Classification and diagnosis of facial pain

Nick S Jones

This is an interesting time to consider the classification of facial pain because of the emergence of new ideas that challenge our understanding of the mechanisms involved. The new hypotheses that have been proposed appear to be of clinical relevance.

Great efforts have been made to categorize facial pain into diagnostic categories based on either the symptoms, signs or aetiology. This approach has classified patients in an effort to understand the aetiology and thus find the most effective treatment for each group. The last specific consensus document was produced 13 years ago (Headache Classification Committee of the International Headache Society (IHS), 1988) and pointed out that it was difficult to have definitions rather than descriptions. Some diagnoses have relatively tight descriptions, e.g. trigeminal neuralgia or cluster headache, but even these have no diagnostic tests. The current alternative classifications (*Table 1*), although laudable in their aim to help research and treatment, are undermined in practice because many patients cannot be classified into one or more distinct diagnostic groups. Treatment selection is then based on the category they resemble closest and the best treatment for that group.

Have there been any advances recently? Ideas from the Copenhagen group (Olesen, 1991; Olesen and Rasmussen, 1995; Bendtsen et al, 1996; Jensen, 1999; Bendtsen, 2000; Jensen and Olesen, 2000) on tension-type headache have resulted in a model that may be relevant to other patients with facial pain. While tension-type headache describes a group of symptoms with some broad but inconsistent features, these theories provide a more inclusive, encompassing interpretation of its aetiology. Essentially these theories expound central sensitization of the trigeminal nucleus from either prolonged nociceptive input from a peripheral injury, surgery or inflammation; pericranial myofascial nociceptive input; or psychological or neurological factors reducing supraspinal inhibition. This broader perspective is a more inclusive method of interpreting the patient's condition. Others have described mechanisms that can produce central sensitization through neural plasticity and tried to explain the phenomenon of hyperalgesia and how pain can persist (Ren and Dubner, 1999; Sessle, 2000).

Overlap between conditions is greater than might appear from current texts on classification. Many patients who could readily be classified into one defined group have additional features such as neuropathic, myofascial, migrainous or supra-spinal characteristics (Eide and Rabben, 1998; Graff-Radford, 2000; Sessle, 2000). A classification that accommodates these variant features might lead to a better understanding of the mechanisms involved and also to a better treatment strategy. The IHS (Headache Classification Committee of the IHS, 1988) classification tried to cater for this by allowing for a patient to have more than one disorder, placed in order of importance. However, this does not allow the addition of other 'characteristics' as opposed to an additional diagnosis which might also help in the patient's management, as there are large areas of overlap between most patients' symptoms and their response to treatment. For example, separating headaches from facial pain is an artificial division as many conditions involve both the head and face.

Facial pain that escapes current classification might be better categorized on the basis of whether it has neuropathic, myofascial, migrainous or supraspinal features rather than placed in a dustbin of miscellaneous or 'not classifiable' (*Table 2*). Nevertheless, these terms do not allow those who have previously escaped classification to be neatly categorized, as these patients often have features of more than one type of pain.

Where facial pain is difficult to classify, it is useful to try and break it down into a combination of elements, be it neuropathic, myofascial, migrainous or supraspinous, rather than calling it

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TABLE 1.
Comparison of the International Headache Society (Headache Classification Committee of the IHS, 1988) and International Association for the Study of Pain (IASP) (1994) classifications

