



A RADIOLOGICAL STUDY OF THE TEMPOROMANDIBULAR JOINT.

Factors affecting joint morphology.

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PRECIS

Patients with symptomatic complaints of the temporomandibular joint constitute a small though significant portion of many oral surgery practices. However, the problems posed by this group are often far greater than its numerical size would seem to warrant. One of these problems concerns the radiology of the joint, traditionally an important investigative procedure in such patients.

There is no single radiograph which allows accurate visualization of all surfaces of the temporomandibular joint. Even combinations of plain film radiographic projections do not satisfy this objective. Consequently a combination of tomographic projections and plain film radiography has been advocated. Such a combination does provide for maximum exactitude in the radiological investigation of the joint but necessitates an increased radiation exposure for the patient. Each tomographic cut alone requires approximately twice the radiation of a conventional radiograph.

In spite of the increased information available from studies utilizing these advanced radiographic techniques, the significance of radiologically determined morphological changes in relation to clinical symptoms and subsequent therapy remains undetermined.

The aim of this study was to investigate the significance of morphological joint changes, visualized on the commonly used panoramic jaw tomograph, in four groups of subjects matched for age and sex. The first two groups had no symptoms related to their temporomandibular joints, one group being fully dentate and the other totally edentulous. The third group consisted of patients who had presented with complaints of pain in or around the temporomandibular joints and the fourth

group comprised fully dentate patients diagnosed as being morbidly obese. Six morphological changes were examined for in both joints of each subject.

The results of this investigation demonstrated that panoramic jaw tomographs provided a satisfactory depiction of the condyle and articular eminence in most subjects. The glenoid fossa, however, was poorly demonstrated in the majority of cases. Morphological joint changes were common in all groups. The incidence of changes was generally higher in the older age groups and in edentulous persons. There were significantly higher incidences of change in the pain group as compared to the pain free groups. No relationship was found between the side on which symptoms occurred in the pain group and the side on which the greater morphological joint change was observed. Negligible differences were found between the obese and non-obese dentate subjects.

DECLARATION

This thesis is submitted in part fulfilment of the requirements for the Degree of Master of Dental Surgery, of the University of Adelaide. Candidature for the degree was satisfied by a Qualifying Examination in 1979.

I declare that it contains no material which has been accepted for the award of any other degree or diploma in any University and, to the best of my knowledge, no material previously published or written by another person, except where due reference is made in the text.

C.B. Muir

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CHAPTER I

INTRODUCTION

Patients with symptomatic complaints of the temporomandibular joint constitute a small though significant portion of many oral surgery practices. However, the problems posed by this group are often far greater than its numerical size would seem to warrant. One of these problems concerns the radiology of the temporomandibular joint, traditionally an important step in the diagnosis of these patients. Not only is it difficult to accurately depict the joint surfaces, but the interpretation of any radiologic changes, their relationship to clinical signs and symptoms, and relevance with regard to treatment, remains a vexed question. The purpose of this investigation is to attempt to determine the significance of alterations in the temporomandibular joints, as seen on a commonly used dental radiograph, the orthopantomograph, in the diagnosis of such patients.

The evolution of concepts concerning the aetiology of temporomandibular joint pain (Chapter IV) is pertinent to a consideration of the radiology of the painful joint. In 1934, when Costen first aroused general interest in what is now known as the myofascial pain-dysfunction syndrome or the temporomandibular joint pain-dysfunction syndrome, the cause of this condition was believed to lie within the joint itself. Mandibular overclosure was considered to produce pressure by the condyle on nerves, blood vessels, and other structures intimately related to the joint, thereby producing the symptoms of the disorder. The overclosure hypothesis was subsequently overthrown to be replaced by the concept that defects in the dental occlusion were the primary cause. More recently, the emphasis has undergone another major shift and abnormalities of muscle and muscle hyper-

activity are now gaining increasing popularity as the factors responsible for the condition. Both of these hypotheses regard symptoms and signs referable to the temporomandibular joint as being secondary to the primary aetiological factor.

At the same time there has been a vast increase in interest and knowledge concerning the form and function of the temporomandibular joint (Chapter II). Linked with this have been investigations into the adaptability of the joint and the diseases to which it is prone. The progress made in these fields has paralleled, and been prompted partly by, the advances related to the myofascial pain dysfunction syndrome. Another important stimulus to research on the joint has been its effect on mandibular growth.

All this has occurred during a period of tremendous upsurge in interest in degenerative joint disease. It has long been recognised that the mandibular articulation is subject to the diseases which affect the body's other movable joints, the most common being osteoarthritis. The unique nature of the temporomandibular joint, in that its articulating elements are functionally linked by the occlusion of teeth, has prompted considerable interest in the aetiology of osteoarthritis in this particular joint, especially with regard to change in dentition. More recently, the effect of the altered muscular activity believed to be present in individuals with the myofascial pain dysfunction syndrome has also been proposed as a possible aetiological factor in osteoarthritis of the temporomandibular joint (Chapter IV).

The comfortable belief that myofascial pain dysfunction syndrome and osteoarthritis of the temporomandibular joint could be

readily distinguished on clinical, and particularly radiological grounds, has been challenged by research published in recent years (Chapter VI). The most significant portions of this research relate to the knowledge that the temporomandibular joint can, and does, adapt to altered environmental circumstances. This is brought about by remodelling within the joint tissues, probably in response to biomechanical stresses, and results in changes in the morphology of the joint components. Such changes are ubiquitous in adult mandibular joints (Chapter V). Similar changes in form can occur in both non-pathological adaptive remodelling and osteoarthritis. Many believe that remodelling merges gradually into osteoarthritis when the adaptive capability of the joint tissues is exceeded, a distinction between the two being made only on the basis of the articular soft tissue destruction seen in osteoarthritis and not in adaptive remodelling. A radiological distinction is clearly problematic as only hard tissue alterations can be detected by this means, and these are already present, to some degree, in all adult temporomandibular joints regardless of the presence or absence of osteoarthritis.

Changes in the dentition have been suggested as important factors in the aetiology of both adaptive remodelling and osteoarthritis in the temporomandibular joint. The masticatory muscle hyperactivity believed to be present in individuals with the myofascial pain dysfunction syndrome should also provide the necessary biomechanical stimulus required for joint remodelling and, as previously noted, it has already been proposed as an important factor in the aetiology of osteoarthritis of the temporomandibular joint.

These inter-relationships are schematically illustrated in Figure 1.

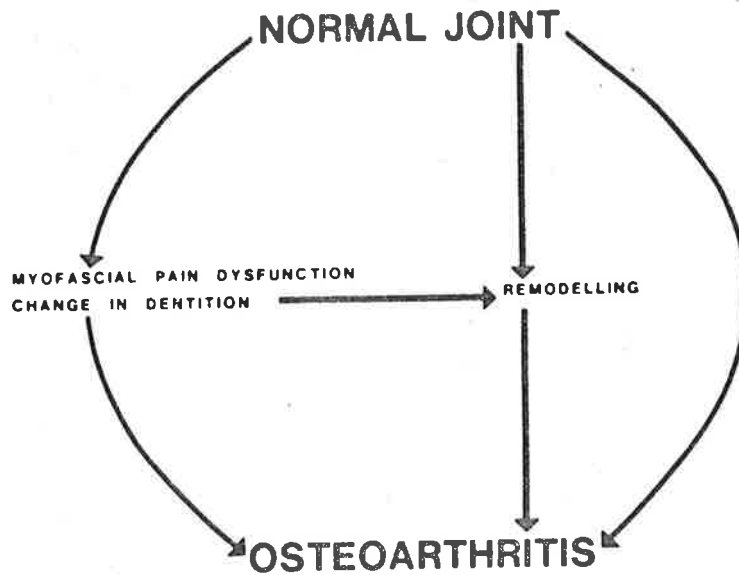


Figure 1.

Until now, little attention has been paid to the possibility that there is a pattern of remodelling alterations, either purely adaptive or associated with osteoarthritis, which occurs specifically in response to the muscular hyperactivity proposed as the basis for the myofascial pain dysfunction syndrome, and is different in form and/or extent to that seen, for example, as a result of changes in the dentition.

The present investigation uses radiological methods in an attempt to examine the relationship between changes in morphology of the joint components and the presence of symptoms believed to be associated with overloading of the joint tissues. The study group is comprised of individuals distinguished by the complaint of pain in or around the temporomandibular joint to the extent that they have sought treatment for this problem. In the absence of a simple and reliable means of distinguishing myofascial pain dysfunction

syndrome from symptomatic osteoarthritis of the temporomandibular joint, the group probably includes some patients suffering from osteoarthritis, also believed to be the result of joint overload, although the majority of subjects were diagnosed by experienced dental specialists as having the myofascial pain dysfunction syndrome. These patients were compared with a control group of asymptomatic individuals, matched for age, sex, and, as closely as possible, for dentition. Comparisons were also made within the control group, between dentate and edentulous and aged and young individuals.

As well as increased mechanical loading on a joint, a number of systemic factors have been related to the aetiology of osteoarthritis. One of these is obesity (Chapter VI). Although traditionally implicated in weight-bearing articulations, a connection between obesity and osteoarthritis in the non-weight bearing sternoclavicular and distal interphalangeal joints has also been noted. The structural similarities of the temporomandibular and sternoclavicular joints, lined not by hyaline cartilage but by fibrous articular tissue, suggested the possibility that there may also be an increased incidence of temporomandibular joint osteoarthritis in obese individuals. Consequently, the present study examined the occurrence in obese subjects, of those radiological features traditionally associated with temporomandibular joint osteoarthritis. This was compared with the occurrence of such features in a non-obese group matched for age, sex, and dentition.

CHAPTER II

THE NORMAL TEMPOROMANDIBULAR JOINT

The temporomandibular joint (TMJ) classified as a ginglymo- (hinge) arthrosis (sliding), is structurally and functionally one of the most intricate, and most used, joints in man. It is unique in that its articulating complexes are functionally linked by the occlusion of teeth as well as through the joint itself (Brown,1965; Schwartz and Marbach,1965).

The articular surfaces are the glenoid fossa and articular eminence of the temporal bone and the articular surface of the condyloid process of the mandible. An inter-articular disc divides the joint cavity into superior and inferior compartments. The joint cavity is enclosed by a capsule which is reinforced by a strong lateral ligament and less firmly by a medial ligament.

2.1 ANATOMY

The Mandibular Condyle

The adult mandibular condyle varies considerably in size and shape. It is roughly elliptical when viewed from above, and generally convex both antero-posteriorly and medio-laterally. Condylar dimensions average 10mm (range 5.5mm - 16mm) antero-posteriorly and 20mm (range 13mm - 25mm) mediolaterally (Oberg et al.,1971). The long axis of the condyle is almost at right angles to the plane of the mandibular ramus.

A thin layer of compact cortical plate comprises the bony articular surface and trabeculae of cancellous bone are orientated perpendicularly to this plate. The intertrabecular tissue consists

of haemopoietic and fatty marrow, the proportions varying with age (Griffin et al. 1975).

The bony surface is covered, not by hyaline cartilage as in most other synovial joints, but by a layer of mature, densely collagenous fibrous tissue. In the region of the convexity of the condyle there is a layer of fibro-cartilage lying deep to the surface fibrous tissue and blending with the bony end-plate.

Between the superficial and deep tissue layers lies an intermediate zone of cells forming a thin but distinct layer which has been termed the proliferative zone (Blackwood 1966a). Although in older joints this zone may be much less obvious than in the growing individual, its cells are capable of proliferative activity throughout life. Posteriorly and elsewhere in areas that do not contact the articular eminence during function, the surface fibrous tissue is applied directly to bone and merges imperceptibly with the mandibular periosteum (Moffett et al., 1964).

Toller (1977) has reported on the ultrastructure of the condylar articular region. Electron-microscopy of the articular surface reveals a dense meshwork of bundles of collagen fibrils interspersed with fibroblasts. The collagen fibre density is far greater than in normal articular hyaline cartilage, and a wavy interlacing of fibres produces an extremely tough and wear resistant structure. Ground substance, lying between the collagen fibrils and the fibroblasts, occupies a very small percentage of the total bulk.

At the articular surface, the collagen ends abruptly. Between the joint cavity and the collagen is a narrow zone of faintly fibrillar material which is more electron dense than ground substance and has a smooth surface. It varies from $1\mu\text{m}$ - $2\mu\text{m}$ in thickness, and may contain a few small diameter collagen fibrils. This zone is termed the lamina splendens.

At the non-weight bearing posterior articular surface of the condyle, the entire surface structure is of a looser texture, the lamina splendens broader, and cellular density higher with more cells near the surface. The fibrocytes retain their integrity very close to the collagen surface and only at its extreme edge do they appear to break up with escape of intra-cellular organelles, followed by nuclear disintegration. This phenomenon is seen virtually only in non-weightbearing zones, there being little or no evidence of the shedding of cells or fibres from the free surfaces in weightbearing areas (Toller,1977).

The Temporal Articular Surface

This consists of the concavity of the articular fossa posteriorly and the convexity of the articular eminence anteriorly. The average dimensions are greater than those of the condyle being 19mm (range 12mm - 23mm) antero-posteriorly and 23mm (range 18mm - 28mm) mediolaterally (Oberg et al.,1971).

The bone of the fossa is generally very thin and covered by a layer of periosteum. Anteriorly, the covering of the articular eminence is much thicker with layers analagous to those described in the condyle.

The Articular Disc

The articular disc is a dense sheet of fibrous tissue filling the space between the opposed articular surfaces. It is roughly elliptical with a thin central area and thickened peripheries, especially anteriorly and posteriorly. Posteriorly the disc splits into an upper lamina attaching to the margin of the squamo-tympanic fissure, and a lower lamina which blends with the periosteum of the posterior surface of the condylar neck. Anteriorly it attaches to the anterior edge of the articular eminence above, and the articular margin of the condyle below. Medially and laterally the disc blends with the capsule and both then attach to the medial and lateral poles of the condyle. Fibres of the lateral pterygoid muscle insert into the anterior and medial edges of the disc.

The Joint Capsule and Ligaments

Anteriorly and posteriorly the joint capsule is thin and composed principally of the anterior and posterior parts of the disc. Medially and laterally it is strengthened by the capsular ligaments. The lateral ligament is especially strong and is termed the temporo-mandibular ligament.

The Synovial Membrane

According to Griffin et al. (1975), all the internal joint surfaces, except for those actually in articulation, are lined by synovial membrane. They also noted that the intermediate and posterior bands of the disc are lined by fibrous synovial membrane, a variety found on articular surfaces subject to pressure.

2.2. EVOLUTION

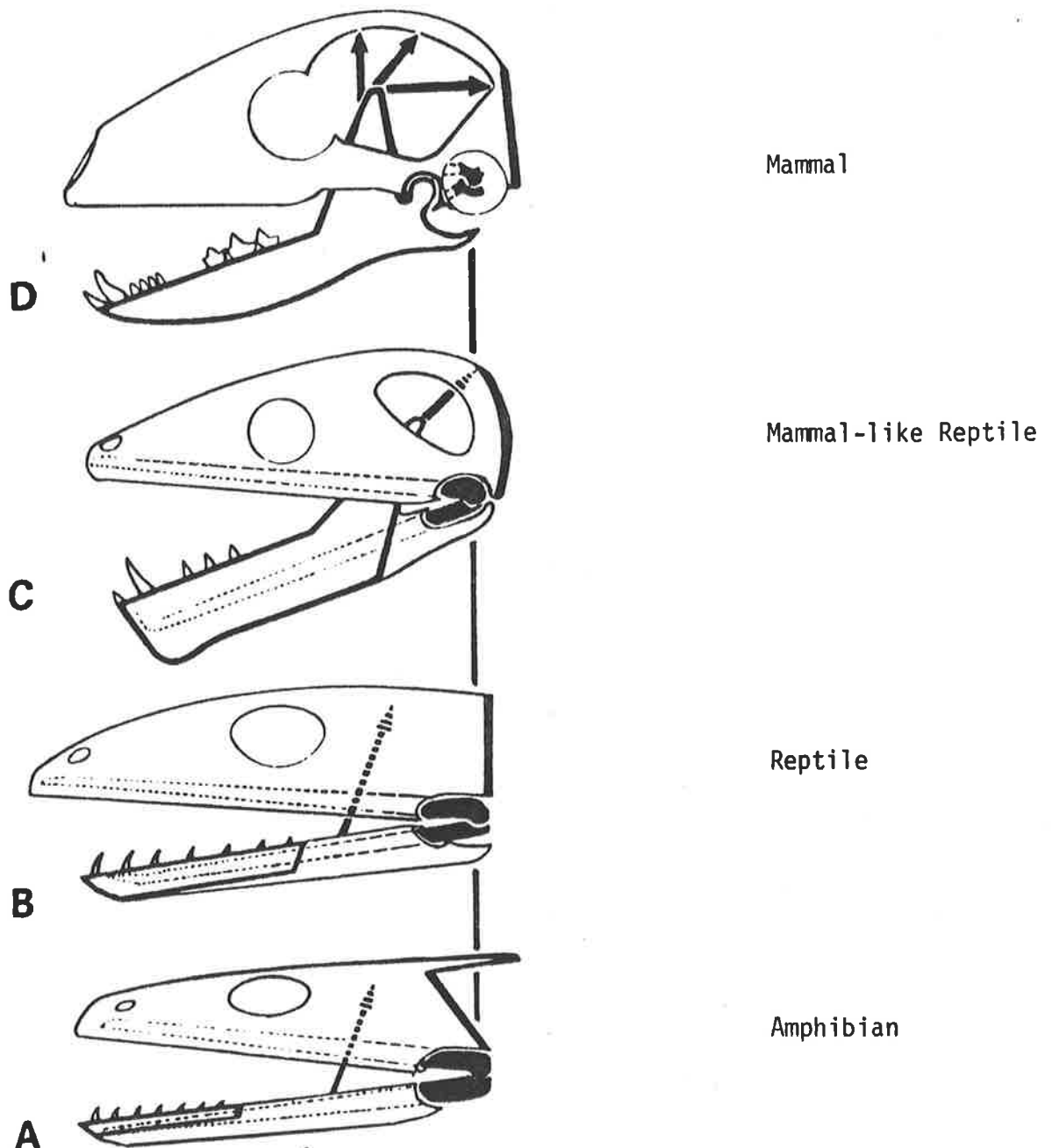
The temporomandibular articulation is regarded as a secondary jaw joint having developed in mammals as a replacement for the primary jaw joint of reptiles. This primary jaw joint existed between the posterior end of Meckel's cartilage and the quadrate bone. In mammals it now functions in hearing as the malleo-incal joint, a development made possible by the formation of a new, secondary, jaw joint just anterior to the ear (Moffett, 1966; Blackwood, 1976).

