A Clinical Lecture

ON

CHRONIC PAROXYSMAL TRIGEMINAL NEURALGIA AND ITS TREATMENT.*

BY

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THE subject of these lectures has been revised and brought up to date in order to present results of my experience of the treatment of this disease during the last twelve years.

Trifacial neuralgia, chronic trigeminal neuralgia, tic douloureux, or neuralgia major of the fifth nerve, as it has been variously described, is an inveterate and violently painful paroxysmal neuralgia, affecting, in the majority of sufferers, one side of the face only. In by far the larger number of sufferers the pain is referred to the territory of the second or the third division of the fifth nerve—that is to say, roughly, the upper jaw and cheek, upper lip and side of the nose for the second division, and the lower jaw, lower lip, and perhaps the side of the tongue for the third division. In a considerable proportion of cases the second and third divisions of the nerve are both affected, and the pain will not cease, or ceases only for a time, if one of the divisions alone is treated. In a small proportion of cases the supra-orbital branch of the first division is involved, scarcely ever alone, but in addition to neuralgia affecting the second division or the second and third divisions. Supraorbital neuralgia alone is usually due to temporarily acting causes, such as influenza, malaria, or other fevers, or to such causes as errors of refraction, frontal sinusitis, migrainous neuralgia, and many others, and it must be distinguished from the chronic paroxysmal and inveterate disease known as trigeminal neuralgia. The pathology of this disease is somewhat obscure. What is certain is that there is no disease to be detected by hitherto known methods, either in the nerve centres of the brain, Gasserian ganglion, or main branches of the fifth nerve. The undoubted fact that efficient interruption of the conduction of afferent sensations along these branches of the fifth nerve gives instant relief to the neuralgia appears to me sufficient proof that such neuralgia is dependent upon stimuli affecting the nerve endings at their periphery.

The results of destruction of the ganglion as compared with neurectomies or alcohol injections below the ganglion differ only in the degree of their permanence—that is to say, operations on the nerve trunks, however complete at the time, are always followed by some re-establishment, sooner or later, of nerve conduction through downgrowth of nerve fibrils from the ganglion cells above. If, on the other hand, the ganglion is thoroughly destroyed by any means, the anaesthesia of the fifth nerve territory is permanent, and the cure of the neuralgia is correspondingly complete.

The nature of these stimuli from the periphery in this neuralgia is a matter of speculation, but it seems highly probable that they are due to septic neuritis of dental nerve filaments, the result of previous dental caries, pyorrhoea, or abscess around the roots; and recently in a case of this chronic neuralgia a piece of the maxilla was removed for me by Mr. Warwick James under the most careful aseptic precautions, and a pure growth of streptococci was obtained.

I have been impressed, in taking the histories of cases, by the number in which the pain has definitely started immediately after dental operations or antral abscess. No other nerve in the body, except the second and third divisions of the fifth nerve, is so liable to chronic infection of its branches, and although in some cases even many years have elapsed between the loss of a tooth and the onset of the neuralgia, yet this does not exclude septic neuritis from dental infection as the cause, and when the infection has thus spread backwards into the dental filaments within the jaw it is easily understandable that removal of the tooth does not cure the neuralgia; indeed, it often makes it worse.

Causes.

During the last twelve years I have seen a large number of cases of chronic trigeminal neuralgia, and I have analysed my records of over 312 cases, 230 in private practice and 82 hospital cases. I have, in addition, seen and operated upon a considerable number of hospital cases whose records are not available or are inadequate. Some curious points come out in my analysis which I have never seen referred to in other writings on the subject. The proportion of women affected to men is 62 per cent. to 38 per cent., or about three women to two men, while of the unilateral cases of neuralgia, which of course form a very large majority, the right side is affected in correspondingly greater frequency—namely, 62 per cent. to 38 per cent. This excess of right-sided cases over left-sided obtains in each group of men and of women, both in private cases and in hospital cases; and, moreover, in each separate year of the eleven years I have analysed a somewhat similar excess of right-sided cases over left is seen. I cannot therefore look upon this difference in frequency of incidence on the two sides as accidental; what its cause can be it is not easy to say.

