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RARE FORMS OF PAROXYSMAL TRIGEMINAL NEURALGIA, AND THEIR RELATION TO DISSEMINATED SCLEROSIS

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Fifteen years ago I published in this *Journal* (Harris, 1935) an account of three cases of "Paroxysmal Neuralgic Tic as a Sequel of Trigeminal Neuritis," which were similar in their mode of onset and course but of very doubtful origin. I have since seen two similar cases, but as one of them became complicated later by the development of disseminated sclerosis the question arises whether this disease can have been the essential cause in the remainder or whether this complication was merely a coincidence. The fact that disseminated sclerosis is so frequently a complication of the common form of paroxysmal trigeminal neuralgia, or trigeminal tic as I prefer to call it, is an important reason for reconsidering the possibility of this disease being the essential cause in the five cases I have met with in which a paroxysmal facial neuralgia has later developed, perhaps several years after a complete temporary numbness with anaesthesia of the whole of one side of the face and tongue, usually with loss of the sense of taste on that side of the tongue. These five cases were seen in 1914, 1927, 1935, 1942, and 1945, and their rarity will be apparent when their number is compared with the total of 1,622 cases of trigeminal tic that I have met with in private practice alone, excluding hospital cases whose notes are unavailable. Of these, 64 were complicated by disseminated sclerosis, or nearly 4%.

In 1940 I published an analysis of 1,433 of my cases, including 377 hospital cases (Harris, 1940). Since then I have seen 566 fresh cases, the two series consisting of 1,318 women and 681 men, or very nearly twice as many women as men. These figures should be large enough to prove the much greater liability of the female sex to trigeminal tic.

Three Earlier Cases Recalled

The three cases of trigeminal neuritis which I described in 1935 may be briefly recalled regarding their essential features. They varied a good deal in their ages—two men, aged 65 and 75, and a woman of 42. In none of these three cases was there any symptom of disseminated sclerosis, though this was specially looked for. The first symptom in all was the sudden onset of numbness of the whole of one side of the face, including the tongue, with anaesthesia so complete that the cheek or lip was bitten; in one case loss of taste on that side was also noted. This numbness lasted in the two men, Cases 1 and 3, for six weeks and six months, followed after three years and seven years respectively by the onset of violent pain in the eye and cheek, in one of the men spreading into the

lower jaw. The pain was described as "a jumping pain, as quick as a flash of lightning—feels like a flaring fire," the pain gradually increasing in intensity until I saw them, when there was no trace of anaesthesia remaining. Case 1, whom I injected in 1914, required a second injection in 1919, and then remained free of all pain, with complete numbness and loss of taste on that side, until his death in 1930, aged 81. Case 3 was injected in March, 1935, and was still free of all pain, with numbness unaltered, when last heard from in August, 1939, aged 80. Case 2, the woman aged 42, was seen in 1932, when her trigeminal numbness gradually faded after lasting four years, and was then replaced by violent trigeminal tic. She was completely cured by Gasserian injection in August, 1932. She lives in Kenya, and to-day is still quite fit and well, 18 years afterwards, and has no sign of disseminated sclerosis, as her doctor brother wrote in August, 1950.

Later Cases

The two cases I have since met with of this curious type were both in women.

Case 4.—A married woman aged 47, whom I saw in March, 1942, gave the history that in 1916, when she was 21, she lost her husband and two brothers in the first world war, and almost at once "began to sleep on her feet," and would walk home in sleep from the village to her home. Within a few months sudden numbness developed over the whole of the right half of the face and tongue, with complete loss of taste on that side. This state lasted for a year, the numbness then passing off. Seven years later a sudden onset of violent neuralgia attacked the whole of the right side of the face "like needles, or a white-hot iron applied to the face." This varied at first so that she might be free for a year, but gradually the pain recurred more frequently until I saw her in 1942. For the previous six or seven years she had had increasing ataxia of the right arm and leg, with bladder weakness, which examination showed to be undoubtedly due to disseminated sclerosis. This disease therefore showed no clinical signs until 19 years after the commencement of the numbness of the face and 11 years after the onset of the trigeminal tic. I injected her right Gasserian ganglion the same day, producing total trigeminal anaesthesia, which was unchanged five days later, with cessation of the pain. A follow-up letter brought the reply, July 3, 1950: "No pain in the face; right limbs more useless; cannot get about."

