Neuralgia and Neuritis.

A Thesis

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Neuralgia & Neuritis

The literature on neuritis is a scant one: some of even the recent text-books on Medicine and Surgery devote not a paragraph to it. Thus Roberts, in his work on the Theory and Practice of Medicine, speaks of it only incidentally and in connection with neuralgia, when he suggests the probability that "in some cases the nerve is more or less congested or inflamed."!

That we know so little of neuritis is, no doubt, due in some measure, to the fact that death from neuritis per se is rare, and that any post mortem evidence of this disease is found, rather than sought for directly, in subjects who have died of some other disease or diseases. Thus the presence of morbid changes in nerves connected with a wound which was followed by tetanus, suggested the source of tetanus itself to be neuritis.

And yet there is no à priori reason why there should not be inflammation of a nerve as well as of other tissues. Not to speak of its anatomical fitness to be the seat of an inflammatory process: the

Fourth Edit. Vol II. p. 342

Neuralgia & Neuritis

very sensitiveness of sensory and mixed nerves and their liability to excess of functional activity: and, in the case of cutaneous and many other nerves, (e.g. the ulnar at the elbow,) their superficial position would be supposed to render them more rather than less liable to influences provocative of inflammatory processes.

Nerves are composed of white and grey fibres, the former being the more numerous. Each white fibre is composed of a central axis (the axis-cylinder of Purkinjé): this axis is surrounded by the medullary sheath (white substance of Schwann): outside this again is the primitive sheath of Schwann. Each axis-cylinder is seen, under the microscope, to present the appearance of longitudinal striation, suggesting that it is itself made up of very fine fibrils. The primitive sheath appears to be homogeneous in structure with nuclei over its inner surface. The grey fibres are more vascular than the white, and are devoid of the white substance of Schwann.

Neuralgia & Neuritis

They are found principally in the sympathetic system.

A cerebro-spinal nerve consists of one or more cords of nerve fibres, called funiculi, surrounded by a common sheath of connective tissue called the neurilemma or perineurium. Each funiculus is surrounded by a reticular sheath proper to itself. From the common sheath processes extend inwards between the funiculi and serve to conduct the bloodvessels that supply the nerve. The bloodvessels of a nerve consist of very fine capillaries which run parallel to the fibres, some being within the funicular sheath, and are connected at intervals by transverse branches. Lymphatics are found in the perineurium: and in it also nervi nervorum are said to be.[1]

Anatomically at least, then, nerves are well fitted to be the seat of inflammatory processes. And this is corroborated if we glance at the processes, analagous to inflammatory, that take place after the division of a nerve.

[1] Quain's Anatomy, Eighth Edit. Vol II. p. 126 et seq.

Neuralgia & Neuritis

First of all there is degeneration of the medullary sheath and probably of the axis-cylinder: the neurilemma generates new cells, of round or spindle shape, which penetrate between the nerve fibres and also between the cut ends of the nerve. From these cells new nerve fibres develope. Billroth, however, inclines to believe that the new fibres develope directly from the divided axis-cylinders.'

It is probable that many symptoms usually referred to neuralgia, and especially when these are intractable to treatment, are due to neuritis. Physiologically, too, what structure in the animal organism is more constantly at work and therefore more liable to be affected than nerves? In all the forms of organic activity in the human being, nerves are involved; and when, from any cause, such activity becomes pathological, nervous action is modified at least: and, it may be, that in more cases than we at present are

' Billroth's Surgery, Vol I. p. 151. New Sydenham Soc. 1877.

able to know, the nerves themselves undergo pathological changes, temporary or permanent.

What relation do neuritis and neuralgia mutually bear? Neuritis is commonly considered to involve neuralgia, and, at some stage of the neuritic process, this no doubt is almost invariably true. But may it not also be probable that neuralgia frequently involves neuritis? Every pain is not neuralgic in the general acceptation of this term: a pain may be momentary and not recur, and we, then, do not speak of it as neuralgic. When does neuralgia cease to be only neuralgia and neuritis begin? Or, to go back a step further, when does neuralgia begin?

"By the term neuralgia," says Bristowe, "is meant pain, for the most part paroxysmal, occurring in the course of nerves and in their area of distribution." Further on he says, "In a large proportion of cases the neuralgia is essentially intermittent; the pains

Neuralgia & Neuritis

come on in paroxysms, lasting probably for a second or two to a minute, rarely longer, which recur every five or ten minutes, day and night, or manifest themselves at longer and more or less irregular intervals. Occasionally they remit for weeks or months together"[1]

The essence of neuralgia, then, would appear to be paroxysmal pain in the course of a nerve, intermittency and periodicity. Now one can conceive a paroxysmal pain as being purely functional, especially if such pain lasts for only "a second or two to a minute": but it is not so easy to conceive such a pain recurring at more or less regular intervals, during perhaps a month or more, and still being only functional. And the difficulty of conception is increased if there be no obvious exciting cause constantly present or constantly applied: for nerves are sensitive only to stimuli of whatever nature.

What is the stimulus, say in a case of tic, the result of cold? The

[1] Practice of Medicine, Fifth Edit. p. 1144

Neuralgia & Neuritis

onset of the pain may well be ascribed to cold: the acuteness of the sensation being legitimately enough referred to the nature of the excitant and its intensity. But the sufferer is removed from the exciting cause; he is transferred from a very cold to a warm atmosphere: any functional aberration, digestive or otherwise, is attended to and corrected; and still the pain recurs at intervals, and probably with increased intensity. Is the pain still functional, only that one stimulus is substituted for another? What is the new stimulus? Is it one external to the suffering organ as the first was; or is it an altered condition of the nerve itself that now serves to excite pain?

"Neuralgias are only symptomatic," says Trousseau[1]: there may, indeed, be no organic lesion; but every neuralgic pain is caused by an immediate stimulus, whether it be by the impoverished and vitiated blood of a chlorotic female, or the degenerate condition of the nerve supplied by

[1] Clinical Med. Vol I. p. 479 New Sydenh. Soc. 1867

such blood, or even by the action of the vitiated blood on the structure which itself has vitiated and so rendered morbidly sensitive.

Where there is neuralgic pain there is a stimulus. But when does the stimulus cease to act on a nerve organically intact, and begin to act on one organically deteriorated? When does the molecular change, concomitant with function, pass into disintegration?

The blacksmith's deltoid enlarges: the molecular changes increase: but it is a hypertrophy that takes place: there is an increase of the normal nutritive activity in the muscle; but so long as the excitant of the increased activity does not become an irritant, inflammation is absent. And, in like manner, so long as the fifth nerve is exposed to only such atmospheric and climatic conditions, as in the course of time it has learned to adapt itself to, the changes in it are still molecular - assuming of course that the general condition of the individual is also healthy.

Neuralgia & Neuritis.

The irritant, in the case of the blacksmith, is really a normal one, one to which his muscle has adapted itself: it is a normal irritant persistently applied. Insert a foreign body between the muscular fibres or subject it to the action of a rheumatic irritant, and myositis, more or less marked, will result. Remove the irritant and the myositis soon will be also removed: in any case its course, unlike that of neuralgia, is continuous.

In the case of the fifth nerve which has been irritated through the excess of cold and damp, remove the immediate irritant; does the pain also go?, Or does it recur at intervals of minutes or hours without the presence of an irritant?

It may be said that nervous phenomena tend to repeat themselves: an action often repeated tends to become a habit: and habits repeat themselves until displaced by some habit stronger than themselves. So, it may be said, the pain tends, like the nervous phenomenon habit, to repeat itself. This would imply that the

Neuralgia & Neuritis

pain, the disease, is essentially a central thing referred to the periphery. But habits, to be repeated, must have an occasion if not a cause. If one goes to a certain place of amusement, the place must be there for him to go to: if one eats and drinks at certain times, the food and drink must be there. So the analogy is not complete: the cold is not there when the pain recurs.

If we look at it from another point of view, and say that the occasion of the pain is a condition of the central sensory cell, is the explanation more satisfying?

In the case of habit, the central cells which condition habit are, no doubt, modified by the impress of the repeated habit. But the habit is not repeated if there is no occasion to call it forth. When one has been in the habit of eating at a certain hour, the desire to eat at that hour continues for some time after the habit ceases. The occasion is not there now, but

Neuralgia & Neuritis

the habit, although it is not repeated, tends to recur. So in the case of pain, could it recur at stated times which are periodic, in the absence of any occasion ?; That is, may neuralgia be the symptom of only a functional modification of the central cell, a symptom which, like habit, tends, up to a certain point, to be intensified, but then, and differing in this from habit, not having the assistance of concomitant volition, tends to become less and less intense, and finally disappears ?