IHS classification	IASP classification
1. Migraine 1.1 Migraine without aura 1.2 Migraine with aura 1.2.1 Migraine with typical aura 1.2.2 Migraine with prolonged aura (more subgroups)	Group V: Primary headache syndromes, vascular disorders, and cerebrofluid syndromes Migraine, common migraine Migraine variants Mixed headache (more in group V later)
2. Tension-type headache 2.1 Episodic tension-type headache (more subgroups) 2.2 Chronic tension-type headache (more subgroups) 2.3 Headache of tension-type not fulfilling above criteria	Group III: Craniofacial pain of musculoskeletal origin Acute tension headache Tension headache: chronic form Temporomandibular pain and dysfunction syndrome Osteoarthritis temporomandibular joint (TMJ) Rheumatoid arthritis of the TMJ Dystonic disorders Crushing injury of head or face
3. Cluster headache (more subgroups) Chronic paroxysmal hemicrania Cluster headache-like disorder not fulfilling criteria	Group V continued: Cluster headache Chronic paroxysmal hemicrania Hemicrania continua Cluster-tic syndrome Syndrome of 'jabs and jolts'
4. Miscellaneous headaches unassociated with structural lesions (more subgroups)	Headache associated with low CSF pressure Post-dural headache
5. Headache associated with head trauma (more subgroups)	Group V continued: Post-traumatic headache
6. Headache associated with vascular disorders (more subgroups)	Group V continued: Temporal arthritis, carotidynia
7. Headache associated with non-vascular disorder (more subgroups)	
8. Headache associated with substances or their withdrawal (more subgroups)	
9. Headache associated with non-cephalic infection (more subgroups)	
10. Headache associated with metabolic disorders (more subgroups)	
11. Headache or facial pain associated with disorder of cranium, neck, eyes, nose, sinuses, teeth, mouth or other facial or cranial structures 11.1 Cranial bone 11.2 Neck 11.3 Eyes 11.4 Ears 11.5 Nose and sinuses 11.6 Teeth, jaws and related structures 11.7 Temporomandibular joint	Group IV: Lesions of the ear, nose and oral cavity ears, Maxillary sinusitis Odontalgia Glossodynia and sore mouth Cracked tooth syndrome Dry socket Gingival disease Toothache of unknown cause Inflammatory jaw conditions Unspecified pain of the jaws Frostbite of face Group VII: Suboccipital and cervical musculoskeletal disorders
12. Cranial neuralgias, nerve trunk pain and deafferentation pain 12.1 Persistent pain of cranial nerve origin 12.2 Trigeminal neuralgia 12.3 Glossopharyngeal neuralgia 12.4 Nervus intermedius neuralgia 12.5 Superior laryngeal neuralgia 12.6 Occipital neuralgia 12.7 Central causes of head and facial pain other than tic douloureux 12.8 Pain not fulfilling 11 or 12	Group II: Neuralgias of the head and face Trigeminal neuralgia Secondary neuralgia (trigeminal) from CNS Secondary neuralgia (trigeminal) from trauma Acute herpes zoster (trigeminal) Postherpetic neuralgia Geniculate neuralgia Neuralgia of nervus intermedius Glossopharyngeal neuralgia (+ from trauma) Neuralgia of superior laryngeal nerve Occipital neuralgia Hypoglossal neuralgia (+ from trauma) Tolosa–Hunt syndrome Shortlasting, unilateral neuralgiform pain with conjunctival injection and tearing Raeder's syndrome
13. Headache not classifiable	Group V continued: Headache not otherwise specified

'unclassifiable'.

DIAGNOSIS

It is essential to take a structured history to reach the correct diagnosis, plan the right treatment and avoid misguided interventions that only complicate the picture. It might be thought that a completely different subgroup of patients will present to each speciality — this is not the case. Most people know that their sinuses lie behind the facial bones so many conclude that the cause of their facial pain lies in their sinuses before they seek medical help. 'Sinus' trouble is an acceptable label in the community while atypical facial pain or 'not classifiable pain' are not. However, rhinosinusitis rarely causes facial pain, even in an ear, nose and throat clinic (see later). Many patients label themselves as having sinusitis when this is not the case.

Facial pain has a special emotional significance: symptom interpretation is often influenced by cognitive, affective and motivational factors. For a few patients, facial pain may be the channel by which they express emotional distress, anxiety or the psychological harm associated with disease, trauma or surgery. It may be the means by which they demand attention or obtain secondary gain. The presence of a marked psychological overlay does not mean that there is no underlying organic problem, but it should make one wary about invasive treatment. If there is a big discrepancy between the patient's affect and the description of the pain, the organic component of the illness may be of relatively minor importance. Pain which remains constant for many months or years, or which extends either across the midline or across defined dermatomes, is less likely to have a physical basis. However, pain associated with clear exacerbating or relieving factors, whose onset was clear cut and whose site does not vary during the consultation, usually has an organic cause. Should the diagnosis remain obscure, re-taking the history at the next consultation may be helpful, or a symptom diary kept by the patient may help.

TABLE 2.
The main types of pain

Characteristics	Migrainous	Myofascial	Neuropathic	Supraspinal
Poorly localized		+++	+	++
Continuous dysaesthesia			+++	+
Fluctuating duration intensity		+++		++ and
Location varies		++		++
Pressure, tight, ache		+++		
Burning			+++	++
Electric, sharp			+++	+
Sensation of numbness,		++	++	++ swelling
History of injury			+++	+
Phantom pain			+++	+
Nausea	+++ pain		+ if severe	
Aura/photophobia	Classical			
More common in women/ factors	++	++	++	++ hormonal
Age	incidence		Increased	
Psychological stress/ tional conflict		++	+	++ emo-
Hyperaesthesia of skin		++	++	
Hyperaesthesia of muscles		+++	++	
Hyperaemia, erythema	+++	++	++	+
Response to neural blockade		++ pain	Peripheral neuropathic effect	Can get placebo
Responds to behavioural		++	+	++ therapy
Responds to tricyclic pressants	+	+ relaxation/ biofeedback	++	++ antide-

A careful history is central to a correct diagnosis. Twelve questions form the basis of an algorithm that will help reach a differential diagnosis.

