FACIAL PAIN*

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As the conditions which cause pain in the facial structures are many and varied, the subject is one of considerable complexity and, because it is not fully understood, even more controversy.

Almost all general practitioners will at some time be confronted with patients complaining of facial pain and in these times of specialization the condition also falls within the province of the ophthalmic and ear, nose and throat surgeons, the physician and general surgeon, the neurologist and neuro-surgeon, and also the orthodontist. As it is not practical to consider, in detail, all aspects of the condition, I propose to deal with it mainly from the point of view of the neuro-surgeon.

In order to introduce some clarity the following main headings are presented as a broad classification and I will develop the various sub-groups in my theme:

1. Local involvement of the facial structures.
2. The cranial nerve neuralgias.
3. Painful vascular disorders of the face.
4. Pain referred to the face from distant structures.
5. Miscellaneous neurological conditions associated with pain in the face.
6. Atypical facial neuralgia.

1. LOCAL INVOLVEMENT OF FACIAL STRUCTURES

Many of the facial structures when directly involved by disease processes present with pain as a major symptom. Conditions in this group include infections of the paranasal and mastoid sinuses, eye conditions, including glaucoma, dental conditions such as peri-apical abscess or an unerupted tooth, diseases of the ear, including otitis and petrositis, and other allied conditions.

It is apparent that treatment directed towards minor infections of the paranasal sinuses rarely alleviates severe facial pain. Infection of the frontal sinus may cause severe pain but the pain originating from the other paranasal sinuses is usually dull and chronic and, unless the physical signs, including radiograms, are unequivocal in demonstrating the presence of a major pathological condition, one would be advised to seek elsewhere for the cause of severe facial pain.

There is a rare condition associated with chronic suppurative disease of the parotid gland. This is Frey's auriculo-temporal syndrome, which is characterized by a burning pain on one side of the face, associated with flushing of the skin and sweating when the patient eats. Between attacks there may be a persistent hyperaesthesia so that the patient may complain of discomfort during shaving or similar stimulation of the skin. This syndrome is due to involvement of the auriculo-temporal nerve and is usually relieved by avulsion of that nerve.

Costen[1] drew attention to disease of the temporo-mandibular joint as a cause of facial pain, and it would be convenient to mention the syndrome at this stage so that the clinical features can be compared with those of other conditions which will be discussed in due course. Most of the patients presenting with this syndrome are over the age of 40 and are usually edentulous. The joint is subjected to excessive strain due to malocclusion or loss of molar support and overclosure of the jaw. It was thought that the symptoms were due to compression of the chorda tympani and auriculo-temporal nerves and pressure on the Eustachian canal, but Gaylor[2] has advanced the theory that they may be due to distortion of the thin tympanic plate, which ends in a free edge at the petro-tympanic fissure and which affects the 5th, 7th, 9th and 10th cranial nerves. The clinical features include ear symptoms and pain and irritative phenomena in other sites. There may be a continuous or intermittent impairment of hearing, with a feeling of stuffiness in the ear and a dull pain. Sometimes there are snapping noises associated with jaw movements, and tinnitus or vertigo may be present. Pain may also occur at the vertex or in the region of the occiput or mastoid process, and there is occasionally a burning sensation in the throat, the tongue and the side of the nose. Tenderness is present over the affected joint. The patient may complain of a dryness of the mouth, and herpes of the external ear or buccal mucosa may be present. X-ray studies of the joint usually show arthritic changes, erosion and abnormal mobility, and the pain will respond to an injection of local anaesthetic into the petro-tympanic fissure.[2] The treatment consists of increasing the vertical dimension of the jaw by various orthodontic procedures.

Malignant growths of the mouth, jaws or pharynx, especially those involving the posterior wall of the maxilliary antrum or the nasopharynx, may cause very severe pain, which can be successfully relieved by interruption of the trigeminal nerve pathways or by section of the nerve in the middle or posterior fossae. Even if the growth has apparently

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been successfully treated by deep X-ray therapy, painful local radiation effects may follow and these will also respond to similar nerve section. If the malignant process affects the glosso-pharyngeal and second cervical nerves as well, then the posterior-fossa approach is indicated because of the accessibility of all three nerves at the same operative exposure. The presence of malignant disease, with its complications, does increase the mortality rate of these operations but it is nevertheless surprising how well elderly and cachectic patients tolerate these procedures.