Again, one might ask, may pain after it has left its impress on the central cell, also recur in the presence of another occasion than cold, say worry or digestive disturbance, or some other cause consciously or unconsciously present ?, It may be so: but even if this hypothesis serve to account for its recurrence, it ignores its periodicity. Why should the occasion be present at a certain time rather than at another ?,

Neuralgia & Neuritis

One might multiply hypotheses and ask again whether, if neuralgia be not functional, it be the result of an inflammatory lesion in the central cell. But this hypothesis also fails to account for its intermittent and periodic character.

If the cause of the neuralgic pain be in the nerve itself, then what is it? Nerve differs from muscle in this, that it is not only the structure irritated but also the one that receives and conducts the sensation of irritation. So long as a sensory nerve continues to convey sensation it is physiologically intact: when it ceases to do so, or, although still conveying it, does so either so as to make the sensation scarcely perceptible or to present it in such a character as to cause it to be mistaken for another sensation, (e.g. touch seems to be pain in the case of a hyperaesthetic nerve,) it ceases to be physiologically efficient.

Sensory nerves are fitted, or

sufficient occasion, to convey the sensation of pain. What change takes place in the nerve during conductivity of the impression? Analogy is often drawn between nervous and electrical conduction. No doubt there is an analogy; but nervous conduction is not electrical. "A galvanic current," says Cleland, "applied to a trunk of nerve supplying a muscle, does not maintain the muscle in contraction; but there is a contraction every time the circuit is completed, and every time that it is broken, so that the muscle can only be kept contracted by a constantly interrupted current. In electrotonus the nerve still performs its functions, but its degree of irritability is altered in different parts of its course. These facts show that nervous influence is not a current of electricity"[1] To this it may be added that nerve force travels much more slowly than electrical - the former at the

[1] Animal Physiology. Cleland, 1877, p. 180

Neuralgia * Neuritis

rate of two hundred feet per second the latter four hundred and sixty two million feet (Vivian Poore).

The intimate changes taking place in a nerve fibre, under the influence of a stimulus, are unknown. Whether the change be merely physical, such as takes place during the conduction of an electric current, or whether the physical character of the change is modified by the vital relations of the organ in which they occur, it is not necessary now to inquire. We may safely assume that the change is a molecular one; the constituent molecules of the nerve adapting themselves fitly to do their work - passing from a state of quiescence to one of activity. And as the molecular activity, in the case of the electric current, is in proportion to the strength of the current: so, no doubt, are the changes in the nerve proportionate to the intensity of the stimulus. Thus, it would appear, that the quality

Neuralgia & Neuritis

of the sensation depends on the degree of molecular activity as well as on the nature of the stimulus. So long, for instance, as the temperature of the atmosphere does not fall below a certain point, a sensation of cold is felt: but when, from either a still greater fall or the continuous application of the same temperature, the stimulus is increased, the sensation of cold passes into one of pain, the molecular elements of the nerve passing at the same time from a less to a greater state of activity.

But in the recurring pain of neuralgia we must eliminate the initial stimulus: what then accounts for the pain? Does the molecular activity recur in the absence of the stimulus that first excited it? And if so, are we any nearer the explanation of the neuralgic pain? Would it not be necessary to ask how and why the molecular activity of the nerve recurred? But, to assume this as the cause

Neuralgia & Neuritis

of the recurring pain, we are again met by the fact that the increased molecular activity implies increased heat, and an increase of heat implies an accelerated circulation. Is neuralgia, then, dependent on an increased molecular activity: and does neuralgia pass into neuritis when the heat generated by this increased activity acts as a local irritant? This is the theory of neuralgia and neuritis respectively, that recommends itself to me. How does it fit to explain the intermittent and periodic character of neuralgia?

Hughlings Jackson refers epileptic fits to discharges of nerve force. Epilepsy differs from neuralgia inasmuch as the symptoms are the phenomena of motor nerves, while those of neuralgia are the phenomena of sensory ones. Here are other differences between the two diseases: the epileptic seizures are not marked by the same periodicity: they may intermit for hours or days, or remit for weeks or months, and the

intervals between successive fits, in the same individual, may vary. In many instances, however, the interval is a fairly constant one: some cases there are in which there is a more or less regular interval of a month, and then, one or more fits occur, to be followed by another similar interval. "Epilepsy, catalepsy certain forms of chorea and many other convulsive affections, pretty frequently assume, not only an intermittent but also a periodic type."[1] Epilepsy bears this further resemblance that, in many cases, it is the result of irritation of sensory nerves. Brown-Sequard says "of all the nervous and other complaints that may be due to an irritation starting from the trunk, branches or ultimate ramifications of nerves, very few, if any, are more frequent than Epilepsy. .... But it is not so well known that an injury to, or a disease of a nerve, not rarely produces epi-

[1] Trousseau's Clin. Med. Vol. I. p. 488. New Sydenh. Soc. 1877

Neuralgia & Neuritis

leprosy"[1] He then goes on to prove his position by showing that, in many cases, the epileptic attacks followed immediately on irritation or injury of a peripheral nerve or on neuralgia: and that, on the removal of the irritation, the fits also ceased.

May not neuralgia, in its way, be due to discharges of nerve force somewhat analogous to those which condition epilepsy? What more likely than that the hyperactivity of molecules should evolve force which again expresses itself by pain, just as the epileptic discharges express themselves by convulsions? The force then, after exhausting itself in a paroxysm of pain, leaves the molecular action of the nerve at its minimum: and so there is an interval until the molecular forces recuperate themselves and again explode.

It is true, indeed, that the nerve discharge of epilepsy appears to be central with its symptoms general,

[1] A System of Surgery, by Holmes and Hulke, Vol II. p. 201

Neuralgia & Neuritis

and that of neuralgia peripheral with its symptoms local. But although the phenomena of epilepsy are general, they may and often do, follow, as we have seen, peripheral sensory stimulation (and it may be observed, that in this it is like tetanus): and it is not inconceivable that the dis-charge of nerve force, although apparently central, may be the continuance or transformation of centripetal sensory force, travelling from the periphery.

How is the localisation of neuralgia to be explained? In those naralgias in which the pain is due to an organic lesion either reflexly or as the result of direct irritation, the question of localisation is answered: but in those cases in which the pain is a symptom "of a general affection or cacheria"[1] the answer is less easy. It is to be doubted, indeed, whether any neuralgia

[1] Science and Practice of Medicine, by Dr. Aitken Second Edit. Vol II. p. 105

Neuralgia & Neuritis

is a symptom of only a cachectic condition. It may be true that in a majority of cases" the condition of a patient at the time of the first attack of neuralgia is one of debility, general or special"[1]. But it is equally true that "often the worst and most intractable cases are in good general health".[2] A cachectic condition, no doubt, renders one liable to neuralgia just as it does to any other disease, by lessening the power of resistance to noxious influences. It is not probable that tic, in a syphilitic subject, is determined by the specific cachexia. The relation of cachectic states to neuralgia is one of predisposition: the onset of the disease is determined by some local condition whether or not this condition be observed. According to Billroth, two thirds of his patients were unable to assign any cause for tic.[3] But not to speak of tic and sciatica,

1. Spender quoted by Aitken. Vol II. p. 101
2. Theory and Practice of Medicine, Bristowe, 5th Edit. p 1146
3. Clin. Surg. by Billroth, New Sydenh. Soc. 1881. p. 65

Neuralgia & Neuritis

the two most frequent forms of neuralgia, the various reflex neuralgias, such as mastodynia, suggest a local exciting cause.

I have spoken of neuralgia passing into neuritis. There are cases, however, where neuritis precedes, in the sense, at least, of causing neuralgia. Dr. Buzzard thinks that, in some instances, inflammation of the sheath of the nerve precedes the neuralgia, "is at least the starting point of the disorder".[1] Here the perineuritis is the local irritant inducing increased molecular action. It is probable enough that hyperaemia of the perineurium, at its onset, might not be followed by neuralgia, this following as the congestion became more marked. For instance one does not feel the pain of Sciatica just at the time of the chill. I lately attended an elderly patient who contracted neuritis of the sciatica while working as a road contractor. He felt himself being chilled, rheumatic pains were general: but the

[1] Quain's Dictionary of Medicine, Art. Neuralgia.

Neuralgia & Neuritis

sciatic pain did not develope until twelve hours had elapsed: indicating, it would seem, that the earlier hyperaemic stage of the sciatic neuritis need not cause neuralgic pain.

Pathology of Neuralgia & Neuritis.