This secondary articulation occurred by means of a bony process which appeared on the mandible and grew superiorly to meet the skull, eventually forming the temporomandibular synovial joint. The gradual appearance of this secondary bony process, the condyle, can be observed in a series of related reptilian skulls, (Fig. 2) and in the mammalian embryo this phylogenetic sequence is faithfully reproduced in the development of the TMJ (Moffett, 1966). A further reflection of this evolutionary change is seen in the later embryonic development of the TMJ as compared with the other joints of the primary skeleton.

2.3 EMBRYOLOGY OF THE HUMAN TMJ

The TMJ arises from two distinct blastemata, the glenoidal and the condylar, which exist at some distance from each other and grow and differentiate at different rates in opposing directions (Levy, 1964). This contrasts with the standard pattern in most other synovial joints, where bones develop from a continuous rudiment which segments at the joint location.

In the seven week embryo, a condensation of mesenchymal cells at the dorsal end of the already calcifying mandibular primordium



Evolution of the mammalian jaw joint.

Heavy outlines represent dentary bone and back of skull. Broken lines represent the original cartilaginous jaws projecting posteriorly to black jaw joints. Arrows indicate muscle vectors.

In the mammal the dentary bone contacts the skull forming the dentary-squamosal (temporomandibular) joint. The primitive jaw articulation now forms the malleolar-incus joint of the ear ossicles.

From Du Brul (1979).

forms the condylar blastema. This is followed about one week later by the glenoidal element. During the ninth and tenth weeks, the first signs of the condylar cartilage are seen in the condylar blastema and two clefts appear in the tissue between this and the developing temporal component. These clefts delineate the inferior and superior joint cavities and the future articular disc. As the cartilaginous condylar mass develops, its lower end becomes encompassed by the membranous bone of the developing mandibular body, and it assumes a typical "carrot-shaped" appearance.

By the twelfth week, the temporal region has developed to recover its lag relative to the mandible (Youdelis, 1966) and the condylar cartilage is clearly defined and extends as far forwards as the crypt of the deciduous second molar tooth. At this stage ossification begins within the condylar cartilage, uniting it to the body of the mandible (Baume, 1962) but the posterior part persists as cartilage to form the condyle. Growth of the cartilage continues by apposition from the covering perichondrium and replacement of the deep surface by endochondral bone formation. During the thirteenth week, the two joint components move into close proximity and the joint cavities proper develop.

By the fourteenth week, due to extensive neurocranial widening, there is a substantial increase in intercondylar width, the condyles themselves expanding in all directions thus giving early indication of their considerable adaptive remodelling potential (Durkin et al., 1979). The adult form of the joint is now recognizable and further modifications are associated with maturation and increase in size.

The joint capsule, medially and laterally, develops by condensation of surrounding mesenchyme in the twelfth week and to this attaches the disc tissue. The posterior part of the capsule develops in the twenty-second week, when the pterygo-tympanic fissure narrows around the remnants of Meckel's cartilage as it passes into the middle ear, thus finally separating the TMJ from its primary predecessor.

2.4 POST-NATAL GROWTH AND DEVELOPMENT

At birth the TMJ is characterised by vascularization of all components, including the condylar cartilage. There is active intra-membranous and endochondral ossification, and a skeletal architecture of fine cancellous bone enclosed by a thick vascular periosteum with no intervening cortical plate.

The temporal part of the joint consists of a rudimentary articular tubercle and post-glenoid process, separated by a shallow glenoid fossa. The mandibular condyle also displays anatomic and histologic immaturity (Wright and Moffett, 1974).

After birth there is very rapid growth of all components of the joint. The condyle grows by endochondral ossification and within the cartilage four cell zones can be detected (Thilander et al., 1976):-

- i. surface articular zone - a dense fibrous connective tissue layer forming the articular surface.
- ii. proliferative zone - a highly cellular layer of proliferating cells which differentiate to become the chondroblasts and chondrocytes of the subjacent third layer.

- iii. hypertrophic zone - a cartilage layer with cells secreting chondroid matrix and then undergoing hypertrophy with a deeper zone of mineralization.
- iv. bone formative zone - a zone of endochondral bone formation.

The major change occurring in the condyle during the first six months of life is a progressive reduction in thickness of the condylar cartilage from between 1.25mm to 1.5mm down to approximately 0.5mm in thickness. This reduction occurs in the hypertrophic zone (Wright and Moffett, 1974). The condyle elongates by means of endochondral ossification and also becomes relocated postero-laterally through a process of periosteal-endosteal resorption and deposition. By three years of age the cartilage has become avascular. Wright and Moffett (1974) suggest that this early vascularity allows nourishment during the rapid growth period when the mandible must accommodate to the developing and erupting deciduous dentition.

Throughout the mixed dentition period, the condyle gradually increases all of its dimensions and, with the establishment of the permanent dentition, its head assumes an adult appearance. During the teenage period an outer bony layer forms which eventually, at about age twenty years, separates the medullary spaces from the overlying cartilage (Ingervall et al., 1976).

The adult contour of the temporal surface of the joint, that is the marked convexity of the articular eminence and concavity of the fossa, first becomes evident at about two and one half years of age but is not fully achieved until the permanent dentition has erupted. As in the condyle, a cortical plate gradually forms over the eminence, reaching completion at about age twenty. At the crest

of the eminence, the articular soft tissue is approximately six times thicker than in the roof of the fossa, and can be differentiated into layers analagous to those previously described in the condyle.

During development in both joint components, the main growth activity occurs in the proliferative zones (Thilander et al., 1976). Unlike chondrocytes in the epiphyseal growth cartilages of long bones, which replenish themselves by mitosis, chondrocytes of the growing condyle show no capacity to divide (Wright and Moffett, 1974), and thus cartilage cells are derived entirely from the germinal layer of the proliferative zone.

The three articular soft tissue zones constitute a growth apparatus in the bony components of the joint which is present throughout life (Sharpe et al., 1965). It is this mechanism which is believed to form the basis for adaptive remodelling within the TMJ (Blackwood, 1966b; Carlsson and Oberg, 1979).

CHAPTER III

RADIOLOGY OF THE TEMPOROMANDIBULAR JOINT

The TMJ is difficult to examine radiographically because of its complex internal anatomy, and its position at the base of the skull. Added to these anatomic problems are difficulties in radiological interpretation imposed by the wide variations in normal anatomy of the hard tissues of the joint, and the fact that the joints may be asymmetrical (Quantrill and Lewis, 1974).

3.1 RADIOGRAPHIC TECHNIQUES

As a general principle, adequate radiographic assessment of any joint necessitates at least two views at right angles to each other (Campbell, 1965; Worth, 1979).

The most commonly used lateral joint projections are the transcranial and transpharyngeal. Antero-posteriorly, either a transmaxillary or transorbital projection is generally employed. Other plain views, such as the reverse Townes and submento-vertical, may also be included.

Tomographs, generally taken in the sagittal and coronal planes, often provide valuable additional information. The orthopantomograph (OPG) (Paatero, 1961), a specialized form of tomograph, displays the temporomandibular articulation accompanied by the dentition, alveolar bone, and contiguous structures, and has enjoyed a massive increase in utilization in recent years.

Arthrography, in which radio-opaque contrast medium is injected into the joint cavities, may also add valuable information especially

concerning the soft tissues of the joint (Campbell, 1965; Katzberg et al., 1980). However, the procedure is painful and may worsen the patient's condition (Campbell, 1970).

Unfortunately, none of the above projections are without their limitations. A number of studies (Lindvall et al., 1976, Carlsson et al., 1968; Bean et al., 1977), in which radiographic changes have been compared with gross and histologic alterations of TMJ components, have shown the popular lateral transcranial projection to be diagnostically unreliable, especially as regards the temporal joint component. Similarly, the transpharyngeal projection, although giving excellent detail of the condylar head (Toller, 1969), is deficient with respect to the temporal surfaces (Ogus, 1975; Hansson and Petersson, 1978). Transorbital and transmaxillary projections are also of limited value with respect to the temporal joint surfaces but do show the central and medial portions of the condyle well (Mohl, 1973) whereas the previously cited lateral projections do not (Madsen, 1966; Weinberg, 1973).

Eckerdal (1973) has demonstrated that even careful tomographic technique in favourable circumstances, accurately reproduces only the central two-thirds of the TMJ, and he notes the occurrence of spurious phenomena, to some extent, in all tomographs. He concluded that a combination of conventional radiographic projections and lateral tomography made "the greater part of the joint accessible for high quality examination". Many authors (Campbell, 1965; Klein et al., 1970; Yune et al., 1973; Omnell and Petersson, 1976; Lindvall et al., 1976; Bean et al., 1977; Worth, 1979) agree with this assessment, and Stanson and Baker (1976) claim a three-fold increase in the detection of TMJ abnormalities using such a combination.

However, the latter authors also note an entrance skin radiation dose two times higher per tomographic exposure than for conventional projections.

It is apparent that in spite of the multiplicity of described techniques, there is no general agreement on a completely satisfactory radiographic method for depicting the TMJ. A combination of techniques will provide for the greatest diagnostic accuracy but against this must be weighed an increased radiation hazard.

3.2 ORTHOPANTOMOGRAPHIC RADIOGRAPHY OF THE TMJ

A number of papers have been written describing the use of orthopantomographic radiography for the diagnosis of TMJ pathology (Tammisalo and Mattila, 1963; Tammisalo, 1964; Uotila, 1964, 1965; Updegrave, 1971; Greig and Musaph, 1973; Quantrill and Lewis, 1974). The technique is particularly valuable as both joints are radiographed "under identical circumstances without the disturbance of shadow from the opposite side and with a definition comparable to that obtained by means of a stationary tube" (Tammisalo, 1964). Consequently, a diagnostically significant comparison of the joints can be made (Uotila, 1965), while allowing simultaneous examination of the dentition, alveolus and contiguous structures.

Both Tammisalo (1964) and Uotila (1964, 1965) believe that the conventional orthopantomographic technique produces a satisfactory representation of the TMJ. Tammisalo (1964) suggests such projections to be "in many respects superior to the corresponding pictures obtained by other methods" and "particularly suited for study of the condylar head". Greig and Musaph (1973) concur, stating that, on the conventional OPG the temporomandibular joints "were frequently well visualized".

In contrast to the moderate enthusiasm expressed, in the aforementioned studies, for the depiction of the TMJ on a standard OPG, Worth (1979) in discussing this projection states that "the detail is too unsharp to provide enough evidence to replace the other projections, so that they should never be the sole means of radiographic study". Blair and Chalmers (1972), however, concluded a detailed comparison of circular tomography, orthopantomography and lateral transcranio-oblique (transcranial) radiography used in the diagnosis of TMJ abnormalities, by stating that "these three techniques are all equally effective in demonstrating features of the temporomandibular joint, provided that the joint can be seen on the relevant radiograph". Uotila (1964) used the OPG as his sole means of radiographic examination of the temporomandibular joints in a large group of patients suffering from rheumatoid arthritis and stated that "The roentgenograms must be regarded as at least equally good as those obtained by the oblique-lateral (i.e. transcranial) technique".

Using a conventional orthopantomographic technique, the central ray does not intersect the temporomandibular joints perpendicularly, but at an angle of about 35° (Tammisalo and Mattila, 1963). Consequently, the joint is not reproduced in perpendicular transverse section, but rather in an oblique lengthwise projection which may distort the size of the joint space and the lateral part of the anterior margin of the condyle (Tammisalo and Mattila, 1963). Therefore an accurate measurement of the true joint space is impossible using this technique. The glenoid fossa is also not seen well enough in most films to be of diagnostic significance (Uotila, 1964; Blaschke and White, 1979).

The general consensus regarding the use of the OPG for TMJ radiography is favourable although, as with all other techniques, it

does have limitations when used alone.

3.3 RADIOLOGY OF THE NORMAL ADULT TMJ

The lateral radiographic appearance of a normal adult human TMJ, with the mandible in a closed position, shows a convex condyle positioned within a concave fossa. The articular eminence forms the anterior, convex, portion of the convexo-concave temporal joint surface and the tympanic plate limits the fossa posteriorly. All components exhibit smooth, even, curves (Uotila, 1965; Updegrave, 1971; Quantrill and Lewis, 1974; Worth, 1979) with a continuous white line representing the cortical plate. An oval radiolucency, the auditory canal, is seen posterior to the tympanic plate.

The occurrence of wide variations in the structure and form of the normal adult TMJ is, however, well recognised (Yale et al., 1966; Updegrave, 1971; Taylor et al., 1972; Quantrill and Lewis, 1974; Worth, 1979). Consequently TMJ radiographs are distinguished more by the variety of appearances they present rather than by their homogeneity.

CHAPTER IVMYOFASCIAL PAIN-DYSFUNCTION SYNDROME

The myofascial pain-dysfunction syndrome associated with the TMJ is a common, yet poorly understood, human affliction. In summarizing the evolution of concepts related to this condition, Greene (1979) states: "Rarely in the history of dentistry have so many laboured for so long, only to end with such extreme disagreement. After more than half a century, the myofascial pain-dysfunction syndrome continues to be one of the most controversial areas in dentistry".

Traditionally Costen (1934), an otolaryngologist, is credited with making this condition well known to the medical and dental professions. However, the first descriptions of facial pain and TMJ disturbance associated with mandibular overclosure were by the dentists Summa (1918) and Prentiss (1918). Costen's syndrome, as the condition came to be known, extended the symptoms reported by the earlier workers to include earache, hearing loss, tinnitus, vertigo, and pain in the side of the tongue. Each of these symptoms was given a specific anatomic explanation related to the basic aetiologic theory of mandibular overclosure with subsequent posterior displacement of the condyles. The displaced condyle was believed to apply pressure to such retro-condylar structures as the auriculo-temporal vessels and nerve and the external auditory meatus, thereby initiating the various symptoms.

Costen's ideas were generally accepted until the works of Sicher (1948) and Zimmermann (1951) which clearly demonstrated the anatomic basis of Costen's syndrome to be unacceptable. Since that time a variety of theories have been proposed to explain the aetiology and pathogenesis of the syndrome, and an even greater number of names

applied to it. For example, De Boever (1979) in his review of "functional disturbances of the temporomandibular joint" cites eight different terms applied by different authors to the condition.

This plethora of terms, and a lack of uniformity concerning the symptoms essential to a diagnosis (Rugh and Solberg, 1979), have led to confusion in the literature and probably retarded progress in elucidating the nature of the condition. In this study the term proposed by Laskin (1969), myofascial pain-dysfunction syndrome (MPD), is employed.

4.1 CLINICAL FEATURES

In the absence of a reliable diagnostic test (Green, 1980), the definitional symptoms of the myofascial pain-dysfunction syndrome include one or more of the following on which there is general agreement (Franks, 1964; Bell, 1969; Laskin, 1969; Griffin and Munro, 1971; Solberg et al., 1972; De Boever, 1979):

1. pain and tenderness in the region of the muscles of mastication and temporomandibular joints - generally unilaterally,
2. sound during condylar movement - clicking or crepitation,
3. limitations of mandibular movement.

Clicking or crepitation in the joint in the absence of other symptoms has not generally been considered sufficient for a diagnosis of MPD (Rugh and Solberg, 1979; Laskin, 1979).

Along with the presence of one or more of these cardinal symptoms, Laskin (1979) believes that certain negative characteristics should also be present for a diagnosis of MPD. He states that "patients with MPD syndrome usually have an absence of clinical,

radiographic, or biochemical evidence of organic changes in the TMJ". This widely held view is, however, challenged by studies of joints obtained at autopsy which show extensive bony changes, including erosion, in the absence of articular soft tissue injury (Carlsson et al., 1968; Bean et al., 1977).

Incidence of MPD

There is general agreement in nearly all reported clinical series that the great majority (70%-90%) of patients with MPD are female (Helkimo, 1979). Agreement on the age distribution of patients seeking treatment has not been so widespread although the 20-40 years bracket has dominated in many studies (Schwartz and Cobin, 1957; Thomson, 1959; Franks, 1964; Carraro et al., 1969; Takada et al., 1971). In some reports, however, the 40-50 year group has been predominant (Gelb et al., 1967; Perry, 1968) while in a few series a fairly even age distribution from young to old adults has been reported (Agerberg et al., 1970; Carlsson and Svardstrom, 1977). Therefore, it may well be that these apparent age differences relate more to the type of specialist, or practice, involved in the study, than the true overall patient population.

Epidemiologic investigations into the incidence of symptoms associated with MPD in whole populations, as opposed to patient groups, have received increasing interest in recent years. This has been especially so in the Scandanavian literature. Agerberg and Carlsson (1972, 1973) used a questionnaire to survey 1215 randomly selected individuals aged 15-74 years in the town of Umea. This revealed that more than one half of those who answered (91% of the total group) had some symptom of dysfunction of the masticatory system, while almost one third had two or more symptoms. The age and sex distribution

of affected individuals in this group was much more even than in most other reported studies. Helkimo (1974), studied symptoms of dysfunction of the masticatory system in Lapps aged 15-65 years in Northern Finland. In this group 43% were aware of symptoms associated with mandibular movement, usually TMJ sounds, and more than half had clinical signs of dysfunction. These findings were equally common in males and females and varied slightly with age.