Case 5.—An unmarried woman aged 62 saw me in September, 1945, on account of violent paroxysmal left facial neuralgia for the past three months, occurring frequently daily and mainly affecting the left lower jaw; she could scarcely eat or talk, or wash her face. She had been edentulous for the past 32 years. Twelve years previously, when aged 50, the whole

of the left side of her face became numbed one day in the summer, so that she could not feel her sister slap her face. This condition lasted for six weeks, and rather more than a year later trigeminal tic started in the left lower jaw and tongue, "like a needle in my face." This neuralgia lasted for two months, and then more or less disappeared, but not completely, for 11 years, when the severe type of tic returned. There was no anaesthesia of the face when I saw her, nor any sign of disseminated sclerosis. Gasserian injection, as in the previous four cases, completely cured her, producing total anaesthesia of the whole of the left trigeminal area. A follow-up letter in June, 1950, brought the reply that she was perfectly well, the numbness remained unaltered, and her limbs were strong, with no unsteadiness. In her case, therefore, disseminated sclerosis can be excluded.

Of these five cases of what I have called trigeminal neuritis one developed disseminated sclerosis 19 years after the appearance of the facial numbness and 11 years after the onset of the trigeminal tic. None of the remaining four cases have shown any sign of spinal disease, and in all of them numbness and anaesthesia of the face on one side was the initial symptom, and lasted a variable time from six weeks to four years, when trigeminal tic appeared after an interval varying from a few weeks to seven years. In every case the trigeminal tic was cured by Gasserian alcohol injection, just as in the common variety of trigeminal neuralgia or in those complicated by disseminated sclerosis.

The Question of Latent Disseminated Sclerosis

It is not uncommon for numbness and anaesthesia of one side of the face and tongue, including loss of taste, to develop as a transient phenomenon in the early stages of disseminated sclerosis (see Cases IV, V, VI in my paper: Harris, 1935), lasting a few weeks or even months, but none of these in my experience has afterwards developed the complication of trigeminal neuralgia; Cases IV and VI died in 1934 and 1947, and neither developed facial neuralgia. It is especially interesting that loss of taste occurred in these cases in addition to loss of pain and tactile sensation, the latter indicating a lesion of the nerve root, which has tactile and pressure fibres running to the principal sensory nucleus and the pain and temperature fibres which turn downwards into the spinal root of the fifth nerve. Such a lesion has been demonstrated post mortem in cases of disseminated sclerosis by Oppenheim (1911) and by Parker (1928), though clinical tests in their cases failed to demonstrate anaesthesia.

The path of the fibres carrying taste sensation is usually now agreed to be via the chorda tympani and the great superficial petrosal nerves, for the tongue and palate respectively, to the geniculate ganglion, and thence by the pars intermedia to join the glosso-pharyngeal fibres in the gustatory nucleus. Yet that this is not the whole story is proved by the demonstration of loss of taste in many clinical cases of fifth-nerve paralysis by competent observers (Gowers *et al.*, 1893), and I have been able to demonstrate immediate loss of taste on the front half of the tongue in dozens of cases of alcohol injection at the foramen ovale (Harris, 1926). Some of these recover the sense of taste after a few weeks or earlier; in others, even with total anaesthesia of the tongue, there may be no loss of taste whatever; and in others, again, in whom there is total and permanent anaesthesia from ganglion injection, the loss of taste may be permanent, as I have seen several times—in one case 24 years after the Gasserian injection.

That this gustatory loss is not produced by spread of the alcohol from the foramen ovale to the great superficial

petrosal nerve and geniculate ganglion is shown by the loss of taste noticed by the patients themselves in several cases of the spontaneous trigeminal anaesthesia met with in cases of disseminated sclerosis. For further discussion on this difficult subject of the gustatory pathway, see Harris (1921).

