Orofacial Pain

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Introduction

In 1965, Melzack and Wall proposed the "spinal gate control theory" hypothesising that incoming pain messages could be modified by inhibitory controls, either at the level of the spinal cord or by the brain.[1] In 1983, a further paradigm shift occurred when Clifford Woolf showed that many of the pain hypersensitivity features that accompanied peripheral tissue injury or inflammation resulted from augmentation of sensory signaling in the central nervous system (CNS)[2] - a concept termed "central sensitization." Central sensitization introduces another dimension, one where the CNS can change, distort or amplify pain, increasing its degree, duration, and spatial extent in a manner that no longer directly reflects the specific qualities of peripheral noxious stimuli, but rather the particular functional states of circuits in the CNS.[3]

Most pain thinking and central sensitization research has focused on synaptic plasticity triggered within the CNS by nociceptive inputs.[3] More recently the influences of glia, gap junctions and membrane excitability have also been recognized. In particular, the glia, their receptors and their secreted signalling factors are now recognised as having a major influence on neural function.[4] The glia, when activated, produce and release a variety of neuroexcitatory substances. Toll like receptors (TLRs) particularly TLR2 and TLR4 have been implicated in glial cell activation.[4]

Central sensitization is characterised by increased (often exquisite) sensitivity to light touch, muscle tenderness, referred pain as well as local reddening and oedema. A large body of experimental evidence and clinical research has shown that migraine, tension headache and temporomandibular joint (TMJ) pain are manifestations of central sensitization.[3] Often, co-morbidities such as anxiety, depression, fibromyalgia, irritable bowel symptoms and low back pain are frequently present.[3] In clinical practice, the challenges lie in not only treating or eliminating peripheral causes of central sensitization, such as infection, ongoing neck problems and neoplasia (rare), but also at the same time searching for and treating factors that contribute to altered CNS pain processing.

Migraine

Current migraine theory centres on sensory processing dysfunction of the brain stem or diencephalic nuclei. Neural events in the brain stem result in ensuing dilation of blood vessels, which in turn results in pain and further neural

activation. Brain imaging studies using positron-emission tomography show that the brain stem particularly the periaqueductal grey matter (PAG) is activated at the beginning of a migraine attack. The PAG is a major gateway to the limbic system and other sensory systems. The amount of light or sound coming into the body does not change during an attack; the brain's sensory response does. The brain is often hypersensitive before an attack, and afterwards the sensory processing mechanisms in the brain stem and limbic system are still hypersensitive.[3,5]

Chronic tension headache

Chronic tension headache is associated with increased tenderness in the head and neck muscles. The severity of the headaches relates directly to muscle tenderness. A common assumption has been the pain in the head and neck muscles causes the headache. The cause was thought to be, and can be, the muscles themselves. However, the neck muscles can also be painful because the spinal cord or brain stem are more sensitive to incoming sensory messages. People with chronic tension headache are often more sensitive not only around the head and neck but also elsewhere in the body, such as in the low back and calves, indicating an overall central sensitization.[6]

Medication overuse headache

In some people, the overuse of medication to treat their headache can make their headaches worse. If a person complains of daily headaches requiring regular pain medication (more than twice a week), the headache could be caused by the medications and, if so, will improve or disappear when the medications are discontinued. Medication overuse headache (MOH) can be a significant clinical challenge. The reality is almost every medication used to treat headache and migraine can cause MOH.[7] The general recommendation is patients come off these medications while under medical supervision.

Dental pain

The tooth is best considered a small body organ so, as with other organs, pain messages from the tooth itself are poorly localised. The dental pulp nerves in the middle of the tooth detect heat and cold only, which registers not as heat or cold but as pain. The hard surrounding structure of the tooth normally protects the inner nerve endings in the pulp from minor stimuli, so only extreme surface irritation (e.g. electrical stimulation or extremes of hot and cold) is sensed, as pain. If the tooth is split, the normal pulp inside may immediately become painful on contact with saliva or air. The stress of chewing or biting tends to open a split. Pain then increases with the additional pressure of biting or chewing. Until the dental pulp becomes inflamed, pain in this situation is often intermittent. Once the pulp is inflamed, it becomes hypersensitive to heat, cold, probing and pressure. Hypersensitivity may range from occasional pain caused by sweets and other minor irritations to an intolerable, throbbing toothache with pain that is difficult to control. Tooth enamel decay, tooth root erosion, a tooth fracture or splitting may immediately identify the problem tooth. Otherwise, clinical identification of the offending tooth may be difficult, if not impossible.[8]