Thus it is apparent from these and other epidemiologic studies that symptoms associated with MPD are probably more common than previously assumed, and are fairly evenly distributed between the sexes and amongst different age groups. The reason that some individuals seek treatment and others do not, however, is yet to be elucidated.

4.2 AETIOLOGY

The condylar displacement theory of MPD, espoused by Costen (1934), is still supported in a number of altered forms (Weinberg, 1972, 1978). De Boever (1979), however, concludes a review of the supporting evidence for this theory by stating that "the mechanical-displacement theory is still unproved and unconvincing".

Two fundamentally different hypotheses have been proposed to replace the now generally discredited ideas of Costen (Yemm, 1979). The first is that irregularities in the dental occlusion generate enough abnormal muscle activity to cause dysfunction. The second, more recent, proposal is that muscular hyperactivity, not necessarily associated with occlusal abnormality, is the primary factor.

The concept that muscular hyperactivity initiates and maintains the clinical condition is common to both hypotheses. This proposal is based on the frequent clinical observation that many patients with MPD have a tendency to clench and grind their teeth (Franks, 1965; Agerberg and Carlsson, 1973; Lindqvist, 1974) and that this can cause pain in joints and muscles (Ramfjord, 1961a; Olsson and Krogh-Poulsen, 1966). These clinical observations have been supported experimentally by Vestergaard Christensen (1971, 1976) in studies requiring normal adults to grind their teeth for a period of 30 minutes. The majority of the subjects in these studies experienced pain or discomfort following the voluntary hyperactivity and the distribution of symptoms was similar to that reported by patients with MPD.

The suggestion that muscle hyperactivity can lead to muscle pain has also been made for non-masticatory muscles. Swanson (1971) considers muscle pain with this origin to be common, and Wolff (1972) has discussed the involvement of muscle hyperactivity and muscle pain in some types of headache.

While there appears to be wide acceptance of muscular hyperactivity as the main underlying factor in MPD, there remains much controversy as to the cause of this hyperactivity.

Occlusal Disharmony Theory

Occlusal disharmony is probably the most commonly suggested factor in the initiation of muscular hyperactivity (Ramfjord, 1961b; Posselt, 1964; Shore, 1976). It seems to have derived its greatest support in this regard, from the clinical observation that alteration of the occlusion in patients suffering from MPD is followed by clinical improvement in a fairly high percentage of individuals

(Ramfjord and Ash, 1971; Dawson, 1974; Shore, 1976). However, this observation has been challenged by the finding that 64% of a series of patients treated by mock equilibration also showed complete or near complete improvement in symptoms (Goodman et al., 1976).

Added to this conflict in clinical observations is a complete lack of experimental support for the contention that malocclusion or premature contact between opposing teeth can reflexly initiate prolonged hyperactivity of the jaw closing muscles (Yemm, 1979). In fact, the experimental evidence available indicates the opposite. Mechanical stimulation of teeth tends to inhibit rather than excite the muscles which close the jaw (Hannam and Matthews, 1968; Kidokoro et al., 1968; Gibbs and Suit, 1973).

This absence of experimental support for the hypothesis of occlusal disharmony as a major initiating factor in masticatory muscle hyperactivity must challenge its credibility. Further uncertainty arises from the inability to find significant differences in the incidence of occlusal abnormality or deficiency in MPD and control subjects (Thomson, 1971). Indeed, it has been suggested that occlusal equilibration may precede the onset of MPD in some patients (Moulton, 1968).

It seems, therefore, that the occlusal irregularities present in patients with MPD may either have been present before the onset of symptoms and be unrelated to them, or alternatively that muscular hyperactivity associated with the condition has actually changed the mandibular movement pattern, thereby creating an occlusal abnormality (Yemm, 1979).

The Psychophysiologic Theory

The lack of scientific support for the occlusal disharmony hypothesis led to the proposal by a number of investigators (Schwartz, 1955, 1959; Franks, 1965; Laskin, 1969) that hyperactivity of the jaw muscles originates in the central nervous system as a response to physical or psychological stress.

The occurrence of increased muscle tension in non-masticatory muscles in humans under stress is well documented (Goldstein et al., 1964; Goldstein, 1965). This phenomenon has also been confirmed in the masticatory muscles of normal humans (Kydd, 1959; Perry et al., 1960; Yemm, 1969a, 1969b, 1971). Further support for the psychophysiologic theory of MPD comes from a variety of clinical observations and experiments.

Numerous studies of large numbers of MPD patients have concluded that this group is characterized by many personality traits typical of patients with psychophysiologic disorders (Lupton, 1969; Fine, 1971; Rothwell, 1972; Pomp, 1974; Schwartz et al., 1979). In addition, Lupton (1966, 1969), from a study of large numbers of MPD patients using psychometric techniques has suggested that these individuals have a tendency to generalized somatic tension as well as tension relieving oral habits. Further evidence of a central abnormality in MPD patients is afforded by studies which show a tendency for this group to have a much higher incidence than the normal population of such stress related illnesses as migraine and backache (Lupton, 1966; Berry, 1969). The high degree of success in the treatment of MPD with placebo drugs, placebo splints and mock occlusal equilibration is a further indication of the role of psychological factors in the aetiology of the condition (Greene and Laskin, 1971, 1972;

Goodman et al., 1976).

Electromyographic studies of MPD patients in experimental stress situations have shown that their masticatory muscles not only respond dramatically to stress, but respond more than other skeletal muscles in the same patient (Johnson et al., 1972; Mercuri and Laskin, 1979). Other studies have produced evidence that patients with MPD generally have hyperactive muscles of mastication and that, when normal muscle function is restored, the symptoms are reduced or eliminated (Munro, 1972; Skiba and Laskin, 1976; Reuben and Laskin, 1977).

Thus, although there are still unexplained areas in the understanding of the aetiology of MPD, the clinical and experimental evidence available at present forms a solid basis for the psychophysiological theory.

4.3 ACQUIRED JOINT PATHOLOGY

The realization that many of the signs and symptoms of MPD are associated with abnormal muscle activity has led to the suggestion that secondary pathologic involvement of the TMJ may occur if the syndrome persists for long enough (Murnane and Doku, 1971; Yemm, 1979; Laskin, 1979). Laskin (1979) believes that an alteration in the position of the jaw as a result of abnormal muscle function "can produce anatomical derangement of the joint structures as well as ultimately lead to degenerative arthritis in the TMJ". He goes on to suggest that constant loading and unloading of the joint, secondary to bruxism or tooth clenching, can accelerate the degenerative process, and that since both joints function as one unit, the changes can involve both joints. This loading and unloading of the joint corres-

ponds to the repetitive impulse loading which Radin et al.(1972) have proposed as the major aetiological factor in osteoarthritis of other synovial joints.

The belief that both joints may be involved by the degenerative process, although symptoms are unilateral in the majority of MPD patients (Greene et al., 1969), is supported by Yemm's (1971) findings that although only one masseter muscle may be painful in an MPD patient, both masseters respond equally to experimental stress. Therefore it seems entirely possible that joint changes secondary to abnormal muscle activity could occur bilaterally in an MPD patient, even though the symptoms are unilateral. An explanation as to the unilateral occurrence of symptoms in most patients was not found in the literature surveyed.

Toller (1977) studied the ultrastructure of condylar articular surface biopsies from ten patients with severe "pain-dysfunction syndrome". He found the appearance of these tissues to be indistinguishable from similar material from patients with frank osteoarthritis of the condyle, and concluded that these findings suggested a relationship between long standing "pain-dysfunction syndrome" and the onset of degenerative joint disease.

4.4 RADIOLOGY IN MPD

It is generally agreed (Goss, 1974; De Boever, 1979) that TMJ radiographs of MPD patients are essential in order to eliminate the possibility of gross pathology such as fractures or tumours. However, the value of radiology for other aspects of the painful joint remains controversial.

Some clinicians (Ricketts, 1964; Weinberg, 1972) believe that changes in the spatial relationships of condyle and fossa can be accurately diagnosed on joint radiographs and are of great importance in the diagnosis and subsequent management of MPD. Others, however, contend that no correlation exists between anatomic relations in the joint and joint disorders (Lindblom, 1960; Taylor et al., 1972). In a group of normal, asymptomatic subjects, no consistent pattern of condylar concentricity could be found by Kovalesski et al. (1976), and Tveito (1974) showed that accurate determination of the real joint space, by radiographic techniques, was fraught with difficulty. The general consensus (De Boever, 1979) seems to be that radiological alteration in the spatial configuration of the TMJ is not important in the patient with MPD, and its association with the largely discredited mechanical-displacement theory of the syndrome renders it doubly suspect.

The other aspect of joint radiology, the significance of which remains unclear, concerns the diagnosis of structural damage in the MPD patient. Laskin (1979) has stated that patients with MPD usually have an absence of radiographic evidence of organic joint changes. However, at the same time he suggests that persistent MPD can ultimately lead to degenerative arthritis in the TMJ. As previously mentioned, this view has been challenged, and will be discussed in the review of the radiology and diagnosis of TMJ osteoarthritis.

CHAPTER V

REMODELLING OF THE TEMPOROMANDIBULAR JOINT.

Remodelling is a process of biological adaptation to altered environmental circumstances and Moffett (1966) has defined articular remodelling as the morphological adaptation of joints in response to biomechanical stress. Although the termination of skeletal growth is usually accepted as occurring at about 20-25 years of age, it is well recognised that growth and remodelling changes occur in the bony skeleton throughout adult life. Such changes inevitably affect the associated articular surfaces which may respond by adaptive remodelling, of both soft and mineralized joint components, in order to maintain harmonious mechanical function (Storey, 1975).

In the jaws as well, there are generally significant alterations in the dentition during the course of life. Thus the temporomandibular joints are probably subjected to greater variations in their articulating elements than any other joint. The slow but continuous remodelling which appears to occur in these joints throughout life is therefore not surprising. Such adaptation seems generally aimed at maintaining the joint in a state of health with acceptable functional capacity. However, where the stimulus to remodelling is excessive, or the adaptive capability of the joint tissues is reduced by age, disease, or other factors, it appears that the resultant structural changes may occur to a pathological extent with subsequent development of osteoarthritis (Blackwood, 1966b; Meikle, 1979).

5.1 INCIDENCE OF REMODELLING

Examination of both autopsy and skeletal material has shown that adaptive remodelling changes are virtually ubiquitous in the adult

TMJ.

Moffett et al. (1964) published the results of a study on 34 temporomandibular joints obtained at autopsy of subjects aged 45-81 years. Every specimen exhibited microscopic evidence of remodelling activity in some area of the articular tissue and subchondral bone, and in many the resultant changes were visible macroscopically. Blackwood (1963) described histologic remodelling changes in most of the adult material examined in his series of 530 mandibular joints obtained at autopsy. In a similar examination of 102 mandibular joints obtained at autopsy from subjects aged 20-93 years, Oberg et al. (1971) noted macroscopic signs of advanced articular remodelling in 57 joints and at a histologic level changes were observed to a much greater extent than this. Macalister (1954), in one of the earliest studies of this type, examined 69 TMJ's from 64 cadavers and found histological abnormalities in 60 of these joints.

Mongini (1972) investigated macroscopic and microscopic remodelling of the mandibular condyle in 100 dry skulls from subjects of both sexes aged between 18 and 67 years at death. Six anteroposterior sections of the condyles were taken from each skull (3 per side - lateral, central and medial), and examined with an incident light microscope. He concluded that, except for the youngest age group of 18-22 years, "remodelling of some kind was a virtually constant finding".

Temporal and condylar joint components were generally observed to have a more or less equal incidence of histologic remodelling changes in the above studies. However, several workers, including Oberg et al. (1971), Bean et al. (1977), Hansson and Oberg (1977), and Wedel et al. (1978), found a greater incidence of macroscopically observable remodelling change in the condyle rather than in the

temporal articular surface.

5.2 THE MECHANISM OF REMODELLING

Articular remodelling has been classified by Johnson (1962) into three categories, progressive, regressive and circumferential. Although Johnson referred to joints lined by hyaline articular cartilage, Moffett et al. (1964) and Blackwood (1966a) confirmed the presence of all three types of remodelling in the mandibular joints.

The following descriptions of the histologic features of remodelling in the human TMJ are based on those given by Moffett et al. (1964) and Blackwood (1966a).

Progressive Remodelling

This results from excessive proliferation and formation of new cartilage with subsequent conversion to subchondral bone, thereby adding length to the end of the bone.

The first changes occur in the cells of the proliferative zone, there being marked hypertrophy with increased matrix production and cellular proliferation. This eventually results in a thickening of the subjacent fibro-cartilaginous layer which is in turn followed by an advance of mineralization into the newly formed tissue. In the normal joint the mineralizing front advances evenly into the fibro-cartilage, its surface remaining parallel to the articular surface. Mineralization continues until the width of unmineralized fibro-cartilage returns to normal.

While these changes occur deep in the articular soft tissues,

there are no apparent alterations in the covering articular layer, the thickness of which remains remarkably constant. When remodelling activity ceases, the proliferative zone also returns to its normal width. Part, or all, of the mineralized cartilage, is eventually replaced through endochondral bone formation, with the result that the subchondral plate of bone advances towards the joint cavity.

In older joints, especially those with histological evidence of osteoarthritis, the above pattern of changes is much more irregular.

Regressive Remodelling

Whereas progressive remodelling seems to be initiated in the articular soft tissue, with associated changes appearing later in bone, the process appears reversed in regressive remodelling. Osteoclastic resorption of subchondral bone and mineralized cartilage is the earliest recognisable change. This continues until a defect filled by vascular undifferentiated mesenchymal tissue is produced. The mesenchyme then differentiates into the same mixture of fibrous connective tissue and fibrocartilage seen in the overlying articular tissue. Eventually, the subchondral cortical plate is replaced at a lower level than before and so a reduction in the vertical dimension of the articular surface is effected.

Throughout this process the surface articular zone remains intact and conforms passively to the changes occurring beneath it. Reduction of the articular tissue to its normal thickness occurs as the surface layers are worn away.

Peripheral Remodelling

Peripheral or circumferential remodelling produces an increased diameter of the chondro-osseous junction. It begins as a thickening of the fibro-cartilaginous zone with an increase in thickness of the mineralised layer extending outwards from the articular region. This occurs in a similar fashion to that seen in progressive remodelling. The newly mineralized cartilage is replaced by bone but there is also simultaneous periosteal bone apposition. In this way osteophytic lipping develops and large outgrowths may form which, in older patients, consist entirely of bone with an intact articular covering.

As with the other varieties of remodelling, the surface articular layer remains intact, and passively follows the changes beneath it, while maintaining a remarkably uniform thickness.

5.3 THE AETIOLOGY OF REMODELLING IN THE TMJ

Carlsson and Oberg (1979) conclude their review of the literature on remodelling in the TMJ with this statement: "All these studies on skull and autopsy materials have shown that remodelling of the TMJ is very common and they strongly indicate that it is related to conditions in the dentition and to functions of the masticatory system". These authors believe that change in functional stresses within the joint, induced by alterations in the dentition or function of the jaws, activates the undifferentiated mesenchyme of the proliferative zone seen in the condylar articular surface and to a lesser extent the articular eminence (Hansson and Nordstrom, 1977).

Brown, (1965) has drawn attention to the unique importance of

the dentition in the mandibular articulation, the interdependence of teeth and the coupled articulation being manifest in the following ways:

- (a) the occlusal surfaces guide the mandible and consequently the condyles during contact movements.
- (b) the teeth meeting in occlusion limit condylar movements after jaw closure.
- (c) pressure sensitive nerve endings in the periodontal ligament are essential units in neural control of jaw muscles.

The only non-functional factor which has been associated with TMJ remodelling is age. There is general agreement from studies which have included juvenile material (Oberg et al. 1971; Mongini, 1972; Hansson and Oberg, 1977) that histological evidence of remodelling is rare below the age of 20 years, this being the stage at which condylar growth ceases. In material from subjects over the age of 20 years, Moffett et al. (1964) and Yale et al. (1966) were unable to correlate age with the extent of remodelling activity. Mongini (1972) states that "the incidence of remodelling increased rapidly between the ages of 18 and 25, after which age has no significant influence". In contrast to these conclusions, Oberg et al. (1971) found gross remodelling in 40% of individuals aged 20-39 years and 60% aged over 40 years. Microscopic evidence of remodelling change was much more common in both groups however. Also, in this study there were very few fully dentate individuals over the age of 50 years and very few edentulous subjects under this age, so that statistically reliable differentiation between age related, and dentition related change was difficult.

Even where age is considered relevant to remodelling changes,

the role ascribed is minor, and the overwhelming opinion among workers in the field is that remodelling depends mainly on factors of a functional or mechanical nature. The following comprise the more important of these.

Dental Abrasion

A number of studies have revealed a close correlation between the degree of dental abrasion and the extent and nature of TMJ remodelling. Abrasion is one of the few associated factors which can be regarded as being of a physiologic, rather than a pathologic or iatrogenic, nature.

Mongini (1975) examined 100 crania from male and female subjects aged 20-53 years, all with complete but variously abraded dentitions. He concluded that there was a definite relationship between the extent of condylar remodelling and the extent of tooth wear. As well, there was an association between the ultimate condylar shape produced by such remodelling and the pattern of such dentitional wear. Similar results were obtained in a survey of Australian Aboriginal skulls by Seward (1976). Brown (1965) stated that gross changes seen in the TMJ of Australian Aboriginal skulls "usually accompany marked tooth attrition". These findings were further supported by Richards (1978), who concluded from a survey of 101 crania of Australian Aboriginals that a significant association existed between the rate and extent of dental attrition, and the degree of bony change in the TMJ.