The close similarity in the attacks of unilateral numbness and anaesthesia of the face and tongue, with loss of taste, in cases which subsequently develop trigeminal tic (trigeminal neuritis), and other cases with precisely similar facial anaesthesia with loss of taste which may precede but usually follow the onset of clinical signs of disseminated sclerosis, but which may not develop trigeminal tic subsequently, is strikingly suggestive of a similar origin in both groups. In neither form is there any motor fifth palsy, and the sensory loss is very suggestive of a lesion of the trigeminal root comparable to the well-known retrobulbar ocular neuritis in disseminated sclerosis, in which the large, scotomata may disappear completely in certain cases after many weeks or months, or, as the motor pareses of the limbs may do, for many months or even years.

I have met with a few cases in which the trigeminal tic developed several years before any clinical signs of disseminated sclerosis appeared. In my paper on trigeminal neuritis I described Case VI, a man of 44 who developed temporary numbness of the right half of the face and of the left leg 18 years before the onset of symptoms of spinal disease suggestive of disseminated sclerosis. No trigeminal tic appeared in his case, and the crossed anaesthesia points almost certainly to a pontine lesion at the level of the sensory nucleus of the right fifth nerve, involving part of the median fillet on that side. He died completely paralysed in 1947, never having had facial neuralgia.

Of the two fresh cases I have described in this paper, it may be suggested that in Case 4 the sclerosis was already present but latent, a view which has been put forward by more than one writer. It raises the question whether in the remaining four of my cases disseminated sclerosis might be lying latent and originated the initial facial numbness and the subsequent neuralgia, in spite of the absence of any signs of spinal disease. I think it must be agreed that the histories of these four cases completely negative this supposition:

Case 1.—This man developed, at the age of 57, complete left facial anaesthesia lasting six weeks and followed two years later by left trigeminal tic. Final relief was given by Gasserian injection in 1919, when aged 70, and he died aged 81, having had no recurrence of pain or any sign of paralysis.

Case 2.—A woman aged 37 developed facial numbness which lasted four years, then trigeminal tic, for which she had Gasserian injection, in 1932, when aged 42. She is still quite well and strong 18 years after.

Case 3.—A man who developed right facial numbness at age 65. Trigeminal tic started seven years later, the numbness having lasted six months. Cured by Gasserian injection, aged 75. Still well and hearty five years later, aged 80.

Case 5.—A woman at the age of 50 developed left facial numbness, lasting six weeks, followed about a year later by trigeminal tic. This was cured at age 62 by Gasserian injection, in 1945. She is still (1950) perfectly well and strong, and the numbness is unaltered.

Disseminated sclerosis may surely be excluded in all these four cases. In Case 4, in which disseminated sclerosis developed 19 years after the onset of the facial numbness and 11 years after the trigeminal tic appeared, it is possible that the spinal disease may be coincidental, and that it has no connexion with the antecedent attack of trigeminal neuritis and subsequent tic.

Classification

The cases of trigeminal neuritis and other cases of neuritis and neuralgia complicating disseminated sclerosis may be divided into six groups:

1. Trigeminal neuritis, a rare form in which paroxysmal trigeminal neuralgia follows weeks, months, or years after a sudden development of temporary numbness and anaesthesia of one side of the face, with no complication of disseminated sclerosis.
2. Cases of disseminated sclerosis which develop trigeminal tic without antecedent numbness. This is the common type, of which I have met with 64 examples. Usually the spinal symptoms antedate the neuralgia, but occasionally the neuralgic tic may precede the spinal symptoms by several years.
3. Cases apparently similar to Group 1—trigeminal neuritis—but in which disseminated sclerosis develops years later, as in Case 4. Is the spinal disease merely coincidental, or is the antecedent trigeminal neuritis dependent on a latent sclerotic patch of disseminated sclerosis?
4. Cases of disseminated sclerosis in which numbness and facial anaesthesia may appear for weeks or months, with loss of taste on the same side, but no subsequent development of trigeminal tic, as in cases IV, V, and VI in my 1935 paper.
5. Cases of disseminated sclerosis in which numbness and facial anaesthesia on one side may be accompanied by numbness or paresis of the opposite arm or leg, suggestive of a lesion of the median fillet at the same time as of the sensory fifth nucleus.
6. Bilateral trigeminal tic is much commoner in proportion in cases of disseminated sclerosis than in uncomplicated cases of the neuralgia. This is suggestive of the scattered pontine patches of sclerosis being apt to irritate the spinal trigeminal roots on both sides.

