia (page 150).

The first author whom we identified as making a definitive link between the two was Downing (30), who described migraines as a ‘neuralgic complaint’ associated with tic douloureux (pages 10-1). Peters and Romberg, both writing in 1853, also described neuralgic
IGNORANCE AND ‘INDETERMINATE’ PAINS

Pains termed indeterminate, ie, pains to which authors of the time could not attribute a definite cause, were classified as neuralgia. Angina pectoris, discussed previously, is known today to indicate another condition. Less well understood in the 19th century, angina pectoris, discussed previously, is known today to indicate another condition. Less well understood in the 19th century, angina pectoris was classified as neuralgia because of the quality of the pain; in fact, Teale dismissed a cardiac-related autopsy finding, preferring neuralgia as an explanation (pages 99-103).

Neuralgia was a term of convenience, allowing a diagnosis to be made when no other explanation for a painful condition could be found. Similarly, other authors diagnosed neuralgia for conditions that did not warrant such a diagnosis anatomically. Sandras, for instance, suggested that muscular abuses could cause neuralgias in the relevant nerves and in nerves unrelated to the abused area (page 325). The fact that nerves in other than abused areas became involved tends to suggest that Sandras used neuralgia as a default diagnosis because he could find no other causal link between two painful symptoms. Neuralgia was becoming a condition recognized by one symptom – pain – and it was offered as a diagnosis based on the presence of that symptom alone. Behan, writing in 1916, recognized that this use of the term neuralgia had tended to become as much the rule as the exception, and cautioned against such use.

headaches. Romberg called them neuralgia cerebralis and noted that they were often misdiagnosed as facial neuralgia (pages 176-7).

These descriptions, together with nearly all the other reports of neuralgic headache, were published between 1841 and 1886, the publishing dates of Valleix and Hammond, respectively. Thus the works of Day (31), who attributed neuralgic headaches to causes such as decayed teeth, excitement, excessive noise, ‘oversuckling’ in women or a “timid and delicate constitution” (pages 226, 227, 274), were not consistent with the popular understanding of neuralgia as delineated by Valleix’s definition, or the suggestion by authors such as Rigal (pages 1, 2) that all neuralgias were caused by lesions of a nerve. The same tendency to distinguish between neuralgia and migraine applies to Dowse, who at least once attributed death to the pain caused by ‘neuralgia of the brain’ (page 85), and to the other descriptions of neuralgic headaches. To explain this, we may suppose that some authors were describing headaches as a neuralgia caused by headache pain in the distribution of the trigeminal nerve, leading to comments such as “the most common of all the varieties of trigeminal neuralgia is migraine” (Anstie, pages 1030-1), but headache pain located in the posterior cranial areas, as migraine often is, could not be linked to the trigeminal nerve. Perhaps then, the connection between neuralgia and migraines resulted from the quality of the pain, which would be more or less violent, as Valleix described neuralgic pain (page 2).

Another interesting feature in the discussion of neuralgic headaches is whether neuralgia was hereditary. Dowse, in discussing megrim and hemicrania, noted that one might often find that those who suffered from neuralgic headaches would often reveal that others in the family suffered similar afflictions; for example, “‘my brother suffers severely from neuralgia, and my sister from epileptic fits’ — so that we have a distinct neurolsisal history in each case where the treatment is nearly, if not absolutely, identical” (pages 106-7). Once Dowse had raised the possibility of a connection in the central nervous system, it is not surprising that others made a connection between neuralgic headaches and ‘nervous’ disorders.

While Peters and Anstie connected neuralgia and nervous disorders, Peters simply categorized neuralgic headaches as one of several types of nervous headache (pages xii-xiii) and Anstie suggesting that within two generations of a patient with neuralgia, one would find other family members suffering from neuralgia, insanity, epilepsy or alcoholism (page 142). Peters (page 1053) and Day (page 227) also suggested emotional causes for neuralgic headaches.

We thus have further evidence that some pains that were called neuralgias were well outside the strict definition of neuralgia, especially in the case of neuralgic headaches. If one also takes into account neuralgias of organs, it appears that the term neuralgia was liable to be applied to any pain in the body that lacked a specific, alternative cause. This pattern was certainly not sporadic in its occurrence. Table 1 shows that 19 of the 28 19th century authors we surveyed cited or suggested the possibility of neuralgic headaches or visceral neuralgias; eight referred to both.