Tooth Loss

From their studies of TMJ autopsy material, both Blackwood (1963) and Moffett et al. (1964), suggested a correlation between the

amount of remodelling activity and the degree of edentulousness. Blackwood (1963) observed that totally edentulous subjects commonly exhibited a typical pattern of joint remodelling due to "a post-normal position of the condyle accompanied by overclosure of the mandible". Takiguchi and Kamijo (1975) also noted typical and extensive changes in both the condyle and articular eminence in edentulous subjects. Oberg et al. (1971) demonstrated statistical significance in the association between tooth loss and changes in the TMJ. Mongini (1972) produced similar results and said that in subjects of similar age, "remodelling increased in extent in keeping with the partial edentulism severity". Other studies on human material by Agerberg et al. (1969) and Kopp (1977b) lend further support to this relationship. However, Ericson and Lundberg (1968) could find no difference in the radiographic signs of joint change in asymptomatic patients with varying numbers of natural teeth. This may possibly be attributed to the inaccuracy of radiographic demonstration of such changes when compared to microscopic examination.

Experimental animal studies are generally in agreement with those on human material. Furstman (1965) removed quadrants of molar teeth from rats and found resultant morphologic changes in the mandibular joints. Breitner (1940, 1941) removed maxillary molars and premolars from adult Rhesus monkeys, with resultant gross and microscopic evidence of remodelling of the condyle and articular fossa. Ramfjord et al. (1971) repeated this experiment but removed mandibular teeth, and although gross changes did not occur, there was histologic evidence of accelerated remodelling activity. More dramatic changes resembling osteoarthritis occurred in rats following removal of their maxillary molars by Cimasoni (1963).

In contrast, Pietrokovski (1970) was unable to demonstrate any microscopic joint changes following removal of opposing pairs of teeth in five adult Rhesus monkeys. However, these animals were left with at least the support of bilateral opposing premolars and this situation may have presented insufficient stimulus for the induction of remodelling activity.

The consensus from human and animal studies is that tooth loss probably plays a major role in the induction of remodelling change in the TMJ.

Alteration in Occlusal Level

Rapid changes may occur in the occlusal vertical dimension as a result of prosthodontic procedures, orthodontics, surgery and major facial trauma. There is, however, very little knowledge available concerning the effects of such change on the TMJ. The observation of typical remodelling patterns associated with total edentulousness by Blackwood (1963) and Takiguchi et al. (1975) has been noted, as has Blackwood's explanation based on overclosure with resultant posterior positioning of the condyle. There were no human studies found on adaptive remodelling associated with an increase in vertical dimension.

Such occlusal alteration has been investigated in animals however. Breitner (1940, 1941) raised the occlusal vertical dimension in a Rhesus monkey by means of cap splints and found resultant bone formation on the roof of the articular fossa and posterior surface of the condyle, and resorption on the anterior surface of the condyle. Similar recent experiments on rats (Lindsay, 1977) and monkeys (Gianelly, 1970; Ruben and Mafla, 1971) have all produced significant remodelling changes in the TMJ.

Orthodontic Forces

Forces applied in orthodontic treatment may act directly, or indirectly via resultant occlusal change, on the TMJ.

In Breitner's (1940, 1941) pioneering investigations, forces similar to those used orthodontically were applied to the dentition of young monkeys. Resultant histological alterations were observed in the condyle and glenoid fossa. Similar studies on growing monkeys (Baume and Derichsweiler, 1961; Janzen and Bluher, 1965; Stockli and Willert, 1971; and McNamara, 1975) and growing rabbits (Gupta et al. 1971) confirm the production of remodelling changes in the TMJ and also suggest that permanent skeletal adaptations may result from such forces applied to young animals. In a human radiological study, Thilander (1965) found condylar alterations in sixty children after chin-cap therapy, also suggesting a remodelling response to such forces.

Similar experiments in adult monkeys have produced different results however. Ramfjord and Hiniker (1966), Hiniker and Ramfjord (1966), Ramfjord, Walden and Enlow (1971), and Blankenship and Ramfjord (1976), reported on the results of anterior, posterior and lateral displacements of the mandible in adult Rhesus monkeys. Neither in short or long term experiments were changes in skeletal relationship produced, but minor adaptive remodelling of the TMJ was observed in all cases. McNamara (1975) supported these findings in a study using adult monkeys. McNamara concluded that major structural adaptability in the joint is dependent on the level of maturation although minor remodelling alterations can occur in the adult animal.

Condylar Injury and Surgery

Change in condylar structure or position may occur

following trauma or surgery.

Lund (1974) and Lindahl and Hollender (1977) have described restititional remodelling of the condyle following its fracture dislocation in children. In these subjects radiographic follow-up after the initial injury often demonstrated complete resorption of the displaced fragment and outgrowth of a new condyle-like process from the fracture site. In adolescents, similar injuries showed a tendency to incomplete remodelling, with partial resorption of the displaced fragment plus outgrowth of a new condylar process resulting in a typical Y-shaped condyle. Moffett (1966) illustrates such a condyle with a second glenoid fossa which had formed to accommodate the anomaly. Where the condyle was retained within the fossa following fracture, remodelling eventually reproduced a normal or near normal joint relationship in the younger age groups. Fracture dislocations of the condyle studied in young monkeys, elicited similar restititional healing (Walker, 1960; Boyne, 1967).

In older adolescents and adults with condylar fractures, remodelling generally results in only minor adaptive changes of the condylar head and temporal joint complex (Lindahl and Hollender, 1977).

Surgical alteration of the condyle or its position has also been shown to stimulate adaptive change. Poswillo (1972) demonstrated regeneration of a functional mandibular condyle in both man and adult monkeys following high condylectomy. Hollander and Ridell (1974) described adaptive condylar remodelling where slight but persistent displacement occurred following orthognathic surgery. In a follow-up of ten patients following unilateral meniscectomy and two patients after unilateral condylectomy, Agerberg and Lundberg (1971) detected

distinct radiographic changes in the operated joints and interpreted these as signs of remodelling. In contrast, Banks and Mackenzie (1975) found very little radiologic evidence of remodelling in the temporomandibular joints of adult humans and adult monkeys following the operation of condylotomy which produces a minor condylar displacement.

5.4 THE RADIOLOGY OF ADAPTIVE REMODELLING IN THE TMJ

The macroscopic and microscopic features of adaptive remodelling in the TMJ are well documented. There have also been numerous studies on the radiographic appearances of osteoarthritis in the TMJ and the often dramatic remodelling associated with this condition. However, there is little information available in the literature concerning the radiographic appearances of non-pathologic adaptive remodelling of the TMJ. This is probably a result of the much greater clinical significance attributed to OA, and the minor nature of many adaptive remodelling changes which mitigates against radiographic detection.

Some of the radiographic changes which may occur during adaptive remodelling can be determined by examining the histology of such remodelling.

The essential radiographic feature of progressive remodelling is sclerosis due to thickening of the cortical plate as the hypertrophied fibro-cartilaginous articular tissue is hypermineralized and then ossified. Cortical bone resorption in regressive remodelling produces a typical erosive appearance, but the ultimate result of both progressive and regressive remodelling is a change in shape with a normal cortical plate, albeit at a new level. Peripheral remodelling, a combination of progressive remodelling and periosteal bone deposition at an articular margin, produces osteophytes and eventually large bony

outgrowths. These have the typical radiographic appearance of a bony excrescence.

"Deviation in form" is the general description applied to the radiographic features of completed remodelling of the TMJ, (Oberg et al., 1971; Hansson and Oberg, 1977). At a more specific level, Lindvall et al. (1976) describe such deviation as consisting of either flattening, osteophyte formation, or the presence of a beak. These authors also regard cortical erosion and sclerosis as radiographically significant in their evaluation of morphologic joint change, and distinguish non-pathologic from osteoarthritic alteration on the basis of the severity of these changes, along with those of deviation in form. Worth (1979) regards faceting or flattening of the normally curved condylar surface as one of the most common radiographic abnormalities, and says that the facet may be smooth or irregular with normal, increased or decreased density. Mongini (1977) also described flattening and flaring as the most prevalent radiographic alteration of condylar shape, but, in addition, notes flattening of the corresponding articular eminence to be an almost constant accompanying feature. As well as the above mentioned characteristics, Hansson and Petersson (1978) include the presence of a concavity in the cortical bone, with a well defined cortical lining, as one of the radiographic features of joint change.

The radiographic diagnosis of adaptive remodelling depends upon the position and extent of associated changes, the radiographic projection employed, the technical excellence of the radiography, and the perception of the radiologist as to what constitutes a remodelling change.

In spite of the histologically proven presence of remodelling activity in each of the 34 joints examined, Moffett et al. (1964)

stated that "the majority of the joints were radiographically normal". Similarly Toller (1973) could find radiographic joint change in only 8 of a group of 100 randomly selected patients without a history of TMJ symptoms, even though epidemiological evidence suggests that remodelling alterations are virtually universal in adult TMJ's.

Site of Remodelling

Radiographic detection of adaptive remodelling in the TMJ is aided by a knowledge of the sites in which alterations most commonly occur.

Moffett et al. (1964) observed TMJ remodelling to be mainly of the progressive and regressive varieties and generally both occurred simultaneously in different parts of the same joint. Oberg et al. (1971), Hansson and Oberg (1977), and Wedel et al. (1978) agreed that remodelling was equally common in the condylar and temporal joint surfaces, although macroscopic change was shown to be more common in the condyle and arthritic changes far more common in the temporal component.

Both Blackwood (1963) and Moffett et al. (1964) reported a definite pattern to the remodelling activity they observed and their findings were in general agreement. Moffett et al. (1964) produced a "map" of the pattern of remodelling sites in the TMJ. (Figure 3).

An important feature of the location of such remodelling changes is their tendency to mainly involve the lateral regions of affected joints (Carlsson et al., 1968; Oberg et al., 1971; Mongini, 1972), as these are the areas most reliably depicted with current radiographic techniques.

Peripheral remodelling is most often situated at the anterior margin of the condyle, probably influenced by the tendinous attachment and pull of the lateral pterygoid muscle.

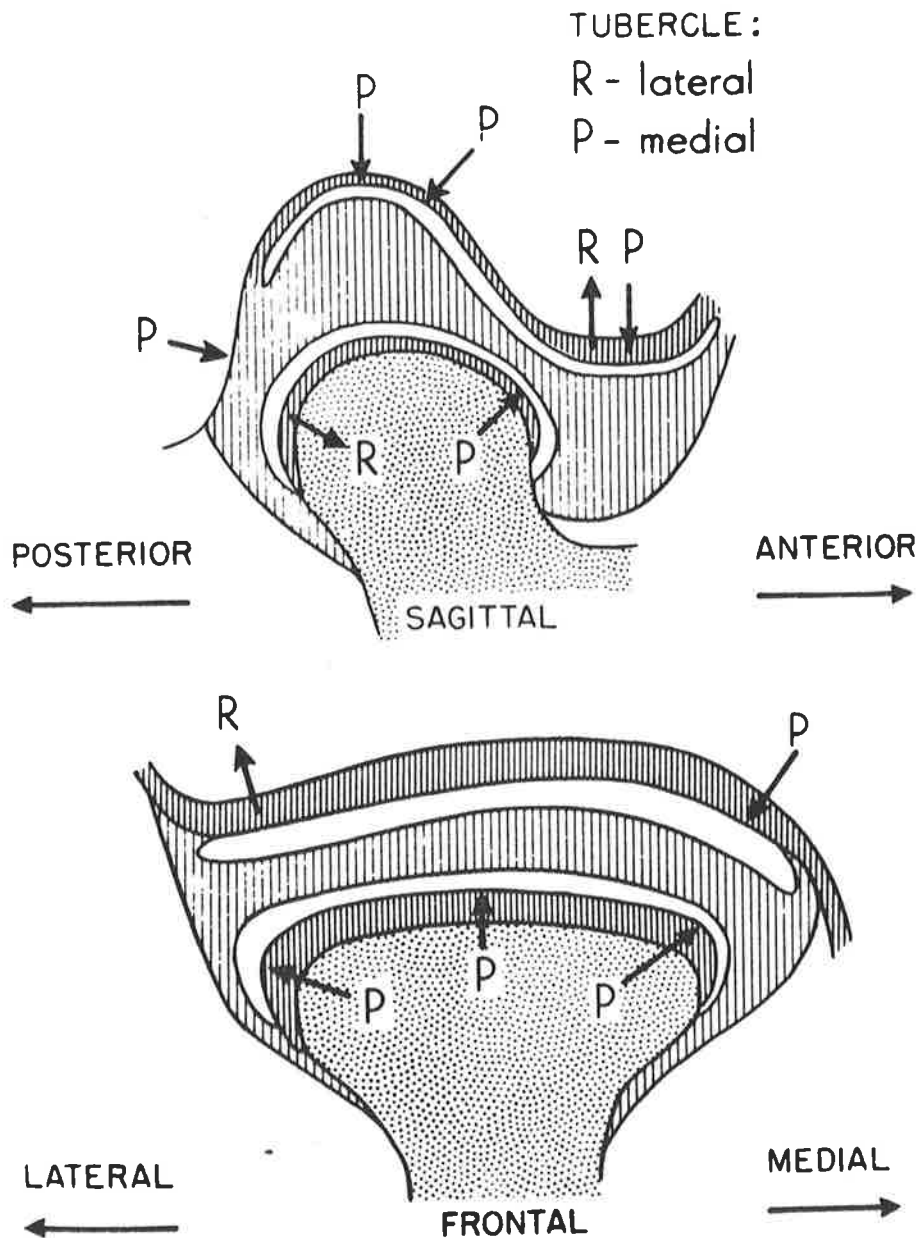


Figure 3: Composite diagrams illustrating distribution of remodelling in thirty adult temporomandibular joints.

Progressive remodelling (P)

Regressive remodelling (R)

From Moffett et al. (1964).

CHAPTER VIOSTEOARTHRITIS OF THE TEMPOROMANDIBULAR JOINT

Osteoarthritis, also known as osteoarthrosis, arthrosis deformans and degenerative joint disease, is a non-inflammatory disorder of movable joints characterized by deterioration and abrasion of the articular soft tissue surface in conjunction with subjacent bony remodelling (Sokoloff, 1979). As articular remodelling in the TMJ gradually merges into osteoarthritis, and since all the mechanisms of adaptive remodelling are active in the osteoarthritic joint, it may be difficult to distinguish between pathologic and merely adaptive change in the joint (Meikle, 1979). Moffett et al. (1964) defined adaptive remodelling as those changes associated with a proliferative response in the articular tissue, and considered osteoarthritis to represent those changes associated with the breakdown of articular tissue: fibrillation, fissuring, eburnation and cystic alterations.

As well as being the most common joint disease in man, osteoarthritis also occurs widely in the animal kingdom having been observed in large and small mammals, animals that swim rather than bear their weight on their extremities, and in birds. It has also been reported as a relatively common finding in certain types of giant dinosaur a hundred million years ago (Sokoloff, 1979) and in Neanderthal man as early as 40,000 B.C. (Schwartz and Marbach, 1965).

Osteoarthritis may supervene on other types of pre-existing joint damage, inflammatory or non-inflammatory, and in such instances is termed secondary osteoarthritis in contradistinction to those forms in which no traumatic or other predisposing articular aetiology can

be elicited. In the latter case intrinsic degeneration of the articular tissue is presumed to underlie the disease development and this is called primary, idiopathic or non-specific osteoarthritis.

As with other synovial joints, the TMJ is commonly affected by osteoarthritis. However, the condition does not appear to constitute a major clinical problem. Toller (1974) believes that osteoarthritis of the TMJ is not often recognised by clinicians and that therefore it must often be clinically silent. He reported that of nearly 2000 patients with TMJ complaints, only 150 or 8% were diagnosed as having osteoarthritis.

Despite its ubiquitous nature, and occurrence in many animals which can provide good models for experimental study, knowledge of the causes and natural history of osteoarthritis is relatively sparse.

6.1 INCIDENCE OF OSTEOARTHRITIS

Autopsy examinations show the first signs of osteoarthritis (OA) appearing around the second decade of life and by age 40 years, 90% of all persons exhibit affected weight-bearing joints (Moskowitz, 1979). Collins (1949) reprints a table of findings from 1002 autopsies showing a 3.1% incidence of OA in the knee of the 15-19 years age group, increasing progressively with age to a 100% incidence in the 80-95 years age group. Moskowitz (1979) cites a radiographic survey of hands and feet which revealed an overall 37% incidence of OA in adults. The rate increased from 4% in persons 18-25 years of age, to 85% at age 75-79 years. A similar survey in the

north of England demonstrated radiographic signs of OA of the hands, feet, back, knees and hips in approximately 50% of adults (Lawrence et al., 1966). Radiographic changes, however, are not necessarily associated with symptoms. Only 30% of subjects with marked radiographic changes in hands, wrists, or knees, had complained of associated pain in a study by Cobb et al. (1957).

Kellgren et al. (1963) and Gordon (1968) have shown an approximately equal sex incidence for OA when all ages are considered. Under age 55 years however, the disease was either equally distributed or slightly more prevalent in males, whereas over this age it was more common and more severe in females.

OA is also the most common disease affecting the TMJ, and, as with other joints, its incidence increases with age. In the most extensive autopsy study to date, Blackwood (1963) found a 40% incidence of degenerative change in temporomandibular joints of subjects over the age of 40 years. He also observed that the disease was rare before the age of 40, and thereafter its incidence increased with age showing a preponderance in the female sex. Macalister (1954) examined 69 mandibular joints from cadavers aged 18-86 years, most being in the older age ranges, and found histological abnormalities in 60 of these joints. The severity of such change increased with age and of 9 joints with extensive lesions, 7 were female. Oberg et al. (1971) in a purely macroscopic study, noted an overall 22% incidence of degenerative lesions in 102 adult joints. No changes were seen below the age of 20 years, and in joints from individuals over 39 years of age, 27% had degenerative lesions and these were more common in females. In the latter study, lesions were most common in the disc and temporal surface, generally sited in the lateral one-third, whereas

Blackwood (1963) observed initial changes occurring most frequently in the posterior region of the condylar articular tissue. Weisengreen (1975) investigated 198 excised TMJ discs, finding no evidence of degenerative change up to 40 years of age, but a 33% incidence thereafter and this was significantly greater in females. Bean et al. (1977) examined 20 mandibular joints from individuals aged 44-90 years and noted a 45% incidence of degenerative lesions. The lesions were more common in the disc and temporal regions than in the condyle.