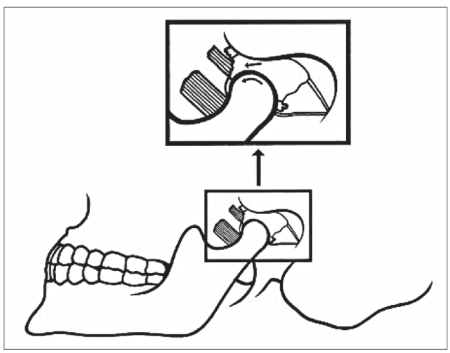
However, pulpal tooth pain does not stay unchanged, as healing can occur. Alternatively, if the pulp dies, the inflammation often spreads from the pulp cavity

to the tooth root and the fibrous tooth joint. Once inflammation affects the fibrous tooth root and the gum, the pain becomes well localised. If the tooth root dies, the pain settles. This is interpreted as the condition having got better. However, an acute abscess can then develop. Pain arising from the ligaments attaching the teeth to the jaws is then readily localisable by the patient, especially when the offending tooth is touched or pressed.[8]

Temporomandibular joint (TMJ) disorders

Two major schools of thought in TMJ pain exist. The first holds that the pain is due to bite abnormalities. No evidence exists that bite abnormalities give rise to a chronic pain disorder or that dental splints correct this.[9] Jaw joint problems occur mainly in women. If bite abnormalities were an important factor, jaw joint problems should occur equally in men and women. The weight of evidence supports the view that chronic TMJ pain is largely due to a variety of psychophysiological forces.[10] The TMJ is lubricated by synovial fluid, which also nourishes the avascular cartilage and cartilaginous disc in the middle of the joint (**Figure 1**). Normally, the joint is held in a relaxed position. Whenever it is compressed, the blood supply to the joint is reduced. If the blood supply is poor, the joint has difficulty manufacturing lubricating fluid, the friction within the joint increases and the cartilaginous disc in the middle of the joint begins to stick. Painless clicking, as the cartilage sticks, is an early symptom of TMJ dysfunction. This is extremely common. There is a click as the disc sticks and moves about on the top of the condyle. If the friction increases, the ligaments holding the disc in place stretch and the disc moves off the condylar head. The joint starts to click on both opening and closing. With time, the disc may be chronically displaced forward. In this situation, the jaw may lock intermittently.[10] The last stage is permanent forward disc displacement and the development of arthritis.

Figure 1. As the jaw joint opens, the condyle rotates on the disc and the disc slides forward on the glenoid fossa



Damage can also occur during surgical procedures, such as dental extraction, that involve prolonged, wide opening of the mouth. Muscles were designed to move and not hold the jaw open for long periods of time. These muscles can often become tight and go into spasm after dental procedures. Similarly, a prolonged period of jaw opening when the blood supply to the joint is itself reduced probably leads to inflammatory changes within the joint. In times of stress, people tend to breath using their upper chest, which tends to lead to a forward head position. Increased pressure in the TMJ can lead to clicking and sticking of the cartilage disc. Hyperventilation is strongly associated with jaw joint disorders. Before

addressing any structural changes within the jaw joint, underlying psychosocial stresses, breathing re-education, as well as musculoskeletal issues, need to be addressed.[10,11] External trauma, such as blows to the joint (e.g. motor vehicle accidents, fights and during sport), can also damage the TMJ. In this situation, patients need referral to a maxillofacial surgeon.[8]

Causes removed from the orofacial area

A history of neck injury can often be overlooked. The nerve supply to the upper neck and head overlaps in the upper spinal cord. Using first principles, neck pain could be referred to the head. Alternatively, by sensitising the spinal cord to incoming pain messages, neck pain could lower the pain threshold to other sensory messages being received from the face and head. After a whiplash injury to the neck, areas well away from the original injury site – from the head to the feet – can be shown to be hypersensitive to normal sensory messages.[3]