<table>
<thead>
<tr>
<th>Author (year)</th>
<th>Cites neuralgic headache</th>
<th>Cites neuralgic organs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Warren (1828)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Graham (1828)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Hall (1830)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Teale (1830)</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Brodie (1837)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Rowland (1838)</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Laycock (1840)</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Valleix (1841)</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Downing (1849)</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Sandras (1851)</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>LeClerc (1852)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Pagis (1852)</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Peters (1853)</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Romberg (1853)</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Garavel (1858)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Laboulbene (1860)</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Axenfeld (1864)</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Anstie (1872/1880)</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Lawson (1872)</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Rigal (1872)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Chapman (1873)</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Cartaz (1875)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Day (1877)</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Rosenthal (1879)</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Dowse (1880)</td>
<td>Yes</td>
<td>Yes</td>
</tr>
<tr>
<td>Allbutt (1884)</td>
<td>No</td>
<td>Yes</td>
</tr>
<tr>
<td>Hammond (1886)</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Tuke (1892)</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>Total citing</td>
<td>12 of 28</td>
<td>15 of 28</td>
</tr>
</tbody>
</table>

This chart does not include passing references to neuralgia or short journal articles on the subject that did not attempt a full investigation of the subject. As a result, some 19th century references that appear in our bibliography do not appear here.
Neuralgia can only be diagnosed by exclusion, and is only justifiable when all other causes having an anatomical basis for the pain production have been excluded, such as pressure from growths, inflammatory exudates, misplaced fragments of bone, etc. The term neuralgia is often a cloak for ignorance. It indicates that the diagnostician has not been able to localize the cause of the painful condition. It is the same as calling a pain in the head a headache, or a lesion of the heart disease. (pages 146,147)

THE ROLE OF THE MIND

Some 19th century physicians thought that mental and emotional conditions could predispose an individual to neuralgia. Downing, for instance, noted that “mental impressions” could be among the catalysts of neuralgia, although he did not delve into the topic (page 4). Sandras, in treating neuralgia as a “nervous disorder” observed that all doctors had seen it occur under the influence of strong emotions (page 324).

Peters explained that those with a “feeble, disturbed, or irritable state of the nervous system” could be susceptible to emotions. These emotions, which included unusual excitement, joy, and pleasure, could induce neuralgic or nervous headaches in such individuals (pages xii-xiii).

Peters had similar views, noting that a condition of the nervous system was the subject of much generalization within the literature, perhaps even more so than in other areas such as indeterminate pains if only because these indeterminate pains did refer to particular problems. Nonetheless, the possibility of mental causes was not ignored, perhaps as a result of concomitant work in related fields.

SEQUENCES OF IDEAS

While the theories of organ neuralgias declined first, to be followed by neuralgic headaches, the idea of general factors and the interest in psychological causes led to a rather more persistent tendency to attribute neuralgia to such etiologies. Later, however, as neuralgia began to be treated as a more specific diagnosis with some demand for evidence of a change in the relevant nerve, whenever possible, the less well characterized pains that had been called ‘neuralgic’ probably were grouped under headings like ‘psychogenic pain’ or ‘psychogenic rheumatism’.

The influence of Valleix, combined with the fact that his view of neuralgia was consistent with the present concept, has made him one of the key figures in the development of this concept. This is not to say that his views were immediately and universally accepted. As has been noted, the concept of neuralgia expanded in the 19th century and apparently was not limited by an acceptance of Valleix’s definition. Similarly, not everyone immediately adopted his theory that neuralgia had a localized cause.

For instance, as described above, French workers Sandras (page 253), Laboulbène (pages 18,104,142) and Axenfeld (page 14), and German worker Romberg (page 244ff) all listed visceral neuralgia. Also, Sandras considered that less localized causes of neuralgia were possible (pages 324,325). Laboulbène pointed to conditions such as temperature and humidity, or emotional disturbances, sex, age and heredity as possible causes for neuralgia other than lesions of a nerve (pages 14,19-22). Like Laboulbène, Axenfeld accepted the possibility of nervous lesions as a cause, but provided a similar list to Laboulbène’s of predisposing causes (page 161).

Although LeClerc understood that nerve lesions could cause neuralgia, as he explained in his 1852 work, he saw the affection as a condition of the entire nervous system. Perhaps as a result of this theory, he believed that dizziness and vertigo, as examples of cerebral disorders, were linked to neuralgia (page 9).

