

Papers and Originals

Trigeminal Neuralgia: the Pain and its Treatment*

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Trigeminal neuralgia is curable and it does not shorten life. The spasmodic pain often becomes extremely severe and makes old age miserable, but fortunately it is always stopped by denervating the affected area. Permanent cure can be guaranteed by cutting the sensory root or by injecting alcohol into the whole of the "Gasserian ganglion" (sensory root). Pain rarely occurs, however, in all three divisions, so that sensory loss can be reduced by cutting only part of the sensory root; selective injection of only part of the ganglion (Harris, 1940) is too difficult and uncertain. Sensory loss can also be restricted by injecting peripheral divisions, but the relief of pain is usually only temporary, for a year or two. Still more conservative treatment by drugs is often tried, but their effect is uncertain and they often fail. One disadvantage of routine peripheral injections or drugs is that temporary success postpones radical cure, which may become necessary later when the patient is old and unfit for operation.

In spite of the possibility of permanent cure, many patients continue unnecessarily to suffer pain, or live in fear of its sudden return, and occasionally they even contemplate suicide. This might happen because operation or Gasserian injection failed to give permanent relief; or because of procrastination that is encouraged by remissions, by hearsay frightening reports, or by persisting misconceptions about complications (which now are very rare); or because treatment is limited mainly to one procedure without making full use of alternative methods, especially operation, which could with benefit be used more often than it is. Despair should never be allowed to develop when dealing with such a curable pain, and it is a great pity when patients refuse further treatment and are resigned to "suffer the pain."

This paper is based on 650 patients treated during 1946-63. The general policy was to produce early permanent cure by fractional sensory root section operation in preference to total Gasserian injection. Many patients had peripheral injection when temporary relief was urgently necessary or when it seemed sufficient. Drugs were sometimes tried. The late results after 1 to 17 years, including re-examination of sensation to evaluate anatomical results, are known in 323 patients who were recently interviewed, and also in other patients, now dead or untraced, who were similarly re-examined in earlier years. Many patients, cured 10 to 15 years previously, had forgotten the pain, while, on the other hand, a few disappointed patients refused to be seen; several patients agreed to further treatment, which, after the lessons of this follow-up, was successful.

The fact that pain sometimes returns naturally gives rise to doubts about the likelihood of permanent cure. An analysis of the causes of failure is therefore more important than the recording of permanent successes, the possibilities of which have been known since the first successful sensory-root operation in 1901 (Frazier and Spiller, 1901), and since the first Gasserian injection in 1910, after which the patient lived 27

years without pain (Harris, 1912, 1940). The follow-up and subsequent anatomical investigations showed that, apart from lack of skill, there were two main causes of failure: (1) the anatomical behaviour of the pain was not at first fully appreciated and allowed for, so that sometimes an inadequate or wrong type of fractional operation was used; and (2) anatomical factors concerning the Gasserian ganglion discovered during post-mortem dissections and injections that were carried out to find reasons for technical failures and certain unexpected late results (Henderson, 1965, 1966). As regards terminology, the old term "Gasserian" injection, which is vague about including the sensory root and is really a misnomer, is retained, but it is understood that permanent total sensory loss is an effect of alcohol on the sensory root inside Meckel's cave and not on the band of nerve cells, or ganglion proper, which is too thin for injection without affecting also the sensory root.

Some of the less-known features of the pain, particularly about its distribution, that are important in early diagnosis and in deciding treatment, and possibly also in relation to causation, are described, followed by a survey of the results of operations and injections with emphasis on the reasons for failures, many of which it is now realized could have been avoided.

The Pain

Description and Frequency.—The pain is usually described as stabbing, shooting, cutting, or like red-hot needles or electric shocks; and mild pain as tingling, pins-and-needles, an "alive" sensation, or aching, the cause of which is suspected when it is of brief duration and is confined to a typical focus. It occurs in spasms that begin with lightning suddenness and last only seconds, and are sometimes followed for a few minutes by a mild aching or burning. A series of rapidly recurring spasms may be described as continuous pain, lasting an hour or longer, and so cause doubt about the diagnosis. The typical history is of periodic attacks, consisting in spasms during several days or weeks, followed by complete remissions of several months or even years. The extremes of history are of pain recurring almost daily for years, or of a few isolated stabs followed by long remissions of several years. The pattern or natural history of attacks and remissions, in conjunction with age, is important when deciding treatment.

Precipitating Causes.—Spasms either occur spontaneously or are precipitated by trigeminal stimuli or distant nervous reflexes. Reflex causes may be unrecognized, and their elucidation sometimes requires careful questioning, especially in senile patients. They are of several kinds:

1. Light touch or tickle in certain localized areas, chiefly the upper lip and ala, the eyebrow, and the gum. Cutaneous triggers may precipitate spasms in another division, but they are usually restricted to the same zone (see below). Distant trigger points sometimes confuse the patient about the actual site of pain, which, before the distinction is explained, may be referred to the wrong

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division and is said to be in the skin (e.g., upper-gum pain induced by touching the eyebrow).

2. Facial movements during eating, talking, or yawning, probably by proprioceptive impulses from contracting muscles because touch may be ineffective.

3. Occasionally other proprioceptive impulses (e.g., orbital pain during eye movements or turning the head).

4. Rarely, auditory stimuli (e.g., high musical notes, a loud noise, or vibration).

5. A general nervous reflex from a sudden unexpected stimulus such as a false step.

6. Attacks sometimes start during an acute emotional disturbance or a debilitating or acute infective illness and rarely after trauma.

Distribution of Pain

It is important to establish the exact origin and spread of the pain. It starts in certain situations or foci and it spreads in certain directions; it does not occur anywhere, and it never affects diffusely the whole territory of the nerve or even of one division. The pain is nearly always deep, and is not in the skin except sometimes in the upper lip, in the eyebrow and adjoining part of the forehead, and in the eyelids.

It became evident that the pain should be regarded as occurring not in nerve divisions, even though it may be confined to one division, but in one of *two zones*, which may be called the mouth-ear zone (Fig. 1) and the nose-orbit zone (Fig. 2). The mouth-ear zone (62%) was affected twice as often (Table I) as the nose-orbit zone (33%); the remaining 5% of patients could not be grouped. This grouping into two zones is supported by the rarity of pain in all three divisions in reported series, and in this series by the rarity of later involvement of the other zone after fractional treatment. The foci within a zone are closely related, and, however localized the pain, the possibility of its later extension inside the zone should be (but was not fully) anticipated.