Sinusitis

According to the diagnostic criteria of the 2004 International Headache Society (IHS), chronic rhinosinusitis (CRS) has not been established as a cause of headache.[12] On the other hand, the American Academy of Otolaryngology, Head and Neck Surgery (AAO-HNS) considers that facial pain or pressure is an important, although not exclusive, consideration in the diagnosis of CRS.[13] While this dichotomy is difficult to explain clinically, advances in our understanding of pain pathophysiology offers potential solutions.[4] TLRs particularly TLR2 and TLR4 have been implicated in glial cell activation. TLR2 and TLR4 recognise and respond to endogenous danger signals such as lipopolysaccaride that are released by damaged and dying cells associated with bacterial infection.[4]

Tenderness to palpation or percussion over the affected sinuses is one of the clinical hallmarks of acute and chronic rhinosinusitis. This sign may equate with the subjective sensation of pain experienced by patients with sinusitis and as such may be indicative of either excessive peripheral or central sensitisation.[14] Bodily pain is increased in patients with CRS awaiting endoscopic sinus surgery and this improves clinically after surgery.[15] Inflammation due to the production of inflammatory substances lowers the sensory threshold in the dorsal spinal cord altering the processing of sensory messages from the surrounding soft tissues and musculature.[14] In recent years the role of the glia in the modulation of pain perception has been increasingly recognized.[4] Increased muscle tenderness in CRS was described by Naranch et al[7] and could potentially be a factor in the association between body pain and CRS described by Chester et al.[15]

A sinus infection does not cause pain; it simply influences sensory thresholds. Some patients with infective sinusitis do not experience pain. A lowering of the sensory thresholds by other causes might also be responsible for the same symptoms. Patients with fibromyalgia and tension headache, who also have nervous system sensory and pain regulation abnormalities, can report exactly the same sinus symptoms. The key to successful nasal and sinus surgery in this situation lies not only in the surgery itself, but in careful patient selection. In patients who complain of chronic sinusitis in the absence of significant infective symptoms, particularly when it is refractory to antibiotic treatment, one should

always consider central sensitization. Co-morbidities such as anxiety, depression, fibromyalgia, irritable bowel symptoms and low back pain should make the surgeon very wary of operating.[3]

History taking

Clinical diagnosis is largely dependent upon an accurate history. The art involves listening to a patient's complaints and in so doing formulating ideas or hypotheses as to the problem. More specific questioning is then needed to support or refute the diagnostic possibilities – the hypothetico-deductive approach. The initial hypotheses generated depend on the clinician's experience the patient's age and gender, and the time course and site of the pain. Acute inflammatory causes are usually relatively obvious. If a tooth is involved, the patient will usually have attempted to make the diagnosis and have seen a dentist. The otolaryngologist becomes involved when the dentist has been unable to find an obvious cause for the pain.

In this situation, the search will be for evidence of infective sinusitis or referred musculoskeletal pain. In a periapical abscess situation, the patient may have had previous hot/cold tooth sensitivity, which has settled. Pressure on the tooth as well as regional gum tenderness helps isolate the offending tooth. Pain from the TMJ may be bilateral, and the patient may complain of ear, neck and temple pain. The pain can be intermittent, throbbing and provoked by jaw movement. Stress and cold wind typically make TMJ pain and tension headache pain worse, whereas heat makes it better. Many patients have symptoms reflecting a sensitised nervous system, such as night sweats, unexplained itch, tinnitus, irritable bowel or bladder symptoms, heavy painful periods and altered sensation on combing their hair, as well as pain problems elsewhere in the body.

Neuralgias are characterised by sudden, intense, lancinating, burning or stabbing pain lasting only from few seconds to less than two minutes. This pain is often triggered by sensory or mechanical stimuli. Trigeminal neuralgia is typically seen in older females, unilateral and located in the second and/or third divisions of the trigeminal nerve. Rarely, pontine tumours or multiple sclerosis need to be considered as a secondary cause. If a cough or sneezing makes the headache worse, a posterior fossa lesion may need to be considered.

Psychological wellbeing

Anxiety and depression have significant associations with migraine, tension headache, sinusitis and TMJ disorders. Depressive symptoms (eg, tiredness, hopelessness, lack of motivation) are important in deciding on treatment and in predicting treatment outcomes. A systems review of the patient often reveals issues with poor short term memory, chest pains, palpitations, mitral valve prolapse, shortness of breath, irritable bowel symptoms, poor sleep quality, cold hands and feet as well as tingling of the hands in an ulnar nerve distribution. Anxious patients typically have difficulty going to sleep whereas depressed patients tend to wake in the early morning.