Peters had similar views, noting that a condition of the nervous system, which he described rather vaguely as “feeble, disturbed, or irritable” caused neuralgic headaches. Alternatively, he did consider...
the possibility of a localized cause, suggesting that nerve damage could provoke neuralgia (pages xii-xiii).

Inman’s 1860 ‘Certain painful muscular affections’ (35) presented a previously unconsidered idea. He suggested that the musculoskeletal system played a role in certain pains through such situations as overstretching or muscle cramps. Such pain was then diagnosed as neuralgic. Inman was among the first to postulate that muscle pain could be referred to the region of the tendinous insertions (page 30ff). However, Inman’s views were not recognized for quite some time, remaining outside the standard discussions of neuralgia; not until 59 years later would Llewellyn and Jones’ discussion of neuralgic pains caused by compression reflect his ideas (36).

In 1872, Mitchell (37) provided a solid study of neuralgia, in which he pointed to local causes including lesions of or damage to a nerve. While he did not include visceral neuralgias, he did consider phantom pain in amputated limbs as neuralgic. But, like others who had recently accepted the idea of localized causes, Mitchell suggested that there could be other predisposing causes, such as rheumatism or influenza (pages 266,360). The difficulty in distinguishing between more diffuse muscular pains and specific nerve lesion pains is apparent even in Mitchell’s approach.

Chapman developed a seven-part theory of neuralgia. In the first part he defined pain as a functional change in the ‘sensory centre into which the affected nerve is rooted’ (page 23) and then went on to suggest that this functional change was the same for sensation as it was for pain, that pain had various degrees of intensity that were proportional to the rapidity of the functional change, that the amount of arterial blood supplied to a sensory centre was proportional to the intensity of the pain, that pain was not necessarily a morbid function and thus could continue after the exciting cause was removed, that neuralgia could be hereditary, and that these six ideas applied equally to general complications of neuralgia (pages 22-4). Chapman also provided a long list of predisposing causes ranging from bowel disorders to masturbation and psychical influences (pages 126,188-211).

Rigal, also writing in 1872, noted that there were two separate approaches to neuralgia in his opinion, favoured by two groups of authors. The first, represented by Valleix, Grisolle and Van Lair, did not deal with lesions of the nerves but instead classified neuralgia as a functional disorder. The second group, which included Chaussier, Axenfeld, Niemeyer and Jaccoud, attached the term neuralgia to any remittent or intermittent pain considered to have a ‘seat’ in the nerve trunks. Rigal, perhaps borrowing from his perception of both groups, preferred to suggest that neuralgia was a condition resulting from the presence of a nerve lesion, and that such a lesion provoked a functional disorder causing neuralgia (pages 1-2,64). As was apparently the custom among those theorizing about the causes of neuralgia, he provided possible causes such as sex, age, constitution, cold, poisons and excesses (page 12).

By the turn of the century, one might have expected that these vague or nonspecific causes would have lost favour in the same way that visceral neuralgias, neuralgic headaches, specific centralized causes and the general expansion of the concept had relatively short existences. However, while the ability of doctors to make specific and educated connections between neuralgia and a particular cause may have illuminated such things as ‘indeterminate’ pain, it did not immediately seem to end suggestions that ‘generalized conditions’ could be major contributing factors in the production of neuralgia.

Gowers (38) firmly separated pain due to a nerve lesion from similar pain without evidence of an organic cause. He related the disorder to changes in the area of the brain cells representing surface regions to which they were connected (page 802). He thought that pain in the region of a viscous, after excluding cases of pain presented by disease of the organ, could be due to dysfunction of both peripheral nerve endings and the central connections.

In 1906 Williams (39) maintained that fibrositis, considered to be inflammation of the nerve sheath’s fibrous tissue, was the most common cause of sciatic and cervicobrachial neuralgia. He also suggested that anemia and eye strain could both provoke these conditions. To put this theory in context, it is worth noting that Williams also explained that eye strain could contribute to the production of “neurasthenia, melancholia, intemperance, drug habits...irritability of temper, dyspepsia, constipation, and sluggish liver” (pages 295-303). This is probably the best example of how difficult it was for the medical profession to maintain a logical position properly supported by evidence.

