DURING the past 15 years, I have had the good fortune to be in a situation that provided clinical experience with disorders of the temporomandibular joint. For evaluation, clinical observations must be related to scientific facts. I will therefore discuss the anatomy and physiology of the temporomandibular joint and associated structures from the only point of view that I can—as a clinician.

Accurate diagnosis and effective management rest upon a knowledge of anatomy and physiology. The importance of such knowledge is illustrated by an examination of the bases for the two most widely practised methods of treatment: the altering of the occlusion of the teeth and the injection of a sclerosing agent into the joint. These methods are based on two different concepts: 'bite closure' and 'hypermotility'.

The premise of 'bite closure' is that loss of posterior teeth or excessive wear changes the positions of the condylar heads. Such 'alteration' at first believed to result in degenerative joint changes (Prentiss, 1918) was extended in time to include a miscellany of symptoms such as deafness, pain, tinnitus and vertigo (Monson, 1921; Costen, 1934; Travell & Rinzler, 1952). Treatment consisted of bite opening by various dental means.

'Hypermotility' believed due to 'lax ligaments' was determined by the symptoms of pain and clicking with a visible bulge caused by the condylar head at maximal mandibular opening. Treatment consisted of the intra-articular injection of a sclerosing agent with the aim of tightening the 'lax ligaments' by fibrogenesis (Schultz, 1937 and 1947).

'Bite closure' and 'hypermotility' are essentially mechanical concepts. The first concerns itself with the position of the condylar head in the closed position, the second at maximal opening. Neither concept has a sound anatomical or physiological basis.

'Bite closure' grew out of actual error as well as erroneous interpretations of anatomical knowledge resulting from the attempt to explain living function in terms of dead anatomy. Hypermotility, on the other hand, evolved as a result of a mistaken understanding of joint physiology. The fact is that motion in the temporomandibular articulation is directed much more by the musculature and much less by the shape of the articulating bones and articular ligaments than in other joints. Hence when mandibular dysfunction occurs it is not caused by 'lax ligaments' but by some disturbance of the intricate neuromuscular mechanisms controlling mandibular movement.

Since treatment stems from concept, concepts lacking anatomical and physiological validity have led to treatment methods which are ineffectual at the best or potentially harmful at the worst. There are a number of reasons for this unfortunate direction of development.

One, and perhaps the most important, is that the temporomandibular joints have been of great interest to dental surgeons studying these joints primarily in
the hope of constructing various devices that would mechanically reproduce
individual mandibular movements. This pragmatic approach emphasised the
uniqueness of the temporomandibular joint.

That the craniomandibular articulations have many unusual features cannot
be denied. Actually the temporomandibular joints consist of a coupled pair of
double joints, each one divided into an upper and lower compartment by an
articular disc. But these are joints, nevertheless, having much in common as far
as basic structure and function is concerned with other freely movable joints.
What is different is not the temporomandibular joints as such but their articulating
complexes. These, unlike any other bones, contain teeth. What is then really
unique is not the joints as such but the physiological system of which they are a part.

The stomatognathic system is a complex and versatile organ system; the
main, but not the exclusive function of which, is mastication. The organs consist
of joints, articulating complexes (the maxilla and mandible), muscles and secretory
glands. These organs comprise the organ system with its functions of mastication,
speech, emotional expression and satisfaction. The mandible is directed in its
movements about coupled double joints by a complex neuromuscular system,
teeth influencing joint function mainly by the stretch receptors located in the
periodontal ligaments.

Any disorder of the temporomandibular joint, or of any other organ of the
system, will interfere to varying degrees with function. The temporomandibular
joint is prone to disorders common to all joints. These disorders consist of
rheumatic disease (mainly rheumatoid arthritis and osteoarthritis), traumatic
injuries (most often fracture of the condyloid process), neoplasms (usually
osteoma) and a non-articular disorder known by such various names as fibrositis,
myositis, non-articular rheumatism and the myofascial pain syndrome.