Trauma to nerves as a result of fracture of the jaws or following dental extraction may result in continuous pain in the distribution of the affected nerve and here again interruption of the affected nerve pathways may be indicated for the relief of pain. It is to be noted that while peripheral nerve section may give temporary relief, the pain can be expected to recur within a few months as the result of nerve regeneration. Destruction of the ganglion cells with alcohol or pre-ganglionic section is not followed by nerve regeneration.

2. THE CRANIAL NERVE NEURALGIAS

(*a*) *Trigeminal Neuralgia*

Trigeminal neuralgia is a nervous disorder with characteristic clinical features. It is one of the few types of facial pain which, if correctly diagnosed and treated, can be cured with certainty, and it is for this reason that the condition should be recognized for what it is, so that prompt treatment can be instituted.

The symptoms consist essentially of a severe momentary paroxysmal pain strictly within the distribution of one or more divisions of the trigeminal nerve in the complete absence of any signs of impaired function of the nerve. It is to be noted that there is no spread of the pain across the midline.

The other cardinal features are that the patient is free from all pain (except occasionally for a mild residual burning pain) between the attacks and that the attacks can often be provoked by external stimuli, such as friction applied to the skin, the exposure of the face to cold water or to a draught, or by the movements of the jaws caused by speaking or eating. It often happens that these stimuli precipitate an attack only when applied to certain areas of the face—the so-called 'trigger zones'—the sites of which are commonly in the region of the naso-labial fold or the skin of the lower lip overlying the mental foramen. The pain may spread from the 2nd or 3rd divisions into the 1st, but it is rare for the 1st division to be primarily affected.

These painful attacks, which usually occur in persons over the age of 60, are best described as having a shooting or stabbing quality, and each spasm rarely lasts for longer than a few seconds or, at most, a minute or two. Initially the spasms occur only occasionally but with the passage of time the intervals become progressively shorter, so that eventually the unfortunate victim may be subjected to bursts of excruciating pain at frequent intervals during the day. When this occurs the patient is in a pitiable plight indeed, and in extreme cases he may refuse to speak and may even deny himself food so that malnutrition, with its attendant evils, may further rob him of his moral fibre to an extent where death itself would offer him his only relief.

The aetiology of trigeminal neuralgia has not yet been convincingly elucidated and, as a spontaneous cure is extremely rare, it is fortunate that interruption of the trigeminal pathways offers certain relief. The disease is not related to dental sepsis and the removal of teeth has no influence on its course. It must be admitted, however, that often in its earliest stages it may present as pain related to the teeth without any of the other characteristic features and it is only after dental extraction has been carried out that the true nature of the condition becomes manifest.

In the early stages, vitamin B12 or Trilene inhalations may be employed. These occasionally have beneficial effects, but because of the proxysmal nature of the disease it may well be that the apparent relief is in actual fact due to the occurrence of a spontaneous pain-free period. Dihydroergotamine has also been used but its evaluation is also subject to the same criticism.

Interruption of the nerve pathways is achieved by surgical section or by the injection of chemical blocking agents such as alcohol, phenol or boiling water[3] into the peripheral nerve or its ganglion. Interruption of the peripheral pathways is only temporary and the pain recurs when the nerves regenerate. Peripheral injection is sometimes used as a diagnostic measure in doubtful cases, or to see how the patient will react to the numbness of his face which follows interruption at the commonly indicated sites. It occasionally happens that the patient experiences unpleasant paraesthesias in the denervated skin (anaesthesia dolorosa) and may complain that these sensations are less tolerable than the original pain.

The most widely employed procedure in cases of trigeminal neuralgia is the surgical section of the sensory root proximal to the Gasserian ganglion through the middle-fossa approach. The approach is usually made extradurally, but in cases of excessive adherence of the dura to the floor of the middle fossa, possibly as a result of previous alcohol injection, the ganglion can be exposed intradurally. Section of the sensory root gives permanent relief and it is possible to preserve those fibres subserving the cornea and conjunctiva in cases where the pain is confined to the 2nd and 3rd divisions. The motor root, supplying the muscles of mastication on the affected side is easily identified and can be spared. The mortality rate of this operation is very low.