A number of studies have reported on the radiographic incidence of OA lesions in the TMJ. The results are not dissimilar to those from autopsy studies, however, there are no generally accepted radiographic criteria for diagnosing TMJ-OA (Carlsson and Oberg, 1979). Only lesions causing extensive bony alterations can be detected radiographically and these may be difficult to distinguish from remodelling changes (Carlsson et al., 1968). A radiographic diagnosis will also exclude many cases, especially of early degenerative change, where soft tissue lesions are present without significant bony alteration (Lindvall et al., 1976). Madsen (1966) cited a 13.5% incidence of what he believed to be arthrotic lesions in 96 symptomatic subjects, 17-90 years old, who were examined radiographically. In a similar study of 80 asymptomatic individuals, Ericson and Lundberg (1968) found a 44% incidence of radiographic change, although they did not designate these changes as necessarily representing osteoarthritis.

Radiographic examinations of patients with symptoms referable to the TMJ by Takada et al. (1971) and Brooke (1977) revealed changes considered to be osteoarthritic in about 10% of TMJ's. In a similar study, Toller (1973) reported an 8% incidence of radiographically

proven "osteoarthrosis" in 1573 patients with TMJ related complaints.

6.2 CLINICAL FEATURES OF TMJ OSTEOARTHRITIS

As previously noted, the presence of osteoarthritic lesions is not necessarily accompanied by clinical signs or symptoms. Consequently, many individuals with TMJ osteoarthritis (TMJ-OA) are probably unaware of the disease and do not require or seek treatment.

Symptoms

The most common presenting complaints are pain in the joint and masticatory muscles, jaw stiffness and difficulty in opening, and joint sounds during mandibular movements (Carlsson, Kopp and Oberg, 1979). Pain and limitation of movement in other joints may indicate a generalized condition.

Clinical Findings

As with the symptoms, clinical findings resemble those associated with uncomplicated MPD (Greene et al., 1969; Carlsson, 1980). There may be tenderness to palpation of the TMJ and masticatory muscles, reduced mobility of the TMJ, and pain and deviation during mandibular movements (Kopp, 1977b). It has been proposed that joint crepitation is a reliable clinical diagnostic sign of TMJ-OA, especially in the later stages of the disease (Toller, 1973; Kopp, 1977b; Carlsson, Kopp and Oberg, 1979). Kopp (1977a, 1977b) compared a group of patients with TMJ crepitation, whom he believed to have TMJ-OA, with another group whom he considered to be suffering from MPD. He was, however, unable to find any significant difference in the signs or symptoms of patients in the two groups. Joint crepitation has been included in a list of MPD symptoms by a number of authors (Goss, 1974; De Boever, 1979) and Findlay and Kilpatrick (1960) were unable to find any

correlation between joint sounds and TMJ pathology.

Osteophytes on the lateral joint margins may be palpable but are usually difficult to differentiate from normal variation (Carlsson, Kopp and Oberg, 1979). Muscular weakness, a common sequela of painful OA in other joints (Bollett, 1969), may be evidenced in a reduced biting force (Helkimo et al., 1975). This is also a feature of MPD (Molin, 1972).

It is apparent that there is no pathognomonic clinical sign or symptom associated with TMJ-OA although crepitation within the joint makes a controversial and generally unsupported claim to such status. However, it can safely be said that a reliable clinical differentiation of MPD and TMJ-OA is not possible at present.

Laboratory Findings

Laboratory findings are generally non-contributory in the diagnosis of TMJ-OA and there is no specific diagnostic laboratory test available (Moskowitz, 1979). However, the exclusion of rheumatoid arthritis by serological testing is important as occasionally the TMJ may be the first articulation affected by the disease (Uotila, 1964.)

6.3 RADIOLOGY OF OSTEOARTHRITIS IN THE TMJ

Radiographic diagnosis of TMJ-OA is severely hampered by the inability of ordinary radiographs to demonstrate alterations in the articular soft tissues, an essential feature of OA. A number of investigations (Carlsson et al., 1968; Oberg et al., 1971; Lindvall et al., 1976; Bean et al., 1977) have found extensive remodelling alterations in the absence of accompanying mutilation of the

overlying articular tissue. Conversely, articular soft tissue destruction may occur in the absence of underlying bony changes.

There are no generally accepted radiologic criteria, specific to TMJ-OA (Carlsson, Kopp and Oberg, 1979; Worth, 1979). However, the radiologic features commonly regarded as significant both in the TMJ and other joints (Hansson and Petersson, 1978; Moskowitz, 1979; Carlsson, Kopp and Oberg, 1979; Worth, 1979) are as follows:

- a. reduced joint space,
- b. osteophyte formation,
- c. erosion of the articular cortical plate,
- d. sclerosis of subchondral bone,
- e. flattening of the normally evenly curved articular surfaces,
- f. sub-cortical "cysts" - rounded areas of bone destruction either superficially or deeply situated beneath the cortical plate.

Such appearances are not pathognomonic of TMJ-OA, or indeed OA in other joints, and may occur in rheumatoid arthritis (Ogus, 1975), ankylosing spondylitis and psoriatic arthritis (Resnick, 1974). As articular remodelling gradually merges into TMJ-OA, and since all the adaptive mechanisms of non-pathologic remodelling are active in the osteoarthritic joint (Meikle, 1979), there is also considerable overlap between the features of OA and those of adaptive remodelling. Although the greatest degrees of remodelling activity are generally associated with OA in the TMJ (Moffett et al., 1964; Oberg et al., 1971), the degree of radiologic change cannot be regarded as an accurate indicator of osteoarthritis (Bean et al., 1977). In addition to the above difficulties, Kopp and Rockler (1978) have shown that even

trained observers may differ substantially in their diagnosis and interpretation of changes in TMJ radiographs.

6.4 DIAGNOSIS

In view of the similarity of symptoms and clinical findings in patients with MPD and those believed to have TMJ-OA, and the inability to accurately diagnose osteoarthritis by radiographic examination, it seems that a reliable differentiation between those two groups is not currently possible. Such a differentiation could, of course, be made histologically or by direct inspection of the joint surfaces, but, at present both processes necessitate a surgical procedure which is not without possible significant complications (Sarnat and Laskin, 1979).

With these problems in mind, the suggestion by Ogus (1979) that MPD and TMJ-OA be considered as one condition under the title of "mandibular stress syndrome" has considerable appeal.

6.5 AETIOLOGY

The aetiology of osteoarthritis is not yet properly understood. The Lancet (1973), in an editorial, likened the disease to "joint-failure" and compared its causation to that of heart-failure. Thus it could develop through an increase in functional demand on a normal joint, or by deterioration in the functional capacity of a joint. If this concept is correct, a variety of aetiological factors could be involved acting via either, or both, of these mechanisms.

There is considerable support for a multiple aetiology in OA. Bollett (1969) stated that "the timeworn but legitimate concept that

there are limited ways in which a tissue can respond to injury applies to osteoarthritis" and instanced the variety of experimental insults which produce degeneration in articular cartilage. Byers et al. (1977) quoted the Subcommittee on Diagnostic Criteria for Osteoarthritis of the 1968 Congress for Population Studies on Rheumatic Diseases, "that the nosologic term osteoarthritis encompasses several pathogenic processes, as yet incompletely understood, each of which results in the appearance of more or less characteristic morphologic changes in articular and periarticular tissues".

Aetiological factors can be classed as systemic or local, depending on the proposed manner of action.

Systemic Factors

(a) Age

Sokoloff (1979) believes that "the most outstanding feature about the occurrence of degenerative joint disease is its relationship to age." Blackwood (1963), Oberg et al. (1971), and Weisengreen (1975) agree that TMJ-OA is rare before the age of 40 years, but increases dramatically in incidence during the fifth decade and thereafter. However, not all joints in the elderly are simultaneously or equally affected and as yet there is no evidence of functionally important age dependent changes in articular cartilage (Freeman and Meachim, 1973).

(b) Heredity

A genetic background to OA has been proposed. Such a predisposition to the disease is established in mice and the genetic character is also manifest by species variability in susceptibility to OA. For example, rats are resistant whereas another rodent, *Mastomys*, develops severe generalized OA by two years of age.

In human studies, Stecher (1955) and Kellgren et al. (1963) have produced good epidemiological support for an hereditary background to OA of the fingers and multiple joint OA, respectively. At a biochemical level, Muir (1977) proposed a genetic determination in the variability of features which might confer better stress resistance to cartilage, thus providing a genetic basis to the development of OA.

Whether a genetic basis to OA in hyaline articular cartilage would also affect the fibro-cartilage of the TMJ is speculative. The possibility has been recognized (Kopp et al., 1976) but is disputed by others (Brooke, 1977). Chalmers and Blair (1974) found no statistical difference between the incidence of TMJ-OA in a control group and a group with generalized primary OA.

(c) Obesity

Because of the obvious extra burden it imposes, obesity has been generally implicated in OA of the weight bearing joints (Kellgren, 1961). A connection has also been suggested between obesity and OA in non-weightbearing articulations such as the sternoclavicular and distal interphalangeal joints (Kellgren and Lawrence, 1958; Silberberg and Silberberg, 1964).

Some experimental (Sokoloff et al., 1960; Walton, 1979) and clinical (Seifert et al., 1969; Goldin et al., 1976) studies, however, have suggested that obesity per se is not a factor in the induction or aggravation of osteoarthritis. Thus the association between osteoarthritis and obesity would appear to be a more complicated problem than has previously been recognised and the role of obesity in the aetiology of OA remains obscure.

(d) Endocrine Factors

An association between diabetes and OA in humans has been reported (Silberberg and Silberberg, 1964) and it is fairly well established that gonadal hormones contribute to OA in mice. However, the sex incidence of OA in joints other than the TMJ is about equal (Gordon, 1968), and there is little evidence that menopausal changes influence the development of OA (Sokoloff, 1979). In contrast, the sex incidence of TMJ-OA is dramatically one sided, varying in different clinical studies as follows:

M:F	1:3 (Hankey, 1954)
	1:4 (Ricketts, 1964)
	1:6 (Toller, 1973)

A preponderance of females with evidence of TMJ-OA is also reported from examinations of autopsy material (Macalister, 1954; Blackwood, 1963; Oberg et al., 1971).

This striking sex incidence, associated as it is with a sudden increase in incidence in the fifth decade, a common time for the onset of menopause, suggests the possibility of a relationship between the aetiology of TMJ-OA in females and an endocrine factor. As yet, however, its exact importance is unknown (Kreutziger and Mahan, 1975).

A relationship between endocrine factors and the aetiology of OA has also been suggested by the use of synthetic steroids in experiments in which repeated intra-articular injections of corticosteroids have produced degenerative joint lesions in rabbits (Salter, 1967) and the TMJ of the Macaca Irus monkey (Poswillo, 1970). Silbermann (1976) produced osteoarthritis-like changes in mandibular joints of mice by the systemic use of pharmacologic doses of triamcinalone diacetate.

Local Articular Factors

(a) Mechanical loading.

Increased mechanical loading of the joint is now widely regarded as the single most important aetiologic factor in OA (Bollett, 1969; Radin et al., 1972, 1973; Br. Med. J. Editorial, 1977; Carlsson, Kopp and Oberg, 1979). Radin et al. (1972) point out that most of the force across a joint is the product of muscular contraction, not weight-bearing, and thereby account for the occurrence of OA in articulations not subject to heavy weight-bearing.

Although the TMJ has been regarded as a non-stress-bearing joint (Robinson, 1946), there is now considerable support for the proposal that, during function, it is subject to load across its articular surfaces (Oberg et al., 1971; Mongini, 1972; Hekneby, 1974; Barbenel, 1974; Kopp, 1977b; Hansson et al., 1977).

As with remodelling, there appears to be a correlation between TMJ-OA and tooth loss (Moffett et al., 1964; Agerberg et al., 1969; Oberg et al., 1971) which could possibly be explained in terms of Hekneby's (1974) calculations showing much greater loading of the TMJ when pressure is applied to premolars rather than molars. Kopp (1977b) studied TMJ crepitation, regarded by some as a sign of OA, in patients with TMJ disorders and found it to be significantly associated with loss of molar support. Conversely, Toller (1973) reported that 70% of 130 patients with a clinical and radiographic diagnosis of "osteoarthrosis of the condyle" had "good or adequate dental status". Neither did Ericson and Lundberg (1968) perceive statistically significant differences in the frequency of radiographic TMJ changes in a group of asymptomatic patients with varying numbers of natural teeth. However, as radiographic change

does not necessarily indicate OA in the TMJ, and vice versa, more credence must be placed in autopsy studies than radiographic studies.

The other major factor which may increase the mechanical load across the TMJ is muscular hyperactivity, and this is now believed to be of considerable importance in the aetiology of OA in the joint. Kreutziger and Mahan (1975) state that female patients with TMJ-OA "show a common and distinctive personality pattern and a marked similarity with certain groups of patients who have psychomatic problems and general somatic tension". As previously discussed, there is also considerable support for the belief that the symptoms of MPD are a direct result of hyperactivity in the muscles of mastication. Toller (1973) observed a history of pain dysfunction symptoms in the affected joint of nearly 50% of 130 patients with TMJ-OA and said that "this strongly suggests some relationship between the occurrence of untreated pain dysfunction syndrome and the later onset of degenerative joint disease". In an ultrastructural study of condyles removed from patients with pain dysfunction syndrome Toller (1977) demonstrated profound structural changes with great similarity to those of frank osteoarthritis. Brooke (1977) suggests that repetitive impulse loading of the TMJ, the aetiologic mechanism for OA proposed by Radin et al. (1972), may occur in patients suffering from the pain dysfunction syndrome and proposes this as a possible aetiologic factor in TMJ-OA. Ogus (1979), on the basis of a belief that both TMJ-OA and MPD are expressions of the same problem, namely "repetitive overload of the joint system", has proposed that they be considered as a single condition.

(b) Joint deformity

Change in the configuration or stability of its components may reduce the functional capability of a joint, thereby producing

a relative increase in the mechanical load. It is probable that traumatic episodes, such as torn ligaments and capsule, a torn or displaced meniscus, detached articular fibrocartilage, intra-articular fracture, and effusion or haemorrhage into the joint, may predispose the TMJ to OA (Worth, 1979). However, in Toller's (1973) analysis of 130 patients with TMJ-OA, only 5% could recall any traumatic incident involving the affected joint. Kreutziger and Mahan (1975) regard trauma as relatively unimportant in the aetiology of osteoarthritis of the TMJ.

Conditions which may affect the joint architecture, impairing its functional capacity, and therefore predispose to osteoarthritis, include, rheumatoid arthritis, pyogenic joint infection, haemophilia with recurrent haemarthroses, metabolic disorders such as gout, and congenital abnormalities such as condylar hyperplasia or hypoplasia (Ogus, 1975; Kreutziger and Mahan, 1975).

A general consensus is apparent in the recent literature, that local factors, especially of a mechanical nature, and possibly in combination with systemic factors, determine the development and progression of osteoarthritis in the TMJ and other joints (Acheson and Collart, 1975).

6.6 OSTEOARTHRITIS AND REMODELLING

Whereas adaptive remodelling consists mainly of alterations in the subarticular layers, OA represents changes associated with a breakdown of the articular surface.

Several studies (Moffet et al., 1964; Oberg et al., 1971) have observed a close relationship between remodelling activity and

OA in the TMJ, with changes in form being virtually always present, and often more extensive, in joints with articular surface lesions. Blackwood (1966b) suggested that, where the demands placed on remodelling activity exceed the physiologic tolerance of the cells in the deeper zones of articular tissue, osteoarthritis may result. As well as excessive functional stimulation, a decrease in the vitality of proliferative layer cells upon which remodelling depends may also initiate OA. Such a decrease could result from systemic factors already mentioned in regard to the aetiology of OA. Moffett (1966) also agrees with this concept.

Radin et al. (1972) explain the close relationship between remodelling and OA by suggesting repetitive impulse loading as the main stimulus to both processes. When subjected to such loading, the joint remodels to better withstand the additional stress. However, if such remodelling also produces bony sclerosis, the affected bone will be stiffer and less effective as a shock absorber for the overlying soft tissue. The latter tissue is less well protected as a result, and if the forces to which it is subjected are increased, there will be a consequent tendency to the development of OA.

Osteoarthritis and Remodelling in the TMJ

Remodelling alterations are evenly distributed between the articulating elements in the TMJ, however, osteoarthritic changes appear to be considerably more common in the temporal region as compared to the condyle (Oberg et al., 1971; Bean et al., 1977). Conversely, macroscopically obvious adaptive remodelling changes were more common in the condyle in both studies. Oberg et al. (1971) describe as not uncommon, the findings of a perforated disc associated with an arthrotic lesion in the corresponding temporal surface while the condyle shows merely marked adaptive remodelling.

Hansson and Oberg (1977) suggest this dissimilarity between the joint components to be a function of differences in their reactive capacities to changes in load. This reactive capacity is in turn dependent on histologic construction. Hansson and Oberg (1977) reported, even in older individuals, the presence of more remnants of proliferative zone undifferentiated mesenchyme in the condyle than in the temporal component. As this tissue is essential to remodelling activity, Hansson and Oberg's (1977) finding could readily explain the seemingly greater adaptability of the condyle, and the greater susceptibility of the temporal surface to OA.

6.7 PATHOGENESIS OF OSTEOARTHRITIS

The descriptive pathology of advanced OA is well known, the end result being a joint deformed by disintegration of articular cartilage, and new bone proliferation at the margins and base of the joint surface. However, the sequence and mechanism of these pathological occurrences remain obscure (Sokoloff, 1979).

The knowledge which does exist, concerning these matters, is almost entirely derived from studies of animal and human joints lined by hyaline articular cartilage, not articular fibrocartilage as is found in the TMJ. Many workers in the field agree with Collins (1949) that the initial lesion in OA occurs at the articular surface. Radin et al. (1972, 1973), however, propose that increased rigidity of the subchondral bone may precede damage to the articular surface. They suggest that the increased rigidity results from trabecular microfractures and bony remodelling initiated by repetitive impulse loading of the joint surfaces.