All disorders of the temporomandibular joint may result in deformity, dys-
function and pain. The anatomical, physiological and pathological bases for these
signs and symptoms in rheumatic disease, fractures and neoplasms are well
known. Discussion, therefore, will be limited to the myofascial pain syndrome—
the most common disorder.

Following extensive clinical investigations of temporomandibular joint
disorders we decided to call this myofascial syndrome the 'pain-dysfunction
syndrome'. In this symptom complex the symptoms are facial pain and mandibular
dysfunction. The pain is usually described as a constant unilateral jawache,
earache, or headache, usually aggravated by mandibular function. The dysfunction
takes the form of poorly co-ordinated or restricted mandibular movements. The
syndrome is found four times as frequently among females as among males.

In 1949, the symptoms were described individually by Foged & Snawdon.
The first investigator considered it an articular disorder while the second held
that the disorder was non-articular, due to fibrositis of the masticatory muscles.
Our own studies also pointed to the conclusion that the most common disorder
of the temporomandibular muscles was indeed non-articular and that it arose in
the musculature. The mechanism, however, remained a mystery.

It did not seem logical that occlusion should be the sole or even the most
important cause. Why should a long-standing and apparently well-compensated
malocclusion suddenly precipitate this syndrome? Rapid or extensive changes in
occlusion through dentistry were another matter. But if not occlusion, what
circumstances were at the root of this non-articular disorder?
The fortunate location of our clinic in a dental school that comprised one component of a large medical centre helped our search. This arrangement facilitated co-operative dento-medical investigations. Such research provided much valuable information and, more important, a broad biological approach to the problem.

In time a number of facts became clear. One was that myofascial syndromes constitute the most common musculo-skeletal disabilities of the shoulder girdle, neck and low back (Bonica, 1953). The most generally accepted concept as to aetiology is that hypersensitive areas exist within the muscles or in the connective tissue. In most cases the precipitating factor is motion that stretches the muscle containing the abnormal foci of pain, setting off a self-sustaining pain-spasm-pain cycle, often persisting after the precipitating cause is gone (Travell & Rinzler, 1952).

The fact that many patients reported the onset of symptoms following rapid or prolonged stretch of the masticatory muscles (after a yawn, a large bite or a long dental appointment) indicated a similar mechanism. The question arose as to why stretch precipitated symptoms in some individuals and not in others. The effect of changes in the physical environment, particularly cold, upon muscle activity is well known. The histories of our patients, however, did not point in this direction. Evidence indicated that it was the individual's emotional state, closely related both to his immediate personal situation as well as his social environment, that played the more significant role.

Psychiatric interviews supported this impression (Moulton, 1959). Moreover, the relation between the state of emotional tension and that of muscle tension has been extensively studied both clinically and electromyographically (MacDougall & Andrew, 1953; Goldensohn, 1959). Jacobsen (1938) has pointed out that when a patient states that he is tense he is describing a muscular state as well as an emotional one.

This research was helpful in trying to understand the aetiology of the pain-dysfunction syndrome. For example, many of our patients reported the onset of symptoms, not following rapid or continuous mandibular stretch, but upon awakening. This suggested the clenching or gnashing of teeth during sleep. Others reported the appearance of symptoms following dental treatment involving rapid changes in occlusion such as may occur in restorative procedures, the selective grinding of cusps, or the insertion of an appliance.

In our view only the uniqueness of the stomatognathic system could provide an acceptable explanation—namely, that teeth with pressor receptors in their periodontal ligaments are embedded in the articulating components of this system. Thus with nocturnal clenching, prolonged contraction of the masticatory muscles can be initiated and maintained, as it were, intrinsically with effects upon the mechanisms involved in the circulatory and metabolic dynamics of skeletal muscle activity. It is believed that these may be responsible for the pain in the involuntary sustained muscle contractions known as 'spasm'.