TABLE I.—*Situation of Principal Pain Foci in 650 Cases*

Nerve Division		Zone	
I	Orbit	77	Nose-orbit .. . 33%
II	Gum-nose .. .	140	
II	Canine .. .	77	
II	Gum-ear .. .	97	Mouth-ear .. . 62%
II and III	Gums .. .	77	
III	Gum or tongue .. .	152	
II	Ear .. .	74	
		217	
		400	

Table I shows the frequency of the principal foci in relation to nerve divisions and zones. Pain started in the upper gum in about 50% of patients, who are grouped according to whether the pain later spread upwards to the orbit (140) or backwards towards the ear and perhaps downwards to the third division (97), leaving 33 patients in whom the pain was still confined to the canine region, or its line of spread was insufficiently described. In addition, 77 patients when first seen had foci in both gums (in approximately half the pain started simultaneously in both gums, and in the remainder, equally, in one gum before the other).

Mouth-Ear Zone (Fig. 1)

Pain in the lower gum began usually in the canine or premolar region, or occasionally in the molar region, but never at the incisor teeth. There might also be simultaneous tingling or pain along the side of the tongue, or at the ear, at first without connecting pain, or in the upper gum, directly opposite and "in sympathy" with the main focus in the lower. Pain might shoot from the gum to the ear and eventually in both directions. Pain did not shoot from the lower canine focus straight across to the upper, but sometimes it went backwards along the lower gum, curving upwards and then forwards to the upper-gum focus.

Pain in the upper gum similarly began in the canine-premolar, or occasionally the molar region, and followed, in reverse, the same pattern as lower-gum pain (i.e., with subsidiary foci at the ear, in the tongue, or in the lower gum, and with spasms shooting backwards to the ear, or turning downwards and forwards along the lower gum).

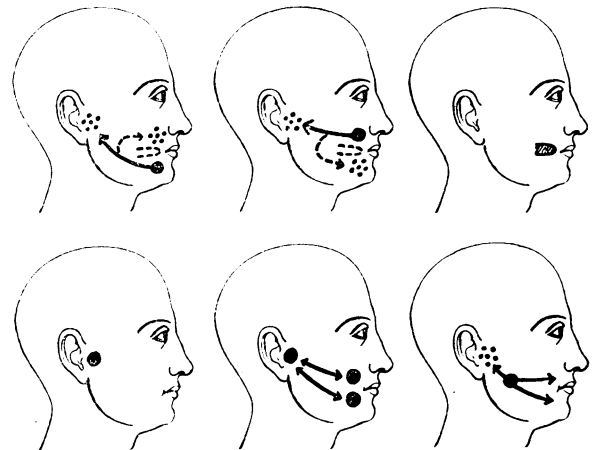


FIG. 1.—Mouth-ear zone, showing principal foci and lines of spread, and subsidiary foci (dotted). The top row shows pain starting in the lower gum, in the upper gum, and confined to the tongue. The bottom row shows pain confined to the depths of the ear, shooting along one or both gums, and starting at the hinge focus between the jaws.

Rarely, in nine patients, pain first occurred only along the side of the tongue, where it remained confined for several months or years before spreading to upper or lower gum and the ear.

Pain at the ear was always deep in front of the tragus or inside the ear; it was not superficial. In two patients it was still confined to the depth of the ear when treated four and six years after the onset (Case 2), but usually it spread forwards into upper or lower gum, or both, and into the tongue; and there might be subsidiary foci anteriorly, with pain shooting both ways along the gums. It never spread to the first division. The deep ear focus thus seems to be related to both second and third divisions, though the trigeminal supply to the depth of the ear is from the third division only.

A few patients described a fifth focus. They had difficulty in locating the pain to any structure, and referred it to the "space deeply between the gums," opposite the anterior border of the masseter (sometimes indicated as the cheek), which several called the "hinge between the jaws"; it may be called the hinge focus. It was the only focus in 13 patients with pain shooting forwards along the gums or backwards and upwards to the ear. Occasionally it was an intermediate stage between the ear and canine foci, or, rarely, the site of displaced pain after infraorbital or mandibular injection; also it seemed likely that pain clearly described as shooting backwards in an arc between upper and lower canines foci passed through this focus.

Pain shooting backwards along either gum sometimes seemed to continue upwards in front of the ear towards the temple; and, rarely, pain started on the zygomatic arch, just in front of and above the ear focus, to which it possibly belonged, and went downwards and forwards along either gum.

In this zone spasms were commonly induced by movements of the mouth, but cutaneous trigger stimuli were only occasionally recorded.

Nose-Orbit Zone (Fig. 2)

Pain started in the upper gum twice as often as around the orbit. At the orbit foci were in the eyebrow (often at the

supraorbital notch); at the medial canthus; at the lateral canthus, with pain shooting always medially above, below, or behind the eye; and, very rarely, in the eyelids. The pain was sometimes described as around and behind the "eye socket," where it may remain localized, but often it went downwards to the front of the upper gum (never to the posterior part of the gum, to the ear, or to the third division); it did not seem to occur in the eyeball, which was said to be surrounded by or "in" the pain. In one patient pain occurred only along the upper eyelid, causing a sensation of electric pins-and-needles in the eyelashes, but during occasional severe spasms it also occurred around the eye as if it was being "gouged out"; this description was used by several patients with pain confined to the orbit. Pain at the orbit thus seems, like pain at the ear, to be related to two divisions.

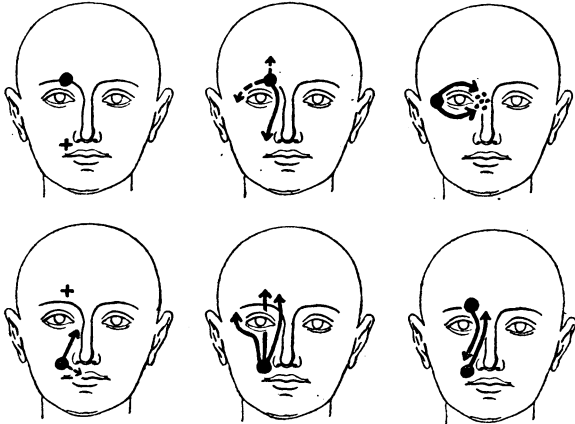


FIG. 2.—Nose-orbit zone. Top row shows pain confined to the eyebrow, with cutaneous trigger (+) on the upper lip, shooting mainly downwards to the upper gum or to the lateral canthus, and at the lateral canthus shooting medially around the eye. Bottom row shows pain starting at the incisor-canine gum and shooting upwards to the orbit and occasionally into the upper lip; shooting upwards past the medial canthus or behind the eye or around the lateral canthus; and shooting upwards and downwards deeply along the junction of nose and cheek.

More often pain started in the upper gum, at the canine or lateral incisor tooth, and observant patients often localized it precisely to the space between these teeth, which is the border of the premaxilla (pain elsewhere in the gums was not localized to a space between teeth). Rarely, pain also occurred on the front of the palate or appeared to shoot medially from the gum into the upper lip. Pain often went upwards, deeply along the junction of the nose and cheek, and then continued either around the inner canthus towards the forehead, but usually curving laterally above the eye, or upwards behind the eyeball, or it turned laterally below the eye and curved upwards to the lateral canthus (the last route was sometimes indicated vaguely from the canine tooth laterally and upwards to the anterior temple, and it was formerly regarded, incorrectly, as second-division pain not related to nose and orbit); pain sometimes went around each canthus alternately.