1. Where is the pain and does it radiate anywhere?

Asking the patient to point with one finger to the site of the pain is helpful, not only because it localizes the pain, but also because the gesture made often relays information about its nature (e.g. patients with myofascial pain give a vague gesture over a broad area; with neuropathic pain, such as post-traumatic neuralgia or trigeminal neuralgia, it is well localized), and the facial expression indicates its emotional significance to the patient.

2. Is it deep or superficial?

Deep pain is dull and poorly localized, but pain from the skin tends to be sharp and well defined.

3. Is the pain continuous or intermittent?

The periodicity of symptoms may be a pointer to the diagnosis, e.g. being woken in the early hours by severe facial pain which lasts up to 2 hours suggests cluster headache.

4. How did the pain begin?

An aura preceding unilateral facial pain or headache is typical of classical migraine.

5. How often does the pain occur?

Recurrent bouts of aching of the ear and jaw with sharp twinges is a pattern characteristic of

temporomandibular joint (TMJ) dysfunction, while monthly premenstrual headaches with nausea are typical of migraine.

6. What is the pattern of the attacks and are they progressing?
The relentless progression of a headache, in particular if associated with nausea or effortless vomiting, is worrying and an intracranial lesion should be sought.

7. How long is each episode?
The stabbing pain of trigeminal neuralgia is short lived with a refractory period.

8. What precipitates the pain?
Trigeminal neuralgia is initiated by a specific trigger point.

9. What relieves the pain?
Tension headaches do not respond to analgesics, whereas patients with migraine often report that lying quietly in a dark room helps.

10. Are there any associated symptoms?
If nausea accompanies the pain, this is characteristic, although not diagnostic, of migraine.

11. How does it affect daily life and sleep?
Should the patient describe a severe unrelenting pain but have an apparently normal life and pattern of sleep, atypical facial pain should be considered in the differential diagnosis.

12. What treatment has been tried and with what effect?
Tension headache and atypical facial pain fail to respond to analgesics; this in isolation does not clinch the diagnosis but is a useful pointer. Chronic paroxysmal hemicrania specifically responds to indomethacin and trigeminal neuralgia to carbamazepine.

TYPES OF FACIAL PAIN

The following are brief descriptions of the common types of facial pain with or without headache in the order used by the IHS classification, omitting groups with headache alone.

Migraine

Migraine primarily causes severe headache, but in a small proportion of patients it can affect the cheek, orbit and forehead. Migraine is a term that is often wrongly used by patients, and the diagnosis needs confirmation by precise questioning. Classical migraine with an aura and visual disturbances rarely affects the face (*Figure 1*). Common migraine, nine times more frequent, is described as sharp, severe and pulsatile in nature and is invariably accompanied by nausea. There is, however, no premonition or photopsia. Stress, diet, premenstrual state and barometric changes can induce it, and it is worth asking about these and other trigger factors. There is often a family history of migraine.

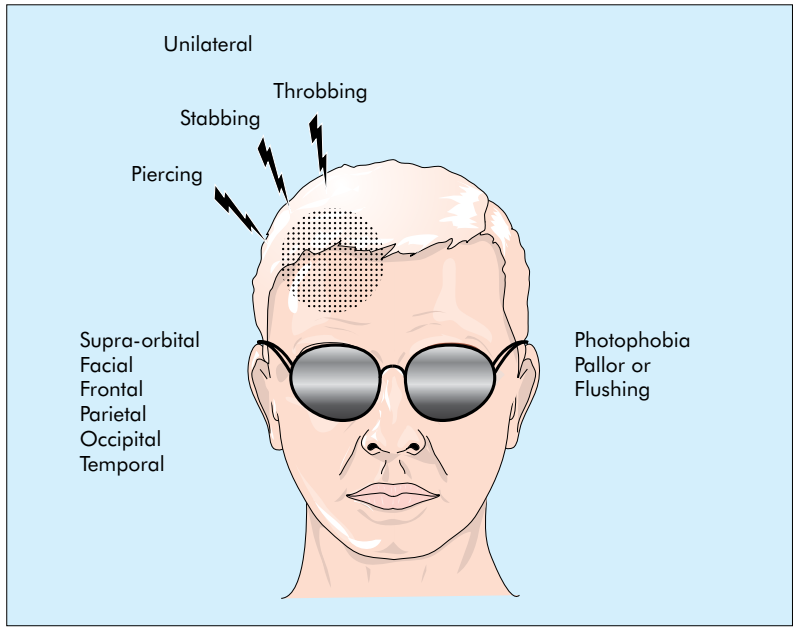

Figure 1. Features of migraine.

Tension-type headache

Tension-type headache is described as a feeling of tightness, pressure or constriction that varies in intensity, frequency and duration. It usually affects the forehead or temple and often has a suboccipital component (*Figure 2*). It may be episodic or chronic (>15 days/month, >6 months) and is only occasionally helped by non-steroidal anti-inflammatory drugs (NSAIDs). Typically, patients are taking large quantities of analgesics of all kinds but say that they have little benefit (Olesen, 1991). It is often associated with anxiety, depression or agitated depression. Hyperaesthesia of the skin or muscles of the forehead often occurs, causing the patient to think that they have rhinosinusitis, as they know their sinuses lie under the forehead.