The type of facial numbness, often with loss of taste, and its subsequent disappearance, appears to be identical in all these cases, and it seems highly probable that the lesion producing the numbness and facial anaesthesia is similar in all. It is probably a patch of neuritis on the root of the fifth nerve at its junction with the pons, a lesion which has been demonstrated at necropsy. This may leave the nerve fibres in an irritable state when the congestion and the anaesthesia disappear. This irritability, combined with sensory pain stimuli from the peripheral nerve branches in the jaws, may precipitate the onset of trigeminal tic (cases of trigeminal neuritis). In cases of disseminated sclerosis the sclerotic pontine patches may similarly act as an irritant to the pain fibres in the spinal root, and, aided by peripheral jaw painful stimuli, trigeminal tic may start. That the source of the stimuli is peripheral, not central, is definitely shown by the fact that in cases of trigeminal neuritis the pain does not begin until the anaesthetic block completely disappears. I have for many years maintained that in the common form of paroxysmal trigeminal neuralgia the source of the pain is in the peripheral nerve fibres in the jaws. There are many arguments to prove this thesis. (1) The anatomical distribution of the pain, and the absence of any anaesthesia or other evidence of lesion of the nerve trunks or Gasserian ganglion. (2) The frequency of cases of severe damage to a jaw by trauma, fracture, or severe dental extractions, followed shortly after by typical trigeminal tic. For example, a woman had a severe molar extraction, the dentist breaking his forceps and leaving a piece of steel in the jaw. Trigeminal tic started within three weeks. Fifteen years afterwards radiographs showed the piece of steel in the jaw. The following is perhaps a better argument: (3) The fact that an anaesthetic block by injection or nerve section instantly arrests the neuralgia, though neuralgic pain due to irritation of the fifth nerve intracranially by a basal tumour will continue in spite of posterior-nerve-root section in front of the lesion.

Anaesthesia of the Third Division

I would like to record here a curious fact that I have noticed during the 40 years or so that I have practised alcoholic trigeminal injection at the foramen ovale. It is that occasionally I have found it exceedingly difficult, and sometimes impossible, to obtain satisfactory anaesthesia of the third division, supplying the lower jaw and gum, tongue, and lower lip. In some of these cases it has been easy to obtain total anaesthesia of the ophthalmic and maxillary divisions of the Gasserian ganglion, and yet repeated attempts to inject the mandibular division may completely or partially fail, and the neuralgia may continue. Were it not that I have known this same difficulty confront six first-class neurosurgeons when operating for posterior-sensory-root resection, I might be content to ascribe my few failures to my own incompetence. One such case was operated on many years ago by the late Sir Percy Sargent and also by Mr. Wilfred Trotter, after I had failed with injection; but the neuralgia continued, and the lower lip, chin, tongue, and lower gum remained sensitive.

The following case is perhaps even more instructive:

A married woman aged 38 was seen by me in April, 1928, with the following history. In 1919, when aged 29, four teeth were extracted from the left lower jaw under a general anaesthetic, the jaw being fractured, as shown by radiographs, and the left inferior dental nerve damaged. Pain continued as a constant ache, and then sharp sudden jabs shooting along the mandible, brought on by eating or by moving the tongue or jaw—clearly a development of true trigeminal tic. Small sequestra continued to come away from the wound in the gum, and as the neuralgia continued she was injected with alcohol by the late James Collier, but no increase of the partial anaesthesia of the lip or gum was produced. She felt better afterwards, and the spasms of pain became less violent; they gradually disappeared during 1920, and she was free for 18 months, as is common in the early history of trigeminal tic. In 1922 she had a recurrence of pain for three months, and was free for six years until 1928, when I saw her. There was then very slight anaesthesia of the chin, lip, and tongue, and I injected her Gasserian ganglion through the foramen ovale in July, getting total anaesthesia of the two upper divisions but no alteration of V3, and the pain persisted. I then sent her to Mr. Hugh Cairns, at the London Hospital (he was not then Sir Hugh). He told me afterwards that at the operation he found the dural sheath adherent, and could not open the arachnoid sheath, but he divided the sensory root so far as possible. Probably the adhesions were the result of the alcohol injections. No further anaesthesia resulted, however, and the pain continued for a time, but she had a long intermission of freedom for 14 years from 1929 to 1943, when in December the neuralgia again became severe. I admitted her to Maida Vale Hospital in March, 1944, and again tried to inject the third division through the foramen ovale, by both lateral and anterior routes, but entirely failed to increase the slight anaesthesia. I then sent her to Mr. Wylie McKissock, at the Atkinson Morley Hospital. He operated through the posterior fossa, dividing the fifth root close to the pons. The upper two divisions had been totally anaesthetic since my injection in July, 1928, but no increase of the anaesthesia of the third division resulted from this last operation, and the pain continued as before when she returned to Maida Vale in September, and persisted unaltered until her death in December, 1946.