What is the pathological evidence as regards respectively neuralgia and neuritis? It is to be remembered that examination for such evidence has been made on only peripheral nerves, except in the case of the optic. That in the various visceral neuralgiae, neuritis or perineuritis is often present, is very probable. But attention has been directed mostly to peripheral nerves in regard to the pathology of their morbid conditions.

In neuralgia pure and simple, one would not expect to find any pathological lesion: and examination of many cases, both by the naked eye and the microscope, confirms this view. Billroth relates the case of a man, aged sixty, who had neuralgia of the left trigeminus, the pains involving "the left cheek

and upper lip, the upper jaw and the teeth." The slightest movement brought on paroxysms of pain which recurred at shorter and shorter intervals. The usual remedies, including morphia in very large doses (two scruples daily), were tried without any lasting benefit. After local blood-letting had failed to give relief Billroth, at the urgent request of the patient, "extirpated the portion of nerve lying in the infra-orbital canal". Three quarters of an inch of the nerve were removed: and "neither with the naked eye nor with the microscope, could anything morbid be discovered in it." This operation having failed to give relief for more than a few days: and the symptoms recurring and now involving the palate, pterygo-palatine fossa and temporal region, Billroth having convinced himself that the disease was confined to the second division of the nerve, decided to operate with the purpose of dividing the second division of the fifth at the

foramen rotundum. The operation appeared to have the desired effect: and the patient was pleased to think that he was cured. In about nine months afterwards, however, the pains again recurred, and continued to increase in frequency and severity. Eighteen months after the last operation, Billroth again operated and cut the second division of the nerve close to the foramen rotundum: he also completely removed the infra-orbital nerve. "Microscopically no pathological changes could be detected" Again speaking of resection of nerves in neuralgia of the fifth he states "we neither could discover anything wrong with the naked eye nor with the aid of the microscope .... The disease appears always to be purely functional"[1]

The testimony of most writers on neuralgia coincides with Billroth's in affirming the absence of any pathological lesion. Aitken indeed does seem to imply some morbid appear-

[1] Clinical Surgery. New Sydenh. Soc. 1881, p. 59 et seq.

Neuralgia & Neuritis

ance in the affected nerve in the direction of either hyperaemia or atrophy.[1] Bristowe believes the lesion, if any, to be in the nerve, or spinal cord or brain[2] Some writers, again, give concomitant lesions as among the pathological phenomena of neuralgia, such, for instance, as extravasations of blood, and nutritive changes in the walls of the bloodvessels.[3] Nothnagel, referring to the alleged affection of trophic nerves in neuralgia, concludes that the nutritive changes observed are due to the implication of vascular nerves giving rise to arterial spasm ("Tropische Störungen bei Neuralgieen" Arch. f. Psychiatrie ii. 29)[4]

The pathology of neuritis, insofar as pathological investigation has thrown any light upon it, is very much that of the neurilemma rather than of the axis-cylinder. And it is not surprising

1. Vol II. p. 104
2. p. 1144
3. Biennial Retrospect of Medicine and Surgery 1869-70. New Sydenh. Soc. p. 117. quoting Mitchell
4. Ibid.

Neuralgia & Neuritis

that a pathological lesion, even if present, should fail to be detected in the axis-cylinder, seeing that the anatomical structure of this organ is not well defined.

In idiopathic neuritis one would expect that the essential elements of the nerve would be affected, and the same probability is credible in the case of cachectic neuritis. Billroth's latest theory of the union of a divided nerve, refers the healing process less to the investing sheath and essentially to the axis-cylinder. And, to reason from analogy one fails to see how the axis-cylinder could escape being involved in a case of well-marked neuritis. In the neuritis of a chlorotic female, as in that of alcoholism, one would expect to find the axis-cylinder primarily affected, just as in degenerative conditions of muscles one finds the cells of the muscular fibres to be affected rather than the connective tissue elements which invest the muscle. In the neuritis of central disease,

Neuralgia & Neuritis

too, one would expect the axis-cylinder the essential part of the nerve- to be involved, And, in point of fact, in parenchymatous neuritis, changes in the axis-cylinder, followed by the disappearance of this organ, have been discovered by Charcot, Perret and Jeffroy who describe the nerve-tubes themselves as presenting a moniliform appearance, and proliferation of the cells of their segments.'

In neuritis the inflammatory appearances may be confined to the perineurium - the nerve fibres then being normal. In perineuritis, pathological anatomy shows the following phenomena:- the perineurium is marked by increased redness and swelling, the latter being the result of serous effusion. If the inflammation proceeds deeply and involves the funicular sheaths as well as the perineural processes intervening between the funicular bundles, the nerve is broken

' Quoted by Brown-Séquard in Holmes and Hulkes' Surgery Vol. II. p. 178.
Charcots' Diseases of the Nervous System, New Sydenham Soc. 1877. p. 23

up by fibro-plastic effusion; and the axis-cylinder undergoes atrophic fatty degeneration as the result of compression (Brown-Sequard). In some cases nerves are found permanently enlarged, and marked by nodose enlargements. In some cases of even interstitial neuritis the nerve tubes escape.[1]

"In its Chronic form", says Brown-Séquard, "neuritis is characterised by the greyish, bluish, or almost violet colour of the affected nerve, which is swollen and offers nodosities. The neurilemma adheres to surrounding tissues."[2]

Clinical Symptoms discussed.

To come now to the clinical point of view. A typical neuralgia may definitely enough mark itself off from any other disease if we accept neuritis: although a doubtful neuralgia is sometimes mistaken for

[1] Jones and Sieveking's Pathological Anatomy. Edited by Payne, Second Edition p. 310.

[2] Holmes and Hulke's Surgery Vol II. p. 178.

Neuralgia & Neuritis

neuritis, as for instance when paresis of muscles exists from the depressant influence communicated to the motor centre by the irritation of the peripheral nerve (Buzzard). But every neuralgia is not a typical one: and the non-typical neuralgias are not easily nor always diagnosed. Intercostal neuralgia and mastodynia are sometimes mistaken for reflex pains, the result of indigestion and vice versa. Either of these affections, again, may be mistaken for rheumatism and again vice versa. How often are Sciatica and rheumatism confounded! And, indeed, failure of diagnosis in such instances is not necessarily due to a want of clinical thoroughness: one is often much at the mercy of his patient, whose history of the onset of his illness and description of his sensations are by no means transparent.

What are the clinical tests of neuralgia? Numbness, anaesthesia, or hyperaesthesia, followed by intense pain which occurs in paroxysms in the

Neuralgia

direction of some nerve; each paroxysm is composed of "momentary shocks following one another in rapid succession" (Bristowe), and may last from one to thirty seconds: the paroxysm may recur at shorter or longer intervals, varying from minutes to many hours: neuralgia is generally unilateral and almost always intermittent.

Suppose these symptoms existed in the sixth right intercostal space, for instance, it would be safe to diagnose neuralgia rather than pleurisy, or rheumatism or peripleuritis: and the diagnosis would be confirmed by the absence of symptoms indicative of these affections. The 'points douloureux' of Valleix (for which, by the way, Trousseau does not express much respect), are also corroborative evidance of neuralgia. That these painful points do exist in some neuralgias is beyond doubt: whether they are to be credited with the diagnostic value claimed for them by Valleix is another matter.

Neuralgia

Trousseau wholly denies the existance of Valleix's tender points in intercostal neuralgia, namely, a point over the angle of the rib, one at the middle, and one at the sternal end. But while depriving us of the aid of Valleix's points in intercostal neuralgia, Trousseau substitutes others of his own which he asserts to be invariably present in true neuralgia. These he names the 'spinous point' and the 'spot of peripheral expansion'. The spinous point is found "by making pressure in succession on the spinous processes of the vertebrae, beginning with the first two immediately beneath the occipital bone, down to the loins. When the tender spot is reached, the patient makes an abrupt movement, and tries to avoid being touched, and even cries out. Pressure on the vertebrae above and below this point gives no pain". The spot of peripheral expansion in e.g. neuralgia of the sixth intercostal space, is over "an antero-lateral portion of the chest corresponding

to the sixth intercostal space".[1] If the spinous point and the spot of peripheral expansion are present, Trousseau considers neuralgia to be proved; it might, however, be too much to say that their absence negatives neuralgia. But to say that this is neuralgia and that is not, is not enough for the purpose of the physician. He knows, indeed, the immediate affection he is to treat: but there is something further which he must also know, if his treatment of the affection is to be successful; and that is the constitutional character and general health of his patient. His ultimate aim must be to treat the patient, even if circumstances such as the severity of the affection, should make his immediate aim the treatment of the neuralgia. For even if it be too much to say with some writers that neuralgia attacks only those who are debilitated from some cause, the experience of most

[1] Clinical Medicine, Vol I. p. 486. New Sydenh. Soc.

