Surgical pathology of the mouth and jaws

R. A. Cawson, J. D. Langdon, J. W. Eveson

7. Disorders of the temporomandibular joints and periarticular tissues

Disorders of the temporomandibular joint can cause various combinations of limitation of movement of the jaw, pain, locking or clicking sounds. Pain, in particular, is a frequent cause of limitation of movement. Few of these complaints are due to organic disease of the joint and many are due to trauma. However, these functional disorders must be considered in the differential diagnosis.

Limitation of movement (trismus)

Trismus may be defined as inability to open the mouth due to muscle spasm, but the term is usually used for limited movement of the jaw from any cause. Inability to open the mouth fully is usually temporary and causes include the following:

Infection and inflammation in or near the joint. The main cause is acute pericoronitis with associated muscle spasm. Mumps can also cause temporary limitation of movement. Rare causes include suppurative arthritis, osteomyelitis, cellulitis and suppurative parotitis. Submasseteric abscess results in profound trismus (Chapter 2), but infection in the pterygoid, lateral pharyngeal or submandibular spaces may also cause varying degrees of trismus.

Mandibular block injections may cause inflammation of the muscles around the joint and oedema, either because of irritation by the local anaesthetic or by introduction of infection. Occasionally, limitation of movement may be more persistent, possibly due to organization of haematoma after the passage of the needle through the medial pterygoid muscle during the injection. If voluntary jaw exercises fail to achieve opening, forced manipulation under general anaesthesia may be necessary. Often the scar tissue can be felt to tear during the forced opening.

Injuries. Unilateral condylar neck fracture usually only produces mild limitation of opening with deviation of the jaw to the affected side, but closing into centric occlusion may be difficult. Bilateral displaced codylar fractures cause an anterior open bite with limited movement. Rarely, a fall on the chin can result in unilateral or bilateral dislocation of the condylar head into the middle cranial fossa and severe restriction of all movements. Less severe injuries frequently result in an effusion into the temporomandibular joint; both wide opening and complete closure are then prevented.

Any unstable mandibular fracture causes protective muscle spasm with limitation of movement. Patients suffering from displaced Le Fort II or III fractures often complain of limited opening, whereas in reality they are fully open with the jaws wedged apart by the displaced middle third. Reduction of the fracture allows closure.

Tetanus and tetany. These are rare causes of masticatory muscle spasm. Trismus (lockjaw) is a classical early sign of tetanus which, though rare, must be excluded because of its high mortality. This possibility should be considered whenever a patient develops acute

severe limitation of movement of the jaw without local cause but has had a penetrating wound, even if small, elsewhere.

Tetany is most likely to be seen as a result of anxiety and hyperventilation syndrome.

Temporomandibular pain dysfunction syndrome. Pain dysfunction syndrome is one of the most common causes of temporary limitation of movement of the temporomandibular joint, as discussed later.

Hysterical trismus. Inability to open the mouth is occasionally the main symptom in disturbed patients.

Drugs. Phenothiazine neuroleptics can cause tardive dyskinesia with uncontrollable, involuntary grimacing or chewing movements. Tardive dyskinesia is typically a result of long-term treatment: unlike drug-related Parkinsonism, there is little response to treatment. Metoclopramide, which has phenothiazine-like properties, can cause limitation of movement of the jaw.

Management

In all these conditions the essential measure is to relieve the underlying complaint.

Permanent limitation of movement (ankylosis)

Management of ankylosis depends on its aetiology and can be classified for surgical purposes as follows:

1. Pseudo-ankylosis (mechanical interference with opening, well away from the joint).

2. False ankylosis (extracapsular limitation of opening).

3. True ankylosis (intracapsular fixation).

Pseudo-ankylosis

Causes include:

- *trauma:* depressed fracture of the zygomatic bone or arch

- *hyperplasia:* developmental overgrowth of the coronoid process

- *neoplasms:* osteochondroma, osteoma, osteosarcoma of the coronoid process

- *miscellaneous:* myositis ossificans, congenital anomalies.

Extracapsular ankylosis

Causes include:

- *trauma:* periarticular fibrosis (wounds/burns); posterior or superior dislocation; long-standing anterior dislocation

- *infection:* chronic periarticular suppuration

- *neoplasia:* fibrosarcoma of the capsule; chondroma or chondrosarcoma

- *periarticular fibrosis:* irradiation; oral submucous fibrosis; progressive systemic sclerosis.