Canine Region of Upper Gum

This is the only place where the affected parts of the two zones are close together. When pain occurs only at that region it is important to look for distant trigger spots, which are common only around the orbit, but in order to be certain which zone is affected it is usually necessary to wait for the appearance of other foci (at the orbit, ear, or lower gum) or until the pain spreads definitely upwards alongside the nose or backwards along the gum; in the meantime severe pain should be treated temporarily by infraorbital injection. Patients sometimes locate the pain definitely to just in front of the canine tooth, or behind it, and this usually corresponds with the future spread of pain,

but it should not be relied on in deciding the type of fractional operation. Less observant patients locate the pain vaguely to the "eye-tooth," and it is possible that this frequent site comprises two adjoining foci (Fig. 3). The front of the upper

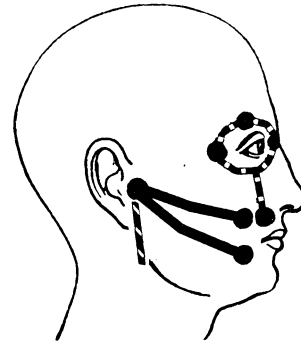


FIG. 3.—Principal sites of trigeminal neuralgia in the nose-orbit and mouth-ear zones; and of glossopharyngeal neuralgia.

gum, ala, and upper lip (i.e., the premaxillary region) is the most active area of the trigeminal territory as regards frequency of pain and of cutaneous triggers.

Displaced Pain

Pain may always remain confined to one focus, or it may migrate after months or years from one focus to another within the same zone, or it may reappear at different foci in rotation. A phenomenon analogous to spontaneous migration of pain sometimes occurred after local denervation of a single focus when the pain was immediately displaced along the usual line of spread to the adjoining area of normal sensation, or further to the next focus, but never to the other zone. Displacement of pain sometimes caused failure after operation or injection.

After 19 infraorbital injections that anaesthetized only the front half of the gum, pain in the mouth-ear zone stopped at the canine focus but immediately appeared where sensation was normal at the premolar-molar region, or further back at the hinge focus. Patients recognized this immediate shift of pain, and, later, its return to the original focus when sensation recovered after several months. Displaced pain was usually mild, and sometimes stopped. Canine pain in the nose-orbit zone (Fig. 4) was displaced upwards to behind the eye after

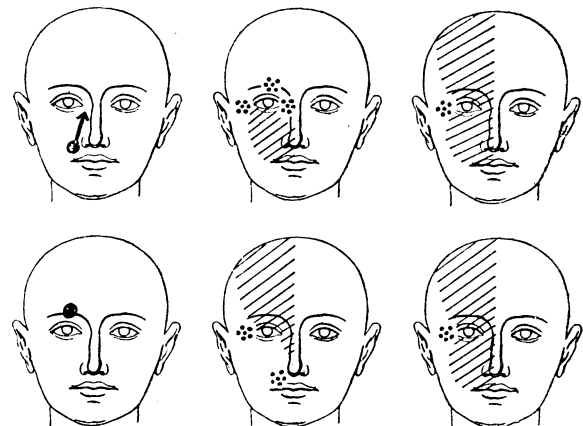


FIG. 4.—Displaced pain in the nose-orbit zone. In the top row, pain starting in the upper gum is stopped by infraorbital injection but starts at the orbit; supraorbital neurectomy stops pain at the eyebrow or medial canthus but not at the lateral canthus. Similarly, in the bottom row, pain in the eyebrow is displaced after supraorbital neurectomy either to the upper gum, where it is abolished by infraorbital injection, or to the lateral canthus.

five infraorbital injections, to the lateral canthus after four, to the eyebrow after four, or to the medial canthus after four (also after three Gasserian injections pain appeared where sensation remained at the medial canthus).

Supraorbital neurectomy (Fig. 4) displaced central eyebrow pain to the lateral canthus in six patients (also after three inadequate medial fractional section operations), to the medial canthus when the infratrochlear nerve was missed in two patients (also after one Gasserian injection), or downwards to the upper gum in four patients. Pain displaced to the upper gum was always abolished by infraorbital injection and was not displaced backwards along the gum, but pain at the lateral canthus cannot be stopped without medial section operation, or Gasserian injection, that produces sensory loss in the first two divisions, including the zygomatic branches of the maxillary nerve.

Mandibular injections were rarely followed by the appearance of pain at the hinge focus or in the upper gum.

Double-zone Pain

In about 95% of patients pain, however severe, remained confined to one zone, leaving either the first or third division unaffected. Shooting pain never definitely went from one zone to the other, but in 30 patients pain later started independently in the other zone. In eight other patients involvement of the other zone was doubtful because of uncertainty about the exact site of pain or its direction of spread. Pain started in the second zone usually within six years; the interval was less than one year in two patients, and longer than 15 years in two patients (20 and 24 years). Four of the 30 patients also had pain on the other side of the face and one had glossopharyngeal neuralgia.

The incidence of double-zone pain remained steady at 4 to 6% for the 224 patients last seen at 10 years, the 112 patients at 15 years, and the 56 patients at 20 to 45 years after onset of pain (and in whom treatment had not denervated the unaffected first or third division). Thus, even after many years, pain, in spite of increasing severity, is unlikely to spread beyond the confines of two divisions. The pain should be regarded as bigeminal and not trigeminal, and when advising fractional operation it may reasonably be accepted that in the rare event of pain appearing later in the other zone it will be treated separately.

Bilateral Pain

Pain occurred on both sides of the face in 32 patients (5%). Bilateral pain usually affected corresponding zones and often it was symmetrical. Symmetrical pain occurred more often in the mouth zone (18 patients), especially in the mandibular division, than in the nose-orbit zone (four patients), so that it was often impossible to treat the second side by full lateral section operation because of making the mouth anaesthetic, which is a great disability. In three patients symmetrical pain was localized to the canine region of the upper gum. In seven patients pain affected different zones, which made treatment easier. Four patients had double-zone pain on one side; they were the most difficult patients to treat. No patient had pain in all four zones. Seven of the 32 patients had disseminated sclerosis.

Pain started on the second side two to ten years after the first in 22 patients, in less than one year in three (in one patient on both sides simultaneously), and in three patients there were long intervals of 21, 24, and 30 years. Pain was already bilateral in 19 patients when first seen.

Several patients with pain in both mouth-ear and nose-orbit zones, whether on the same or opposite sides, had noticed that pain in the mouth was induced by eating and talking but not

by touching the skin, whereas pain around the orbit and down the nose seemed more superficial and was more often triggered off by touch. The pains usually occurred at different times, but sometimes simultaneously, depending upon the type of stimulus.