Alcohol injection of the ganglion requires considerable experience for its adequate performance and has the additional disadvantage that relief is not always permanent because of the difficulty in securing adequate destruction of the ganglion. Furthermore, it is not always possible to preserve corneal sensation, and a neuroparalytic keratitis may subsequently develop. This may, however, be offset by a prophylactic cervical sympathectomy. However, it is an important consideration that a ganglion block does avoid an operation and its employment is especially indicated in the aged or where all three divisions are involved. Surgical section is advisable when it is desirable and feasible to spare corneal sensation and in cases where ganglion block produces only short-lived relief, or where it has failed entirely.

Taarnhøj[4] has recently advocated decompression of the trigeminal nerve as it courses over the petrous ridge. In this procedure sensation of the face is undisturbed, but the operation has not been widely accepted and has not been carried out for a sufficiently long time to assess the permanency of its effects.

If it is suspected that a lesion in the posterior fossa may

be a possible cause of the trigeminal pain, the nerve can be exposed *via* the posterior fossa. The operation is not difficult but it does nevertheless carry a slightly higher mortality rate than the middle-fossa approach.

The descending root of the trigeminal nerve extends well into the medulla and may be severed in this region. Tactile sensation is preserved in the face and the danger of a neuroparalytic keratitis is reduced but the possibility of other side-effects and a higher mortality rate rather tends to offset the advantages. This operation is indicated in young persons, and in cases where the condition is bilateral and where one side of the face has already been rendered anaesthetic by a previous retrogasserian neurectomy.[5]

(*b*) *Glossopharyngeal Neuralgia*

The pain in this relatively rare condition is similar to that of trigeminal neuralgia but is situated in the region of the throat at the base of the tongue with radiation down the side of the neck, in front of the ear and to the back of the lower jaw. Exceptionally the pain may begin deep in the ear.

The attacks tend to be triggered by swallowing, coughing or protruding the tongue, and can be temporarily abolished by spraying the back of the tongue and pharynx with a surface anaesthetic. Malignant growths in the pharynx must be excluded. The paroxysms of pain can be terminated by sectioning the glossopharyngeal nerve in the posterior fossa but, if the ear is also involved, it is advisable to section the upper rootlets of the vagus nerve as well.[5]

(*c*) *Superior Laryngeal Neuralgia*

While pain in the region of the larynx is more often due to tuberculosis or cancer, a paroxysmal neuralgia may occur where no obvious pathological lesion is present. All these conditions respond to alcohol blocking or section of the superior laryngeal nerve. Harris[8] has injected alcohol into the internal laryngeal nerve. The paroxysms of pain are situated in the region of the thyro-hyoid membrane, with radiation to the side of the neck and up towards the lower face and gums and downwards towards the thorax and shoulder. Any irritation of the throat, such as swallowing or coughing, may trigger off the pain. Stimulation of the internal branch of the superior laryngeal nerve as it passes above the pyriform sinus or as it pierces the thyro-hyoid membrane may also provoke a paroxysm, and spraying the pharynx with a surface anaesthetic, or procaine blocking of the nerve, may produce temporary relief. Tenderness over the point where the nerve pierces the thyro-hyoid membrane may help in the diagnosis.[7]

(*d*) *Geniculate Neuralgia*

It was long thought that the facial nerve was purely motor in function but it has now been conclusively demonstrated that the nerve and its branches carry sensory fibres; the nervus intermedius is the sensory root and the geniculate ganglion is the sensory ganglion. Ramsey Hunt, during his observations on herpetic inflammations of the ganglion, has shown the cutaneous distribution to include parts of the pinna, the external auditory canal and the tympanic membrane.

Even in the absence of herpes, disturbances of the ganglion can lead to a severe deep lancinating pain in these regions, with radiation to the mastoid and retro-orbital and paranasal structures. Stimulation of parts of the ear may sometimes trigger off the pain and there may be a trigger zone in front of the ear.