Sokoloff (1979) marries the theories, suggesting both to be

probably true and intimately related, with the relative importance of cartilage degeneration versus bony changes varying in different joints and different types of OA.

Histology of osteoarthritis

The following description is based on a review by Toller and Glynn (1976) of OA in the TMJ. Apart from the last stage, it is in general agreement with the description of the disease in other joints by Sokoloff (1979).

Stage I. Fibrillation

The earliest histologic change consists of irregularity and then loss of the surface layers of articular soft tissue with collagen bundles fraying off into the synovial cavity. While this occurs, the cartilaginous zone of the articular soft tissue hypertrophies and mineralizes, resulting in consolidation of the articular end plate.

As the disease continues, the articular surface becomes progressively denuded and clusters of chondrocytes, a characteristic of OA, are seen. Eventually the underlying cortical plate, thickened by ossification of mineralized cartilage, is completely exposed, and this eburnated surface may apparently articulate successfully with the joint meniscus.

Stage II. Perforation

In some areas, thinning of the bony end plate may occur to such an extent that punctate failures associated with trabecular micro-fractures result, thereby allowing ingress of synovial fluid under pressure. This violation of the marrow spaces may induce the formation of small cyst-like spaces, lined by fibrous tissue, which slowly enlarge with reactive new bone formation at their periphery. In these

regions the marrow becomes fibrotic and there is generalized trabecular bone destruction.

Stage III. Erosion

Attempts at bony repair of perforations in the articular cortical plate may occur and can be successful, If not, however, the defect may enlarge with more loss of trabecular bone. Fibrosis of the marrow may occur throughout the sub-articular region and complete destruction of large areas of the bony end plate are seen.

Stage IV. Repair

There is no histological evidence of complete repair of a massively eroded articular end plate by new bone formation, leading to a satisfactory cortical surface. However, Toller and Glynn (1976) believe that such a result is seen in many serial radiographs of the disease in the TMJ. They also suggest the likelihood of reformation of the fibrous articular tissue in conjunction with such new bone growth, and cite clinical evidence of concomitant reduction in symptoms in support of their claim.

Biochemistry of Osteoarthritis

The only studies available on this topic refer to hyaline articular cartilage and not articular fibrocartilage. Hyaline articular cartilage is composed of chondrocytes enveloped by a highly hydrated matrix of proteoglycans and collagen. Cartilage carries load by virtue of the interaction of its collagen, proteoglycan, and bound water. The proteoglycan is hydrophilic, thus retaining the water, and collagen retains the proteoglycan.

Muir (1977), from the study of an animal model which closely resembles the human disease, proposes three phases to the biochemical

changes occurring in OA.

Phase I: An increased water content is seen in the focal areas which will later manifest the first signs of degeneration. Also at this stage, proteoglycans are more easily extracted from the cartilage than normally.

Phase II: Focal erosion occurs and is accompanied by some loss of water and proteoglycans. Elsewhere the localized changes of Phase I spread throughout the joint cartilage.

Phase III: Severe erosion develops in focal sites and in these areas water content decreases and there is marked loss of proteoglycan. Throughout the remaining cartilage, the changes seen in Phase II become more marked. Only at this stage would the disease be recognized by its histopathology as OA.

The cells respond to osteoarthritic destruction by a brisk reparative reaction with increased synthesis of protein and glycosaminoglycans (Mankin and Lippiello, 1971). This reaction tends to keep pace with the disease until it progresses from moderate to severe, at which time reparative mechanisms fail and the cartilage is rapidly destroyed (Mankin, 1976).

Whether proteoglycan loss is the result of damaged or abnormal collagen allowing it to leak away, or an enzymatically mediated attack, is controversial. However, the consensus opinion as summarized by Sweet et al. (1977) is that "enzymatic activity is unlikely to be the sole, or even the major factor in the pathogenesis of progressive osteoarthritis and the biochemical changes are much more compatible with collagen and matrix disruption due to focal overloading".

Contrarily, Toller and Glynn (1976) base their theory for the pathogenesis of TMJ-OA on enzymatic damage caused by lysosomal proteases. These are released by the synovium during digestion of "wear and tear" material in the synovial fluid but cannot penetrate normal cartilage. These authors propose that penetration occurs in areas of the articular surface subject to alternate pressure and relaxation, especially if muscle loading across the joint surfaces is excessive. Once such penetration occurs, the articular tissue is slowly broken down with the eventual production of an osteoarthritic erosive lesion.

CHAPTER VIIMATERIAL AND METHODMATERIAL7.1 SUBJECTS

The subjects of this study were 340 female patients from the Dental Department of the Royal Adelaide Hospital. They were separated into four different groups (Table 1.). The first two groups were distinguished by the absence of symptoms related to the temporomandibular joint region, Group I consisting of fully dentate persons, while those in Group II were totally edentulous. Group III comprised approximately equal numbers of dentate, partially dentate and edentulous subjects, all of whom had presented for treatment of pain in the region of the temporomandibular joint. There were 100 subjects, aged from 20 to 69 years, in each of these three groups. The five age brackets, from 20 to 29 years through to 60 to 69 years, were equally represented with 20 subjects in each. Group IV consisted of 40 patients diagnosed by specialist physicians as being morbidly obese. These subjects were aged between 20 and 39 years and there were 20 in each of the two ten year age brackets.

Persons with systemic joint disease such as rheumatoid arthritis, gout, psoriatic arthropathy, or generalized osteoarthritis, were excluded from the study. A history of systemic steroid medication was also grounds for exclusion from the study.

7.2 GROUPS

I. Dentate: Subjects were considered eligible for this group if they had lost no more than one molar or premolar tooth in each quadrant. The presence or absence of third molar teeth was disregarded, as were

TABLE 1.GROUP DISTRIBUTION AND AGE RANGES OF SUBJECTS

GROUP		DENTITION	N	AGE RANGE (YRS)
PAINLESS	Group I	DENTATE	100	20-69
	Group II	EDENTULOUS	100	20-69
PAINFUL	Group III	MIXED	100	20-69
OBESE	Group IV	DENTATE	40	20-39

TABLE 2DISTRIBUTION OF SUBJECTS IN PAIN GROUP BY DENTITION

AGE RANGE (YRS)	DENTITION		
	DENTATE	PART. DENTATE	EDENTULOUS
20-29	17	1	2
30-39	13	6	1
40-49	5	11	4
50-59	1	7	12
60-69	0	6	14
TOTAL	36	31	33

premolar teeth extracted for orthodontic purposes when the resultant spaces had been closed. Preference was given to those with complete dentitions, but in the older age groups such individuals were not common. In order to provide a suitable control group for comparison with the morbidly obese group, obviously overweight individuals were excluded from the 20 to 39 years age range. The first 20 suitable subjects encountered were selected in each ten year age bracket.

II Edentulous: The first 20 completely edentulous subjects encountered, provided the general criteria were satisfied, were selected in each age bracket. Recently edentulous patients were excluded by rejecting all those whose radiographs showed evidence of tooth sockets. The majority of subjects were selected from the Dental Department prosthetic waiting lists and were wearing full dentures at the time of their initial examination. In general, the older the patient, the longer the period of edentulousness.

III. Pain: Subjects for this group were selected from amongst patients who had been examined and treated for complaints of pain in the region of the temporomandibular joint. All subjects had been examined by one of three specialist clinicians, two oral surgeons and a periodontist, each with a particular interest in disorders related to the TMJ. Orthopantomographs taken at the time of initial presentation were available for each subject.

The first 20 subjects encountered, who satisfied both general and group criteria, were selected for each ten year age range. The great majority of subjects had been diagnosed as suffering from the myofascial pain dysfunction syndrome although in several instances a

diagnosis of temporomandibular joint osteoarthritis, based on radiologic findings, had been made. However, in all subjects the symptomatology and clinical findings were similar and typical of the pain dysfunction syndrome.

No attempt was made to choose subjects with a particular type of dentition and in general the younger individuals were fully dentate and the older ones totally edentulous with a scattering of partially dentate individuals in between (Table 2).

IV. Obese: Obese patients must be at least 70% above their calculated ideal weight to be considered for jaw wiring treatment, and only then as a last resort following failure of more conservative measures. The majority of referred patients are in the 20-40 years age group and all have an orthopantomograph taken as part of their assessment of suitability for jaw wiring. Potential candidates are also carefully questioned and examined regarding possible TMJ symptoms and pathology.

To minimize variables associated with the dentition, only fully dentate subjects were selected. The first 20 suitable subjects, in each of the two ten year age ranges, were selected for inclusion in the study. None of these patients had complained of any symptoms in the region of the TMJ although the absence of such complaints was not a prerequisite for inclusion in the study group.

7.3 RADIOGRAPHS

Standard panoramic jaw tomographs* were used exclusively in this study. Details of exposure are: 60-80 kV., 225 m A-sec, anode-film

*Siemens ORTHOPANTOMOGRAPH unit; Palomex OY, Finland.

distance 46cm., medium diaphragm. Kodak X-OMAT S films (XS-5), 15.2cm x 30.5cm, in a PALOMEX metal curved cassette were used. Processing was carried out in a PAKO 14X automatic processor using ILFORD chemistry.

An evaluation of film quality was not made at the time of subject selection. However, on subsequent radiological evaluation, a number of subjects had to be deleted from the study because one or both joints were inadequately depicted. One of the most common reasons for this was the superimposition of patient identification data over a joint.

METHOD

7.4 RADIOLOGICAL ANALYSIS

All radiographs were examined in a quiet, darkened room, using the same fixed intensity illumination. The viewing screen was masked except for a 5cm.x 5cm.square, just large enough to contain one temporomandibular joint area on an orthopantomograph (OPG), Radiographs were examined in a random order and not according to the study group or age group. Each joint was evaluated and scored independently of its contralateral companion and the subject's name, age, and clinical data, were unknown to the examiner at the time. At the start of each examining session, a three minute acclimatization period was allowed prior to the commencement of scoring. A session was limited to one hour's duration in order to minimize observer fatigue.

I. Radiological changes under investigation: The following radiological changes in the condyle and articular eminence of each joint were scored and recorded:

- Osteophyte - local outgrowth of bone arising from a mineralised joint surface.
- Erosion - local area of rarefaction in the cortical plate of a joint surface.
- Flattening - loss of an even convexity or concavity of the joint outlines.
- Sclerosis - thickening of cortical bone on a joint surface.
- Concavity - concavity in the bone contour with a well defined cortical lining.
- Sub-cortical "cyst" - rounded radiolucent area which may be just below the cortical plate or deep in trabecular bone.

Evaluation of the articular fossa, as well as the eminence, was initially intended, but the definition of this region on most films was unsatisfactory to the extent of precluding a reliable assessment.

An attempt was also made to evaluate the size of the condyle and the height of the articular eminence in each joint.

II. Method of scoring: Each of the six radiological changes were scored in both joint components examined according to the following scale:

- 0 - no demonstrable change
- 1 - mild change
- 2 - gross change.

The size of the condyle and height of the eminence were assessed as being normal, above average, or below average.

Where doubt existed concerning the score for a particular feature, the lesser score was assigned.

Standard radiographs, previously selected for each feature (Figures 5-15), were available at all times for comparison with the films being evaluated. An orthopantomographic depiction of a normal TMJ in a 20 year old subject is illustrated in Figure 4.

III. Reliability of Scoring and Recording: The reproducibility of the scoring and recording methods employed was assessed by a double determination procedure. Forty radiographs, 12% of the total, were randomly selected by a non-clinical member of the laboratory staff not involved in the study. Each radiograph was re-examined and the result recorded two months after the initial examination had been completed. The scores obtained on the two separate occasions were then compared.

7.5 DOCUMENTATION

The scored data for both joints in each subject were recorded on one sorter card*. Following completion of this recording, the subject's name, age, and study group, were determined and placed on the card.

Additional relevant clinical information for the pain group was also recorded on the subject's sorter card, and was as follows:

- a. side of symptoms:- left, right, both
- b. type of dentition:- dentate, partially dentate, edentulous.

*"Invicta" sorter cards; Sands and McDougall (Aust.)



Fig. 4: Normal temporomandibular joint as seen on a standard orthopantomograph.

Note the evenly curved outlines and absence of cortical defects or sclerosis.

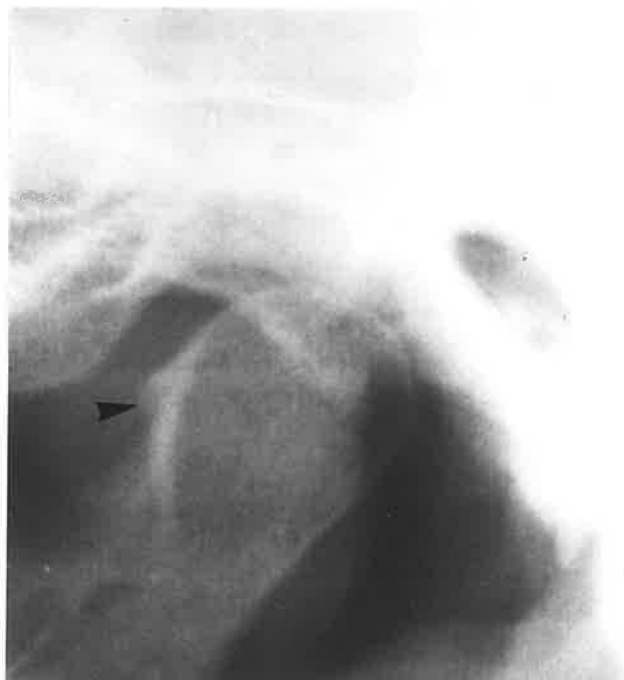


Fig. 5: Osteophyte (arrow) - grade 1.

Note that radiological abnormalities other than that specifically selected as an illustrative example of the particular feature indicated in the respective legends are present in many of the photographs (Figs. 5,6,7,8,10,12,13,14,15).

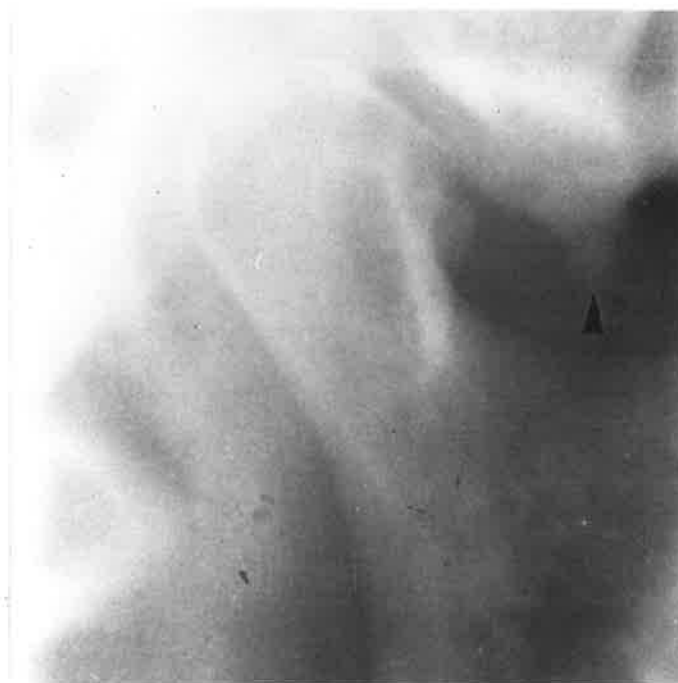


Fig. 6: Osteophyte (arrow) - grade 2.

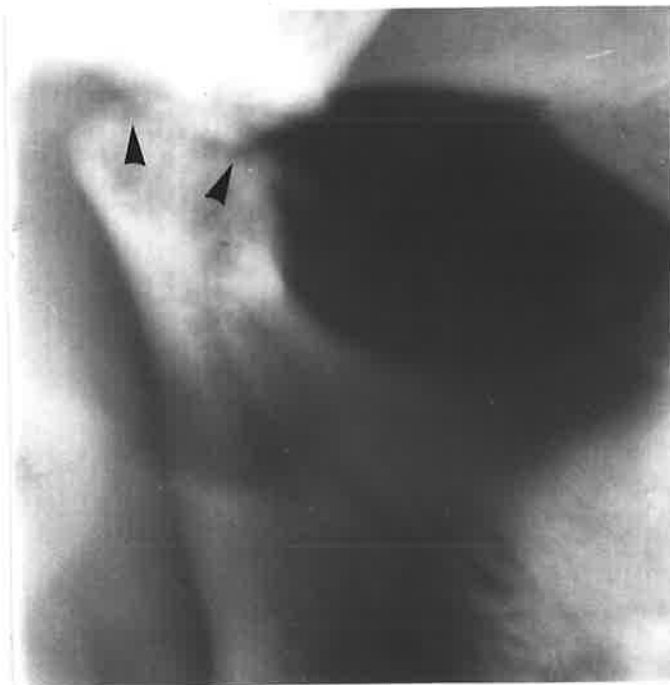


Fig. 7: Erosion (arrows) - grade 1.

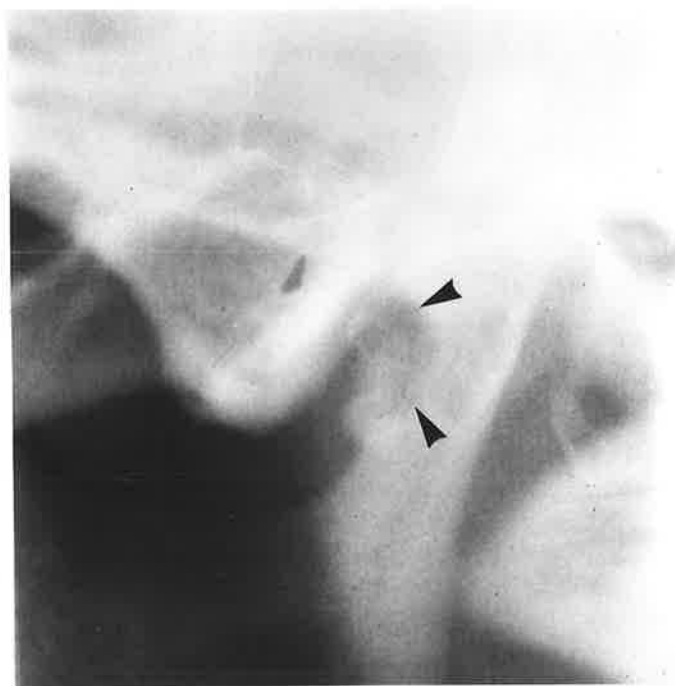


Fig. 8: Erosion (arrows) - grade 2.