Thinking along similar lines we reached the conclusion that the onset of the pain-dysfunction syndrome following dental treatment involving occlusion is not due as much to the excessive afferent stimuli caused by the procedure itself (the great adaptability of most dental patients is known) but rather to a change in proprioceptive input in a patient with a hyperexcitable central reflex mechanism.
An examination of our observations and clinical experiences in the light of what is known of the anatomy, physiology and pathology of the stomatognathic system led to the following hypothesis:

No single cause exists for the pain-dysfunction syndrome. A number of aetiological factors, including the occlusion of the teeth, seems to be responsible. What the patient does with his occlusion in reacting to stress seems to be more important than any malocclusion that he may have. Malocclusion, by mechanically increasing the amount of force or altering its direction, can make the chance of injury likely. More important than the type of malocclusion, however, is the amount and kind of muscular activity together with the reaction of the individual patient to such activity. Changing an existing occlusion by either grinding or restoration was frequently associated with the onset of the pain-dysfunction syndrome. In some individuals change in proprioception, no matter how slight, seems to be more important than a long-standing malocclusion, no matter how irregular.

This hypothesis was used as a basis for further investigations and from these evolved diagnostic procedures and treatment methods. The anatomical and physiological bases for these will not be considered.

Generally speaking, the diagnoses of so-called temporomandibular syndromes have been based on a meagre history, a detailed study of the occlusion of the teeth and the use of radiology according to the familiarity of the individual practitioner with the techniques involved and the importance he attributed to the information they may provide.

A complete history is of primary importance. Thoroughness in taking the history, in addition to providing important clues to the physiological mechanisms involved, will do much to control the tendency to treat the patient as if he were a mere assemblage of organs and tissues.

The examination proper must be as comprehensive as the history is complete. Physical examination must encompass not only the teeth but also the joints and musculature. The fact that the teeth are visible facilitates examination. It should be kept in mind that any pain arising in the oral cavity, whether from a carious or erupting tooth or from the periodontal tissues, may cause mandibular dysfunction—especially restricted opening. Difficulties arise regarding interpretation of the occlusion of the teeth because of the various methods of examination based on different concepts. Too often the physiological fact is overlooked that the purpose of an occlusion is to function for the patient, not to please the dentist.

Though the maxilla and mandible, joints and muscles cannot be actually seen, observation is still possible. For example, a front view of the face may reveal asymmetry following condylar fracture. Prominent contours near the angles of the mandible are often seen with masseteric hypertrophy. A profile view may reveal the micrognathia that accompanies Still's disease.

Such findings are signs of altered structure but important observations are also possible concerning function. These are provided through a study of mandibular posture and movement.

With spasm, mandibular movements are usually restricted, particularly during opening, with deviation of the mandible toward the symptomatic side. Spasm may also alter the relation of the mandible to the cranium at rest. The usual complaint of the patient is that the teeth do not feel as if they interdigitate properly.

The function of the temporomandibular joints cannot be seen during exam-
ination as easily as mandibular movements. Although in thin individuals the anterior movements of the condylar heads during mandibular opening are visible, in most individuals palpation is necessary. If translation does not occur and only hingelike movements take place, rotary instead of anterior movement of the condylar head can be felt by the palpating finger within the auditory meatus. Palpation in front of the tragus will disclose the bulging of the condylar head under the skin as it moves forward. The absence of this bulging points to restriction of movement. Radiology, of course, clearly shows the range of condylar movement. Palpation may also reveal tenderness over the temporomandibular joint areas. This seems to point to the 'capsulitis' described by Bell (1960).

During palpation of the temporomandibular joint areas crepitus or clicking may be heard. Such sounds, however, can be better determined through the use of a stethoscope. Bailey (1954) writes that fine crepitations are present in many subacute and chronic joint infections, coarse crepitations usually signify osteoarthritis and a constant click a sign of a displaced cartilage. It is doubtful if this is true of clicking of the temporomandibular joint.

For the examination of muscles, aside from determining the presence of hypertrophy and occasional masseter fasciculation, observation is of little value. Palpation, on the other hand, provides important information as to the size, the state of contraction or the presence of painful areas. The internal pterygoid muscle, since it forms the slanting medial wall of the pterygomandibular space, may tell much as to the state of contraction.