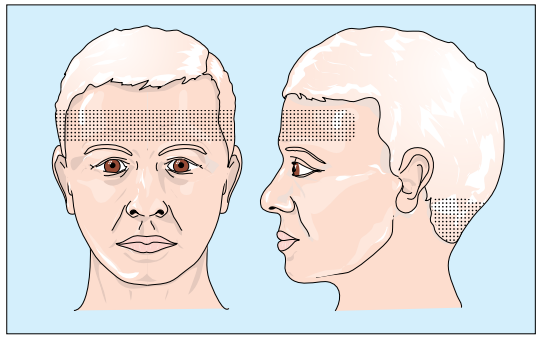

Figure 2. Localization of tension-type headache.

Cluster headache
This typically presents with a very severe unilateral stabbing or burning pain which may be frontal, temporal, ocular, over the cheek or even in the maxillary teeth (*Figure 3*). Pain is therefore facial and 'headache' is a misnomer. Nausea is absent but frequently there is rhinorrhoea, unilateral nasal obstruction, lacrimation and sometimes conjunctival injection. It is most common in men between 20 and 40 years of age. The patient is awakened in the early hours, often walking around in distress, with the pain lasting between 30 minutes and 2 hours. It may be precipitated by alcohol intake. Myosis or facial flushing may be seen.

Chronic paroxysmal hemicrania
Chronic paroxysmal hemicrania is an excruciating pain occurring in women at any time of night or day. It can affect the frontal, ocular, cheek or temporal regions and last 30 minutes to 3 hours. The patient can experience several episodes in 24 hours, and nasal congestion, lacrimation and facial flushing can all be a feature.

Associated pains
Headache or facial pain associated with disorders of the cranium, neck, eyes, ears, nose, sinuses, teeth, mouth or other facial or cranial structures.

Eyes
Uncorrected optical refractive errors can cause headaches, but their importance is exaggerated. Optic nerve disease results in reduced acuity and colour vision. Pain on ocular movement suggests optic neuritis or scleritis. It is vital to recognize acute glaucoma that may cause severe orbital pain and headache. The patient may see haloes around lights, and circumcorneal injection can occur as well as systemic upset, especially vomiting. This requires urgent treatment, as vision is rapidly lost. Pain is a feature of periorbital cellulitis, which may present with lid swelling and erythema if it is preseptal and with chemosis and proptosis. Orbital pain can also be caused by uveitis, keratitis, dry eye syndrome and convergence insufficiency.

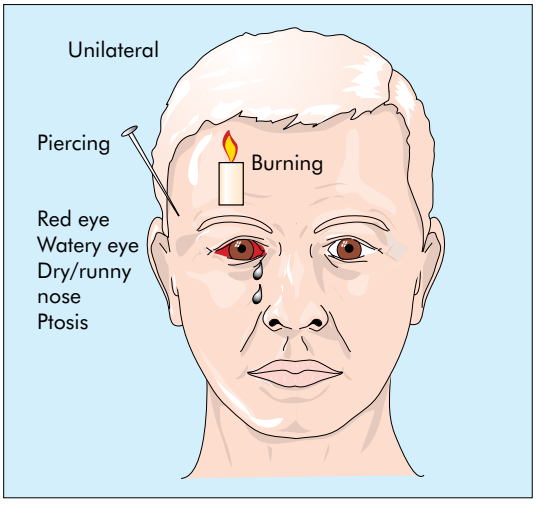

Figure 3. Features of cluster headache.

Ears
Earache without any hearing loss and with a normal eardrum is usually caused by referred pain from the TMJ, tonsil, tongue base or hypopharynx. These should be examined.

Nose and sinuses
Patients not infrequently complain of 'sinus', believing that they have sinusitis. This often used term should be treated with scepticism. Acute infective sinusitis causes severe pain that is usually well localized over the affected sinus, follows an upper respiratory tract infection, and the patient has a temperature. However, chronic sinusitis is often painless, causing nasal obstruction as a result of mucosal hypertrophy and a purulent discharge throughout the day (not just a collection in the morning that is usually the result of postnasal mucus stagnating in a mouthbreather or snorer that has become discoloured by local commensals).

An acute exacerbation can cause pain but this rarely lasts more than a few days. Symptoms of a dull ache behind the eyes, affecting the lower part of the forehead, under the nasal bridge or either side of the nose are often not related to sinus disease and are caused by an extension of tension-type headache. The term used to describe this is midfacial segment pain, and it is described later as it is not in the IHS classification.

Facial pain without any nasal symptoms is unlikely to be caused by sinusitis.