Irradiation

The penetrating power of modern sources such as cobalt 60 or linear accelerators are such that high doses can damage deep tissues without a superficial burn. Radiation involving the region of the masticatory muscles, for the treatment of a maxillary tumour for instance, can lead to fibrosis of muscle and fibrous adhesions to the surrounding fascial layers, producing a fixed mass bound to the jaw. When this happens there is severe limitation of opening the mouth or complete ankylosis.

Treatment is difficult and may involve division of muscle attachments from the jaw or section of the angle or body of the mandible to produce a false joint. Bone surgery is complicated by the risk of osteoradionecrosis and infection (Chapter 2).

In children, irradiation can result in inhibition of condylar cellular activity with subsequent inhibition of mandibular growth.

Oral submucous fibrosis

Oral submucous fibrosis is a disease which produces changes similar to those of scleroderma but limited to the oral tissues. The disease is virtually only seen in those from the Inidian subcontinent.

In their review, Pillai et al (1992) conclude that the aetiology is unknown, but suggested factors have been irritants (chillis) in the diet, betel (areca nut) chewing, genetic factors or a questionable relationship to progressive systemic sclerosis. However, there are no immunological findings to suggest any relationship between oral submucous fibrosis and systemic sclerosis.

The main contributor is thought by Jayanthi et al (1992) to be the chewing of a preparation *(pan)* which typically consists of areca nut, tobacco and lime wrapped in betel leaf. While tobacco may possibly contribute to the premalignant potential of this disease, similar changes are not seen in the oral mucosa from long-term use of oral tobacco in other countries where the typical response is keratosis (Chapter 9).

Experimentally, an alkaloid component of the areca nut, arecoline, can induce fibroblast proliferation and collagen synthesis and it may penetrate the oral mucosa to cause progressive crosslinking of collagen fibres. Susceptibility to submucous fibrosis also appears to be related to specific HLA types. The fact that oral submucous fibrosis is an almost specifically Indian disease also strongly suggests a genetic factor, and the use of areca nut is mainly restricted to that subcontinent. It is probably the tobacco content of the betel quid, rather than an areca nut, which induces epithelial atypia and any tendency to malignant change (Chapter 10). Pillai et al (1992) conclude, therefore, that the aetiology of this disease is multifactorial.

Clinically, symmetrical fibrosis of such sites as the buccal mucosa, soft palate or inner aspects of the lips is characteristic. The overlying mucosa may be normal or there may be a vesiculating stomatitis. Fibrosis causes extreme pallor of the affected area which becomes so hard that it cannot be indented with the finger. Ultimately, opening the mouth may become so limited that eating and dental treatment become increasingly difficult and tube feeding may become necessary.

Microscopy

The subepithelial connective tissue becomes thickened, hyaline and avascular, and there may be infiltration by modest numbers of chronic inflammatory cells. The epithelium usually becomes thinned and may show atypia. Underlying muscle fibres undergo progressive atrophy.

Investigation

In patients from the Indian subcontinent, with these lesions and especially with the characteristic histological changes on biopsy, the diagnosis can rarely be in doubt. Scleroderma should be readily differentiated by the dermal and visceral involvement, the less severe oral changes and the autoantibodies.

Management

Treatment is unsatisfactory. Patients must stop the habit of betel chewing and intralesional injections of corticosteroids may be tried in associated with muscle-stretching exercises to prevent further limitation of opening, but the benefit is not great. Wide surgical excision of the affected tissues including the underlying buccinator muscle, together with skin grafting, can be carried out, but is likely to be followed by relapse. Borle and Borle (1991), in comparing treatments in 326 patients, concluded that injections could be hazardous and conservative treatment by topical applications of such preparations as corticosteroids or vitamin A derivatives, and oral iron, were as effective. However, no form of treatment is more than palliative and in some cases operative treatment may become unavoidable. Jayanthi et al (1992) suggest that the most important measure is prevention and that use of pan should be forbidden in patients. This may be initially effective, but fibrosis often recurs even though the patient complies. Regular follow-up is important because of the premalignant potential of this disease.

Progressive systemic sclerosis (scleroderma)

Systemic sclerosis is an uncommon connective tissue disease characterized by widespread subcutaneous and submucous fibrosis. Though the most obvious feature is the progressive stiffening of the skin, the gastrointestinal tract, lungs, heart and kidneys can also be affected. Unlike oral submucous fibrosis, the prognosis is poor as a result.