Painful areas in the masticatory musculature are most often located in specific areas. Common sites are the anterior portion of the origin of the superficial part of the masseter muscle as it arises from the lower border of the zygomatic arch and the anterior portion of the origin of the temporalis muscle in the temporal fossa. A painful area is sometimes found intra-orally at the insertion of the temporalis muscle.

For intra-oral palpation with full mandibular opening the probing finger passes from the anterior border of the ramus to its medial surface to reach the deep temporal ligament. If the finger continues posteriorly and medially it reaches part of the origin of the internal pterygoid muscle, an area often painful to palpation. The insertion of this muscle on the medial surface of the ramus of the mandible between the mylohyoid groove and the angle is sometimes involved.

Anatomical and physiological considerations are as important in treatment as in diagnosis. The most widely practised method of treatment is occlusal therapy. The rationale for such treatment is based largely upon the assumption that malocclusion mechanically affects the joint. During the discussion so far it has been pointed out that there is no scientific basis for the two 'vertical deviations'; bite closure and hypermotility. There is, moreover, no reason as yet to believe that 'horizontal deviations' are mechanically injurious to either the temporomandibular joints or their musculature.

Recently physiological explanations have been offered. In substance, the mechanism is thought to be one of 'excessive stimuli' caused by occlusal interferences through the stretch receptors in the periodontal ligaments. Although this may very well be true there are a number of considerations that should be kept in mind before embarking upon occlusal therapy. One is that motor response is not determined entirely by the nature of afferent stimuli. The state of excitability of the central reflex mechanism may play an important, and in some cases, a
determining rôle. This view is supported by the number of patients seen with the pain-dysfunction syndrome with normal occlusions but under great emotional stress.

The problem of occlusal therapy is further complicated by the fact that spasm in the masticatory muscles may alter the relation of the mandible to the cranium, creating in fact transitory occlusal deviations. It thus seems evident that occlusal therapy, never a simple mechanical procedure under any circumstance, becomes much more complex in the presence of mandibular dysfunction. What has been stated so far does not apply to occlusal abnormalities resulting from dental treatment. In such cases mechanical treatment may have so violated anatomical and physiological principles as to exceed anyone's capacity to adapt and the indication for occlusal correction is evident.

Occlusal therapy, even of long standing apparently well-compensated occlusal disharmonies is undoubtedly beneficial in some cases. But this, in my opinion, attests to the competence of the individual practitioner rather than the existence of an adequate scientific rationale. Moreover, the preoccupation with treatment of the occlusion has obscured the fact that other types of effective therapy are available. These methods may be grouped as psychological, pharmacological and physical.

An essential, indeed an unavoidable component of all therapy, is the psychological. The patient is a seeker and the dental surgeon or physician is in the same sense a helper. Thus the nature of this seeker-helper relationship can either relax the patient (with relaxation of the musculature) or alarm him (with an increase in muscle tension). In fact, a practitioner's injudicious words may so disturb the patient as to create a new and additional disorder—iatrogenic disease. Particularly with myofascial syndromes, having as they do a large tensional component, reassurance by means of careful explanation is of the greatest importance. The ability of the individual practitioner to relax his patients may explain the success of a particular method of treatment in some hands and not in others.

The psychological extends into the pharmacological through the placebo effect known to exist with the administration of any drug. Sedatives, analgesics, muscle relaxants, anaesthetics and corticosteroids are the drugs most useful in the management of the pain-dysfunction syndrome.

Sedatives are used to make the patient more composed and tranquil by achieving a lesser degree of cortical excitability. Analgesics lower the perception of pain; muscle relaxants are prescribed in an effort to decrease the tonicity of the musculature. Local anaesthetics are utilised for the purpose of interrupting the spasm-pain-spasm cycle that perpetuates the syndrome and to lessen or eliminate the pain that may accompany exercise. Corticosteroids depress inflammation and are administered intra-articularly in capsulitis.

Although drugs are useful adjuncts in the management of the pain-dysfunction syndrome, physical methods are more fundamental in therapy. William C. Mackenzie (1918) wrote: 'the muscle fibre is delicate, sensitive and responsive. It can be coaxed but not driven'. Physical methods are used in order to coax painful contracted muscles to relax.