Imaging: Plain X-rays have a poor specificity and sensitivity in sinusitis management. Computed tomography (CT) is not much better, as about 30% of asymptomatic people have false-positive changes in one or more of their sinuses on CT scanning (Bhattachayya et al, 1997; Jones et al, 1997). Rigid endoscopy provides more accurate information about the extent of sinus disease. However, patients with facial pain who have no objective evidence of sinus disease (endoscopy and CT negative) and whose pain does not respond to medical antibiotic or steroid therapy aimed at treating sinonasal disease are unlikely to be helped by surgery for more than a few months (Acquadro et al, 1997; Ruoff, 1997; Tarabichi, 2000; West and Jones, 2001). It seems that surgery can alter central neuronal activity in a third of these patients for up to 12 months, but in a third the pain is worse and in the remaining third there is no difference.

Previous theories implicating contact points as a cause of facial pain have been discredited, as they are found as frequently in an asymptomatic

population as in those with pain, and these patients often respond to low-dose amitriptyline.

Plain sinus radiographs have no role in diagnosing chronic rhinosinusitis.

CT scans show changes in 30% of asymptomatic individuals.

Occasionally, following a nasal fracture, pain or paraesthesiae can persist over the nasal bridge. The cause of this is unclear; it may be the result of a neuroma in the scar tissue but seems to be influenced by the degree of distress the patient continues to feel about the insult they have received. Why some people develop neuropathic pain after an injury while others do not may centre on whether central neural plasticity occurs to inhibit peripheral neural changes that might otherwise lead to dysaesthesia, hyperaesthesia or chronic pain instead of central sensitization or downregulation of central inhibition (Ren and Dubner, 1999; Sessle, 2000). Peripheral regenerative or deafferent changes may influence the trigeminal brainstem sensory nuclear complex just as nerve compression, sympathetically-mediated pain and neuroma formation can cause neuropathic pain.

Other diseases of the nose or sinuses: Carcinoma of the maxilla is rare. Patients unfortunately often present late when the disease has spread beyond the confines of the sinus. Unilateral bloody purulent nasal discharge is the most frequent presentation. Less common symptoms are infraorbital paraesthesiae, loose teeth or ill-fitting dentures, proptosis, deformity of the cheek, nasal obstruction or epistaxis. Pain is a late feature.

Nasopharyngeal carcinoma is also rare but presents most commonly in young adults from the Far East. It often presents with cervical lymphadenopathy and middle ear effusions. However, its spread can involve the fifth and sixth cranial nerves, causing facial pain or a lateral rectus palsy.

Tolosa–Hunt syndrome (recurrent painful ophthalmoplegia) occurs equally in both sexes at any age. It presents with gnawing unilateral orbital pain with relapsing and remitting paralysis of the third, fourth and sixth cranial nerves. Occasionally, there is paraesthesia of the forehead. It is caused by a lesion in the region of the cavernous sinus or the superior orbital fissure. It should be differentiated from ophthalmoplegic migraine, painful diabetic oculomotor palsy and malignancy. Although the condition often responds to steroids, this is not diagnostic.

Raeder's paratrigeminal syndrome presents as an intense sharp pain or ache around the ophthalmic division of the trigeminal cranial nerve, with associated myosis, ptosis and facial hypoaesthesia in the same area. The corneal reflex is reduced, but there is no reduced sweating as in Horner's syndrome. A lesion near the base of the middle cranial fossa at the medial border of the Gasserian ganglion is responsible, caused by a carotid aneurysm, metastasis or local neoplastic invasion.

Teeth, jaws and related structures

Afferent fibres from the dental pulp, being small and unmyelinated, produce poorly localized pain, which often radiates to surrounding structures but rarely crosses the midline. However, dentinoenamel defects produce a sharp, usually well-localized excruciating pain, often followed by a dull ache. This may be caused by cervical erosion or a lost or cracked filling, and induced by temperature change, osmotic or mechanical stimuli.

Pain becomes more localized once the periodontium is involved. The periodontium may be affected either by the formation of a periapical abscess involving the periodontium at the tooth's apex or by infection in a pocket around the tooth where there has been long standing gingival and periodontal inflammation. Acute pulpitis causes a dull ache with exacerbations of excruciating pain and often radiates or is referred to the adjacent ipsilateral jaw. Chronic pulpitis causes a dull, difficult-to-define ache, which may be worse when lying flat. Clinical examination usually reveals a carious tooth or leaking restoration, and toothache is initiated or exacerbated by hot and cold stimuli.

'Phantom tooth pain' is said to follow a dental extraction with incomplete osseous repair. Necrotic bone and neural elements have been found in some patients with this syndrome, but in most it is a form of atypical facial pain (see later). All too often the pain precedes the extraction which has been done in part through pressure from the patient believing that their pain is dental when it was a form of atypical facial pain.