Systemic sclerosis is a connective tissue (collagen vascular) disease. There are circulating antinuclear antibodies in about 50% of patients, antinuclear antibodies or antibodies to RNA. An antinuclear antibody Scl 70 appears to be restricted to this disease, but is found in only about 20% of patients. Other laboratory findings are a normochromic anaemia and a raised ESR.

Clinically, women between the ages of 30 and 50 years are predominantly affected. Raynaud's phenomenon is the most common early manifestation, often associated with arthralgia. A hallmark of the disease is involvement of the hands, causing such changes as atrophy of or ischaemic damage to the tips of the fingers and contractures preventing straightening of the fingers.

The skin becomes thinned, stiff, tethered, pigmented and marked by telangiectases. The head and neck region is involved in over 75% of patients and, in a minority, symptoms start there. Narrowing of the eyes and taut, mask-like limitation of facial movement (Mona Lisa face) can give rise to a characteristic facial appearance. The lips may be constricted (fish mouth) or become pursed with radiating furrows. Occasionally, involvement of the periarticular tissues of the temporomandibular joint, together with the microstomia, may greatly limit opening of the mouth. Involvement of the oral submucosa may cause the tongue to become stiff and narrowed (chicken tongue), but the clinical effects on the oral soft tissues are tpically relatively minor compared with those seen in oral submucous fibrosis. Widening of the periodontal membrane space is another abnormality characteristic of systemic sclerosis, but seen in fewer than 10% of cases. The mandibular angle may be resorbed or rarely these is gross extensive resorption of the jaw. Sjögren's syndrome also develops in a significant minority.

Microscopy

There is great thickening of the subepithelial connective tissue, degeneration of muscle fibres and atrophy of minor glands. The collagen fibres are swollen and eosinophilic. There are scattered infiltrates of chronic inflammatory cells which are frequently perivascular, and arterioles typically show fibro-intimal thickening of their walls.

Complications and prognosis

The main disabilities caused by systemic sclerosis are dysphagia and pulmonary, cardiac or renal involvement. Visceral disease, particularly dysphagia and reflux oesophagitis, are common and are sometimes the initial symptoms. Pulmonary involvement leads to impaired respiratory exchange and, eventually, dyspnoae and pulmonary hypertension. Cardiac disease can result from the latter or myocardial fibrosis and these are potential contraindications to general anaesthesia. Renal disease secondary to vascular disease is

typically a late effect; it leads to hypertension and is an important cause of death. The overall 5-year survival rate is approximately 70%.

No specific treatment is available. Immunosuppressive drugs are ineffective. Penicillamine may be given to depress fibrous proliferation but can cause loss of taste, oral ulceration, lichenoid reactions and other complications. Symptomatic measures such as those described for oral submucous fibrosis can be used to prevent undue limitation of jaw movement. Complications such as renal failure are managed by conventional means.

Localized scleroderma (morphoea)

Morphoea is characterized by similar tissue changes to systemic sclerosis, but limited to a single area of skin and without visceral disease or systemic effects. A typical manifestation is involvement of the side of the face causing an area of scar-like contraction like a sword wound *(coup de sabre)* and there may be atrophy of the underlying bone. Facial hemiatrophy may be the result from morphoea starting in childhood.

Intracapsular ankylosis

Important causes include:

- *Trauma:*
    - Intracapsular comminuted fracture. Intracapsular fracture of the condyle disorganizes the joint. Bleeding may be followed by organization and bone formation. Early mobilization of such injuries should prevent bony ankylosis.
    - Penetrating wounds.
    - Forceps delivery at birth.

- *Infection:*
    - Otitis media / mastoiditis.
    - Osteomyelitis of the jaws.
    - Haematogenous - pyogenic arthritis.

    Acute pyogenic arthritis is exceedingly rare but, if treated inadequately, ankylosis follows.

- *Systemic:*
    - Juvenile arthritis.
    - Psoriatic arthropathy.
    - Osteoarthritis (rarely).
    - Rheumatoid arthritis (rarely).

- *Neoplasms:*
    - Chondroma, osteochondroma, osteoma, sarcoma, fibrosarcoma, synovial sarcoma.
    - Metastases to the condyle.

- *Miscellaneous:*

- Synovial chondromatosis.

Management

These conditions are described in previous or subsequent sections. If ankylosis cannot be prevented, the objectives of surgery are to establish joint movement and function, to prevent relapse, to restore appearance and occlusion in the adult and to achieve normal growth and occlusion in the child by interceptive surgery and orthodontics.