Case 1 (Fig. 5)

The patient was aged 54 when first seen in April 1939. Pain had started 20 years previously in his right upper gum and slightly in the lower, shooting towards the ear; peripheral procedures had given only temporary relief. In August 1939 I cut the lateral part of the sensory root, preserving the motor root, and the pain was permanently cured. In 1943 pain started in the left upper gum and very slightly in the lower (i.e., in a distribution symmetrical with the previous right pain); infraorbital injection gave temporary relief. In October 1948, before agreeing to his demand for operation (he had mentioned suicide), I tried mandibular injection to observe the effect of bilateral sensory loss in the mouth, but the alcohol produced total trigeminal anaesthesia. The mouth was then virtually anaesthetic and he had to suck soft food through a tube or from a spoon in front of a mirror for several months until, luckily, mandibular sensation gradually returned and he resumed normal eating; the total sensory loss in the first two divisions remained permanently.

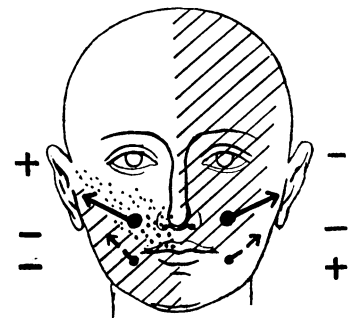


FIG. 5.—Case 1, showing the sites of pain, which started on the right side in 1919 and was cured by lateral fractional root section in 1939; and started on the left side in 1943 and was cured by mandibular (Gasserian) injection in 1948. The sensory chart shows total (shaded by lines) and marginal partial (dotted) cutaneous sensory loss (+ and - indicate sensation on the eye and the gums).

When last seen in October 1963 he was happy and grateful, in marked contrast to the earlier years of misery. There had been no pain on either side, and no trouble with eating or with the insensitive left eye or from numbness apart from temporary troublesome paraesthesiae after his wife died. On the left side of the nose there was a temporarily healed ulcer, which since injection had been intermittently active and had destroyed part of the ala.

Comment.—It is of interest that during 44 years pain never occurred in the normal right first division, whereas after 24 years it started in the corresponding distribution on the left side. The fact that pain is just as likely to arise later on the other side of the face as in the unaffected zone on the same side is one reason why fractional procedures are preferred to total.

Other Factors

Age of Onset.—Pain started most commonly in the fifties, also often in the forties and sixties; caution is necessary in making the diagnosis in younger patients, but in 9% it started before the age of 40 years (in 1% between 16 and 29).

Heredity.—The rare occurrence of a familial history was mentioned by Harris (1940). There were three pairs of sisters; one of the six had bilateral pain. None had disseminated sclerosis.

Examination.—It is necessary to exclude any impairment of sensation, which never occurs in trigeminal neuralgia, even apparently in patients with disseminated sclerosis, and to test vision if treatment is likely to denervate the eye. General examination includes possible evidence of disseminated sclerosis, and assessment of general fitness for operation. Only pain sensation was tested, a sharp white-headed pin being used; it seems that a blunt response to pin-prick is insufficient for spasms which require at least slight sharpness.

Diagnosis.—The pain is so distinctive that diagnosis is obvious from the history alone if the patient can give a reasonable description or if spasms are witnessed. Pain from local lesions in the sinuses, eye, mouth, teeth, or ear is usually easily excluded, but a few days' observation may be necessary when there is doubt about neurosis, which is a common cause of facial pain. Functional pain is generally described as a constant ache, usually in the skin, but sometimes with superimposed shooting or stabbing that may cause uncertainty.

Disseminated Sclerosis.—Spastic paraplegia was present in 23 patients (3.5%); it always preceded the onset of trigeminal pain, usually by several or many years, except in one patient with simultaneous onset at 35. Pain started at an earlier age, and six patients were under 40. It was bilateral in 7 (30%), compared with the overall incidence of 5%, but only one patient had double-zone pain on one side. The distribution and behaviour of the pain and the results of treatment were the same as in other patients. There was no impairment of trigeminal sensation. Post-mortem examination on two patients who died after operation revealed plaques of demyelination scattered throughout the brain and involving the trigeminal nerve within the pons.

Glossopharyngeal Neuralgia.—This must be mentioned because it closely resembles trigeminal neuralgia, and has the same features of intermittent, brief severe spasms started by trigger mechanisms. It is a much rarer disease; there were only 10 cases in 18 years, giving a relative incidence to trigeminal neuralgia of 1.5%; three of the ten patients had trigeminal neuralgia (0.5%). The pain is usually maximal in the throat, but it may be localized to the ear.

Aetiology

Trigeminal neuralgia has a unique, stereotyped distribution, which presumably is related to causation. The foci are small enough to be called spots, and the pain spreads only by occurring at certain new sites, or by shooting along certain lines between foci, in a manner which, together with the invariable absence of sensory impairment, is quite different from the manifestations of known lesions that affect trigeminal sensory pathways in the nerve or in the brain.

The fundamental cause of trigeminal neuralgia and the mechanism of pain production remain a mystery. Necropsies on untreated patients are very rare, and no definite histological changes have yet been demonstrated (apart from the finding of plaques in cases with disseminated sclerosis). Many theories about possible peripheral or central causes have been suggested. Briefly they include, peripherally, (a) a dental cause, because pain is common in the gums (sometimes leading to unnecessary removal of healthy teeth) and occasionally starts after tooth extraction or sepsis (Harris, 1940), but pain at the orbit is not explained, and (b) compression of the nerve, but this would be expected to produce prolonged paraesthesiae or pain and usually some sensory loss; and, centrally, (c) a sensory epileptic discharge (epileptiform neuralgia—Trousseau, 1868), because of the intermittency, brevity, and explosive character of the pain, (d) a degenerative, post-inflammatory, or arteriosclerotic lesion in the brain stem which causes demyelination and allows a short-circuiting of benign stimuli of touch or of muscle movement into pain pathways (Dott, 1951), and (e) Kugelberg and Lindblom (1959) suggest that the sensitive mechanism is situated in the brain stem in relation to the descending trigeminal nucleus.

The occasional association of disseminated sclerosis seems to be more than coincidental; but whether there is a causal relationship remains a vexed question, and it is doubtful even though plaques affecting trigeminal pathways in the brain stem have been found pathologically. Trigeminal neuralgia is rare (1%) in patients with disseminated sclerosis (Rushton and Olafson, 1965), which is a painless disease, and it is very rare

in patients who previously had temporary numbness and sensory impairment on the face due, presumably, to demyelination (which causes paraesthesiae but not pain).