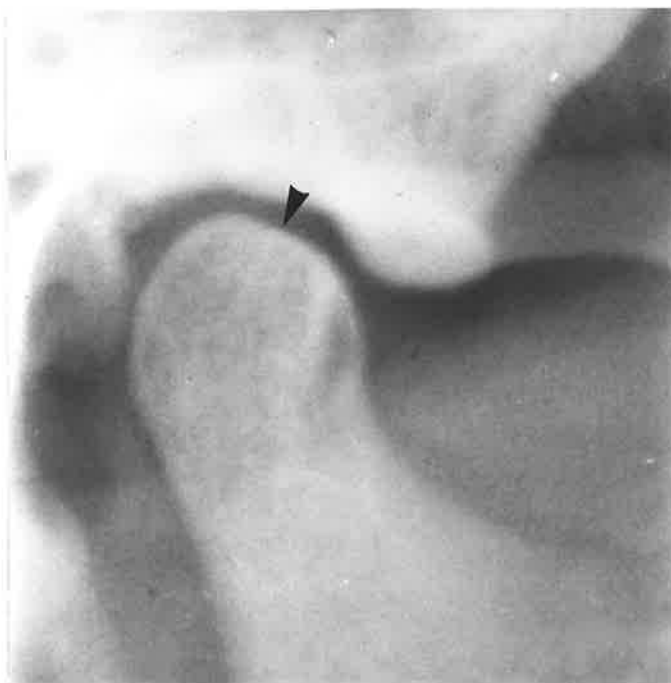


Fig. 9: Flattening (arrow) - grade 1.

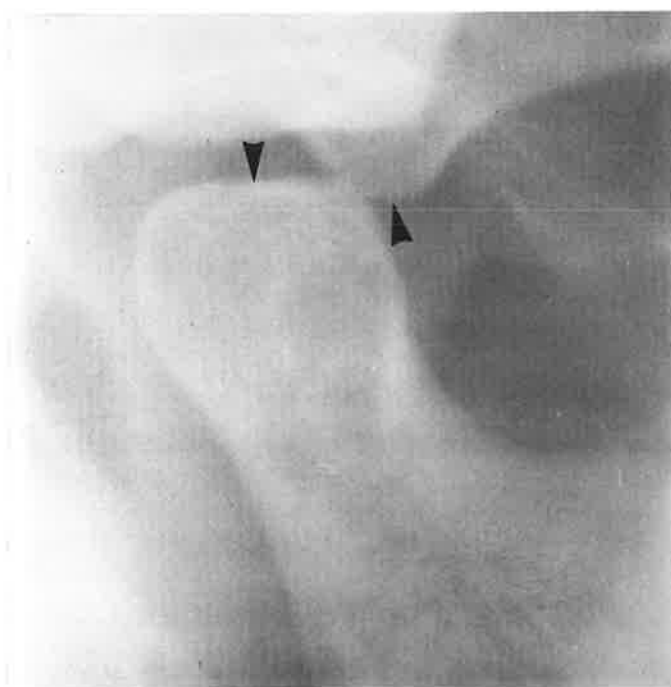


Fig. 10: Flattening (arrows) - grade 2.



Fig. 11: Sclerosis (arrow) - grade 1.



Fig. 12: Sclerosis (arrows) - grade 2.



Fig. 13: Concavity (arrow) - grade 1.

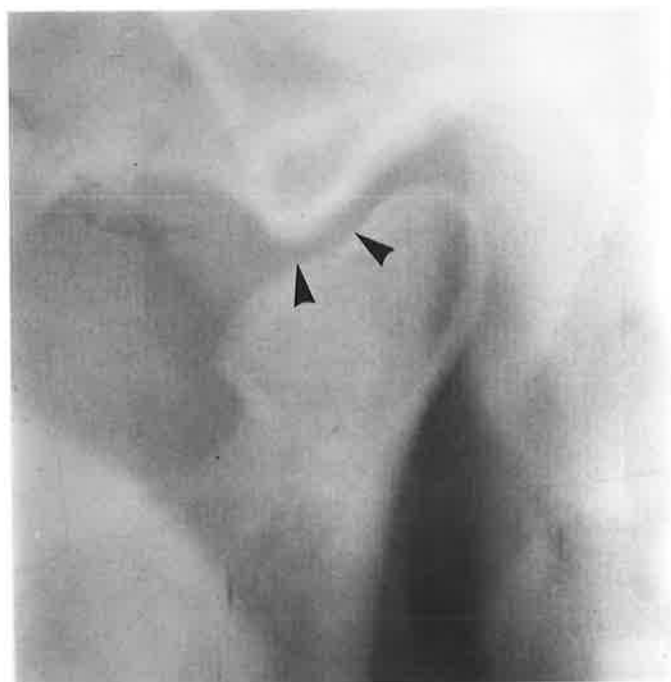


Fig. 14: Concavity (arrows) - grade 2.

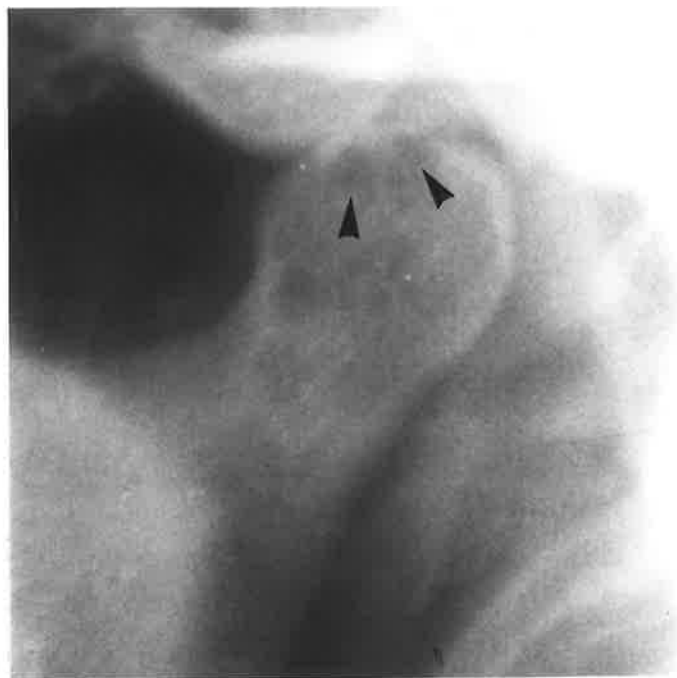


Fig. 15: Sub-cortical "cyst" (arrows) - grade 1.

There were no radiographs found in which this feature was scored as grade 2.

7.6 DATA ANALYSIS

Statistical

Grouped data were compared using the chi-square test in one of two forms. For four-fold contingency tables of the form

a	b	a + b
c	d	c + d
a+c	b+d	N

the statistic χ^2 was calculated using Yates correction in all cases so that the formula was:

$$\chi^2 = \frac{N (1ad - bc) - \frac{1}{2}N}{(a+b)(c+d)(a+c)(b+d)}$$

For other contingency tables with more than one degree of freedom chi-square was calculated as follows:

$$\chi^2 = \frac{(O-E)^2}{E}$$

where O is the observed occurrence of a feature and E is the occurrence predicted by the null hypothesis. The number of degrees of freedom for a contingency table of dimensions Y by Z is calculated to be (Y-1)(Z-1). Results were interpreted using probabilities obtained from the standard tables, with a probability of less than 0.05 ($p < .05$) being regarded as statistically significant in all instances.

Radiological Index:

An index of radiological findings was constructed for each joint in the pain subjects. A similar procedure to that used by Kopp and Rockler (1979) was employed although the radiological findings contributing to the index in the present study varied slightly from that used by these investigators. The index included the following abnormalities:

osteophytes, erosions, flattening and cortical sclerosis. The presence of any of these four features in the condyle and articular eminence of a given joint contributed one unit to the score. Thus each joint could be allotted from 0-8 points.

Each radiological feature in the TMJ, as well as the radiological index, was analysed for any association with symptoms in the pain group.

RESULTS8.1 CLARITY OF RADIOGRAPHS

The majority of orthopantomographs examined provided satisfactory diagnostic detail of both temporomandibular joints and in some this detail was excellent. However, not all films were of an acceptable standard and Table 3 gives the distribution of subjects included in the study and the number deleted because their radiographs did not depict the joint surfaces with adequate clarity.

Although it was originally intended to evaluate the condyle, articular eminence and glenoid fossa in each joint, detail of the fossa proved to be poor in the majority of orthopantomographs. Consequently the examination was restricted to the condyle and articular eminence. It had also been proposed to score the height of the articular eminence and the size of the condyle as average, below average or above average. However, because of the difficulty in locating the exact position of the fossa in many instances, it was often impossible to accurately define the height of the eminence and thus these scores are not included in the results. The great majority of condyles were considered to be of average size and because of this, and the presence of some doubt as to variations in the magnification of the condyles on different films, these results have also been excluded.

8.2 RELIABILITY OF SCORING

The results of the double determination, in which 40 randomly selected films (12% of total) were re-examined two months after the initial examination, are given in Table 4. These figures indicate scoring concordance values varying between 89% and 100% with the lowest values of 89% and 90%, resulting from the scoring for flattening and

TABLE 3AGE AND GROUP DISTRIBUTION OF SUBJECTS INCLUDED IN STUDY

AGE RANGE (YRS)	GROUP			
	DENTATE	EDENTULOUS	PAIN	OBESE
20-29	20	18(2)	20	20
30-39	19(1)	19(1)	19(1)	19(1)
40-49	19(1)	20	20	
50-59	20	20	18(2)	
60-69	19(1)	20	20	
TOTAL	97(3)	97(3)	97(3)	39(3)

Numbers in parentheses indicate subjects excluded because of unsatisfactory radiographs.

TABLE 4SCORING CONCORDANCE - DOUBLE DETERMINATIONS

CONCORDANCE	RADIOLOGICAL FEATURE											
	Osteophyte		Erosion		Flattening		Sclerosis		Concavity		Cyst	
	C	E	C	E	C	E	C	E	C	E	C	E
Concordant present	10	0	3	1	46	16	51	41	5	1	1	0
Concordant absent	67	80	77	79	25	61	21	38	74	79	78	80
Discordant	3	0	0	0	9	3	8	1	1	0	1	0
Overall Concordance (percent)	96	100	100	100	89	96	90	99	99	100	99	100

C = Mandibular condyle

E = Articular eminence

Total number of joints = 80

sclerosis respectively, in the condyle. The concordance values for scoring of the articular eminence are higher than those for the condyle except in the case of erosions where values for both condyle and eminence equal 100%.

8.3 RADIOLOGICAL FINDINGS

The majority of subjects in each of the four study groups were found to exhibit one or more of the six radiological features under examination. Table 5 gives the distribution of subjects for whom no radiological changes were noted in either one or both joints.

The number and percentage frequency of subjects exhibiting radiological changes in the two joint components examined, and as a total for overall occurrence in a subject, are given in Tables 6, 7, 8 and 9. For example, in the 20-29 years age group of dentate subjects, eleven individuals exhibited flattening of one or both condyles, seven individuals exhibited flattening of one or both eminences, and thirteen individuals altogether exhibited flattening in at least one of the joint surfaces evaluated. The total percentage frequencies of affected subjects are graphically presented in Figures 16-21.

The frequency occurrence of each radiological feature in individual joint components, as opposed to subjects, is shown in Tables 10, 11, 12 and 13. For example, in the 20-29 years age group of dentate subjects, sixteen condyles exhibited flattening as did eleven articular eminences and thus twenty-seven joint surfaces, out of a total of the eighty surfaces examined, exhibited this feature. The total percentage frequencies of affected joint surfaces are graphically presented in Figures 22-27, with subdivisions indicating the percentage frequency of radiological features allotted a score of 2 (gross change).

TABLE 5

DISTRIBUTION OF SUBJECTS WITH NO RADIOLOGICAL CHANGES IN ONE OR BOTH JOINTS.

AGE RANGE (YRS)	<u>NO. OF SUBJECTS WITHOUT RADIOLOGICAL CHANGE</u>							
	Dentate ¹		Edentulous ¹		Pain ¹		Obese ²	
	Unilat.	Bilat.	Unilat.	Bilat.	Unilat.	Bilat.	Unilat.	Bilat.
20-29	5	1	6	1	2	0	3	1
30-39	6	2	2	0	1	0	3	0
40-49	2	0	0	0	1	0		
50-59	0	0	1	0	1	1		
60-69	0	0	3	0	0	1		
TOTAL	13	3	12	1	5	2	6	1
Unaffected joints (percent)	9.8%		7.2%		4.6%		10.3%	

¹ N=97 ² N=39

TABLE 6

FREQUENCY ANALYSIS OF RADIOLOGICAL FEATURES, BY SUBJECTS - DENTATE GROUP

RADIOLOGICAL FEATURE		NO. OF SUBJECTS EXHIBITING FEATURE					Total ³
		AGE RANGE (YRS)					
		20-29 ¹	30-39 ²	40-49 ²	50-59 ¹	60-69 ²	
OSTEOPHYTE	C	7(35)	2(10)	2(10)	5(25)	4(21)	20(21)
	E	0(0)	0(0)	1(5)	0(0)	0(0)	1(1)
	Total	7(35)	2(10)	2(10)	5(25)	4(21)	20(21)
EROSION	C	1(5)	0(0)	1(5)	2(10)	0(0)	4(4)
	E	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
	Total	1(5)	0(0)	1(5)	2(10)	0(0)	4(4)
FLATTENING	C	11(55)	12(63)	14(74)	17(85)	14(74)	68(70)
	E	7(35)	5(26)	7(37)	6(30)	9(47)	34(35)
	Total	13(65)	14(74)	18(95)	18(90)	14(74)	77(79)
SCLEROSIS	C	12(60)	13(68)	19(100)	19(95)	18(95)	71(73)
	E	10(50)	4(21)	8(42)	12(60)	11(58)	45(46)
	Total	15(75)	13(68)	19(100)	20(100)	19(100)	86(89)
CONCAVITY	C	4(20)	0(0)	7(37)	1(5)	2(10)	14(14)
	E	0(0)	1(5)	2(10)	0(0)	0(0)	3(3)
	Total	4(20)	1(5)	8(42)	1(5)	2(10)	16(16)
CYST	C	0(0)	0(0)	1(5)	0(0)	0(0)	1(1)
	E	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
	Total	0(0)	0(0)	1(5)	0(0)	0(0)	1(1)

C = condyle, E = Eminence, % frequencies in parentheses

¹ N=20 ² N=19 ³ N=97

FREQUENCY ANALYSIS OF RADIOLOGICAL FEATURES, BY SUBJECTS - EDENTULOUS GROUP

RADIOLOGICAL FEATURE		NO. OF SUBJECTS EXHIBITING FEATURE					Total ⁴
		AGE RANGE (YRS)					
		20-29 ³	30-39 ²	40-49 ¹	50-59 ¹	60-69 ¹	
OSTEOPHYTE	C	5(28)	5(26)	4(20)	6(30)	8(40)	28(29)
	E	1(5)	0(0)	0(0)	0(0)	1(5)	2(2)
	Total	6(33)	5(26)	4(20)	6(30)	9(45)	30(31)
EROSION	C	1(5)	1(5)	1(5)	1(5)	1(5)	5(5)
	E	0(0)	0(0)	0(0)	1(5)	0(0)	1(1)
	Total	1(5)	1(5)	1(5)	2(10)	1(5)	6(6)
FLATTENING	C	11(61)	13(68)	18(90)	18(90)	14(70)	74(76)
	E	5(28)	1(5)	10(50)	10(50)	7(35)	33(34)
	Total	14(78)	13(68)	19(95)	19(95)	17(85)	82(85)
SCLEROSIS	C	7(39)	14(74)	14(70)	15(75)	19(95)	69(71)
	E	8(44)	5(26)	12(60)	11(55)	7(35)	43(44)
	Total	11(61)	16(84)	18(90)	18(90)	19(95)	82(85)
CONCAVITY	C	0(0)	4(21)	4(20)	6(30)	4(20)	18(19)
	E	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
	Total	0(0)	4(21)	4(20)	6(30)	4(20)	18(19)
CYST	C	1(5)	4(21)	2(10)	1(5)	3(15)	11(11)
	E	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
	Total	1(5)	4(21)	2(10)	1(5)	3(15)	11(11)

C= Condyle, E = Eminence, % frequencies in parantheses

¹ N = 20 ² N = 19 ³ N = 18 ⁴ N = 97

TABLE 8

FREQUENCY ANALYSIS OF RADIOLOGICAL FEATURES, BY SUBJECTS - PAIN GROUP

RADIOLOGICAL FEATURE		NO. OF SUBJECTS EXHIBITING FEATURE					Total ⁴
		AGE RANGE (YRS)					
		20-29 ¹	30-39 ²	40-49 ¹	50-59 ³	60-69 ¹	
OSTEOPHYTE	C	7(35)	5(26)	10(50)	8(44)	9(45)	39(40)
	E	0(0)	0(0)	0(0)	1(5)	1(5)	2(2)
	Total	7(35)	5(26)	10(50)	8(44)	9(45)	39(40)
EROSION	C	3(15)	1(5)	1(5)	3(17)	5(25)	13(13)
	E	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
	Total	3(15)	1(5)	1(5)	3(17)	5(25)	13(13)
FLATTENING	C	16(80)	15(79)	19(95)	17(94)	18(90)	85(88)
	E	7(35)	9(47)	5(25)	7(39)	10(50)	38(39)
	Total	17(85)	18(95)	19(95)	17(94)	18(90)	89(92)
SCLEROSIS	C	13(65)	16(84)	18(90)	16(89)	17(85)	80(82)
	E	9(45)	10(52)	10(50)	10(55)	14(70)	53(55)
	Total	18(90)	17(89)	20(100)	17(94)	19(95)	91(94)
CONCAVITY	C	6(30)	2(10)	4(20)	6(33)	6(30)	24(25)
	E	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
	Total	6(30)	2(10)	4(20)	6(33)	6(30)	24(25)
CYST	C	1(5)	1(5)	0(0)	1(5)	3(15)	6(6)
	E	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
	Total	1(5)	1(5)	0(0)	1(5)	3(15)	6(6)