Neuralgia

observers goes to prove that it finds its nidus most frequently in those who, so to speak, afford the necessary fabulum for its sustenance. Most of the neuralgias met with in my own limited experience have been in females: and I can recall only one case out of many of which I have notes, where there was no obvious constitutional defect. This patient, Mrs S- aged 40, came under my care for the second time on monday 8th March 1886. Three months previous to this date, she had been treated by me for the same affection in the same nerve. She is a stout, florid woman, married: no children. Except for slight 'colds' she has never troubled the doctor. The family history, as given, is devoid of any neurotic tendency. In her case, the pain involved the second division of the right trigeminus: and the point of irritation was referred to a diseased molar tooth in the right upper jaw. Not that she felt any pain in the tooth:

Neuralgia

but the popular connection of neuralgia with decayed teeth led her to reject, respectively, the theories of 'bad blood' and 'indigestion' proferred by her female friends, and to make the diseased molar the delinquent. Hot applications over the right cheek and Chloride of Ammonium in fifteen grain doses thrice daily were exhibited. The paroxysms, which occurred about every four hours and lasted a "minute or two", became less severe; and on Saturday 13th she was left with a sense of aching in the right face.

This was a typical enough case of neuralgia: the intermittency, paroxysmal attacks, the seat of pain, Valleix's point over the infraorbital foramen, and Trousseau's painful point over the second cervical vertebra all serving to suggest the diagnosis. The attack was attributed to a chill contracted on sunday the 7th.

This patient presented the unusual combination of severe neuralgia

Neuralgia

with otherwise good health. The fact, however, that she supplemented her husband's earnings by charing, legitimately enough suggests the hypothesis that, at the time of the neuralgia's onset, she was suffering from more or less nervous exhaustion, the result of overwork. And it was on this hypothesis that Chloride of Ammonium was given.

A physician who diagnoses neuralgia in his patient, and who does not wait to diagnose something more, will be very likely to mislead himself and to dis-appoint his patient. To leave out of account the neuralgias for which no external cause can be detected, it becomes the scientific physician to ask why, from half a dozen persons exposed to the same chill, neuralgia chooses only one for its seat. Here are few adults without one bad tooth: and a decayed tooth will not serve to explain away the

Neuralgia

cause of an intercostal neuralgia. And even if we press some local defect into the service of explaining a solitary neuralgia, how are we to explain one that repeats itself year after year and often at the same period of the year? There are cases, which prove themselves to have been caused by local irritation as a decayed tooth, by disappearing on the removal of the tooth. But it is well, instead of considering the tooth and neuralgia in the relation of cause and effect, to ask whether both are not the result of some less obvious condition.

In the case of an intercostal neuralgia, what is the local condition that invites the attack? Here too a weak point, locally, is suggested: for why otherwise should the affection be seated in the sixth rather than in the fifth intercostal space, or in the fifth in preference to any other? Or why again, should the pain choose the sciatic nerve in preference to the intercostal, or the trigeminus

in preference to both? Rheumatic neuralgia prefers the sciatic and occipital nerves: anaemic the gastric and intestinal. So that while it may be too much to say that any neuralgia is wholly due to a local or a general cause, it is as obviously true that, in the majority of instances, there is a general condition to be considered. Neuralgia, as has been well said, is a symptom, not a disease.

What are the cachectic states frequently met with in patients suffering from neuralgia? A personal or family history of neurotic tendencies naturally suggests itself; syphilis, which in so many and various ways affects the nervous system, is often present; chlorosis and anaemia; the rheumatic and gouty diatheses; the diathesis resulting from exposure to malaria.
In one manifesting neurotic tendencies, the ultimate cause of

Neuralgia

the affection is frequently central as in Locomotor Ataxia: the cachectic condition may however manifest itself in peripheral lesions which again give rise to neuralgia.

There are cases where neither a central origin nor peripheral lesion can be predicated. I am now, and have been for the past nine months, attending a female patient of marked syphilitic cachexia. During the whole of this time she has been confined to bed and is very weak. Mrs. P— is the wife of a publican, aged 44 years, and the mother of three children. She has been in ill health during the whole of her married life: six years ago she suffered from a pelvic abscess. She now suffers from a constant and most profuse endometric discharge which is accompanied by no pain. At times the monotonous character of her illness is interrupted sometimes by occipital neuralgia which is accompanied by excru-

Neuralgia

iating pain, making the patient feel "as if she should lose her senses," at other times by a similar affection of the right trigeminus. The paroxysms recur every ten or fifteen minutes at the beginning of the attack: she is hardly out of one, as she feels, before another is upon her. Each attack lasts about two days: during the trigeminal attack the corresponding side of the face is much flushed and the conjunctiva of the same side congested. Quinine, Croton-Chloral, Chloride of Ammonium, and the other drugs usual in this affection fail to influence the pain: hot opiate applications locally applied and morphia internally diminish the severity of the paroxysm. The patients' room is maintained at a comfortable and fairly constant temperature day and night: the bed is protected from draughts by a screen. On no occasion have we been

able to refer an attack to a local irritant, unless we assume it to be reflex from the uterine disease. But the irregular occurrence of the neuralgia corresponds to no defined period in the uterine disease. The drug from which the patient feels herself to derive marked benefit is iodide of potassium, and this drug cannot be intermitted without a concomitant change for the worse in the patients' condition.

In such a case as this where there is an absence of any evidence of central disease or peripheral lesion or irritation, what is the relation of the cachexia to the neuralgia? First of all, is it legitimate to assume that because the cachexia is there, it has therefore a causal relation to the neuralgia? When a definite syphilitic lesion exists as, for instance, disease of the bones through whose foramina the divisions of the fifth pass, or a syphilitic tumour pressing

Neuralgia

on a nerve, the relation is obvious. But it is true that neuralgias do occur in syphilitic patients for which no other cause can be assigned than the specific one: and it appears justifiable, in such cases, to consider this as the ultimate cause. A disease which may affect any structure in the body: which tends, during its course from its earliest to its latest historical epoch, to engender many disturbing and exhausting lesions, sufficient any one of them to undermine the general health: and, at last, leaves a general impression of itself on the whole animal economy, adding its own qualification to the personality of the individual: such a disease must show itself in some way, and at times in the nerves in preference to other tissues. Its mode of action is, in all probability, similar to that of chlorosis and anaemia, in causing a want of resistance to an

increasing and morbid molecular action in the nerve, or a want of inhibitory power in a nerve centre

Gout too has a causal relation to neuralgia: indeed Sir James Paget asserts that severe pains, often attributed to gout, are in truth neuralgic. Such pains occur in the chosen seats of gout, as the heel and external ear, and so are readily and generally accepted as the inflammatory pains of that disease, without much care to define whether the pain is proportionate to the gout. "Gouty neuralgia", he says, "is more sudden than others, more fitful, more quickly affected by errors of diet, indigestion and other causal disturbances."[1]

In the case of gout occurring in one of the usual seats of the gouty paroxysm, and in a gouty subject, the causal relationship

[1] Sir James Paget's Clinical Lectures and Essays, Lect IV, p. 383. Second Edit.

Neuralgia

Of gout to neuralgia may be a local one as well as a general; but, in neuralgia of the sciatic or brachial nerves, gout plays a similar part to syphilis and other diathetic states. Such diseases become part of one's self: not always showing themselves, and now and again even forgotten by their subjects: but they seldom allow one to rise to the ideal standard of health; ever ready to show themselves just when most likely to be forgotten. This is true especially of gout. I know several gentlemen who are constantly accusing gout of invidious attacks; the ball of their great toe is racked with pain; and still there is, in the words of Paget, pain "altogether disproportionate to the other signs of inflammation." It may well be that, as in the case of gout, we may find rheumatism mistaken for neuralgia. In not a few instances of chronic or oft-repeated illness, the patient takes the

libaty of diagnosing his own disease, And perhaps the physician is at times too unwary and ready to assume that the patient is right. The mistake is an easy one: the patient, say, has suffered for many years from rheumatic attacks: and one is apt to ask who should know his own sensations so well as the sufferer. But it is well to remember that the patient is prejudiced in favour of his mindful visitor, who has stuck faithfully to him for so many years.