Lesions involving sensory pathways in the brain produce sensory loss and only very rarely pain that is prolonged, diffuse, and quite different from trigeminal neuralgia. It is unlikely that a lesion in the thalamus or thalamocortical connexions could produce such localized pain, but there may be an abnormality in the brain stem affecting second relay neurones that connect with peripheral neurones from the sites of pain. On the other hand, the invariable abolition of pain by interrupting any part of the trigeminal nerve would not be expected to stop central pain, and it seems that intact peripheral neurones are essential in the mechanism of pain production even when pain occurs (apparently) spontaneously or is induced by extra-trigeminal stimuli.

As no specific morphological lesions have been demonstrated, it seems reasonable to postulate a physiological abnormality to explain the trigger mechanism which is such an important feature of trigeminal (and glossopharyngeal) neuralgia. Afferent impulses, whether tactile or proprioceptive or from a distant source, must act centrally, possibly by stimulating physiologically abnormal neurones in the descending trigeminal nucleus. Electrophysiological studies in animals (King, Meagher, and Barnett, 1956) suggest that the pain may be due to abnormal electrical potentials in the nerve and in the descending nucleus, resulting in conversion of normal benign impulses into severe pain. The distribution of pain indicates that only certain neurones are ever affected, and, whether pain originates centrally or reflexly, it would seem that their involvement is related to a peripheral factor.

The pain does not follow nerve branches and is not strictly related to their distribution, and it rarely affects the sensitive lips; it occurs mainly in deep structures. In view of the similarity between its distribution and the lines of the embryological facial clefts (Fig. 3), it is tempting to speculate whether there is a developmental factor that determines the distribution of abnormal sensory neurones or is atavistic, reverting to a primitive type of sensation. A developmental abnormality might explain the sites of pain along the gums on either side of the buccal cleft which extends backwards to the depth of the ear, or along the line of the naso-orbital cleft which extends around and behind the eye; it might also explain the preponderance of pain at the premaxillary or snout region, where developmental defects occur, also the stereotyped sites of new foci and of displaced pain within the zone corresponding to one cleft, and perhaps also the marked tendency for bilateral pain to be symmetrical; it would also offer a possible explanation for the strikingly similar pain of glossopharyngeal neuralgia (related to the third branchial arch). There is as yet no definite indication of the site or the nature of the cause of trigeminal neuralgia, and it is possible that there are several contributing factors involving both central and peripheral neurones.

Operations on the Sensory Root

Operation is preferred to Gasserian injection when the patient is in good health, because of the advantages of painless procedure, of preserving sensation on the eye or in the mouth, and of fewer sequelae and fewer recurrences, but there is a slight risk in old patients. There were 246 operations (in 240 patients), all except one (Case 2) in the middle fossa: 62 were extradural until 1951, when, because of the risk of facial paralysis, which occurred in six patients, the intradural approach was adopted for 183 operations (without facial paralysis).

Mortality.—Six patients died after operation, mostly from myocardial or cerebral vascular attacks after one to three weeks. Two patients had disseminated sclerosis. This mortality of 2.5% is too high (there have now been 79 operations since the

last death), but five patients with a long history had refused further injections. All except one were aged 69 to 78; on the other hand, many patients in the seventies, and even eighties (the oldest was 87), had successful operations. The mortality, considering age and severity of pain, is not unreasonable, and with stricter selection it is negligible—for example, Cushing (1920) recorded 332 operations with 0.6% mortality, Frazier (1931) 654 operations during 30 years (he operated on 50% of patients) with 0.26% mortality; and several other large series with mortality rates around and below 1% have been reported. The negligible risk of early operation should not detract from its great value, and even poor-risk patients may reasonably be accepted when other treatments fail.

Complications.—Temporary motor-oculi paresis followed two extradural operations, and three patients had hemiparesis, which soon disappeared.

Keratitis and Tarsorrhaphy.—An insensitive eye must be carefully supervised, and, when necessary, promptly treated to prevent loss of vision, which need never occur. A drop of sterile paraffin is instilled twice daily for three months, after which, with ordinary care, there is rarely any trouble. The eye is never covered, and shielded spectacles were stopped early in the series. The patient is instructed to report immediately if the eye becomes red or vision is blurred. Occasionally during the first week the eye suddenly becomes red and the cornea is roughened. If local treatment fails lateral tarsorrhaphy should not be delayed, particularly when the eye is dry. Tarsorrhaphy produces rapid improvement, and, when not delayed, the eye returns to normal and the eyelids are opened in three months; the condition need not be permanent unless the patient is uncooperative. The eyelids were partially closed in five patients (four had extradural operations and two had facial paralysis) out of 51 with insensitive cornea about one week after operation; vision returned to normal and there was no further trouble. Follow-up revealed one patient in whom later keratitis developed after several years; tarsorrhaphy had been delayed several weeks and corneal scarring permanently reduced vision to finger-counting.

Lateral fractional section	181
Middle fractional section	16
Medial fractional section	20
Total section (middle fossa, 6; posterior fossa, 1)	7
		224
Abandoned	14
Decompression	2
Postoperative deaths	6
		246

Fourteen operations were abandoned without cutting the sensory root, mainly because of reluctance to divide large anomalous vessels passing between temporal lobe and middle fossa, or when a satisfactory view of the root was obscured by bleeding from dural veins; there should be no hesitation in stopping the operation, as there are alternative treatments. Two decompression operations were tried in 1954 but the pain continued.

The operation results are based on 224 root sections (Table II). The whole zone should be denervated, always including the upper gum, to prevent pain occurring after several years elsewhere within the same zone. This necessitates cutting at least four-fifths of the bundles and sparing only one bundle on one side of the root—either a large lateral or a medium-sized medial bundle (Fig. 6). The rearrangement of nerve fibres in the root plexus close to the ganglion results in slight overlap to each division, and a marginal area of partial sensory loss after fractional operations; this area may include the upper gum when too few bundles are cut, as had occurred in 16 of the 24 patients with recurrence of pain. An upper-gum focus with partial sensation may remain dormant for several years before pain recurs. Restriction of sensory loss to

one division was occasionally deliberate, but when the root bundles were poorly seen it was unintentional. Sometimes the wrong type of operation was used before it was realized that pain extending from the upper incisor-canine gum upwards to the orbit, or to the lateral canthus, belonged to the nose-orbit zone and required medial section instead of lateral.

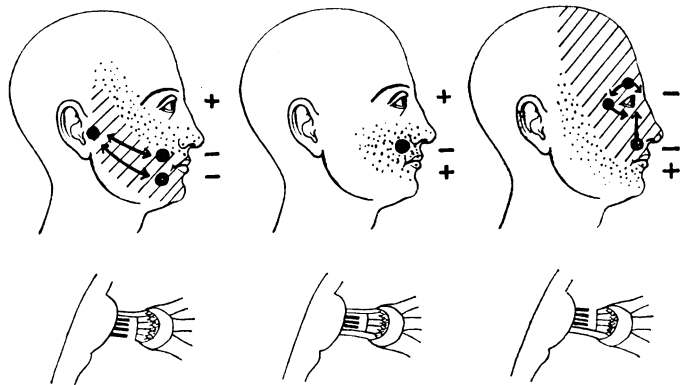


FIG. 6.—Operations of fractional sensory-root section. Above, the areas of total (lined) and partial (dotted) sensory loss (+ and - indicate sensation on the eye and the gums); below, the amount of sensory root cut and the degenerated root bundles (black). Left to right are lateral, middle, and medial fractional sections.