I do not know whether any statistics are available of the relative frequency of dental caries on the two sides. Other factors, too, have to be taken into account which may act as the exciting cause for the origin of trigeminal neuralgia. I have already spoken of dental operations originating the pain in a certain number of cases, such as fracture of teeth and difficult stump extractions. In ten of my cases severe *chill* to the face, as by motoring in a cold wind, was the immediate precursor of the original spasms of pain; in five cases intense *emotion* immediately preceded the first attack. A woman was shocked by being told that her child, who was playing in the roadway, had been run over by a motor car and taken to the hospital. She immediately hurried off to the hospital, and on reaching there three-quarters of an hour later heard that her child was dead; at the same time a spasm of violent neuralgia struck her in the right lower jaw, the pain continuing at intervals until I saw her two years later, and was able to arrest it by injecting the third division of the fifth nerve with alcohol.

Still another cause of onset of this terrible neuralgia is a *blow* upon the jaw or face; this occurred in six of my cases. For example, a young woman, aged 27, was stooping to pick something off the floor when another girl's head struck her violently on the lower jaw, making her teeth clash and rattle, as she said. About three hours later, when she had just mounted on to the top of an omnibus, she was suddenly seized with the most excruciating pain in the face, which made her behave so wildly that she said she was like a mad woman, and had to be taken off the bus. The paroxysm lasted altogether some ten minutes, but recurred again and again in the manner characteristic of trigeminal neuralgia until I saw her and injected the third division of the nerve some years later.

Such suddenness and violence of onset of the spasms of pain are extremely characteristic of this peculiar form of neuralgia, and I have notes of some forty-six cases in which this was the case. One of apparently the most severe cases of pain I have ever seen began in this way. She was a cook, aged 45, who in her spasms of neuralgia lost all control, and screamed and threw herself about. Six years previously her first attack occurred, without any warning one night at 3 a.m., when she was awakened with the most awful agony in her face. Her screams awoke the whole household; a doctor had to be sent for, and she was kept under morphine almost continually for three weeks. She got no relief from any drugs until I injected the second and third divisions of the fifth nerve with alcohol; this gave her complete relief for two years. Recurrence and reinjection with alcohol again gave her two years' freedom from pain, when I injected her Gasserian ganglion with alcohol, since when she has been completely free for several years.

In other patients the pain may at first be mistaken for dental neuralgia, but the spasms of pain gradually increase in severity, in spite of the removal of teeth, until at last it is realized that the neuralgia is of the graver inveterate type.

Age.

The age of onset of the disease varies very widely in my experience, the oldest being 85 when the disease

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commenced. In two cases, both females, the severe neuralgia began at the age of 17; one of them had suffered from neuralgia of the third division for twenty years when I saw her, temporary relief only having been obtained by a division of the inferior dental nerve. Alcohol injection of the third division ten and a half years ago gave her complete relief, which still persists. The other patient was a lady aged 70 when I saw her for the pain; she had suffered almost constantly for fifty-three years; in her case injection of the ganglion was done under chloroform two and a half years ago, causing total anaesthesia of the fifth nerve and complete relief of all pain, which should be permanent.

The onset of the neuralgia is rare under the age of 30, though I have seen eleven other cases commence before the age of 30, at the ages of 20, 21, 22, etc. The average age of onset in 205 of my cases is almost exactly 50. Once the disease has started it is practically certain that permanent cure can only be obtained by destruction of the nerve trunks involved, or of the ganglion itself. In only two instances have I known the neuralgia disappear spontaneously, after existing a number of years. One was the father of a lady whom I injected for typical neuralgia, and she told me that her father suffered from the same spasmodic pains from the age of 65 until he was 90, when the pains gradually left him, so that he was free from the neuralgia during the last twelve years of his life, for he died at the age of 103.

In a number of cases a considerable interval may elapse between the first onset of the neuralgia and its subsequent establishment. A lady had severe spasmodic pain in her left lower jaw at the age of 45, lasting for nearly two years, when it left her for thirty years, recurring severely after exposure to a cold wind when driving. After the pain had continued for nine weeks I injected the left Gasserian ganglion. I have notes of similar intervals of twenty years, seventeen years, nine years, seven years, and so on.