Heat is of particular value. By altering the amount of vasodilator substances held in the tissue spaces heat causes vasodilation. Thus heat can increase the local tissue immunity, augmenting the white corpuscles in the region and causing
phagocytosis to become more active. Generally speaking, heat has a relaxing effect and is often used preliminary to other forms of physical treatment such as exercise (Bierman & Licht, 1952).

In exercise, the ideal is voluntary performance against resistance. Sherrington’s (1906) principle of reciprocal inhibition states that ‘where two muscles would antagonise each other’s action the reflex-arc, instead of activating merely one of the two, causes when it activates the one depression of the activity (tonic or rhythmic contraction) of the other. The latter is an inhibitory effect’. Thus, when the depressor muscles are made to open the mandible the elevator muscles are inhibited. By having the patient open the mandible against resistance he is able to more easily stretch the reflexly inhibited antagonists. Utilising these physiologic principles, exercise aims to restore normal mandibular function by improving one or more of the three basic qualities of muscle function: power—the ability to contract, elasticity—the ability to give up contraction, and co-ordination—the ability to co-operate with other muscles in proper timing and with appropriate power and elasticity (Kraus, 1950). In the management of the pain-dysfunction syndrome it is the improvement of elasticity and co-ordination that are important.

Massage, with the changes it produces through reflex reactions, has wide application in physical medicine. However, this form of treatment must be applied to the masticatory muscles with great care because of the danger of trauma arising from the fact that these muscles are relatively thin and lie directly over bone. The only safe form of massage in the treatment of the pain-dysfunction syndrome is that of effleurage—a light stroking movement (Bierman, 1959).

Before completing the discussion of anatomical and physiological fundamentals in the treatment of the pain-dysfunction syndrome, the place of surgery should be mentioned. In certain temporomandibular joint disorders, ankylosis for example, surgery provides the only effective method of treatment. Other indications may be the correction of deformities, some types of condylar fractures and recurrent dislocation. In the management of the pain-dysfunction syndrome, however, since all evidence points to the fact that the disorder is extra-articular, there is no scientific rationale at the present time for surgical intervention.

From what has been said thus far it seems evident that the anatomical, physiological and even pathological foundations for the diagnosis and management of the pain-dysfunction syndrome are not fully developed. One may speculate as to the reasons—is the slow development due to the fact that no scientific knowledge of the temporomandibular joints and associated structures was available until recently? This is not so.

John Hunter, whose museum, or his great unwritten book as it has been called, is located in the Royal College of Surgeons, published in 1771 his monumental work, ‘The Natural History of the Human Teeth’. In this volume Hunter described in almost contemporary terms the anatomy and physiology of the temporomandibular joints. So astute were Hunter’s observations that they included the statement that ‘... fear produces a vibratory contraction of all the voluntary muscles while the will is doing all it can to stop it’ (Kobler, 1960).

Why, one may ask, has this really modern book been almost entirely ignored in the search for a scientific basis for the management of temporomandibular joint disorders? One reason has been stated, the emphasis that the dental profession has placed upon technology as opposed to biology. But there is another and possibly more compelling reason. This is the existence of an all too prevalent
attitude, probably due in part to the pressure of our times, that holds that the essential in practice is effective therapy and that somehow or other, somewhere, an infallible technique can be picked up without the discipline of science, the trouble of observation or the doubts engendered by thought.

In the introduction to his life of John Hunter, Paget (1897) wrote: 'If we try to find, in Hunter's mental character, the facts to which may be ascribed his great influence in the promotion of medicine and surgery, I think it may justly be assigned to the degree in which he introduced the exercise of the observant scientific mind into the study and practice of surgery'.

John Hunter is quoted as having said, 'I love to be puzzled'. It is my opinion that if in the management of temporomandibular joint disorders we would try to repress the urge for spectacular action and develop in its place our powers of observation, we would be unavoidably directed to that state of puzzlement which enriches the scientific source of any clinical field.

REFERENCES