Lateral Section

After 33 (18%) of 181 operations some sensation remained on the chin, and there was recurrence of pain in seven patients (out of 121 followed up) who had moderately sharp sensation on the lower gum, and in whom operation was sometimes difficult because the root was short and the dural incision was on the (post-fixed) ganglion. In such cases the short lateral bundle is partly hidden under the petrosal sinus, where it may be missed in spite of careful searching with an angled hook, whereas a long root facilitates the accurate selection and division of nerve bundles. Lateral sections, especially if modified for third division pain only, and total sections, are probably more certain (and are now being tried) in the posterior fossa, where the mandibular fibres are on the presenting surface of the nerve, but accurate denervation of the upper gum is less certain; and for recurrence in the lower gum this approach is better than Gasserian injection (Case 2). There were 18 recurrences; seven were in the lower gum or ear, ten in the upper gum, and only one, five years later, was at a new focus at the orbit in the other zone.

Middle Section

This operation for pain confined to the upper gum is not always permanently satisfactory, because the sensory loss is too restricted, and later on pain may start elsewhere. Nevertheless it is occasionally a useful conservative operation, especially for bilateral pain. Second division (mouth-ear) pain does not occur in the cheek or upper lip, which need not be anaesthetized. It seems to be a fortunate anatomical arrangement that the sensory-root bundles in Meckel's cave supply, in the order in which they are cut from the lateral side, the skin of the lower jaw, the lower gum, the upper gum, and then the cheek, so that cutting appropriate middle bundles abolishes pain sensation in both gums with sparing of useful partial sensation on the cheek and lips.

Preservation of ophthalmic sensation was an important aim in 197 operations (181 lateral and 16 middle sections). Corneal sensation was lost after 26 operations (13%), mostly after extradural operations, when the root was sometimes avulsed instead of cut. In addition, corneal sensation was unavoidably lost after 18 medial and seven total sections. Altogether, therefore,

corneal sensation was preserved after 173 out of 224 operations (i.e., fractional procedures prevented any possibility of eye trouble in 77% of patients).

Medial Section

This satisfactory operation is easy in the middle fossa, where the curved medial border of the root is clearly separate from the medial wall of Meckel's cave. When pain was confined to the first division sometimes only half, or less, of the root was cut, which was insufficient to anaesthetize the lateral canthus and the upper gum, with the result that 4 out of the 16 patients had recurrence of pain.

Total Section

The whole sensory root was deliberately cut in only 7 patients. There was no return of sensation or of pain and no eye trouble after 7 to 14 years. In addition, after 10 lateral sections (eight were extradural) there was unintentional near-total anaesthesia with absent corneal reflex, and sparing of moderate sensation at the medial canthus only.

Case 2.—A woman aged 52 was first seen in September 1946 with a six-year history of spasmodic pain deep at the ear focus. Mandibular injection followed by lateral root section, which evidently missed a few lateral fibres, and two Gasserian injections, which added total permanent anaesthesia in the first two divisions but not in the third, where slight sensation still remained, all failed to cure the pain deep in the ear (believed to receive fibres from the lateral end of the ganglion). In February 1949 the whole sensory root was cut in the posterior fossa. The pain immediately stopped and had not recurred when she was examined 14 years later.

Late Results

Late results are available for 158 operations after 1 to 16 years. There was no return of pain after 134 operations (85%). There were five early failures with continuation or recurrence of pain within 12 months, and in 11 patients recurrence was delayed five to nine years, while 51 patients remained free of pain after 9 to 16 years.

Gasserian Ganglion (Sensory Root) Injections

Gasserian injection was sometimes used when patients refused operation or were poor surgical risks, or for pain at the orbit or for urgent relief. The aim was always to produce total anaesthesia, because I never felt able to inject only a selected part of the ganglion. There were 217 procedures (in 165 patients), but 21 were abandoned without injection for various reasons, mainly inability to enter the foramen ovale. It is, however, easier to pass a needle through the foramen than to be sure of entering the ganglion and sensory root. Radiography was not used, but its use, as described by Penman (1953), is no doubt helpful during difficult injections.

Immediate total sensory loss, which was obtained in 81% of 196 actual injections, does not always remain permanently, because it may be due to (a) transient nerve block, which disappears within a few hours (many injections were completed within ten minutes and were not prolonged by waiting for possible return of sensation), or (b) axon destruction in the nerves distal to the ganglion followed by regeneration and return of sensation in 12 to 18 months, as well as to (c) the effect of alcohol on ganglion cells or sensory root, producing central axon degeneration and permanent sensory loss. It

cannot be known until 12 to 18 months later whether sensory loss will remain permanently.

The late results were assessed after first injections because repeat injections usually reproduced the same result (only 5 out of 35 were successful). Of 86 patients who were examined after 1 to 17 years 56 (65%) remained free of pain. Nearly half the recurrences (14 out of 30) started immediately or within 12 months, 24 started within three years, and the longest interval was seven years; while 23 patients were free of pain after 7 to 17 years.

Re-examination of sensation showed, however, that only 33% of the 86 patients had total anaesthesia (Henderson, 1965; Fig. 2). The most frequent result was the return of sensation in the mandibular division only, in spite of persisting total sensory loss in the first two divisions (this accidental fractional result was ideal for pain in the nose-orbit zone, but unfortunately the reverse fractional result with mandibular sensory loss was rare). These results were surprising, because the lateral part of the ganglion is nearest to the foramen ovale and is in the line of the needle. This finding, and in other patients recovery of sensation in all divisions, suggested that alcohol often had not reached the ganglion, and it seemed likely that the explanation was anatomical rather than technical; also, there was a striking difference between the ease in obtaining a permanent result in some patients and the great difficulty or impossibility in others.

These unexpected results led to a series of post-mortem dissections of the Gasserian ganglion and injections of coloured alcohol (Henderson, 1965, 1966). Briefly they showed: firstly, variations of 10 mm. in the antero-posterior position of the ganglion, which cannot be known clinically or by x-ray examination; secondly, the long, thin ganglion cannot be adequately injected without alcohol bursting into the sensory root; and, thirdly, the presence of a constant dural venous sinus in the foramen ovale, into which alcohol was sometimes injected and stained indirectly the maxillary and ophthalmic nerves—this is believed to occur when sensation returns after Gasserian injections. The ganglion is not, as is sometimes incorrectly illustrated, a thick, compact structure on top of a short foramen, where it would be easily injected; the ovale canal is about 10 mm. long.