Heredity.

I have noticed heredity as a factor of trigeminal neuralgia in five cases. I have already referred to one patient, a lady of over 60, whose father suffered for over twenty-five years from this form of neuralgia, and then outlived it. In another instance I have treated a mother over 70 years of age, who suffered for many years with bilateral neuralgia, mostly of the third division. Injection of the nerve at the foramen ovale gives her about two years' freedom from pain. One of her daughters at the age of 36 began to suffer in a similar manner with spasmodic neuralgia of the right third division. I saw her for this soon after the pain began, and recommended drug treatment and rest; this, however, failed entirely. I did not see her for three years, the pain having continued all that time, and she told me she had been twice injected by another physician without any relief, as the nerve could not be found. I then injected the third division at the foramen ovale, causing complete anaesthesia of its distribution and immediate cessation of the pain, from which she has now been quite free for three and a half years. In my other three patients—two men and one woman—it was the mother in each case who was said to have suffered from typical trigeminal neuralgia for many years. In investigating such histories one must be careful to distinguish between migraine and the true trigeminal neuralgia.

Chill.

I have already referred to chill as being the exciting cause of the first spasms of the neuralgia in ten cases. One illustration will suffice. A woman, aged 50, drove twenty-five miles in an open dogcart through a blizzard, snow, and icy cold wind to Exeter. The same evening, about 9 o'clock, while she was having supper, intense neuralgia struck her lower jaw, the pain being on the same side as had been exposed to the wind. This was the beginning of a severe case of trigeminal neuralgia which continued for several years until I saw her. In numerous other instances exposure to a cold wind or draught will restart the paroxysms of a neuralgia which has become dormant, and, indeed, cold wind or draught may be said to be the worst enemy of these subjects of neuralgia.

Although chill has such a bad effect upon trigeminal neuralgia it is surprising that subjects of this disease have

not special immunity during hot weather, and it is extremely common for patients to say that the time of year makes no difference to their pain, and it is waste of time and money to send such patients for treatment to a hot climate.

"Warnings."

I have referred to the absolutely sudden onset of the disease in numerous cases. Three of my cases have described a warning by which they recognize the imminence of a period of severe attacks. A lady, aged 64, had suffered from right lower jaw neuralgia for ten years, made worse by extraction of the teeth. Alcohol injections by Horsley gave no relief. As she says no numbness of the face was produced, the nerves were no doubt not injected. She says she has always noticed a burning sensation on the top of the head for some days before the neuralgia attacks commence. Alcohol injection, in May, 1916, of the second and third divisions relieved her pain entirely, the relief still persisting. In another case, a man whom I injected at the age of 39, with neuralgia of the left second and third divisions, seven years ago, used to get a warning, for a day or two before the neuralgia attack, of constipation and sensation of swelling in the gums.

Another patient, a lady aged 54, says she always gets a shivering sensation along the right cheek before the pain starts. She had lost all her teeth twenty-seven years before the pain began.

Character of the Pain.

The suddenness of onset of neuralgia is often likened to an electric shock; one patient thought she had bitten on to a needle, another that she had broken a tooth. Some speak of a stinging pain at the commencement. Very many describe the pain as hot, "like a lot of red hot bradawls being pushed up," "flickering," "hot," "like daggers," "like a hot knife or needles being run into the forehead." In others the pain starts with a tacking sensation or tapping, scarcely painful at first, but rapidly getting worse, until the pain "explodes along the nerve like fireworks or hot electric wires." The severity of the attacks must be witnessed to be appreciated, and patients get little sympathy or credit for their suffering, except from others who have seen them in it. Some patients lose all control of themselves in the attacks, screaming out, fighting, and struggling. In others the jaw becomes fixed, perhaps wide open, and saliva may trickle from the mouth.

One patient described the commencement of her attack as having suffered for two days with severe agony in a tooth; she thought such pain was impossible, had two teeth out, but was no better for a week; since then she has been subject to repeated attacks in which the pain stings into the right side of her tongue. She calls it a "hellish pain," and says it has destroyed her nerve, and she feels during the attacks as if she could smash everything, or commit suicide. Indeed, before the remedy was devised for this pain in the operation of gasserectomy, and later by alcohol injection of the nerve trunks, suicide was not a very uncommon ending for sufferers from this dreadful disease.