Simple division of bone and fibrous tissue at the articular surface (osteoarthrotomy) is seldom indicated and results in early relapse. Osteoarthrectomy, in which a block of bone is removed either as a condylectomy or gap arthroplasty, is more effective and is the usual technique employed in adults. Relapse is prevented by interposition of a temporalis muscle flap, silicone rubber or metal. In the growing patient, joint reconstruction using a free costochondral bone graft gives better results as in many causes the graft will grow with the patient, preventing subsequent facial deformity.

Other techniques sometimes used are ramus osteotomies (Ward's technique) in which a false joint is created in the ramus. Relapse is prevented by interposition of a cobalt-chrome cap or angle ostectomies (Esmarch's technique) in which a section of bone is removed at the angle and bony union prevented by the interposition of masseter and medial pterygoid muscles. Occasionally, the joint is reconstructed with a cobalt-chrome or titanium prosthesis articulating in a silicone rubber articular fossa. However, long-term results with such prostheses have been disappointing and have resulted in much litigation.

Arthritis and other causes of pain in or around the joint

The main causes are as follows:

- *Injury.* Dislocation, joint effusion or fractures of the neck of the condyle can cause pain of varying severity, but surprisingly often, fractures in this region pass unnoticed.

- *Infection and inflammation.* Acute pyogenic arthritis and osteomyelitis are rare but exceedingly painful.

- *Rheumatoid and other arthritides.* The temporomandibular joints tend to be much less severely affected than other small joints, although if specifically asked, many patients with rheumatoid arthritis admit to temporomandibular joint symptoms, but overall, pain is not conspicuous. Pain associated with osteoarthritis of this joint is even more uncommon.

- *Vascular disease.* Cranial arteritis is an important cause of ischaemia and pain in the masticatory muscles while chewing.

- *Muscle spasm.* Probably the most common causes of pain in the region of the temporomandibular joint is the so-called pain dysfunction syndrome, as discussed later.

- *Salivary gland disease.* Painful conditions of the parotids (inflammatory or neoplastic) can cause pain in this region.

- *Ear disease.* Otitis externa, otitis media and mastoiditis are potent causes of pain referred to the temporomandibular joint.

Rheumatoid arthritis

The main feature of this common multi-system disease is chronic inflammation of many joints, pain, progressive limitation of movement, varying degrees of constitutional upset and immunological abnormalities.

Rheumatoid arthritis is the only important inflammatory disease of the temporomandibular joints, but is nevertheless an infrequent cause of significant symptoms there.

Clinical features

Women are affected, particularly in the third and fourth decades. Loss of weight, malaise and depression are common. The smaller joints are mainly affected (particularly those of the hands), and the distribution tends to be symmetrical.

The main symptoms of temporomandibular joint involvement are crepitus and limitation of movement, but severe pain is surprisingly uncommon. The temporomandibular joints are never involved alone and the other affected joints usually dominate the picture. Among 100 patients with rheumatoid arthritis sufficiently severe for them to attend a large rheumatology centre, Chalmers and Blair (1973) found 71% to have clinical abnormalities of the temporomandibular joints (compared with 41% for controls), while 79% showed radiographic abnormalities compared with 34% for controls. In spite of the fact that these were middle-aged patients with long-standing disease, pain was *not* significantly more common than in control patients. The main clinical abnormalities were limitation of opening or crepitus, both of which were considerably more common than in other patients.

Radiography

Flattening of the condyles with loss of countour and irregularity of the articular surface are typical findings. The joint space may be widened by exudate in the acute phases, but later narrowed. The underlying bone may be osteoporotic and the margins of the condyles may be irregular. There may be limitation of condylar movement.

Microscopy

There is proliferation and hypertrophy of the synovial lining cells and infiltration of the synovium by dense collections of lymphocytes and plasma cells. The inflammatory cells are often arranged as focal aggregates with germinal centres. There is effusion into the synovial fluid which contains neutrophils and fibrinous exudation from the hyperaemic vessels on to the surface of the synovial membrane. A vascular, inflamed mass of granulation tissue (pannus) spreads over the surfaces of the articular cartilages from their margins and is

followed by death of chondrocytes and loss of intercellular matrix. Fibrous adhesions form between the joint surfaces and the meniscus. The meniscus may eventually be destroyed and inflammatory changes in the ligaments and tendons can lead to fibrous ankylosis or loss of stability of the joint.

Management

Diagnosis is based on the clinical, radiographic and autoantibody findings. Serum IgG is usually raised and a variety of autoantibodies particularly rheumatoid factor and antinuclear antibodies may be detected.