Temporomandibular joint dysfunction: This is most commonly unilateral (90%) and usually occurs in young adults with a history of bruxism, clenching, trauma, recent dental work, anxiety, enthusiastic kissing or cradling the telephone between the jaw and the shoulder. Another contributing factor is poor occlusion, as occurs in crossbite, or in a partially edentulous patient without an appropriate denture, or in someone with a completely edentulous mouth whose dentures are very worn or have been made with an inadequate vertical height resulting in overclosure.

Pain is caused by pterygoid spasm and is described as a deep, dull ache that may masquerade as toothache or earache. There is often a superimposed sharper component that may radiate down the jaw or over the side of the face or temple (*Figure 4*). It is often necessary to ask whether chewing exacerbates symptoms, as this is rarely volunteered. Spasm may be initiated by a

reflex mechanism to avoid an undesirable pattern of malocclusion. Anxiety lowers the threshold for this mechanism, and it often occurs in people under stress. Clicking of the TMJ is an unreliable sign, whereas pain on palpation of the insertion of the lateral pterygoid is a better indicator. This can be demonstrated with the gloved little finger where the lateral pterygoid muscle can be palpated at the most posterior end of the upper buccal sulcus. Trismus and deviation of the jaw from the midline on opening, as well as evidence of malocclusion or a high shiny spot on a filling, should be sought. Radiographs are normally of little help in making the diagnosis but may show degenerative changes in rheumatoid arthritis or gout where there is a suspicion of an arthropathy.

Eagle's syndrome produces pain which may be felt in the lateral wall of the pharynx, the mandible, the floor of the mouth or the side of the neck. A nagging discomfort lasting seconds to minutes is precipitated on opening the mouth or head turning. An elongated styloid process with a calcified stylohyoid ligament may be palpated laterally or via the tonsillar fossa and can be seen on an oblique radiograph.

Glossodynia is characterized by a burning sensation in the tongue and there is often disordered taste or the sensation of a dry mouth. The oral cavity should be inspected for signs of ulceration or erythroplakia and the cause of these investigated and treated. Local irritation, lichen planus, diabetes mellitus, candida, serum iron deficiency, vitamin B_{12} deficiency, irritant mouthwashes, a drug reaction, denture component sensitivity and galvanism should be excluded. In some patients, no cause can be found and this group is over-represented by women over 50 years old, often with a cancer phobia, a history of an emotional disturbance or a precipitating major life event.

Cranial neuralgias, nerve trunk pain and deafferentation pain

Trigeminal neuralgia is more common in women over 40 years of age with a peak incidence between 50 and 60 years. Patients complain of paroxysms of agonizing lancinating pain induced by a specific trigger point. Repetitive bursts can be triggered usually with a refractory period of more than 30 seconds. In more than a third of sufferers, the pain occurs in both the maxillary and mandibular divisions, while in a fifth it is confined to the mandibular region and in 3% to the ophthalmic division. A sufferer can always localize the trigger zone but is reluctant to demonstrate it.

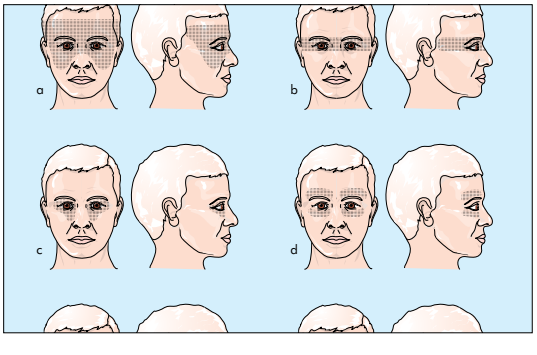

Figure 4. Distribution of pain in temporomandibular joint dysfunction.

Typical trigger sites are the lips and nasolabial folds, but touching the gingivae may also trigger pain. Some patients report that firm pressure over the trigger point helps and delays a further bout. A flush may be seen over the area in question, but there are no sensory disturbances in primary trigeminal neuralgia. Remissions are common, but it is not unusual for attacks to increase in frequency and severity. Histology shows proliferative and disorganized changes in the myelin sheath of the nerve involved. Janetta (1976) found that a percentage had vascular compression of the trigeminal nerve. However, this has been noted in many normal cadavers. Beware the dental pain that can mimic trigeminal neuralgia, particularly a fractured tooth or exposed cervical dentine. Secondary trigeminal neuralgia is attributable to a discernible pathological cause. In patients under 40 years old, it is most commonly caused by multiple sclerosis, while over this age a tumour, aneurysm or meningioma can cause the pain.