Examination

The way a patient walks, the ease with which a patient sits and a patient's posture while sitting can all give vital clues. People who are depressed and

people with neck and shoulder pain often sit in a slumped posture with their head forwards and their shoulders rounded. People who are highly stressed tend to take small breaths, largely in their upper chest, and talk rapidly. They may yawn and sigh during the interview.

Palpation for muscle tenderness, and excessive reddening afterwards, provides important physical information about the state of the central nervous system. Sometimes there are subtle differences in swelling and redness between the two sides of the face. Pectoralis minor, trapezius, levator scapulae, sternocleidomastoid, the suboccipital muscles, masseter and temporalis are usually tender when examined using appropriate palpation techniques. Jaw joint and associated muscle tenderness together with limited and jerky jaw movements and clicking of the jaw joint may be found on clinical examination. The teeth can be examined for excessive wear (bruxism) or percussion tenderness. Alterations in facial sensation are best detected by comparing moving light touch between the two sides of the face in the three divisions of the trigeminal nerve.

An examination of areas away from the head and neck, such as the hands, abdomen and low back, often provides useful, additional information. Anxious people often have brisk reflexes and active bowel sounds. Frequently, their abdomens are diffusely tender. The low back, extensor forearm muscles and calves are often tender to palpation in pain patients as well. Patients with unilateral facial pain are often found to be tender all down that side of the body. The muscles may be shortened on that side.

Investigations

Radiology

Further urgent investigation and neurological evaluation are warranted for patients presenting with facial pain together with:

- grossly disturbed facial sensation
- facial palsy
- hearing loss and disturbed balance
- dysphagia
- dysphonia (huskiness) or
- dysarthria.

Diagnostic imaging tests (e.g. plain x-rays, MRI, axial CT scan) may help determine or exclude a cause of pain. The choice and timing of test varies according to clinical suspicion and the findings on physical examination.

Blood Investigations

Certain blood tests can be useful in evaluating patients presenting with tension headache and/or sinusitis.[16] Some patients, particularly females, will have an iron deficiency. Vegetarians and the elderly can have undiagnosed vitamin B12 deficiencies. Low vitamin D levels are frequently found in facial pain patients and these are also associated with poor immune function. People of any descent who have dark or brown skin, and females in particular, are often at significant risk of vitamin D deficiency while living in countries with temperate climates, particularly during winter. Blood tests for thyroid function are occasionally useful in tension headache patients, particularly those with associated depression. In a

person aged over 50 years with a rapidly developing headache, an erythrocyte sedimentation rate (ESR) test is mandatory (**Table 1**).

Table 1: Diagnostic criteria for temporal arteritis

• new headache
• onset at age ≥50 years
• abnormalities of the temporal artery on clinical examination
• raised ESR (≥50mm/h) or C-reactive protein >5mg/L
• abnormal findings on biopsy of temporal artery

Management

Depending on the clinical diagnosis a wide range of treatment options are available to help patients with orofacial pain. Psychological interventions such as cognitive behavioral therapy and relaxation work can be extremely useful.[17,18] Drugs that are commonly useful are Gabapentin and low dose Amitriptylline at night. Care is needed in prescribing opiates and benzodiazepines.

Conclusions

The view that migraine, tension headache and sinus headache represent discrete clinical conditions is not in keeping with basic clinical science. Around the head and neck other factors apart from sinus infection can also be related to facial pain. These factors cannot be neglected and also need to be considered as part of the diagnostic work-up. In chronic orofacial pain patients a multidisciplinary approach is often needed.

Red flags in facial pain

Further investigation and/or specialist referral is indicated when:

• the pain is new or has significantly changed
• there is significant associated nausea and vomiting
• the pain is unusually severe or persistent
• there is accompanying fever
• the pain is made worse by coughing, sneezing or a change in position
• there is a change in strength, coordination or senses
• there is drowsiness, difficulty thinking or concentrating
• the headache is progressively getting worse
• the headache wakes the patient from sleep
• the headache occurs for the first time in childhood or after age 50

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