Generally speaking, though rheumatoid arthritis is a common disease, it is not a major cause of temporomandibular joint pain, and radiographic changes appear to be more severe than the symptoms. The mainstay of treatment is the use of non-steroidal anti-inflammatory drugs, but any gross abnormalities of occlusion, such as overclosure, should be corrected to reduce stress on the temporomandibular joints.

Osteoarthritis

Osteoarthritis has long been thought to be a degenerative (wear-and-tear) phenomenon, particularly affecting chronically overstressed joints such as the hips and knees in elderly patients. However, it is now clear that osteoarthritis is a systemid cisease: trauma is only a contributory factor, but heavy stress on affected joints is the cause of pain.

Osteoarthritis of the temporomandibular joint is a post mortem finding in many elderly patients, is occasionally seen by chance in radiographs, but is not a cause of significant symptoms. Nevertheless, the conviction that osteoarthritis is a significant cause of temporomandibular joint pain, even in young persons, is widely held in the USA, to justify surgical interference. However, the argument that the temporomandibular joint is prone to osteoarthritis because of the heavy stresses it has to bear is hardly consistent with the types of operation that are used. It would be unlikely, for example, that excision of the head of the femur would be appropriate treatment for osteoarthritis of the hip.

In the rare event that a patient has pain associated with osteoarthritis of the temporomandibular joint, any factor contributing to stess on the joint should be relieved. Non-steroidal anti-inflammatory analgesics in generous doses are the main line of treatment. A high condylar shave is also effective in reducing symptoms either by denervating the joint or by decompressing the condylar head.

The concept of temporomandibular joint 'internal derangement' is also open to a variety of interpretations. Some regard it as an underlying cause of pain dysfunction syndrome, others that it is a contributory cause of temporomandibular joint osteoarthritis. After an International Conference on Temporomandibular Joint Surgery, Goss (1993) commented disarmingly that 'it was noted that facts are commonly not allowed to interfere in discussions of TMJ problems'. Although some degree of consensus was reached at this conference it was apparent that neither the pathogenesis nor the effectiveness of surgical treatment of this condition were firmly established. It therefore remains too controversial a subject to discuss here.

Other types of arthritis

Many other types of arthritis can affect the temporomandibular joints but rarely do so. They include psoriatic arthritis, the juvenile arthritides, gout, ankylosing spondylitis, Lyme disease and Reiter's disease (Könönen, 1992). Psoriatic arthropathy can cause ankylosis of temporomandibular joint (Miles and Kaugars, 1991; Koorbusch et al, 1991). Some of the variants of juvenile arthritis such as Still's disease are severe and disabling. Destruction of the condylar head, severely limited opening and secondary micrognathia have been reported by Larheim and Haanaes (1981). Management is essentially that of the underlying disease.

Cranial (giant cell) arteritis

Cranial arteritis occasionally causes ischaemic pain in the masticatory muscles in elderly patients and should be considered in patients over middle age who complain of headache and pain on mastication.

Clinically, women, usually after the age of 55, are predominantly affected. The disease may start with malaise, weakness, low-grade fever and loss of weight. Severe throbbing headache is the most common symptom. The temporal artery frequently becomes red, tender, firm, swollen and tortuous. In 20% of patients, there is ischaemic pain in the masticatory muscles, comparable to and often misnamed jaw claudication (claudication is, literally, *limping*). More important is involvement of the ophthalmic artery causing disturbances of vision or sudden blindness. Polymyalgia rheumatica may be associated. There is then weakness and pain of the shoulder or pelvic girdles associated with the febrile symptoms. The erythrocyte sedimentation rate is usually greatly raised.

Microscopy

Inflammation involves the arterial media and intima. The infiltration by mononuclear cells is typically associated with multinucleate cells. Intimal damage leads to formation of thrombi, which usually become organized, and there may be severe damage to the internal elastic lamina, sometimes going on to complete destruction. Healing is by fibrosis, particularly of the media, thickening of the intima and partial recanalization of the thrombus. Lesions skip short lengths of an artery and a biopsy should be at least 3 cm long.

Management

The possibility of complications, particularly blindess which develops in up to 50% of untreated patients, makes it essential to start treatment early. Systemic prednisolone (starting with 60 mg/day) should be given on the basis of inflamed scalp vessels and a high (> 70 mm/hr) ESR. Corticosteroids are usually quickly effective and should be continued until the ESR falls to normal.