Postherpetic neuralgia affects an eighth of patients suffering from herpes zoster infection. Thankfully, two thirds of these recover in the first year. It is more common in the elderly, and if it persists for more than a year, it is unlikely to resolve. It causes a persistent intense burning or lancinating pain, and where there is a sensory loss there is more likely to be associated dysaesthesia. Patients often become depressed and irritable. Histology shows demyelination, together with a disproportionate loss of large nerve fibres, and this may allow increased transmission in nocioceptive fibres through the dorsal horn, thus causing pain. Zoster appears to have some effect at the suprathalamic level, as tractotomy at a more peripheral level is of little help.

Mental nerve neuralgia can mimic trigeminal neuralgia by producing sharp pain in the lower lip and chin when the lower premolar area is touched in the edentulous patient. It is the result of exposure and irritation of the mental nerve branch of the inferior alveolar nerve in a long-standing edentulous patient whose alveolar bone has atrophied. Direct pressure from either a denture or finger can initiate this unpleasant sharp pain.

Glossopharyngeal neuralgia: This is rare, being 100 times less common than trigeminal neuralgia. A stabbing pain is felt in the tonsillar region and

ipsilateral ear (rarely in the base of the tongue and angle of the jaw). It is precipitated by swallowing or talking, and bouts last for weeks or months with a tendency to recur.

Other causes of persistent pain of cranial nerve origin: Stretching the arterial tree which supplies the proximal portions of the cranial nerves and the dura within 1 cm of any venous sinus induces a headache but can also cause facial pain. The supratentorial vessels and dura refer pain to the ophthalmic division of the trigeminal nerve. Infratentorial structures refer pain to the distribution of the glossopharyngeal and vagus nerve and the upper three cervical nerve dermatomes. Space-occupying lesions, e.g. meningiomas, angiomas and intracerebral metastases, can induce facial pain by irritation of the trigeminal nerve along its intracerebral course.

Facial pain not fulfilling groups of 'headaches and facial pain involving the head and neck' or 'cranial neuralgias, nerve trunk pain and deafferentation pain'

'Atypical facial pain, atypical odontalgia': Atypical facial pain is not a dustbin term, and it has many distinguishing features that make it a diagnosis in its own right, not one to resort to in despair. It should only be made reluctantly when organic causes have been excluded. It is often complicated by surgical procedures that have been performed in an attempt to alleviate symptoms that have been misdiagnosed.

The description of symptoms often does not correlate with the patient's affect; there can be unusual associated factors, exaggerated responses to the pain and often psychological factors or an excess of unpleasant life events. While psychological factors are always important in any patient's interpretation of facial pain, in this condition they play an overwhelming role. Pain is typically deep and ill-defined, changes location, is unexplainable on an anatomical basis, occurs almost daily and is sometimes fluctuating, sometimes continuous, without any precipitating factors and is not relieved by analgesics. Often more specific questioning about symptoms results in increasingly vague answers. The pain does not wake the patient up, and while the patient reports that they cannot sleep, they often look well rested. It is more common in women over the age of 40 years and typically lasts many months. A proportion has symptoms of depressive illness or anxiety neurosis or has problems adjusting to the difficulties which life presents. Some patients appear to be so used to their pain that its loss would force them to dramatically reappraise their life. Confrontation is counterproductive, while sympathetic discussion, close liaison with the GP and possibly psychiatric help may be beneficial. Tricyclic antidepressants are often of help, in particular when there are symptoms of endogenous depression, e.g. loss of appetite and interest in life, self neglect, early morning waking and fatigue.

The description of hysterical pain is often vague with unusual neurological symptoms, such as weakness and paraesthesia. No physical lesion can be found and the distribution of neurological loss does not conform to the known anatomy. Pain occurs throughout the day although fluctuating in severity. It enables the patient to obtain some personal benefit, often by avoiding an unwanted task.

NON-CLASSIFIABLE FACIAL PAIN

Although not in the IHS classification pain can be characterized as to whether it has neuropathic, myofascial, migrainous or supraspinal features.

A further group of patients whose symptoms have evaded classification to date are those with symmetrical facial (with or without forehead) pain which has all the same characteristics as tension-type headache but a different distribution of pain (*Figures 5a–h*). 'Midfacial segment pain' avoids