That this applies in uterine diseases, we have the authority of Clifford Allbutt. He asserts that he has had, over and over again, patients come to him who had been treated for every conceivable uterine displacement, and often for obscure uterine disease. A course of arsenic dismissed the pains. This is surely very suggestive. One is so apt to think of neuralgia only when it is typical in seat, subject and symptoms. One

Neuralgia

is so apt to have an ideal case for every disease, and to miss anything short of his ideal: forgetting that the ideal is seldom attained. An acute rheumatism may fulfil all the conditions of the text-book; recurring attacks of chronic rheumatism are not so obvious. An ideal is well if we remember that it is only the general expression of particular phenomena that occur in a majority of cases: it is misused when one tries, not to apply the ideal to every case affording some or most of these phenomena, but to make every case fit the ideal. In neuralgia, especially, is it not true that we are too prone to associate it with certain sensory and mixed nerves to the exclusion of others? The fifth, the sciatic, the gastric: why not the first and second? Why again should we neglect neuralgia merely because rheumatism is present? We speak of rheumatic neuralgia: but if we find that rheumatism itself is

Neuralgia

a frequent visitant, we forget the neuralgia. I have been for the past six months attending a man for subacute rheumatism: a year ago I attended him four months for the same disease. He is a grave-digger, aged thirty four. During the greater part of the six months he has been confined to bed. Almost every joint has been swollen:- shoulder, elbow, hand, knee, foot: feet and hands been most frequently the seat. Now and again (three times during the six months) he has been able to get up, having only a feeling of stiffness and slight pain in the hands and feet. He has not left his bedroom: but just when he is about to venture forth, he is invariably attacked with what he designates 'rheumatism in the eye'. Now it is one eye, now the other. I always find that the so-called eye-rheumation is supraorbital neuralgia with marked conjunctivitis, probably either

Neuralgia

the result of, or followed by, perineuritis as the pain tends to become continuous. He has it in the left forehead and eye now.

Then in Clifford Allbutts uterine neuralgias: it seems strange that neuralgia should escape gynaecologists of high standing, remembering the tendency of the female generative organs to be associated with reflex symptoms due to them, such as mastodynia.

On the other hand we may have uteralgia associated with organic disease of the uterus. Trousseau relates some very interesting cases illustrative of this: the following one is quoted from him. "Thus I saw a lady in the year 1845... ... who was affected with a cancer of the inner wall of the uterus. She had, every day, paroxysms of awful pain, recurring at exactly the same time; the pain was seated in the hypogastrium and radiated to the kidneys, to the buttocks, and to the thighs,

Neuralgia

along the branches of the principal nerve trunks. It lasted from three to four or five hours, and then ceased, to reappear on the following day at exactly the same hour"[1]

He relates another case in which there was cancer of the inner wall of the uterus, and in which the intermitting and periodic pain was so intolerable that, during the paroxysm the patient "rolled on the floor" in agony. A peculiarity of this case was that the pain recurred "from half an hour to three quarters of an hour later each time, so that in the space of a month or more, the hour of attack had gone round the clock."

What was the relation of the neuralgia to the cancer? Was the cancer the local irritant causing the neuralgia, and the primary cause of it: or were both cancer and neuralgia symptoms of a constitutional tendency which realised

[1] Clinical Medicine. Vol I. p. 489. New Sydenh. Soc. 1867

itself, first of all, in a local lesion - the cancer, and secondly, and as a result of the first, in neuralgia?

That the central nervous system may be involved in neuralgia, apart from neuralgias immediately due to central disease, is proved by the occurrence of reflex neuralgias. Take the case of knee joint pain in hip joint disease, which may be reflex through the common origin of the associated nerves, or direct through their peripheral connection; or again the familiar example of mastodynia in uterine disease which also may be accompanied by occipital neuralgia. It is noteworthy that nerves involved in a secondary neuralgia are often of the same plexus as in the case of neuralgia of the anterior crural, which gives two articular branches to the knee joint and of which the long saphenous nerve is a branch, following irritation of the obturator in hip-joint disease. It is observable, too,

Neuralgia

that, even when the implicated nerves are not of the same plexus, there is often an organic connexion between the parts supplied by these nerves as when irritation of the uterine nerves is followed by breast neuralgia. It is difficult to trace the connexion between uterine disease and occipital neuralgia. Here the nerves concerned are from wholly different parts of the spinal cord: the great occipital being the internal branch of the second cervical nerve, and the uterus being supplied by branches of the hypogastric plexus. The only conceivable connexion between these nerves is a central one.

The motor system also bears evidence to the implication of the nerve centres in neuralgia. Neuralgia is sometimes accompanied by powerlessness of muscles. Whether this is due to mere functional inertia (passive lesion of Charcot), motor paralysis, irritation of trophic nerves, or mere nerve-irritation (Brown-Sequard), does not

appear to be decided.

Charcot draws a marked distinction between passive and trophic lesions. Passive lesions he believes to be due to functional inertia: trophic lesions to irritation of nerves or nerve centres followed by vaso-motor derangement.[1] He goes so far as to affirm that nutritive processes are not at all dependent on the existence of a nervous system. In support of this view he cites the case of plants and many animals, such as the protozoa, in which no nervous system can be defined: and, in the higher animals, he shows that, under certain pathological conditions, cell proliferation takes place in cartilage and epithelium which possess no nerves. His strongest argument, perhaps, is the experimental one which shows that, on the destruction of nerves supplying a part, or even on the destruction of the spinal cord

[1] Diseases of the Nervous System. New Sydenh. Soc. 1877. p. 9 et seq.

itself, peripheral parts will continue to be functionally efficient for "a considerable time" at least.

The clinical evidence, in favour of the non-essentiality of the nervous system in nutritive processes, is supplied by the cases in which, from mere want of use, as e.g. in the case of a diseased limb from hip joint disease, wasting takes place from inaction. M. Charcot agrees with M. Charles Robin that "nutrition is a general property of anatomical elements, be they animal or vegetable" (Journal de l'anatomie 1867. pp. 276-300). But surely if functional inertia is due to "suppression of function" of the nerves supplying the part, it is due immediately, at least, to nerve action, or rather to the want of it. Whether we seek to account for this muscular powerlessness by the hypothesis of trophic nerves which fail in their nutritive attendance, being reflexly affected by the irritation

Neuralgia

or injury of the sensory nerve; or by the supposition of paralysis of the motor nerves either reflexly or by proximity to the involved sensory nerve (as in a mixed nerve like the sciatic): or by nerve irritation only, the nervous system is not eliminated.

The argument from the case of plants proves too much. If they have no nervous system, then of course their nutrition is independent of it. But a plant is not an animal: and plant life is other than animal.

That plants exist without a nervous system proves, indeed, that life, ~~plant~~ life at least, is possible without nerves: but that is all. It does not prove, in the least, that animal life also is so possible. One might go further and ask whether there be not in plants a something which fulfils, in them, the purposes served by a nervous system in animals. And, of late years, observers have been led to believe that, even in the lower forms of animal life such as

the medusa, there is indicated the presence of a definite nervous system. That functional inertia has an important influence on the non-development of muscle is obvious. Not to speak of the hammersmith's arm and the professional swimmer's thigh, one sees, in prolonged inaction from sciatica, an atrophy of the muscles of the thigh. This may be from functional inertia: but may it not as probably be from vaso-motor paralysis or irritation: and may not the paresis be due to a combination of motor and vaso-motor deficiency: in short, a provision of nature, in obedience to her own wise law, to allow the painful limb to remain at rest? The centripetal sensory irritation of the sciatic may well suggest to the motor centre that, in the interests of the organism of which they form a part, the offending limb should rest: and the vaso-motor centre may, from the same consideration,

agree to modify its wonted activity, even if it neglect the economic argument that to afford the same nutritive supply, to an organ at rest as to one at work, would be Extravagance on its part.

As to the irritation[1] of a nerve causing paralysis, Charcot supports the theory from the analogous cases of trophical lesions, such as herpes, occurring in neuralgia. Section of a nerve does not, at once, induce loss of electric irritability and atrophy of a muscle, but only after some time and gradually as in the case of functional inertia. Rhamatic irritation of the seventh nerve, on the other hand, is followed by comparatively immediate paralysis. The immediate and essential cause of the paralysis, Brown-Séquard and Charcot hold to be, not in the nerve, but some change

[1] Irritation of motor fibres is here meant and considered under neuralgia with reference to this affection in a mixed nerve followed by irritation of its motor fibres.

in the contractile substance of the muscle. Brown-Séquard tied the ischiatic nerve in a rabbit, and, on the application of both poles, the muscles contracted to galvanism: galvanisation of the nerve produced no effect. He then tied the aorta below the origin of the renal arteries. On the application of galvanism now, whether to the nerve or muscle, there was no response. On the removal of the ligature again, the muscles readily contracted under galvanism: the ischiatic nerve gave no response, it was permanently paralysed: thus showing, according to him, that the difference between the paralysed and the contracting muscle, was just the difference between non-nutrition and nutrition (Journal de Physiologie' t ii, p. 77, 1859'). So far well: obviously enough, if the contractile substance of a muscle be inefficient, it cannot contract.