So long as patients are able to take food they do not lose weight or suffer notably in health, but as soon as the neuralgic spasms are sufficiently frequent and severe to prevent the taking of food there is danger of rapid loss of strength from starvation, and it may be necessary for the patient to suck liquid nourishment through a straw or tube on the other side of the mouth, and to write instead of speak, because the spasms of pain are started by attempts at eating and talking.

Treatment.

Only a comparatively small number of sufferers from trigeminal neuralgia get relief from drugs. In a few gelsemium gives relief, though with the majority even morphine in large doses fails. Practically the only two methods are gasserectomy and alcohol injection. In my opinion the former operation should never be undertaken until alcohol injection has been thoroughly tried. During the last ten years I have injected the Gasserian ganglion with alcohol through the foramen ovale in 63 cases; in 31 the anaesthesia has remained total and no recurrence of pain has taken place. In many of the remainder partial

ganglion anaesthesia has persisted though pressure could be felt, yet the relief from pain appears to be equally good.

In many cases, though total ganglion anaesthesia is produced at the time of injection, yet the anaesthesia of the first and second divisions wears off, perhaps completely, in from ten minutes to an hour, though the third division remains totally anaesthetic. This is no doubt due to the ganglion cells having been shocked, and not destroyed, by a partial infiltration only. In such a case pain is liable to recur after twelve months or two years, as in a case of ordinary injection of the nerve. With total destruction of the ganglion there is the same liability to keratitis as with the operation of gasserectomy, but if the lids are kept carefully closed by strapping for the first week and the conjunctival sac washed out with boracic lotion twice a day, I have found little or no trouble with my later cases. The lids, however, should be kept closed from the beginning, and it is unwise to wait and see whether keratitis develops, as then it will be necessary to keep them closed for a much longer time.

Hutchinson's partial gasserectomy avoids the danger of keratitis, but is not a certain permanent cure, as I have seen three such cases relapse with neuralgia.

Bilateral Neuralgia.

Bilateral trigeminal neuralgia is comparatively rare, though I have seen and injected 25 bilateral cases, usually at different dates, but on several occasions I have injected both sides, one after the other.

Gasserectomy is an impossible operation for bilateral cases of trigeminal neuralgia, owing to the necessary destruction of the motor branch of the fifth nerve on each side and consequent jaw-drop. Alcohol injection has the advantage that the nerve injury is less complete, and the motor nerve fibres regenerate. In one case I produced jaw-drop for two and a half months, which recovered completely later. On December 10th, 1919, I injected the left Gasserian ganglion in a woman of 49, for neuralgia of the left upper jaw, whose right Gasserian ganglion I had previously destroyed by alcohol injection seven years previously. She was still totally anaesthetic over the whole right fifth distribution, just as much as in any case of gasserectomy, though in her case the motor branch had recovered well, and she had quite good right temporal, masseter, and pterygoid muscles. Injection of the left Gasserian has now produced in her case total anaesthesia of the whole of the face and front half of her head, though she can chew fairly well by means of the muscles on the right side, and those on the left side will doubtless recover also later. So far as I know, bilateral destruction of the Gasserian ganglia has never previously been done.

Method of Injecting Alcohol.

As regards the methods of practising alcohol injection, details have already been published and need not here be repeated, though I would like to insist on the advisability of using the method of trial and test. That is to say, when the needle is thought to have struck the nerve inject a few drops of 2 per cent. novocain, and, after waiting half a minute, test the skin distribution of the nerve for anaesthesia. If none has developed do not inject alcohol, but try again to find the nerve. As soon as anaesthesia appears to novocain injection, then complete by injecting about 1 c.cm. of 90 per cent. alcohol, when total anaesthesia to touch, pin, and pressure will develop almost instantaneously if the nerve at the foramen ovale or foramen rotundum has been correctly hit.

Results.

In almost every case, as soon as anaesthesia develops from the injection, the neuralgic spasms cease, and they can no longer be started by any chewing movements or rubbing the face.