Pain dysfunction syndrome

Pain dysfunction syndrome is probably the most common cause of pain in the temporomandibular joint region. It predominantly affects young women and typical features, in varying combinations, are pain, clicking sounds from the joint and limitation of movement.

Despite intensive investigation over several decades and a voluminous literature, no organic disease of the temporomandibular joints has been convincingly demonstrated. However, a significant number of patients have been noted by Buckingham et al (1991) to have hypermobility syndrome.

Aetiology

The female to male predominance is nearly 4 to 1. Most patients are aged between 20 and 40 years. Older persons are rarely affected. There is no evidence that the disease is progressive and goes on to produce permanent changes, or that there is disease of the joint itself. Symptoms may be sufficiently troublesome to justify treatment, but in the majority of patients symptoms resolve after two or three years, even without treatment.

Malocclusions have been held responsible, but the evidence is unconvincing. Lack of posterior occlusal support sometimes appears to be a contributory factor. Trauma, particularly from minor injuries, is a possible factor, but the evidence is anecdotal. However, some abnormal habits seem to contribute. An obvious one is habital grinding, particularly at night time. The habit of holding a telephone receiver between the shoulder and chin for prolonged periods, to free the hands for writing, and the lateral deviation of the chin to grip the telephone can result in instability of the meniscus on the opposite side. Violinists occasionally have similar trouble. Woodwind and brass players sometimes develop temporomandibular joint symptoms when their embouchure involves abnormal posturing of the mandible. Other patients aggravate their symptoms by habitually and forcefully biting into hard food; in the susceptible patient this too can lead to instability of the meniscus, particularly in patients with a deep overbite. Modification of these habits can lead to relief of symptoms.

Electromyographic and arthrographic studies suggest that incoordination of the upper fibres of the lateral pterygoid muscle attached to the anterior margin of the joint meniscus results in inappropriate movements of the meniscus during opening and closing. This incoordination results in the characteristic clicking and locking.

Patients with pain dysfunction syndrome tested by means of standard psychological scoring methods also appear to have significantly higher scores for neuroticism, anxiety and related factors affecting muscle tension, and significantly lower pain thresholds than controls. Psychiatric factors probably underlie pain dysfunction syndrome in many cases and, in older patients especially, more serious underlying psychopathology has been reported (Kaban and Belfer, 1981).

To summarize, pain dysfunction syndrome typically affects only a restricted group of the population. It has little organic basis, is self-limiting and does not cause permanent damage or degenerative arthritis later in life. Defective neuromuscular coordination and areas of fatigue or spasm in the masticatory muscles appear to be the main cause of the symptoms.

Clinical features

The onset is usually gradual, but a few patients ascribe the onset to violent yawning, laughing or some similar incident. Pain is usually one-sided and typically a poorly localized dull ache made worse by mastication. The condylar region is the main site. Pain is typically

felt in front of the ear, but sometimes felt lower down the ramus, occasionally at the angle of the jaw and, more rarely still, in the neck or occiput.

Limitation of opening or 'locking' of the jaw in the partially open or closed position is frequently associated. A clicking sound associated with deviation, when the mouth is opened or closed, is common. Defects of occlusion can often be found, as they can in asymptomatic patients.

Management

In view of the absence of objective signs, diagnosis is largely by exclusion. Rheumatoid or other arthritis should be excluded by appropriate investigation. Tenderness or swelling of the joint suggests organic disease. Trigeminal neuralgia should be suspected in patients of middle age or over with severe pain, since it can occasionally be triggered by movement of the jaw and is also unassociated with any detectable abnormalities. Trigeminal neuralgia is subject to spontaneous remissions and if remission follows the fitting of an appliance then the wrong conclusions can be drawn. However, the pain of trigeminal neuralgia is far more acute than that of pain dysfunction syndrome and stabbing in character.

Movements of the head of the condyle can be felt through the overlying skin or with a finger in the external auditory meatus. With practice, abnormal excursions or asymmetrical movement of the condyles or crepitus may be detected.

Lateral radiographs of the joints should be taken with the jaw in the open and closed positions, to make sure that movements are equal and not excessive in either direction. The main value of radiographs is to exclude organic disease shown by increased size of the joint space or damage to or deformity of the joint surfaces. Signs of hypermobility should also be sought.