It is assumed that permanent total anaesthesia results only from the effect of alcohol on the sensory root inside Meckel's cave, and it became clear that many injections had not been deep enough (when the ganglion was post-fixed). It is probable that more patients would have had permanent anaesthesia if injections had been made more boldly to a depth of 20 to 25 mm. instead of 10 to 15 mm. (Henderson, 1965) into the sensory root after withdrawing cerebrospinal fluid from Meckel's cave, but this requires skill.

Medial Canthus Sensation.—Sometimes there was sparing of good sensation in a semicircle of about 1 in. (2.5 cm.) at the medial canthus, which is easily missed on examination because the forehead is anaesthetic; it presumably resulted from sparing of the medial end of the ganglion. Pain in the nose-orbit zone occasionally started there after injection; the area may be difficult to anaesthetize by further injection, but it is easily denervated by sensory-root operation.

Nerve Palsies.—Six slight palsies of the oculomotor and two of the abducens nerves were mostly transient, and all recovered completely. One glossopharyngeal palsy, after failure to enter the foramen ovale, caused slight difficulty in swallowing which disappeared in a few months.

Keratitis and Tarsorrhaphy.—Insensitve eyes caused more trouble after injection than after operation, and the onset was often rapid—during the first two days. Tarsorrhaphy was required in 13 (19%) out of 69 patients with absent corneal sensation. Follow-up revealed one patient in whom tarsorrhaphy, two months after injection, had been delayed three weeks and the eye became blind. Gasserian (and mandibular) injections

can be done in outpatients, but it is essential to examine an insensitive eye often during the first 10 to 14 days, if necessary by admitting the patient.

Nasal Ulceration.—In 12 (18%) patients out of 66 with permanent anaesthesia on the cheek and nose an ulcer appeared on the alar margin of the nostril, usually from one to six months after injection. The ulcers, with slight bleeding and crust formation, were resistant to local treatment, became indurated and chronic, and usually extended by eroding the nostril; ulceration never followed infraorbital injection. (An ulcer has occurred after a medial section operation since the end of the series.) The cause is uncertain, but self-inflicted trauma in response to paraesthesiae may be important (Schorstein, 1943).

Paraesthesiae.—Most patients soon become accustomed to the numbness and are not troubled by it, especially younger patients who are fully occupied, which is one reason for preferring early radical treatment. Occasionally the numbness is unpleasant, but only rarely is it distressing or intolerable, and then usually in old, lonely, worried, or neurotic patients, and particularly when they do not read or have no interests after retirement. Troublesome paraesthesiae seem to depend mainly upon personality and domestic circumstances (Case 1), and they are less troublesome when sensory loss is limited by fractional operation. Patients are never treated during remissions, and it is rarely necessary to make a preliminary injection to test the patient's reaction to numbness before making it permanent.

Peripheral Procedures

Temporary relief of pain is sometimes sufficient, and even preferable to radical cure, for example during the first attack, or when pain is limited to the upper gum, or when the patient is very old or unfit for operation, or when total anaesthesia after Gasserian injection is undesirable (for example, poor eyesight), or when pain is bilateral, or as a diagnostic test.

Mandibular Injection.—Injection of the nerve at the foramen ovale is comparatively easy and is especially useful for bilateral pain; but it should be avoided when an insensitive cornea is undesirable, because there is a slight risk of alcohol spreading to the Gasserian ganglion. Sensory loss was obtained after 208 out of 227 injections (in 165 patients). After 33% of 164 followed-up injections there was also unwanted sensory loss in the first and second divisions, which in most cases disappeared either in a few hours or after 12 to 18 months; but after 8% of injections it remained permanently (Case 1), even though mandibular sensation returned to normal (Henderson, 1965; Fig. 3). As expected, pain usually recurred after 12 to 24 months, but 10% of patients were still free of pain five years after injection in spite of return of sensation.

Infraorbital Injection.—It is essential to anaesthetize the gum, and alcohol is injected as far as possible along the infraorbital canal in the hope of reaching the posterior alveolar nerves to the molar gum. Sensory loss, however, is usually limited to the front half of the gum, where a focus in the nose-orbit zone is easily denervated; but a focus in the premolar-molar region often escapes. Injections numbered 596 (in 287 patients). There were many failures, due to (a) inability to enter a small scarred foramen, (b) anaesthesia on the cheek only, (c) inability to denervate the molar gum, and (d) immediate displacement of pain backwards along the gum or upwards to the orbit. Pain usually returned within 6 to 24 months; only five patients were still free of pain after five years.

Supraorbital Neurectomy.—Cutting the supraorbital, supratrochlear, and infratrochlear (which is apt to be missed) nerves through an incision in the eyebrow is sometimes useful for pain confined to the eyebrow and forehead because the operation can be done under local anaesthesia. There is a strong tendency for supraorbital pain to be displaced to the lateral canthus or to the upper gum, and full medial-root section is often required for permanent cure (Fig. 4). Injection of the

supraorbital nerve may be surprisingly difficult, and it is not advisable to try injecting the supratrochlear and infratrochlear nerves near the medial canthus. There were 62 supraorbital procedures (in 49 patients), mainly neurectomies, most of which were followed by recurrence of pain within three years; only two patients were still free of pain after five years.

Maxillary Injection.—This injection is potentially dangerous because the maxillary nerve in the pterygopalatine fossa is not far from the optic nerve. During the first two years several injections were made for molar pain, but the procedure was abandoned after alcohol reached the optic nerve and made the eye blind.

Drug Treatment

Many drugs have been tried since Fothergill (1773) described "a painful affection of the face." Tegretol (carbamazepine), the drug at present in vogue, was introduced four years ago and is now widely prescribed in the hope that it will effectively control the pain. Preliminary reports, and a small personal experience, show that about half the patients are relieved of spasms within 24 hours, but the relief may not continue and the drug has often no effect. Frequently there are unpleasant side-effects, such as giddiness, vomiting, unsteadiness, skin rash, or mental confusion, which may be severe; one death from acute aplastic anaemia has been reported (Spillane, 1964), and others have occurred.

Drug treatment is difficult to evaluate in a short period because of natural remissions, and, remembering the disease is usually progressive, it seems that as yet no drug is likely to stop the pain for 10 or 20 years. Tegretol, in certain circumstances, may reasonably be tried under careful supervision, and occasionally it may suffice when the attacks of pain are infrequent, are mild, and of short duration; it should not, however, be continued indefinitely without full consideration of possible difficulties of radical cure later on if the drug ceases to be effective. A drug should be safe and free from side-effects, otherwise the long-term taking of tablets is no improvement on the safe and more certain results of temporary peripheral procedures or, better still, permanent cure.

