

MORE ABOUT...HEADACHES

CRANIOMANDIBULAR DYSFUNCTION AND HEADACHES

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Temporomandibular disorder (TMD) is a term reflecting ailments of the temporomandibular joint (TMJ), the masticatory and cervical muscles. The skeletal entity involves an intra-articular inflammatory process. In support of this are increased levels of cytokines, prostaglandins, and 5-HT found in the synovial fluid of affected TMJs.¹ The pathophysiology of craniofacial muscle pain is unclear; therefore treatment is not causally directed. Masticatory muscle pain is a localised expression of a spectrum of myofascial disorders, with many similarities between TMD, tension-type headaches, and fibromyalgia. The prevalence of TMD pain among adolescents is 0.7 - 7%; women are more commonly affected than men in the ratio 3:1, with the highest incidence in the 20 - 40-year age group.⁵ Typical signs of TMD include joint noise, tenderness of masticatory muscles and limitation in the range of motion of the mandible. Patients often relate these signs and symptoms to headache, earache and sinusitis.

This article is limited to the differential diagnosis of the myofascial pain syndrome (MPS) and internal joint derangement (IJ) as contributing causes of craniofacial headaches.

Myofascial pain accounts for over 60% of TMD cases. It is characterised by tender areas (trigger points) involving the masticatory muscles as well as superior constrictor, sternomastoid and the posterior cervical muscles. Current theories hypothesise that macrotrauma (whiplash injury and facial trauma) or microtrauma (clenching or bruxing) disturbs normal or weakened muscle through injury or sustained contraction.

The MPS also has a psychophysiological component in that stress influences the pain and extent of this condition. At a biochemical level, patients with MPS

have significantly higher levels of catecholamines and 17-hydroxysteroids than symptom-free patients.

In addition to muscle tenderness, headache is a frequent symptom; concomitant symptoms may include blocked ears, tinnitus, difficulty with swallowing and painful limited mouth opening. Although myofascial pain is a functional disorder, it can lead to organic changes in the TMJ.

Treatment should be directed toward management rather than a definite cure inasmuch as the definitive causes of a psychophysiological disorder may be more difficult to eliminate. The ability of the patient to understand and accept the basis for their pain is essential in dealing with the problem.

Reduction in pain and muscle spasm can be achieved by guiding the patient in home heat physiotherapy. Ultrasound, electrogalvanic stimulation and postural correction are also indicated in selected patients. A non-steroidal anti-inflammatory drug (NSAID) is extensively used; in combination with an anxiolytic (benzodiazepines) it is more effective than the NSAID alone. Amitriptyline in low doses (10 - 25 mg/day) has proved beneficial. Injection of local anaesthetic at trigger points has been prescribed with varying degrees of success. Painful TMJs respond to occlusal appliance therapy in conjunction with the treatment modalities described.

The response to occlusal appliance therapy is mediated via alteration in the patient's muscle activity patterns. Patients in denial regarding stresses and anxieties will be refractory to all forms of treatment. Referral to a psychologist or psychiatrist is essential to rehabilitate emotional distress and cognitive behaviour patterns.

Internal derangement is an abnormal relationship of the articular disc to the mandibular condyle, fossa and articular eminence.⁵ The signs and symptoms are similar to those of myofascial pain syndrome (MPS), with joint noise a common feature.

Two forms of disc displacement are recognised:

- **Disc displacement with reduction.** The disc is displaced in the closed-mouth position but reduces to a

normal relationship to the mandibular condyle, fossa and articular eminence during opening or protrusion. The joint noise produced is described as clicking.

- **Disc displacement without reduction.** Similar to the above, but the disc does not reduce to a normal relationship during mandibular movements (closed lock, no click).

Mouth opening is restricted to 20 - 30 mm (normal: 40 - 50 mm) interincisal with deviation of the jaw to the affected side on opening. Pain is reportedly worse with 'closed-lock' due to compression by the condyle of the innervated and vascular posterior attachment ligament. Crepitus is considered to represent advanced disease and occurs as a result of movement across irregular surfaces and suggests a perforation of the disc.

There are no proven aetiological factors for internal derangements of the TMJ. Probable factors include microtrauma due to parafunction, whiplash neck injury and direct trauma to the mandible. As in MPS, women are affected more than men.

Diagnostic imaging is essential in the management of TMD. This includes panoramic and transpharyngeal films, tomography, CT scanning and MRI. Arthrography-video fluoroscopy for dynamic evaluation of the articular disc is an excellent diagnostic modality but is invasive. Patient signs and symptoms as well as the intuitive needs of the clinician drive the choice of diagnostic imaging. The management of internal joint derangement is, in the main, identical to those patients suffering myofascial pain.

Surgical interventions are reserved for patients who do not respond to conservative, reversible treatment. Surgical procedures include needle arthrocentesis, arthroscopic lavage and arthrotomy or open arthroplasty.

Conclusion

Temporomandibular joint dysfunction is a common and frequently seen problem. Understanding the pathophysiology and its progression is essential to management.

References available on request.