Jelliffe (40) asserted that neuralgia arose from a lesion in nerve trunks, sensory ganglia or viscus. But conditions that might affect the individual on a larger scale than a nerve lesion, such as anemia, toxins, hereditary predisposition or affections of visceras, also remained as causes (pages 769-70). Behan, writing in 1916, attributed neuralgia to damage of the nerve trunks, the influence of cold or meningeal diseases of the spinal cord. Nonetheless, he considered that excessive indulgence in alcohol, tobacco, drugs and sexual activity, as well as neurasthenia, old age and heredity were all contributing factors (pages 140,141,143).

As noted, Llewellyn and Jones (page 11) combined some of their own theories of how compression caused neuralgia with the more well known and accepted theories. They did not suggest any generalized contributing factors.

It becomes evident in the early 20th century that doctors were examining physical influences on nerves in an attempt to pinpoint the cause of neuralgia in damage to or interference with the relevant nerve. The tendency to include broader potential causes – virtually a hold-over from century-old discussions of ‘general disorders’ and ‘nervous disorders’ – did not disappear, as if the focus on minute damage to specific nerves left doctors worried that they would not see the forest for the trees. However, this tendency did decline as the 20th century progressed, although the psychological option remained popular (41).

CONCLUSIONS

Neuralgia symptoms accepted today are strikingly similar to those encountered in the early understanding of neuralgia, but between these times changes occurred. For instance, the term neuralgia has been applied to various types of headache pain and to certain organic conditions. Several theories about the causes of neuralgia were suggested in concert with this varying use of the term. These theories often focused on the site of the condition’s origin. While this was usually seen as being located in either the peripheral nerves or the central nervous system, some authors believed that it was located in organs or elsewhere.

There appears to be a certain progression in the development of
these theories. To begin with, early American and English authors, such as Warren (pages 1-6) and Graham (pages 256-7), suggested that neuralgia’s cause was to be found in nerve trunks and branches. Following further entrenchment of this idea through the works of Laycock and Valleix, the ‘local cause’ theory appeared to become the most widely accepted throughout the 1800s and into the next century.

Nonetheless, other theories, such as the suggestion that the cause of neuralgia, while located in the nerves, was central rather than peripheral, did arise. In 1830 Teale located its cause in nerve roots and the spinal cord while Anstie leaned towards the nerve roots as the probable site.

Other arguments surrounding the concept related to whether afflictions of the viscera, as well as migraines, cluster headaches and nervous headaches, could be considered as neuralgia proper. A large number of authors in the latter half of the 1800s believed such a consideration justified, and this seems to have promoted the use of the diagnosis of neuralgia to such an extent that it began to be used as a label for what might be termed indeterminate pains to which no cause could be ascribed. As a result, the concept labelled by the term neuralgia was greatly expanded. The expansion was not sustained and did not persist much into the 20th century. Later authors accepted a more limited use of the term previously delineated by those such as Valleix, which was a reasonable approximation of the modern concept.

ACKNOWLEDGEMENTS: We thank Alpha Academic for permission to draw substantially upon the longer article in the *History of Psychiatry* (1994;5:429-74) on which this present report is based.

REFERENCES


With two divergent suggestions for the causes of neuralgia, the overriding factor behind the diagnosis was the quality of the pain, rather than its origin. Perhaps, as well, neuralgia provided an easy and attractive label when other more accurate diagnoses were not available due to a lack of knowledge.

While this permitted headache pain, visceral pains and ‘indiscriminate pains’ to be categorized as neuralgias, the process was short-lived because neuralgias of the head or organs could not be justified as true neuralgias. More interesting and innovative theories, such as Tuke’s exploration of psychical pain (pages 835-7) or Inman’s review of muscle pain (page 30f) appeared, but they did not receive as much attention as the more readily accepted views of localized causes, and thus remained detached from discussion.

It is also likely that, as understanding of the nervous system increased, the indiscriminate diagnosis of indiscriminate pains as neuralgia became unacceptable. Perhaps some of this use was diverted to a psychological explanation of pain, a common practice (38,41).

The early 20th century provided the same combination of theories suggesting localized causes and generalized conditions, but the expansion that occurred in the 19th century did not recur, probably because of an increased knowledge of anatomical processes. Thus, random pains or visceral pains cannot now be termed neuralgias simply as a default diagnosis, although we still have problems knowing how to classify conditions that do not fit easily, case by case, with obvious anatomical and physiological evidence.