Conclusions and Summary

A survey of early results of operations and injections in 650 patients, and of late results, showed the marked contrast between the lives of patients who are permanently cured and of those who continue to suffer recurring pain. It seems necessary to emphasize that, as has been known for many years, permanent cure can usually be obtained without risk or complication, and that treatment should be carried out more often than it is at present; many patients wished the cure had been made earlier.

Cutting the sensory root under vision, as early as possible before the patient becomes unfit for operation, was preferred to blind Gasserian injection, and the results were better; and partial section of the root was preferred to total section because pain rarely affects all three divisions. The late results of fractional operations showed that sometimes insufficient root bundles had been cut, partly because the anatomical behaviour and future spread of the pain was not at first fully appreciated; this mistake can be avoided.

Post-mortem dissections and injections were made to discover reasons for certain surprising anatomical results of Gasserian injections. They revealed anatomical explanations for some injection difficulties and recurring failures, and it became clear that injections had not been deep enough always to reach the sensory root, and that alcohol in front of the ganglion had sometimes produced total sensory loss that did not remain permanently.

The indications for, and the results of, various peripheral procedures that give temporary relief are summarized, and drug treatment is mentioned.

There is no single treatment for all cases of trigeminal neuralgia. Some patients will not tolerate injection and others are afraid of operation. There are individual considerations in every case, and the best treatment may be operation, or Gasserian injection, or peripheral-nerve injection, or tablets; all have a place in proper management and all should be considered by the surgeon who has experience of all methods and is familiar with the Gasserian region.

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REFERENCES

- Cushing, H. (1920). *Amer. J. med. Sci.*, **160**, 157.
 Dott, N. M. (1951). *Proc. roy. Soc. Med.*, **44**, 1034.
 Fothergill, J. (1773). *Med. Obs. Inquir.*, **5**, 129.
 Frazier, C. H. (1931). *J. Amer. med. Ass.*, **96**, 913.
 — and Spiller, W. G. (1901). *Philad. med. J.*, **8**, 1039.
 Harris, W. (1912). *Lancet*, **1**, 218.
 — (1940). *Brain*, **63**, 209.
 Henderson, W. R. (1965). *Ann. roy. Coll. Surg. Engl.*, **37**, 346.
 — (1966). *J. Anat. (Lond.)*, **100**, 905.
 King, R. B., Meagher, J. N., and Barnett, J. C. (1956). *J. Neurosurg.*, **13**, 176.
 Kugelberg, E., and Lindblom, U. (1959). *J. Neurol. Neurosurg. Psychiat.*, **22**, 36.
 Penman, J. (1953). *Lancet*, **1**, 760.
 Rushton, J. G., and Olafson, R. A. (1965). *Arch. Neurol. (Chic.)*, **13**, 383.
 Schorstein, J. (1943). *J. Neurol. Psychiat.*, **6**, 46.
 Spillane, J. D. (1964). *Practitioner*, **192**, 71.
 Trousseau, A. (1868). *Lectures on Clinical Medicine*, vol. 1, p. 105. London.

Results of No-loop Gastrectomy for Duodenal Ulcer

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The operation of partial gastrectomy for duodenal ulcer has been in general use in this country for many years. In most cases satisfactory relief has been obtained from the burden of the ulcer, but the quality of this benefit has been modified by the vulnerability of the stoma to recurrent ulceration, by the so-called postgastrectomy syndromes, and by some reduction in the level of general nutrition. The latter is due partly to the inadequacy of gastric digestion during the transient passage of the meal through the remnant of the stomach, but also to the virtual exclusion of the duodenum and upper jejunum from the path of the food that is brought about by the Polya type of gastrectomy.

By practising a rather extensive gastric resection the incidence of stomal ulceration has been controlled, though at the cost of accentuating the difficulties of nutrition. The most elusive problem, however, has been that of the postgastrectomy syndromes. This has not been made easier by the word "dumping," which has been stretched from the conception of overloading of the jejunum distal to the gastric anastomosis to cover the quite different problem of obstruction on the proximal side of the stoma. In mild cases this may involve no more than postprandial nausea and sweating, but in more severe cases the discomfort persists between meals, with distension, loose stools, and attacks of bile-vomiting. The final state can be one of complete misery and malnutrition—even tuberculosis.

The inexplicable and tantalizing question about these obstructive manifestations is why they should occur in one patient and not in the next—patients who, so far as one can tell, have undergone the same surgical procedure. The evidence suggests that they are due to the displacement of the proximal loop of jejunum from under the mesocolon up to the gastric stump in order to make the anastomosis. This attachment creates a variable degree of obstruction, partly by simple kinking, partly by rotation effects at the stoma (Stammers, 1954; Tanner, 1954; Hermon Taylor, 1964). The intensity of the symptoms depends on the degree of obstruction, but if dilatation and stasis allow abnormal bacterial growth to occur malabsorption may follow in the whole of the distal intestine (Stammers, 1961; Orr, 1964). *The appropriate name for these phenomena is therefore not the postgastrectomy syndrome but the loop-*

gastrectomy syndrome—a phrase which places the responsibility for them on the jejunal loop proximal to the anastomosis rather than on the gastric resection as such.

The reality of this conception was illustrated in one of our patients who was himself a surgeon with experience of gastrectomy. He had already had a partial gastrectomy for duodenal ulcer and two revision operations for "dumping" at which, however, the jejunal loop had been retained. He described two quite different types of pain—one after eating too much, and the other, "a more distressing type radiating into the back and chest, which seemed to have no anatomical basis." At his final operation the jejunal loop was detached from the stomach and returned to its anatomical position and a no-loop anastomosis was made (see below), with subsequent relief from the symptoms. The special interest in this case concerns a naso-oesophageal catheter which had been guided through the stomach and the stoma into the duodenum as a safeguard against postoperative distension. Before this tube was removed some days later it was used to study the function of the parts under x-ray screening. It was found that the injection of 20 ml. of Gastrografin into the duodenum produced an acute discomfort which the patient recognized as the same as that which had been associated with his jejunal loop. The pain was relieved by withdrawing the fluid from the duodenum. The catheter was then drawn back into the stomach, where a similar injection now produced no sensation, and the medium passed on into the efferent jejunum. This patient is quite convinced that the nauseating discomfort he used to experience with his jejunal loop was due to duodenal distension.

The "No-loop" Operation

The object of this paper is to show that a design of gastrectomy which does not employ a jejunal loop for the anastomosis (Hermon Taylor, 1959) is not only free from this obstructive syndrome but also, by restoring a measure of duodenal function, affords some protection of the stoma from gastric acid and a better level of nutrition than the standard operation. Fig. 1 shows how the "no-loop" anastomosis functions in this way and indicates the difference from the loop gastrectomy (Fig. 2). By freeing the fundus of the stomach from the spleen the anastomosis can be made to the duodeno-

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