I have given the names of the surgeons, who did their best to help me, so that there may be no doubt that the operations were done with the utmost skill; but how to explain the escape of the mandibular division from its numerous medical and surgical assaults is beyond me. It is, however, a further illustration of my thesis that occasionally the mandibular nerve is extremely difficult, if not impossible, to destroy.

Medullary Thrombosis

A fairly well known and interesting syndrome is that produced by thrombosis of one of the posterior inferior cerebellar arteries. This vessel, the largest branch of the vertebral artery, which is the first branch given off from the subclavian, winds backwards round the upper part of the medulla between the vagus and spinal accessory nerves, and over the restiform body to the under surface of the cerebellum, where it divides into two branches, external and internal. The latter supplies the middle lobe of the cerebellum and the side of the medulla. Hence thrombosis of this vessel will involve the spinal descending root of the trigeminal nerve (uncrossed) and the spinothalamic tract (crossed) ascending to the thalamus, causing analgesia of the face on the same side and of the trunk and limbs on the opposite side. Paraesthesiae and pain may occur in both these areas, and occasionally the facial neuralgia may be severe and shooting in character, resembling tabetic pains. It is not affected by light touches or by movements of the face, as in eating or talking, as is the case in true trigeminal tic, nor is this form of neuralgia amenable to Gasserian injection. Vertigo and vomiting and nystagmus may be early symptoms, with incoordination of the arm and leg on the same side and unsteady gait due to the cerebellar infarction.

Pontine Thrombosis

Thrombosis higher up in the pons, above the level of the decussation of the pyramids and of the ascending sensory tracts forming the median fillet, is apt to cause hemianaesthesia of the face, limbs, and trunk on the opposite side. The following case suggests the effect of a small pontine lesion involving the trigeminal fillet more than the spino-thalamic fibres, while the main sensory fillet close by must have escaped.

Case 6.—A woman aged 43 was sent to me in January, 1944, with a history of sudden onset of pain ten years before in the left upper jaw, across the cheek, down the mandible, and into the tongue, with pain in the left hand, the left fingers being contracted. The face felt at first as if on fire, and facial neuralgia has continued at intervals ever since, though the pain in the left hand faded and disappeared after four years. The facial neuralgia continued mainly in the left cheek, jaw, and side of nose, with momentary sharp shoots in the left lower jaw many times daily and occasionally at night. Two years later the neuralgia was limited entirely to the cheek and side of nose. There was no objective sensory loss to touch or pin-prick anywhere, and no motor weakness, though the left forearm jerk was slightly increased. The plantar reflexes were flexor.

This case was sent to me as trigeminal neuralgia for Gasserian injection, but as investigation showed the lesion to be intrapontine, and localized to the neighbourhood of the right median fillet, no alcohol injection was indicated. The type of pain was similar to that met with after posterior inferior cerebellar thrombosis, and differed from the pains of trigeminal tic in not being started by the movements of eating and talking or by light touches.

The neuralgic pains in these medullary lesions resemble tabetic pains somewhat closely, and the sclerotic lesions of the posterior columns and roots in tabes may be compared with the sclerotic lesions of intramedullary and intrapontine fillet thrombosis.