The condition responds to section of the nervus intermedius if, at operation under local anaesthesia, the same pain is produced by stimulation of that nerve. If this association cannot be definitely demonstrated, it is advisable to section other nerves which supply the ear, namely the glossopharyngeal and the upper rootlets of the vagus.[8]

3. PAINFUL VASCULAR DISORDERS OF THE FACE

(*a*) *Facial Migrainous Neuralgia*

The facial pain in this condition has special characteristics with regard to the periodicity of its onset. The many cases of facial pain described under the names of Horton's histaminic headache, ciliary neuralgia, petrosal neuralgia, and Sluder's sphenopalatine neuralgia, suggest that these are merely synonyms of the same condition.

Facial migrainous neuralgia occurs predominantly in males, and consists of paroxysms of the sudden onset of a strictly unilateral, burning, constant pain in the supraorbital region or in or behind the eye and temple. Occasionally there is radiation to the face, jaw and neck. Each paroxysm of pain rises rapidly to a peak and then, after maintaining this level for from a few minutes to two or three hours, gradually subsides to disappear completely, leaving the patient free from pain. There is at least one attack in every 24 hours, the attacks being mainly nocturnal and often occuring with clock-like regularity, awakening the patient at the same hour each night.

This state of affairs may last up to 2 months, and is then followed by cessation of the pain with complete freedom for several months or even years before the attacks again reappear.

About half the patients have associated signs of autonomic disturbance in the way of congestion of the conjunctiva, swelling of the peri-orbital tissues, blockage of the nasal passage, and dilation of the temporal vessels.

The condition differs from migraine in that the attacks are of shorter duration and, although nausea may occur, there is rarely vomiting or visual or sensory disturbances. In addition, true migraine lacks the autonomic changes and the marked regularity of the attacks.

Treatment has been carried out in the past with varied results by histamine desensitization, by section of the greater superficial petrosal nerve[9] or by injection of the sphenopalatine ganglion. Sir Charles Symonds[10] has recently shown that the paroxysms can be prevented by the regular administration of intramuscular ergotamine tartrate until the patient enters his pain-free period, when the injections can be discontinued.

Dott[2] has stated that interruption of the trigeminal nerve gives certain relief.

(*b*) *Temporal Arteritis*

This disorder is part of a generalized vascular disease[11] occurring in elderly patients. A subacute inflammatory process involves the media of the arteries, and giant cells are present. The characteristic clinical features include fever and sweating, with anorexia and joint and muscle pains. Headache, which is severe and constant, is a major feature and visual disturbances are common. Temporo-mandibular pain occurs fairly frequently. When the superficial temporal

arteries are involved they are acutely tender and may become thrombosed, so that the pulsations are decreased. The disease tends to run a slow course and finally becomes arrested, leaving a variable degree of disability.

Relief may be obtained by the use of vasodilators or potassium iodide in large doses. Excision of small portions of the affected arteries may afford prolonged freedom from pain.

4. PAIN REFERRED TO THE FACE FROM DISTANT STRUCTURES

Diseases involving the heart and lungs may cause pain which radiates to the face, as may also pathological processes involving the cervical spine.

In cases of prolapsed intervertebral disc or cervical spondylosis the patient may present with orbital or peri-orbital pain with radiation to the temple or towards the vertex. There may be pain in the mastoid region or at the occiput.[12] The pain is constant, with variations in intensity, and is affected by emotional disturbances or by changes in the weather. Movements of the neck may influence the pain, and also any actions which raise the cerebrospinal-fluid pressure such as coughing or straining at stool.

Delmas, Laux and Guerrier, as quoted by Neurwith[12] have described the presence of sympathetic fibres in the ventral nerve roots C5, C6, C7, C8 and T1 and, as the pain often corresponds to the fields of arterial distribution of the external carotid system, it is supposed that the facial pain can be explained on a basis of sympathetic irritation. In any patient with facial pain of undetermined aetiology, and especially if there is evidence of cervical root involvement with motor and sensory signs, it is advisable to carry out radiological studies of the cervical spine and, if indicated, to institute treatment with traction or by means of a cervical collar.

5. MISCELLANEOUS NEUROLOGICAL CONDITIONS ASSOCIATED WITH FACIAL PAIN

Neurological conditions having facial pain as one of the symptoms are numerous, and include tabes dorsalis, disseminated sclerosis, intracranial aneurysms, posterior-inferior-cerebellar thrombosis and post-herpetic neuralgia. In these conditions the history and physical findings will give a clue as to the diagnosis, and treatment where indicated can be carried out.