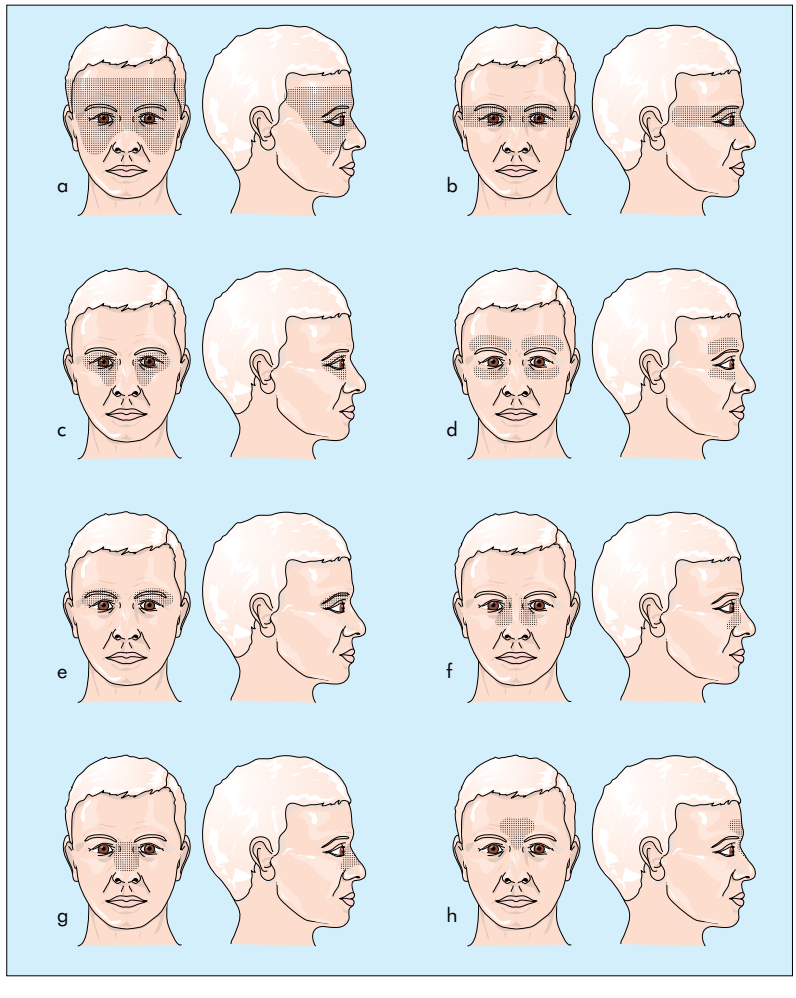

Figure 5a–h. Distribution of pain in midfacial segment pain.

the use of the term tension used in tension-type headache and avoids the confusion which that term causes with the patient. The pain usually affects the nasal area, around or behind the eyes or the cheeks, and not uncommonly involves the forehead. It is described as a pressing or aching pain, similar to the feeling of constriction or tightening. It can affect each area in isolation or in combination, but it is usually symmetrical unless there has been trauma or surgery to one side when the features of atypical facial pain can be superimposed on a background of midfacial segment pain.

The pain is usually persistent, but it can be intermittent and is usually present on waking. It does not worsen with routine physical activity and rarely interferes with the patient getting to sleep. To make matters more complex, the stimulus of a genuine acute sinus infection may exacerbate the symptoms, with a return to the background faceache on resolution of infection. Indeed, an episode of acute rhinosinusitis very occasionally appears to have been the initial trigger for the onset of symptoms in the first instance.

Symmetrical facial pain involving the bridge of the nose, either side of the nose, behind the eyes, the supraorbital or infraorbital margins and/or the forehead is often caused by midfacial segment pain, an extension of tension-type headache which affects the face.

It is hardly surprising that patients (and doctors) interpret all their symptoms as being related to their sinuses. Patients often describe tenderness on lightly touching the skin of the forehead or cheeks, along with hyperaesthesia of the skin and soft tissues in these areas. It is important to note that the tenderness is felt on light touching of the skin and soft tissue, and there is no further pain on deep palpation of the underlying bone.

Sufferers are often taking a considerable number of over-the-counter analgesics, despite saying they help little if at all. In the author's experience, the only simple analgesic of even little benefit is ibuprofen. The current first-line prophylactic treatment of chronic tension-type headache is low-dose amitriptyline at night for 6 weeks in the first instance, and this should be tried as a first-line treatment in these patients. A quarter of patients with midfacial segment pain have migrainous features associated with exacerbations of their pain, and approximately the same proportion have a history of migraine. There is also a suggestion that there is a downregulation of central inhibition from supraspinal impulses as a result of psychological stress and emotional disturbances. It is of interest that if surgery is mistakenly performed as a treatment for midfacial segment pain, the pain may sometimes abate temporarily, only to return after several weeks to months. It is as though the surgical stimulus alters the 'balance' of neuronal activity in the trigeminal nucleus for a short time. It is the author's belief that rhinological surgery should be discouraged in patients with midfacial segment pain, as the pain only helps a third temporarily; in a third it makes no difference, and in third the pain is made worse.

KEY POINTS

- Many patients' facial pain cannot be categorized into discrete diagnoses.
- There is often an overlap between the symptoms used to define many conditions with facial pain.
- It helps to be aware of the categories of pain — migrainous, myofascial, neuropathic and supraspinal — and of what treatment helps each of these.
- A new category of facial pain, midfacial segment pain, is described — this is a form of tension-type headache of the face.

CONCLUSIONS

Diseases that cause facial pain are not limited by the speciality on the sign of the outpatient clinic, and it is therefore absolutely essential to be aware of the alternative diagnoses.

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