Paroxysmal Supraorbital Neuralgia, Spontaneous and Post-traumatic

Under this heading I do not wish to include the sinus headaches of antral and frontal infections, migrainous neuralgias, or the post-traumatic dural headaches following concussion and subdural haemorrhage.

Case 7.—A man when aged 49 received a severe blow on his right eye from a squash ball which made him "see stars" badly. About three weeks later, when driving his car, he was attacked by sudden violent pain in the right eye, and had to stop, the pain lasting an hour. There was no congestion of the eye, no lacrimation, and no vomiting. Similar attacks recurred for a few weeks until he was fitted with glasses for reading. Then he was quite free for four years, when similar intermittent attacks recurred, two or three daily, lasting about an hour, for three or four weeks. Another intermission for two years occurred until three weeks before I saw him in December, 1946. He was having much domestic worry at that time, to which he ascribed the return of the pain, although the seizures were quite sudden. Partial inner Gasserian injection, causing analgesia of the two upper trigeminal areas, cured the paroxysmal attacks, and he has been free for the last three and a half years, except for some constant aching in the right eye lately. This is possibly aggravated by heavy business work and family worry, and also by the strain of driving daily 46 miles to and from business.

The diagnosis of the cause of these sudden attacks of violent pain is difficult. They closely resemble post-traumatic ciliary neuralgia, but without the ocular congestion and lacrimation usual in that condition. The pain is referred to the eyeball rather than to the supraorbital area, and as nothing wrong can be found with the eye I class it as a post-traumatic ciliary type.

Paroxysmal Trigeminal Tic Affecting the Supraorbital Area Only

Case 8.—A man aged 54 was sent to me a year ago on account of violent neuralgia which had been recurring in spasms for the past seven weeks. His first attack had occurred 23 years ago, at the age of 31, the pain starting suddenly at the inner canthus of the left eye and shooting up over the forehead. These spasms, lasting 10 to 15 minutes, recurred at intervals for the following fortnight, and since then at intervals of four or five years. He describes the pain as being hot, "like a firework," and almost unbearable, and for seven weeks it had been worse than ever. I realized this was a true case of trigeminal tic affecting only the ophthalmic division, although it has persisted in that division until the present time, the longest period I have known this type to be limited to the first division without invading the maxillary area. Usually after three or four years or less this spasmodic neuralgia spreads to the second division, the pain running down the side of the nose and into the upper jaw and cheek to the upper lip. The patient would not agree to a Gasserian injection, and I therefore gave a local procaine-and-alcohol injection to the supraorbital notch in May, 1949. This caused only partial numbness, the nerve not having been well hit, and the spasms continued for another two weeks, then disappearing until June, 1950, when violent spasms of pain recurred as before. He again asked for a supraorbital injection rather than Gasserian, and this time I obtained total anaesthesia of the left half of the forehead, eyelid, and scalp as high as the vertex. The painful spasms, however, persisted for several days unless he kept perfectly still in bed, light touches on the left side of the face starting off the pain, and he now admitted that the left upper lip was sensitive, a touch on it starting the supraorbital pain. This proved at long last that the second division was becoming involved.

Although it is quite common for the first onset of pain in trigeminal tic to involve both the ophthalmic and second divisions of the nerve at the same time, it is rare for this type of paroxysmal neuralgia to affect the supraorbital area or the eye alone. When this is the case I have known it continue thus for three or four years, and even once for nine years, but this last case, in which the neuralgia was limited to the inner canthus of the eye and the supraorbital area for 23 years before showing signs of involvement of the second division, is unique in my experience. At the same time it should be noted that total anaesthesia of the

supraorbital nerve area did little or nothing to relieve the pain, indicating that the source of the neuralgia was deeper and would require an inner Gasserian alcohol injection or a fractional section of the inner half of the sensory root to cure it.

Glossopharyngeal tic, though very much rarer than the trigeminal form, perhaps in the order of 1 to 100 cases, is now better known since I first described it (Harris, 1921). It is still often confounded with trigeminal tic or with otic or geniculate neuralgia, but its definite characteristics should be sufficient to distinguish it. The pain is usually felt on swallowing, and often runs into the ear, which may become so sensitive that the patient will leave the ear for years unwashed. Touches on the tonsil will often start the pain, which is referred to the throat and ear and to the back of the cheek in front of the ear. The painful paroxysms recall the trigeminal type, and the only cure is avulsion of the nerve.