Arthrography, magnetic resonance imaging or direct arthroscopy via a fine endoscope can all be used to demonstrate an unstable meniscus which is often displaced anteriorly. These investigations are invasive or costly or both, but merely serve to confirm the clinical findings. They should be reserved for patients resistant to conservative management or when there is real doubt about diagnosis.

Treatment options may be considered under six main headings:

- reassurance
- use of appliances
- muscle training and modification of habits
- occlusal equilibration
- antidepressive treatment
- surgery.

Many other forms of treatment (sufficient in fact to fill large textbooks) have been recommended, but this is merely another reflection of how little is known about this disorder.

Reassurance. Once the diagnosis has been established, reassurance alone is sometimes effective. A benzodiazepine may be helpful for its anxiolytic and muscle relaxant action, but should only be used for a limited period. However, there is wide individual variation in patients' response and the risk of dependence should be borne in mind.

Overlay appliances. In many cases an acrylic overlay appliance covering the occlusal surfaces of the teeth is effective. This allows free occlusion without cuspal interference and tends to relieve abnormal grinding habits. It is not uncommon for these patients to be over-closed with a deep anterior overbite and locked occlusion. This also can be managed by means of an appliance, using an anterior bite plane, but full occlusal cover is essential to prevent over-eruption of the posterior teeth.

Overlay appliances should be worn day and night in severe cases and may have to be replaced or built up as necessary, as they wear down during use. The appliances should be firmly fitting and retained by means of cribs. Patients usually accommodate quickly to these appliances which, after a day or so, interfere little with speech or mastication. In many cases, the relief of pain or other symptoms is a strong impetus to continue wearing the appliance.

Overall, the use of these types of appliances is probably the most satisfactory form of treatment. They are usually effective and are much less time-consuming than other methods.

Muscle exercises and modification of habits. There is no firm evidence that this time-consuming method of treatment produces any more satisfactory results than the use of overlay appliances.

Antidepressive treatment. Pain dysfunction syndrome may sometimes be part of the spectrum of atypical facial pain. Psychiatric assessment and antidepressive treatment is appropriate and successful in such cases.

Occlusal equilibration. Objective evidence of the value, other than a placebo effect, of detailed occlusal analysis and equilibration has yet to be established.

Surgery. In the USA in particular, surgery is widely used. Over the years many techniques - closed condylotomy, open condylotomy, high condylar shave, condylectomy, capsular rearrangement and reattachment of the joint meniscus - have been advocated. In the short term most of these procedures provide symptomatic relief. This may be due to a placebo effect, surgical denervation of the joint or perhaps modification of some defect such as a dislocated meniscus. However, in the long term the relapse rate is high, and surgery should therefore be reserved for those patients with objective organic joint disease who have failed to respond to more conservative management.

To summarize, pain dysfunction syndrome frequently responds to a relatively simple type of occlusal appliance, and in some patients this may be usefully combined with antidepressive treatment. Harris et al (1993) have reviewed the prosthetic and psychiatric aspects of management of this complaint, and possible medicolegal problems. Surgical interference should generally be avoided.

'Costen's syndrome'

This syndrome was said to comprise headache, ear symptoms (tinnitus or deafness) and burning pain in the tongue and throat and was ascribed to overclosure causing excessive backward movement of the head of the condyle.

This syndrome does not exist, but the term 'Costen's syndrome' persists in medicine and is often thought to be synonymous with pain dysfunction syndrome.

Condylar hyperplasia

Condylar hyperplasia is a rare, usually unilateral, overgrowth of the mandibular condyle. It causes facial asymmetry, deviation of the jaw to the unaffected side on opening, and a crossbite. The condition usually manifests after puberty and is slowly progressive. Pain in the affected joint is variable. Serial models and radiographs are used to monitor the condition. An isotope bone scan confirms the diagnosis by showing greatly increased bone activity in the affected condyle. If the condition is still active at the time of diagnosis, an intracapsular condylectomy should be performed to destroy the active growth centre. If the disease has stabilized - usually at the end of puberty or shortly afterwards - corrective osteotomies should restore the occlusion and facial symmetry.

Neoplasms

Tumours of the temporomandibular joint are rare, but may arise from the condyle - either the bone or articular cartilage - or from the joint capsule. Metastatic tumours such as those from the breast, prostate and thyroid occasionally metastasize to the condylar head (Chapter 6).