The duration of the cure rarely lasts less than twelve months, if good anaesthesia has been obtained, and in the majority the relief from pain lasts from two to three years. Very many of my cases have been quite free for four or five years, one six and a half years, another seven years, another nine years after nerve injection only, while I expect my cases of ganglion injection to be permanent cures, just as if gasserectomy had been done. No trophic

effects on the skin are ever seen. Keratitis is a risk if the cornea is anaesthetic after injection of the ganglion, but this may always be avoided by proper precautions.

THE SIGNIFICANCE OF ACIDOSIS IN CERTAIN NERVOUS DISORDERS.*

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ACIDOSIS, as evidenced by the presence of acetone bodies in urine, is frequent in cases of acute delirium, confusional and stuporose states, certain melancholic conditions, and in epilepsy.

The following notes are taken from two recent deaths resulting from extreme acidosis:

1. E. J., a mischievous, weak-minded boy of impulsive tendencies, aged 15, admitted in September, 1917. He was well nourished, and took his food very well indeed. He had had no fits previously. During the night of December 30th, 1919, he had a severe typical epileptic fit, and lapsed into a condition of cerebral irritation, with paresis of his legs and conjunctival injection. Specific gravity of urine 1024; no albumin or sugar, but strongly positive to acetone. He was put on alkaline treatment and improved, the acetonuria disappearing, and he continued to take his food well. On January 10th he again developed strong acetonuria, which did not yield to alkaline treatment, and he died at 9.40 a.m. on the 16th.

Post-mortem Examination.—Intense minute injection of the pia arachnoid in a patchy fashion, especially marked over sulci and along veins, one large patch extending over the vertex of the left hemisphere; numerous puncta cruenta on section of the cerebral tissue; strong acetone reaction in cerebro-spinal fluid and blood. Microscopical examination showed minute vascular engorgement, diffuse and central chromatolysis of the neurone body, and degenerative changes in nuclei—in other words, primary degeneration of the nerve cell. The suprarenal glands showed some congestion and fat deficiency; other organs apparently normal.

2. J. H., a well developed man, aged 49, admitted on February 4th in a state of acute confusion; had been ill for six weeks prior to admission. He was, on admission, in a state of restless noisy excitement, continually wet and dirty, rendering it impossible to collect his urine for examination. He was put on milk and other extra diet, which he took satisfactorily. He was extremely feeble when admitted, became gradually weaker, and died at 2.30 a.m. on the 10th.

Post-mortem Examination.—Cadaver in good condition, marked opacity and thickening of the pia arachnoid, with much recent minute injection and patchy ecchymotic areas. Brain tissue showed numerous puncta cruenta on section. Acetone reaction strongly marked in cerebro-spinal fluid, which was in excess in pericardial fluid and in the blood. Microscopic examination showed engorgement of vessels, with some minute extravasations, diffuse and central chromatolysis of cells, nuclear changes, and absence of pigment. Suprarenal glands softened, scanty fat; liver somewhat fatty; other organs normal.

In mental cases it is the exception to find *post mortem* a normal transparent pia arachnoid. Secondly, in long-standing cases of epilepsy almost the only change met with may be a thickening and opacity or milkiness of the pia arachnoid; this thickening and opacity I regard now as most probably due to congestive attacks resulting from acid intoxication. These congested areas are most pronounced over the vertex where the membrane is thickest and the underlying neurones mainly motor.

Thirdly, after death in status epilepticus there is intense congestion of the membranes and the cells show the profound structural changes of primary degeneration—structural changes which I have shown to be also present in the cases mentioned. It is of interest to note here that the meningeal appearances in these cases are very similar to many I have seen in the Balkans as a result of malignant malaria, and in which the symptoms indicated intense toxæmia, no doubt due to deficient oxidation as a result of the destruction of vast numbers of erythrocytes and also to the haemoglobin and oxyhaemoglobin set free in the plasma behaving as weak acids. Addison, Lusk, and Graham consider that the rise in heat production in severe anaemias is due to the pathological production of lactic acid from carbohydrates in consequence of an inadequate supply of oxygen to the cells. Recently I have had two cases of confusion following malignant malaria contracted at Salonica, one of whom had definite acidosis periodically while here.

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