' Quoted in Charcot's Diseases of the Nervous System p. 44.

Neuralgia

But surely this is only a part of the Explanation - not the whole: the contractile substance of the muscle is inefficient - but the nerve was cut. First the cut nerve: then the incompetency of the muscular substance in which the terminal twigs of the nerve ended. But there was an intermediate process which was the mediate cause of the incompetency - the cutting off of the blood supply to the muscle. Muscular paralysis here, therefore, is not the result of one but of three causes, namely, firstly the dividing of the nerve, secondly, the stopping of the nutritive fluid, and, thirdly, the incompetence of the starved contractile substance. This looks more like the way in which nature is wont to work; not by an isolated and independent force but through many correlated forces, whether or not by the "transformation and equivalence" of forces. And the truer theory of the causation of paralysis, in irritated nerve conditions, would

be a combination of various theories: one that would involve the sensory, motor and vaso-motor nerves as well as the muscular contractile substance.

But even if we agree with Charcot and Brown-Séquard that the immediate cause of the paralysis is nutritional, is it not also and essentially nervous: for is not the circulatory directly under the control of the nervous system? If we suppose the vaso-motor nerves, as a consequence of the nerve-irritation, to assume the condition of spasm, so cutting off the blood-supply from the muscular fibre, surely this is in consequence of the hyperactivity of the vaso-motor centre following that irritation. And it does not seem scientific consideration of a pathological process to hold that, because one fact is the last link in the causal chain, therefore the earlier links are to be denied any contribution to the effect.

Neuralgia

Rather would it seem to be true that the nervous system is the essential factor in the causation. For, to accept Brown-Séquards experiment as proving that the divided ischiatic nerve, after the ligature was removed from the aorta, failed to respond to galvanism, and that still the muscular fibre did respond: what is thus proved but only this, namely, that the divided nerve of a muscle failed to control that muscle in the presence of the blood supply necessary for its own and the muscles' nutrition, while the muscular fibre in the presence of the same nutritive fluid and in the presence of an incompetent nervous supply, yet responded? The aortic ligature did artificially what the vaso-motor spasm naturally does - cut off the blood supply. Removing the ligature was equivalent to regenerating the cut nerve.

Further it is to be remembered

that, in the experiment, the galvanic current was applied to the muscle only a short time after the blood supply was severed: so that all we know is that the muscular fibre appeared able to respond to the current up to this time, while the divided nerve failed to do so. It would be interesting to know, and it is legitimate to ask, whether, up to this time, the muscular fibre may not have retained unused some of the nervous electricity formerly supplied to it. The nerve terminations were still in connection with the muscle: and it is not improbable that the terminal ends of a nerve may be storehouses of nervous electricity (if one may use the term), which requires only a stimulus suitable, in order to be discharged. Charcot himself bears witness that, in an injured nerve, the electric excitability persists for a longer time in the distal, than in the central end of the nerve. On the

recovery of the nerve, the excitability returns more quickly in the distal than in the central end; and, if the lesion be slight, the distal end does not wholly lose its excitability. These phenomena were exhibited when the ischiatic nerve of a rabbit was injured (crushed) with a forceps'. Brown-Séquard's experiment would be more conclusive, if it were shown that, after a lengthened period, and at a time when this stored electricity may be supposed to have been expended, the muscle still responded to galvanism.

The nutritional theory of paralysis has its valuable side even if it is only a partial explanation. It shows the importance of the blood in nerve irritation and nerve lesions: and so serves to indicate how rheumatic, syphilitic, anaemic and other diathetic conditions are so frequent factors in the pro-

[1] Ibid. p. 46.

Neuritis

duction of nervous phenomena.

Paralytic conditions however, though possible in neuralgia, are more likely to occur in neuritis: and it is even probable that the neuralgia in which paralysis occurs is the result of a neuritic or perineuritic process. Here the irritation is greater: there is the probability of the extension of the anatomical lesion to the nervous centres: and a motor nerve may be deprived of all functional competence and be even destroyed. In a mixed nerve it is hardly conceivable that, in neuritis, either the sensory or the motor portion should escape. There is the likelihood too that either by the same irritant as causes neuritis of the motor or mixed nerve, or by direct extension of the inflammatory process from the nerve primarily affected, or by a secondary extension from a nerve centre implicated directly from the primary

nerve, or again by a sympathetic affinity, the vaso-motor nerves may become affected. If these nerves themselves are neuritic, one must, if he accept the nutritional theory here, account for the muscular paralysis by their paralysis: if perineuritic, by their spasmodic contraction.

Perineuritis may serve as an irritant to induce paralysis either, if in a sensory nerve, reflexly by involving the motor centres or the vaso-motor nerves, or by a mixed nerve being involved in both its motor and sensory fibres. Charcot holds that, in a divided nerve, paralysis does not so soon occur as in an irritated one eg. by a contusion. This would imply that paralysis should occur earlier in perineuritis than in neuritis; and, if true, would be a valuable diagnostic index of these two conditions. Erb and Ziemssen however declare that the results of complete section and mere irritation

Neuritis

of a nerve are not appreciably different: and they are supported in this conclusion by the experiments of Vulpian (Archives de Physiologie t. iv, 1871-72 pp. 757, 758), and the histological observations of Neumann, Ranvier and Eichorst.'

In the presence of a paralysis caused by a morbid condition of a peripheral nerve, how are we to know that it is the result of neuralgia, or perineuritis or neuritis?

In paralysis with neuralgia, and following it in its onset, we have the typical neuralgic paroxysm and the intermittency. In this case we must assume the absence of neuritis - at least to begin with - and that the paralysis is reflex. For instance, if after well marked neuralgia of the infra-

'Note by Dr. Sigerson in Charcot's Diseases of the Nervous System. New Syd. Soc. 1877 p. 50

orbital nerve, paralysis of the seventh occurs, we may infer that the paralysis is reflex from the neuralgia, or that both are the result of the same cause as damp or cold. In sciatic paralysis if the pain comes on gradually, and although abating now, and again becoming intense, does not intermit, we may infer neuritis or perineuritis. Probably, too, paralysis following neuralgia is more sudden than in the case of neuritis. In perineuritis of a superficial nerve there is swelling along its course and, it may be, nodosities: and in any neuritic condition, whether of motor or sensory or mixed nerves, pressure along the course of the nerve is painful as well as movement of the parts to which the nerve is distributed: the pain is said to be centripetal. If the neuritic condition is so far advanced as that the axis-cylinder

of the nerve is paralysed, it will not react to galvanism. Trophic troubles are in favour of neuritic conditions as is proved by post mortem evidence; so also are local inflammatory symptoms and general febrile disturbance. Trophic troubles may be absent in neuritis: so may paresis and paralysis: but their presence is in favour of it. According to Nothnagel, cutaneous sensibility is of importance as a diagnostic point between neuritis and neuralgia. In neuralgia early hyperaesthesia is common, giving place later to anaesthesia; anaesthesia appearing in the course of a few days is in favour of neuritis'.

One would expect the electric condition of a nerve to throw light on the diagnosis. In

' German Clinical Lectures, 'Diagnosis and Pathology of Neuritis', New Sydenh. Soc. 1877 p. 215. Translated by Dr. Joseph Coats.

neuritis if the nerve fibre disintegrates we have no response to the electric current: in early perineuritis there is hyperexcitability. It is to be noted that a penetrating perineuritis, that is, one in which the perineural processes intervening between the funiculi and involving the sheath peculiar to the funiculus itself, produces symptoms as marked as those of an essential neuritis of the axis cylinder itself.

Neuritis may occur in a sensory, motor, or mixed nerve. In neuritis of a sensory nerve there is absence of intermittency, there may be "spontaneous pains": and if there are trophic lesions as herpes zoster, atrophy of the hair or nails, or ulceration of the skin, and lastly impaired sensibility, all the symptoms having a definite relation to a nerve, the diagnosis is confirmed. Nothnagel quotes a case of Weidner's in which a

Neuritis

Zoster developed in the region of the first branch of the fifth nerve along with signs of inflammation in the eye and acute fever: there were paroxysmal pains in the region of the nerve. Post mortem examination, five years later, showed inflammation of the first branch of the trigeminus. In my own case of the grave-digger related at page 46, there was doubtless rheumatic perineuritis of the supraorbital nerve.