Though otherwise rare in the facial skeleton, osteochondroma (Chapter 5) is probably the most common tumour of the condyle or coronoid process, as reviewed by Kerscher et al (1993) who found 30 reports of coronoid osteochondromas, while Forssell et al (1985) were able to find 26 examples of condylar osteochondromas up till then. Osteoma and chondroma and their malignant counterparts may develop, but apart from their interference with joint function and disturbance of occlusion, their behaviour and management are the same as when they arise elsewhere in the jaws. Condylectomy may be required for a neoplasm such as an osteochondroma and an external approach may be used.

Synovial sarcoma is an exceptionally rare tumour and, paradoxically, arises more frequently in the soft tissues adjacent to joints, such as tendons or tendon sheaths, rather than in joint cavities, or in soft tissues unrelated to joints (Chapter 13). White et al (1992), in reporting a synovial sarcoma of the temporomandibular joint, could find only two earlier reports.

Loose bodies in the temporomandibular joints

Loose bodies are rare in the temporomandibular joints compared with other joints. The following types of loose bodies are recognized:

- *Fibrinous:*
  tuberculosis or haemorrhage
  pyogenic arthritis

- *Fibrous:* tuberculosis, syphilis or haemorrhage

- *Cartilaginous:* fragments of meniscus (1-3 bodies)

- *Osteocartilaginous:*
  osteochondritis dissecans (1-3 bodies)
  intracapsular fractures (1-3 bodies)
  osteoarthritis (1-10 bodies)
  synovial chondromatosis (50-500 bodies)
  rheumatoid arthritis
  tuberculosis or syphilis

- *Foreign bodies:* following trauma or surgery.

Osteochondritis dissecans

This is the main cause of loose bodies in joints, most commonly in the knee. It is believed to result from trauma causing an area of subchondral bone to undergo avascular necrosis with subsequent degenerative changes in the overlying articular cartilage. This area subsequently separates to produce one or several loose bodies. In the temporomandibular joint, the patient has discomfort and episodes of locking. The loose body may be seen on tomography, arthrography or arthroscopy. Surgical removal of the loose body, which is always in the lower joints compartment, relieves the symptoms.

Synovial chondromatosis

In this disease, foci of cartilage develop in the synovial membrane; it is thought to be a benign neoplasm. Synovial chondromatosis rarely affects the temporomandibular joint, but can cause swelling and limitation of movement, with deviation to the affected side on opening. Conventional radiographs may show radiopaque masses in the region of the joint capsule, but if no abnormality may be seen, MRI should reveal multiple cartilaginous nodules and distension of the capsule. Both upper and lower joint compartments are involved and exceptionally rarely the process may extend into the cranial cavity (Sun et al, 1990).

Microscopically, there is proliferation and atypia of chondrocytes in the subsynovial connective tissue, with formation of microscopic or larger nodules of cartilage. These nodules enlarge, and may calcify and escape into the joint cavity. The appearance of the chondrocytes is such that it is possible to mistake the lesion for a chondrosarcoma if the pathologist is not made aware of the clinical history and operative findings. Fujita et al (1991) demonstrated immunhistochemically that most of the nuclei of the chondrocytes were inactive and that malignancy could be excluded.

Removal of the loose bodies and the whole of the affected synovium are curative. Incomplete excision can be followed by recurrence.

Dislocation

The temporomandibular joint may become fixed in the open position by anterior dislocation; this is due to forcible opening of the mouth by a blow on the jaw, or during dental extractions under general anaesthesia. In the latter case, the condition should be noticed immediately. It must be corrected before the patient recovers consciousness, by pressing downwards and backwards on the lower posterior teeth. Occasionally a patient will dislocate spontaneously while yawning. Epileptic patients sometimes also dislocate during a fit.

Occasionally the dislocation remains unnoticed and, surprisingly, a patient may tolerate the disability and discomfort for weeks or even months. In these cases, effusion into the joint, following injury, becomes organized to form fibrous adhesions. When this happens, manual reduction may be impossible and open reduction, with division of adhesions, must be carried out.

Recurrent dislocation

Recurrent dislocation of the temporomandibular joint is more common and is typically seen in adolescent girls and young adults. It is a typical feature of floppy joint syndromes, notably Ehlers-Danlos and Marfan's syndromes or the benign type with no underlying systemic disorder.

Techniques advocated for the management of recurrent dislocation have included surgical reduction of the articular eminence (eminectomy), augmentation of the eminence (by bone grafting or by down-fracture of the malar arch using the Dautrey procedure or down-fracture of the eminence with interpositional grafting) and capsular plication to limit condylar movement. Augmentation of the eminence by bone graft or down-fracture of the eminence are overall the most successful procedures.