In post-herpetic neuralgia the lesions probably involve both the peripheral and central nervous pathways and treatment directed towards the peripheral pathways alone is not likely to succeed. In severe cases where peripheral alcohol injections have failed one may have to resort to prefrontal leucotomy or other allied procedures, but it is always worth while to try the effect of deep X-ray therapy first.

6. ATYPICAL FACIAL NEURALGIA

It is unfortunate that, all too frequently, patients present with severe facial pain and yet the clinical features are not covered by those described in the foregoing groups. For want of a better term, these patients are said to be suffering from 'atypical facial neuralgia'.

It is considered by many that this type of facial pain is psychogenic in origin, but there are certain features which seem common to all these unfortunate sufferers and this tends to suggest that there is an organic basis for the pain. Admittedly there is a psychogenic overlay in many of the patients, but this is possibly merely the reaction to a demoralizing and debilitating condition. Probably the condition is due to irritative lesions involving the thalamus or thalamic connections in the brain or brain stem. Dott[2] believes them to be vascular in the older age-groups and neuro-degenerative in the younger.

The disturbance of sensation known as 'thalamic over-reaction', although rare in a fully developed form, is well recognized and is thought to be due to damage to the lateral nucleus of the thalamus. In this condition the pain is deeply situated and is constant and gnawing. Although the threshold for pain is raised, the stimulus, when effective, arouses sensations of a peculiarly unpleasant character and all sensory modalities evoke a disagreeable sensation. A particular stimulus applied to the affected side causes a more severe reaction than when it is applied to the unaffected side, and the precise localization of the stimulus is impaired.

In patients with atypical facial neuralgia these characteristics are present in a very modified form but do nevertheless serve as a basis for the pain usually described. These patients complain of a constant, ill-defined, deeply situated and poorly localized pain. They often describe it as being drawing or crushing or tearing. The pain is often bilateral and cannot be related to any particular nerve and there are no 'trigger zones'. These patients often over-react to their pain emotionally and drug addiction is common. Careful examination always fails to reveal any local or intracranial cause for the pain.

The treatment of atypical facial pain is always difficult and, more often than not, unsuccessful. Dott[2] has stated that on no account should any peripheral nerve injection or operation be done and many other neuro-surgeons, from bitter experience, also hold this view. Some cases do, however, respond to interruption of the peripheral pathways for a limited period until nerve regeneration takes place and in these cases a retrogasserian neurectomy may be indicated. Sympathectomy has been tried, with discouraging results. Psychotherapy may be of value and should always be tried before any surgical procedure is advised. If the patient has deteriorated to such an extent that he is already unable to hold his place in society, prefrontal leucotomy may be indicated, but here again the results are variable and relapses are frequent.

The question of Sluder's sphenopalatine neuralgia is always raised in a discussion of facial pain. Sluder described a pain of wide distribution starting at the root of the nose and extending on to the face and head and sometimes as far as the arm and finger-tips. The pain does not appear to have any specific characteristics, and it is difficult to know in any case presented as one of Sluder's syndrome that Sluder himself would agree with the diagnosis. For this reason the diagnosis of Sluder's neuralgia as a definite type of atypical facial pain is best avoided. Sluder has claimed successes in the treatment of atypical facial pain by cocainization or extirpation of the sphenopalatine ganglion, but this has not been the general experience. However, the injection of alcohol or hydrocortisone into the ganglion is still a common practice and this will no doubt continue until a more specific form of therapy has been advanced for this difficult condition.

SUMMARY

Facial pain is discussed under the following headings:

1. Local involvement of the facial structures
2. The cranial nerve neuralgias:
   (*a*) Trigeminal neuralgia
   (*b*) Glossopharyngeal neuralgia
   (*c*) Superior laryngeal neuralgia
   (*d*) Geniculate neuralgia
3. Painful vascular disorders of the face:
   (*a*) Facial migrainous neuralgia
   (*b*) Temporal arteritis
4. Pain referred to the face from distant structures with special regard to cervical pathology
5. Miscellaneous neurological conditions associated with pain in the face
6. Atypical facial neuralgia with a note on Sluder's syndrome.

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