Sometimes treatment is valuable as a diagnostic. Nothnagel had a case, apparently of neuritis in the left sciatic of a young girl. The pain occurred in paroxysms, thus simulating neuralgia, but at one spot behind the great trochanter there was continuous pain. He considers neuritis to have been proved by the fact that leeches relieved the circumscribed pain, and on the girl herself applying twenty leeches to the spot, she

Neuritis

became quickly and perfectly well. Treatment is not always so successful. I saw the case of a man aged sixty five, in the practice of Mr. J. Winter Dryland of Kettering, which was evidently neuritic in character and in which treatment gave little relief. Pain followed the course of the left sciatic and it was continuous. There was general febrile disturbance at first with loss of appetite and sleeplessness: latterly there was incapacity of motion in the affected limb. The usual internal remedies were given: the limb covered with cotton wool: blisters were applied along the course of the pain, and finally Corrigan's cautery was used with great relief at the time. In no case was the relief more than temporary: and, for three months, the patient could not move his left leg without severe pain. The left thigh became

Neuritis

smaller than the right.

Neuritis may simulate not only neuralgia but other conditions which are usually associated with central disease; and, on the other hand, there may be present, in certain diseases, symptoms which indicate neuritis but which are found not to be due to this lesion.

Neuritis, especially in its multiple form, may simulate central disease. Dr. Buzzard, in his Harveian Lectures', states the case of a single woman, aged twenty four, who was sent to him. She suffered "from loss of power of power in the right hand with agonising pain. ... . The right hand and forearm had a soddened, puffy, helpless appearance, with swollen fingers and puffy discolouration of the skin

' Lancet, Oct. 28th. 1885

Neuritis

"in patches which here and there looked glossy.' Her immediate illness commenced in the preceding august- six months previously - with pain and swelling in the middle finger which gradually extended to the others, and, for some months past, her hands had been quite useless. The pain was so constant and severe that she could scarcely ever get sleep at night ..... Temp. 100°F. Power of extending wrist moderately good; flexion could not be done. There was slight power of flexing the last joint of each finger, and an equally slight power of extending it, and this applied also to the last joint of the thumb. There appeared to be no power of the intrinsic muscles of the thumb and fingers. Examined electrically the thenar muscles did not respond to either form of electric excitation: but the

' See interesting case of Herpes Zoster and Paralysis of Motor nerves, quoted from D. Arch. f. Klin. Med. oct. 1885. in Practitioner Feb. 1886 p. 128

Neuritis

"muscles of the front and back of the forearm were excitable by induced currents, though only when a considerable strength was employed. Warmth was felt as well by the right hand as by the left, but cold was felt best by the left (unaffected) hand. ... ... The patient was forced to keep the limb covered up; the air would start pain, and conveyed a burning, smarting sensation. There was a more or less constant feeling of numbness in the fingers." Blisters were applied without any appreciable result: melancholia developed and the patient was removed to an asylum. Ultimately the hand recovered. There was no suspicion of syphilis, but a history of doubtful rheumatism was given.

In this case obviously, motor, sensory and vaso-motor nerves were involved. The hand was more affected than the arm, a point on which Dr Buzzard

Neuritis

lays much stress as indicating the peripheral character of the affection. Generally, too, the paralytic phenomena follow pain - a point of diagnostic value.

He relates the case of another female, aged fifty six, who became affected with loss of power and numbness in the left arm. There was no indication of central disease. The left arm gradually improved. A month after the first attack she lost all power in both legs: this soon passed off. Several such attacks occurred, now in one limb now in another, indicating the peripheral character of the affection. There was a family and personal history of alcohol. In such cases Dr. Buzzard found a gouty or other diathesis marked: he considers gout, by the local action of urate of soda, to be a potent local irritant of nerves.

He concludes that "there is now ample evidence that a more or less widely spread paralysis

Neuritis

"may depend upon a degeneration of the nerve fibres themselves, most pronounced towards the periphery, and independently of any recognisable change in the nerve centres or roots. Such cases may occur in connexion with chronic alcoholism, diptheria, enteric fever, syphilis, tuberculosis"

Pitres and Vaillard have found neuritic changes in peripheral nerves in cases of typhoid fever, and the possibility of this explains paralytic changes in typhoid which were wont to be ascribed to central nervous disease.[1]

Hun considers that alcoholic paralysis, as well as the neuralgic pains and cutaneous symptoms accompanying it, is due to degeneration of peripheral nerves. Dr. de Watteville, on the other hand, believes that these symptoms and the degeneration of the peripheral

[1] Lancet, January 2d, 1886.
Lancet, April 3d 1886, where similar changes in tuberculosis are referred to

Neuritis

nerves themselves, are due to a molecular alteration in the nerve cells of the anterior horns.[1] Westphal also bears witness to the existence of multiple neuritis and its liability to be referred to central disease.[2] He irregular distribution of the symptoms suggests the peripheral nature of the lesion. He gives one case, a male aged thirty two, in whom there was paralysis and wasting of the muscles of the feet and toes, paresis of the muscles of the calf, along with paralysis of the muscles supplied by both radial nerves. But all the branches supplied by any one nerve were not equally affected: for instance the branches of the radial distributed to the triceps and supinator muscles were not affected, nor was the branch

1. Lancet, September 5th 1885.
2. Practitioner, January 1886, p. 50 et seq.

Neuritis

of the peroneal supplied to the tibialis anticus.

Dr. Lamby draws attention to certain cutaneous paraesthesia, in the form of numbness, or tingling, or a sense of pressure on a nerve trunk, occurring in one or more extremities, sometimes confined to one or both upper or lower limbs, at other times present in all the extremities or in the upper and lower extremities of the same side and accompanied by a loss of power in the affected limb. The sensations are peculiar in that they are paroxysmal and tend to occur in the night. He attributes the affection to gastric disorder. Sinkler (New York Medical Journal, July 26th. 1884) believes the conditions to be caused either by hyperaemia of the cord or of the affected nerve.

In all these cases diagnosed as multiple neuritis, there was an

Lancet, Sept. 5th 1885.

absence of the girdle pain common to locomotor ataxia and other central nervous diseases; there was generally a combination of sensory and motor symptoms so irregular in character and distribution as to exclude central disease. A very common combination is the simultaneous affection of the nerves of the forearm and the leg. In some cases the paralysis is permanent and the reaction of degeneration well marked.

So far I have spoken of neuritis as the primary disease, not only as to causation, but also as indicated by its clinical symptoms. This condition, however, may play another part in which, although etiologically primary, it is clinically of only secondary significance. Neuritis has come to be looked upon as either directly or reflexly the origin of other nervous diseases. Tetanus, according to

Neuritis

some authorities, may be caused either reflexly by the irritation of neuritis, or directly by the extension of the disease to the nervous centres. The fact that tetanus more frequently results from punctured, torn, and lacerated wounds than from incised ones: and that further these wounds are more frequently peripheral in the hands and feet, and so exposed to irritation of the wounded nerves, goes far to show how the disease may result from neuritic conditions of a nerve. In many cases of tetanus post mortem evidence has proved neuritis. Dr. Clifford Allbutt relates several cases in which the injured nerves showed distinct signs of inflammatory action. In two cases the posterior tibial nerve was bathed in pus.'

Curling, Froriep, Rokitansky, Erichsen, Lepelletier, Trousseau, Lockhart Clarke, bear witness to inflammatory

' Lancet. Feb. 25th. 1871. p. 270

Neuritis

changes in nerves involved in a wound followed by tetanus. Froriep in seven cases found "tumefaction and reddening of isolated tracts, extending from the wound to the spinal cord"[1] indicating that the process pursued its centripetal course per saltum. Brown-Sequard supports the theory of the peripheral origin of tetanus by the alleged frequency with which the muscles in the neighbourhood of the wound are earliest attacked; and by the fact that he and others have found pressure on the wounded part or on the cicatrix to induce the tetanic spasms during periods of relaxation. As to the alleged frequency of the early implication of the muscles in the vicinity of the wound, the only three cases of tetanus that have come under my own notice do not bear out the statement. In one case, that of a boy aged fourteen, whose right auricle was

[1] Jones and Sieveking's Pathol. Anatom. p. 311

almost wholly separated by contact with a threshing machine and in whom the wound healed by second intention, the earliest spasmodic indications were in the muscles of the face and neck on the side opposite to the injured auricle. In the case of a little girl, aged seven years, the first complaint of pain was in the muscles of the back. The origin of her case was obscure. Fourteen days before the onset of the lumbar pains she was exposed to a damp chill (in May 1885) while lying on the grass. Minute inquiry brought forth the fact that she had bruised her right thumb about a week previous to the chill: but inspection showed no signs of this, although she complained of slight irritation at the tip. There was no post mortem in either case. In the case of a horse belonging to a friend which, with the purpose of converting him to a

Neuritis

hunter, had been "docked" and a month afterwards, from a chill it was supposed, became afflicted with tetanus, the earliest muscular symptoms appeared in the abdominal muscles, and were described by the groom as "hardness over the stomach". The slightest touch on the wounded tail induced tetanic spasms. Amputation was suggested here above the seat of the wound in order to remove irritation, but the suggestion was not carried out.