The nerve may be irritated by a basal tumour, and trigeminal tic may also be closely simulated by intracranial pressure from an acoustic neuroma or other basal tumour, such as an intracranial aneurysm of the internal carotid artery.

Summary

Five cases of a form of trigeminal neuritis are described in which paroxysmal trigeminal tic follows at varying intervals after a period of total unilateral facial numbness.

The question whether the cases of trigeminal neuritis described are due to latent disseminated sclerosis is considered disproved.

Paroxysmal trigeminal tic occurs in a considerable proportion of cases of disseminated sclerosis, without previous numbness and anaesthesia of the face.

In many cases of disseminated sclerosis temporary numbness of one side of the face may occur, with anaesthesia and loss of taste on that side. These cases do not later develop paroxysmal trigeminal tic.

Loss of taste, either temporary or permanent, on the front of the tongue is commonly found in unilateral fifth-nerve paralysis in addition to the spontaneous cases of numbness in trigeminal neuritis and in disseminated sclerosis.

Analysis of 2,000 cases of trigeminal tic shows that this neuralgia occurs about twice as often in women as in men.

The lesion producing the numbness and anaesthesia of the face in trigeminal neuritis is probably a root neuritis of the fifth nerve close to or just inside the pons.

The frequency of trigeminal tic, unilateral and bilateral, in disseminated sclerosis is almost certainly due to local irritation of the trigeminal spinal root by the diffuse patches of sclerosis in the pons.

Rare forms of trigeminal pain due to medullary or pontine thrombotic lesions are described.

An occasional difficulty—even impossibility—in producing total anaesthesia and cure of third-division neuralgia, either by injection or by surgical root resection, is described.

Reasons are given for ascribing the origin of trigeminal tic to peripheral causes in the end-nerve filaments in the jaws.

Paroxysmal trigeminal tic of the ophthalmic division is compared with post-traumatic supraorbital pain resembling ciliary neuralgia.

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PROLONGED TREATMENT OF RHEUMATOID ARTHRITIS WITH A.C.T.H. ALONE AND WITH GOLD*

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From the first publications of Hench *et al.* (1949) and Thorn *et al.* (1949) the following conclusions can be drawn: (1) adrenocorticotrophic hormone (A.C.T.H.) has a very favourable effect on the symptoms of rheumatoid arthritis, (2) the symptoms reappear when the administration of A.C.T.H. is discontinued, and (3) with the dosage used untoward reactions appear. When, in October, 1949, a certain quantity of A.C.T.H. ("cortrophine," Organon†) was put at our disposal, we set ourselves to search for a maintenance dose which would keep the rheumatic process under control but cause no untoward reactions; and to inactivate the rheumatic process with A.C.T.H., and during its administration to begin with one of the approved medical therapies, in the hope that no relapse would then occur after discontinuation of the A.C.T.H. injections.

Here, therefore, we concern ourselves exclusively with clinical application. The theoretical bases and the pathophysiological and chemical problems of A.C.T.H. therapy have been dealt with in the comprehensive survey with extensive bibliography of Boland (1950).

Plan of Study

Three women and two men with rheumatoid arthritis, and one man with ankylosing spondylitis, were selected for treatment. The diagnosis was based on the results of the clinical data, biopsies, x-ray photographs, serology, and other non-specific data such as sedimentation rate (Westergren); haemoglobin, etc. In view of the fact that we were striving to find an effective treatment for rheumatoid arthritis, complete rest in bed was prescribed for all the patients for a fortnight before and after the beginning of the A.C.T.H. treatment. This was followed by a period of graded re-educative remedial exercises and mobilization. Spectacular exertions, of which some patients were already capable after a few days, were thus intentionally avoided.

During the pre-treatment period a thorough physical examination was carried out, with special documentation of the condition of the joints (ergometry, measuring, photographing, and filming of the swellings and of limitations of function). Special laboratory investigations were made of kidney function (concentration, phenol red, urea clearance), liver function (galactose-tolerance test, van den Bergh reaction, prothrombin index, cholesterol

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