In one of Nothnagel's cases, on the other hand, in which tetanus followed injury by an axe to the last phalanx of the left thumb, the wound itself was painless: but pressure over a point about the middle of the internal aspect of the forearm, over the median at the hollow of the elbow, and over the radial at the styloid process of the radius was painful: and in the latter case there were slight contractions of

the thumb. He diagnosed neuritis of the median and radial nerves; post mortem evidence of this was, however, wholly negative.

Neuritic conditions of peripheral nerves are sometimes indicated in other nervous conditions such as epilepsy, chorea, hysteria and even hydrophobia. The similarity of epileptic seizures to those of tetanus are in favour of the theory that epilepsy may result from peripheral irritation, especially in cases of obvious peripheral lesions or other cause likely to induce reflex motor excitation. The effect of exposure to draughts in the induction of tetanic spasms; and of drinking or the attempt to drink, in the induction of hydrophobic convulsions, suggest a similar liability to be affected by external agents. Marlet ('Press Méd. Belge 1869. 237)' relates a case of epilepsy with seizures re-

' A Biennial Retrospect of Medicine and Surgery for 1869-70. New Sydenh. Soc.

resembling those of hydrophobia in a man who had been bitten four days previously by a mad dog. The recent investigations of Pasteur however, indicate that hydrophobia is a toxaemia rather than a neuritis.

Nothnagel relates a case of Virchow's in which a soldier, after being shot in the upper arm, exhibited symptoms indicating injury of the median nerve. After a time, and preceded by various cerebral symptoms, epileptic fits occurred. Virchow excised a portion of the median nerve and found interstitial neuritis. The epileptic fits ceased after the excision.[1] Billroth relates a case of injury to the sciatic nerve followed by epileptic fits which ceased on the closure of the injured nerve without excision.

That neuritis may induce chorea and hysteria is supported by the fact that counter-irritation over the injured

[1] German Clinical Lectures 1877, New Sydenham Soc. p. 232

nerve has sometimes induced the disease to disappear.

Diagnosis

Briefly to consider the diagnostic points that must be kept in view with reference to neuralgia and neuritis. The main points are the course and character of the pain in both diseases: and its obvious connection with a nerve or nerves. A neuritis of the sciatic might simulate a myalgia: the myalgia mostly disappears when the affected muscles are at rest; the neuritic pain is aggravated by motion, but does not disappear nor is it much alleviated by rest. Then a myalgia will probably be indicated by a similar affection in other muscles or subacute pains in joints. So neuralgia in the great toe may simulate gout; but the pain is too severe for any form of gout other than the acute, and the absence of local and general febrile disturbance negatives this.

Neuralgia & Neuritis

There is lastly a disease accompanied by severe pain where neuralgia or neuritis might not suggest themselves: and, for the purpose of diagnosis, the process of exclusion aided by the foregoing considerations and collateral trophic and other phenomena, is the best method to adopt.

Prognosis

The prognosis in a neuralgia, due wholly to local irritation, depends on the nature of the local irritant. If due to the pressure of a tumour, the nature of the tumour, especially as to its removeability, governs the prognosis. a fibroma pressing on the brachial nerves gives a more favourable prognosis than a uterine one. If the neuralgia is due, not to a personal source of irritation, but to one external as cold or damp, then the possibility of removal of the individual from the irritant, guides the prognosis. If, again, the

Neuralgia & Neuritis

pain follows from an external irritant or one deficient, from any cause, in resistant power, the removability of this cause governs the prognosis: the prognosis of the neuralgia is very much that of the conditions that induce a want of resisting force. In neuritis more or less similar considerations present themselves. The nature of the local irritant and of the general constitutional conditions are the factors to be considered. Here, moreover, there is in addition to be considered the position of the affected nerve. Neuritis is more readily influenced by local treatment than neuralgia is: and the locality of the neuritic nerve may make local treatment efficient or comparatively inefficient.

In serious affections associated with a neuritic condition of a nerve, the neuritis is not so much what we are called upon to prognose. In tetanus following neuritis, the prognosis is bad

Neuralgia & Neuritis

not because the neuritis, per se, is serious, but because it has in this case been followed by tetanus. Here our efforts are directed not primarily to cure the neuritis, but to cure the tetanus by removing the neuritic source of irritation.

Treatment

The treatment of neuralgia resolves itself to two first principles; namely, the removal of any local irritant and the treatment of the general health, and specially any phase of it that may be defective so as to give to the patient a deficiency of resisting force to noxious influences.

The pain must first be removed: but one must not forget that the pain is only a symptom, and that, when it is removed, we have rid the patient of only a sign of his disease, not the disease itself. The pain cannot wait for general or diathetic treatment; and while it

is present, the general irritability that accompanies it, would counteract any possible good effect of more general treatment.

To remove the pain, we must know, if possible, the local condition causing it: to see that, after it is removed, it does not again recur, we must know the general cause, predisposing or otherwise, of its presence.

First of all then, let something be done for the pain: opiate applications locally applied and morphia either by the mouth or subcutaneously. Cold applications are not indicated: ice, applied in the by Billroth, aggravated the pain. Hot applications sometimes are alleviative: the affected part should be protected from cold. Any obvious source of local irritation is to be removed. Then as early as can be done, let general and diathetic treatment be adopted - according to the indications. Steel and specially arsenic are most useful in

Neuralgia & Neuritis

anaemic conditions: quinine in malarial and typically periodic neuralgias: and it must be remarked generally that quinine is the most potent immediate agent to stop the neuralgia for the time. Chloride of Ammonium is indicated in depressed nervous conditions: I have no experience of tonga. Judicious dieting must go along with general treatment.

If notwithstanding the means adopted pain still persists, more heroic measures may be indicated. It may be necessary to cut down on the affected nerve and stretch or even excise a portion of it. This stretching or excision must be on the proximal side of the point of irritation. If the dental branches of the infraorbital are affected, excision of this nerve may cure: but if the source of irritation is on the proximal side of the infraorbital foramen the operation would fail. Billroth operates subcutaneously and cuts

through the nerve in several contiguous places. Stretching is by no means always successful nor is excision as witness the case related by Billroth'. Acupuncture sometimes is successful especially in perineuritis (Fayor) In paralysis associated with ritter neuralgia or neuritis the galvanic current must be applied three or four times weekly.

As to neuritis diathetic tendencies are most common. In acute neuritis or perineuritis, specific treatment should be tried at once, just as it should be in acute rheumatism or acute gout. But at once, also, local treatment must be tried. Leeches, blisters, the cautory, followed by opiate applications, and, if the pain is very severe, opiates internally. In some forms of neuritis, even at the very commencement, iodide of potassium is indicated. I had a case of a working mason, aged thirty years, who went to bed

ante p. 122 et seq.

Neuralgia or Neuritis

at night all right, and awoke in the morning with paralysis of the whole right side of the face. He had a sense of numbness but no pain. As there was a specific history (not of himself so much as of his wife) iodide of potassium was exhibited in doses latterly of twenty grains thrice daily. The constant current was applied thrice weekly. For the first six weeks there was no appreciable improvement; then gradually he found some power of closing his right eye and was able to whistle in an abortive kind of way. In three months he was cured.

In conditions involving neuritis and to which neuritis has, or is supposed to have, a causal relation, local treatment should be energetically pushed. In tetanus from a wound, for instance, it might be well to raise a portion of the nerve above the seat of irritation and on the proximal side

of any neuritic appearance. In a case of tetanus following injury of the infraorbital nerve, Larrey cut the nerve across and a cure followed: Murray cut across the posterior tibial in a case of tetanus in a youth who had trodden on a rusty nail; cure followed.[1] Or even amputation of a limb might be undertaken as a means to cure tetanus or epilepsy probably arising from a neuritis.

Brown-Sequard[2] quotes a case of hydrophobia communicated to him by Stokes, in which the application of a tourniquet above the bite relieved the symptoms; and amputation of the limb was suggested but not carried out.

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High Street,
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[1] Erichsens 'Science and Art of Surgery' p. 565
[2] Lectures on the Central Nervous System, appendix p. 261.