C = Condyle E = Eminence % frequencies in parantheses

¹ N = 20 ² N = 19 ³ N = 18 ⁴ N = 97

TABLE 9

FREQUENCY ANALYSIS OF RADIOLOGICAL FEATURES, BY SUBJECTS - OBESE GROUP

RADIOLOGICAL FEATURE		NO. OF SUBJECTS EXHIBITING FEATURE		
		AGE RANGE (YRS)		
		20-29 ¹	30-39 ²	Total ³
OSTEOPHYTE	C	3(15)	1(5)	4(10)
	E	0(0)	0(0)	0(0)
	Total	3(15)	1(5)	4(10)
EROSION	C	0(0)	2(10)	2(5)
	E	0(0)	0(0)	0(0)
	Total	0(0)	2(10)	2(5)
FLATTENING	C	17(85)	12(63)	29(74)
	E	8(40)	7(37)	15(38)
	Total	17(85)	16(84)	33(85)
SCLEROSIS	C	14(70)	14(74)	28(72)
	E	6(30)	2(10)	8(20)
	Total	15(75)	15(79)	30(77)
CONCAVITY	C	4(20)	1(5)	5(13)
	E	0(0)	1(5)	1(3)
	Total	4(20)	2(10)	6(15)
CYST	C	0(0)	0(0)	0(0)
	E	0(0)	0(0)	0(0)
	Total	0(0)	0(0)	0(0)

C = Condyle E = Eminence % frequencies in parentheses

¹ N = 20 ² N = 19 ³ N = 39

FIG. 16

FREQUENCY (%) OF OSTEOPHYTES - BY SUBJECTS

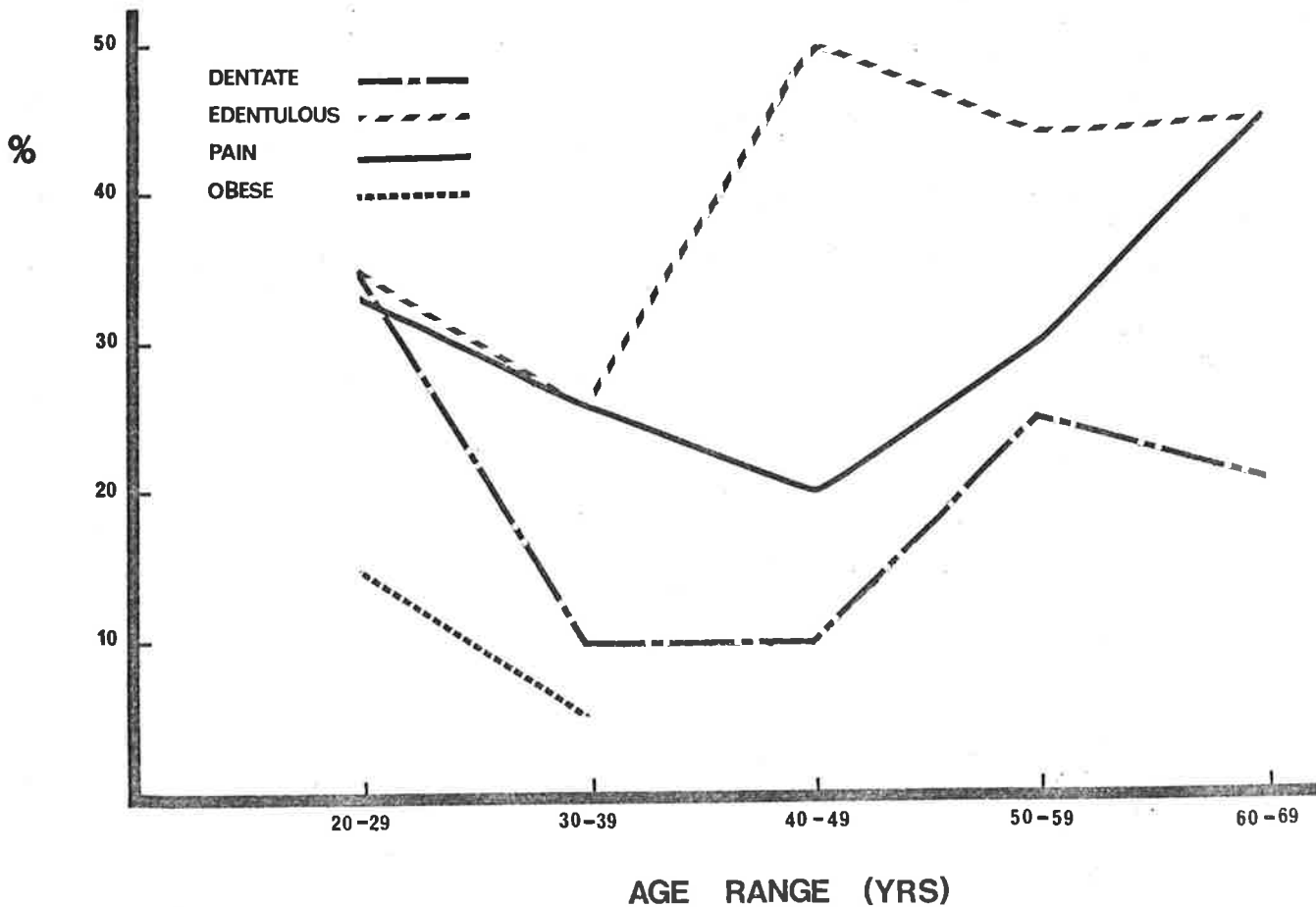


FIG. 17

FREQUENCY (%) OF EROSIONS - BY SUBJECTS

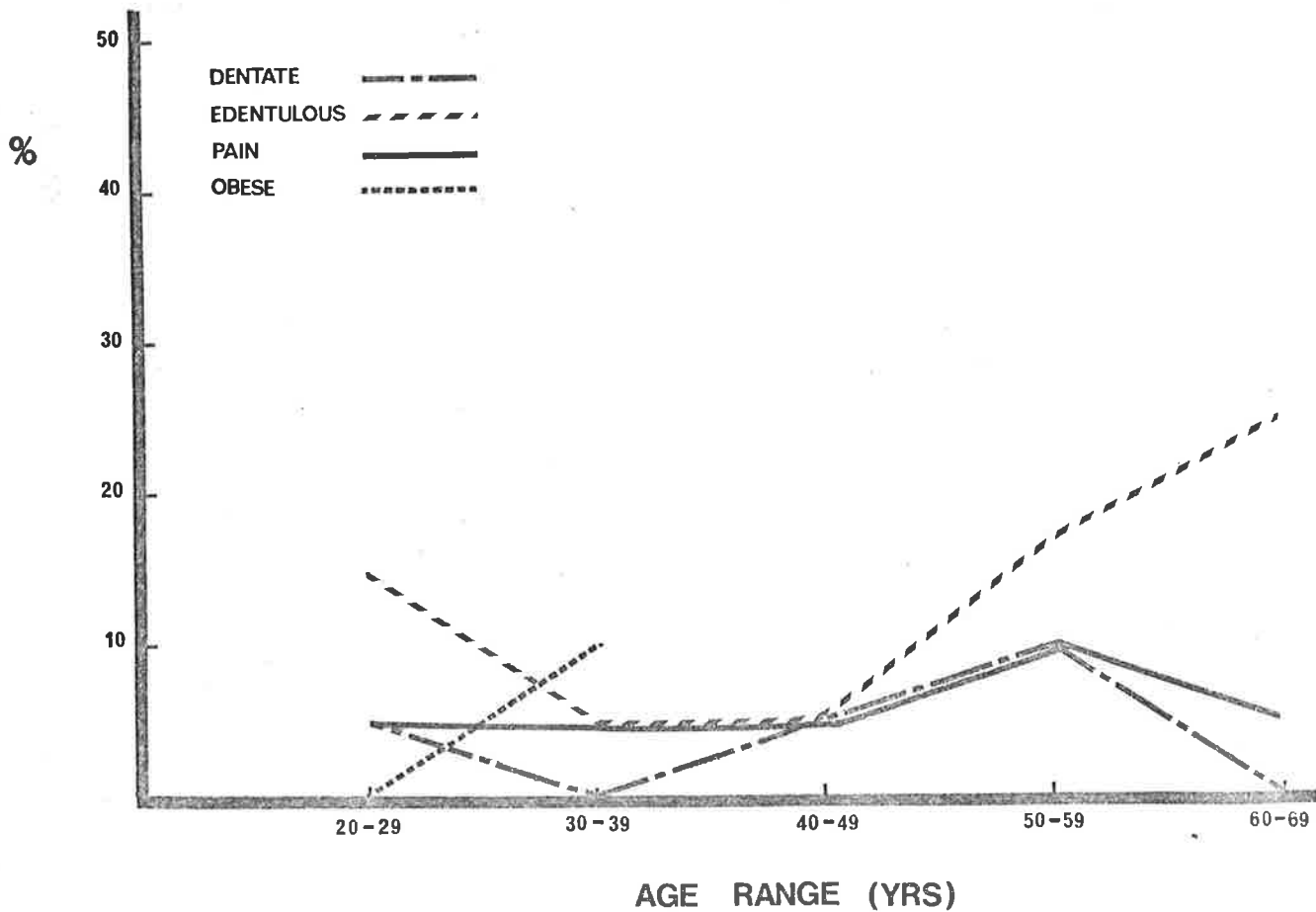


FIG. 18

FREQUENCY (%) OF FLATTENING - BY SUBJECTS ^{91.}

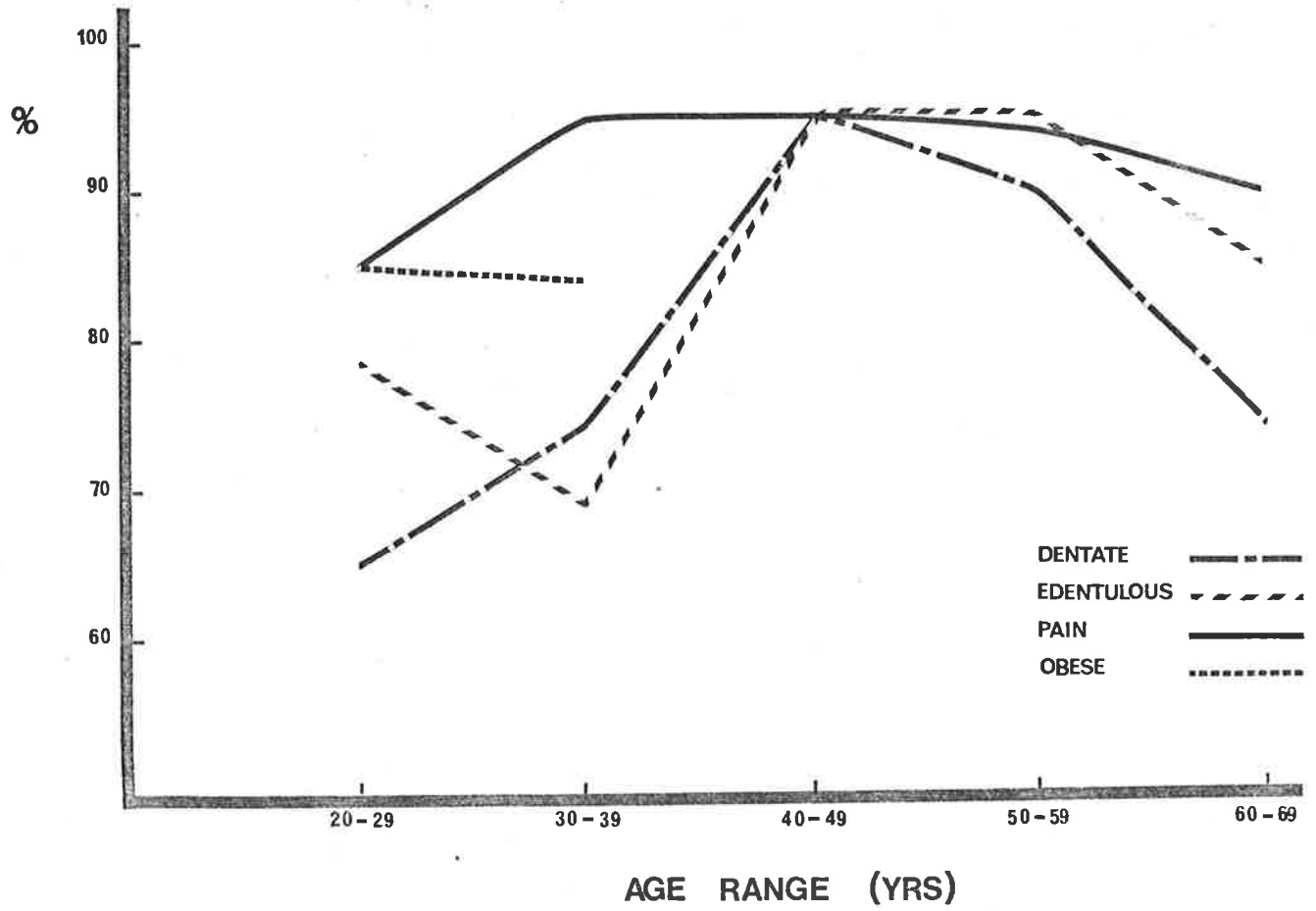


FIG. 19

FREQUENCY (%) OF SCLEROSIS BY SUBJECTS

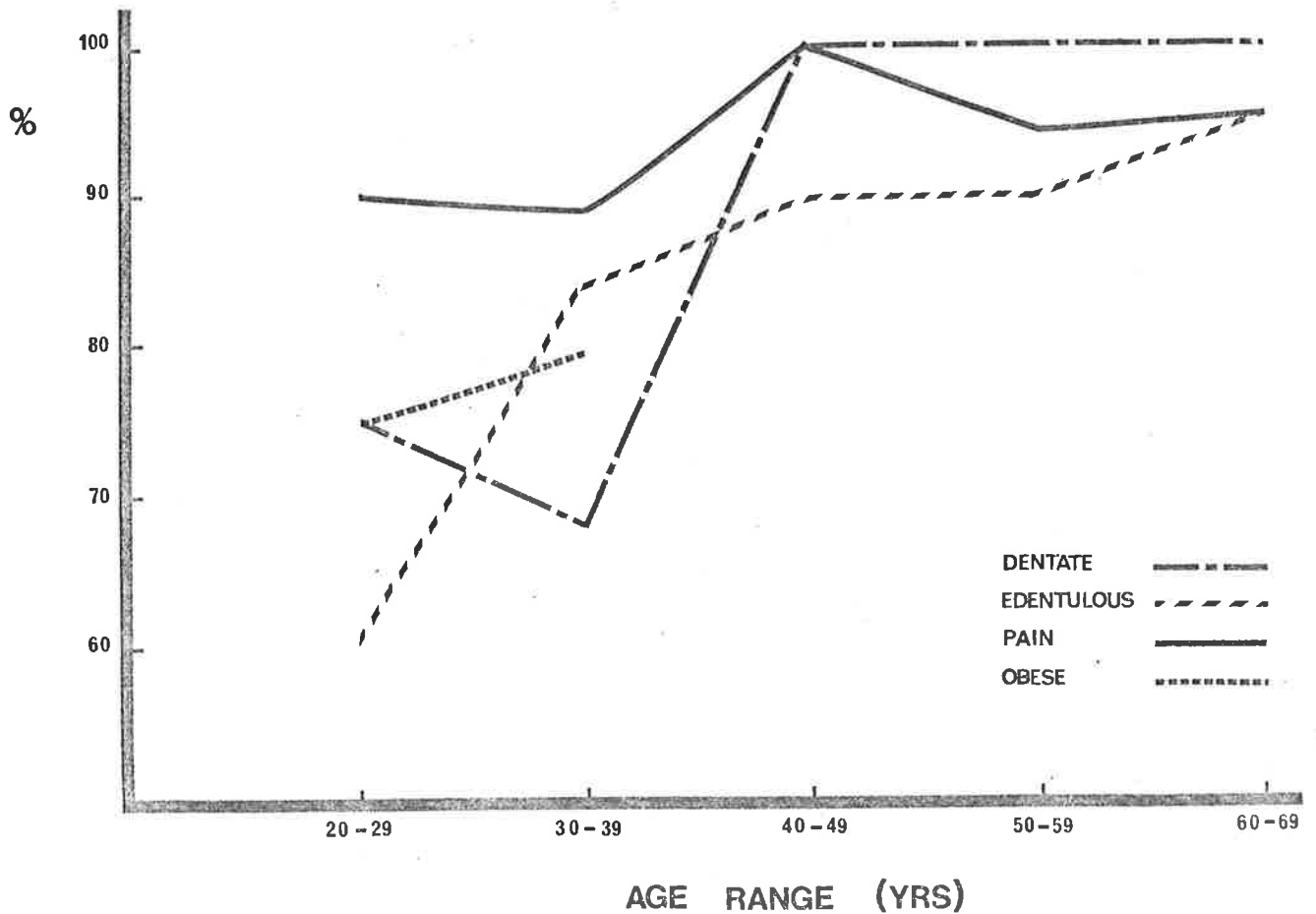


FIG. 20

FREQUENCY (%) OF CONCAVITIES - BY SUBJECTS

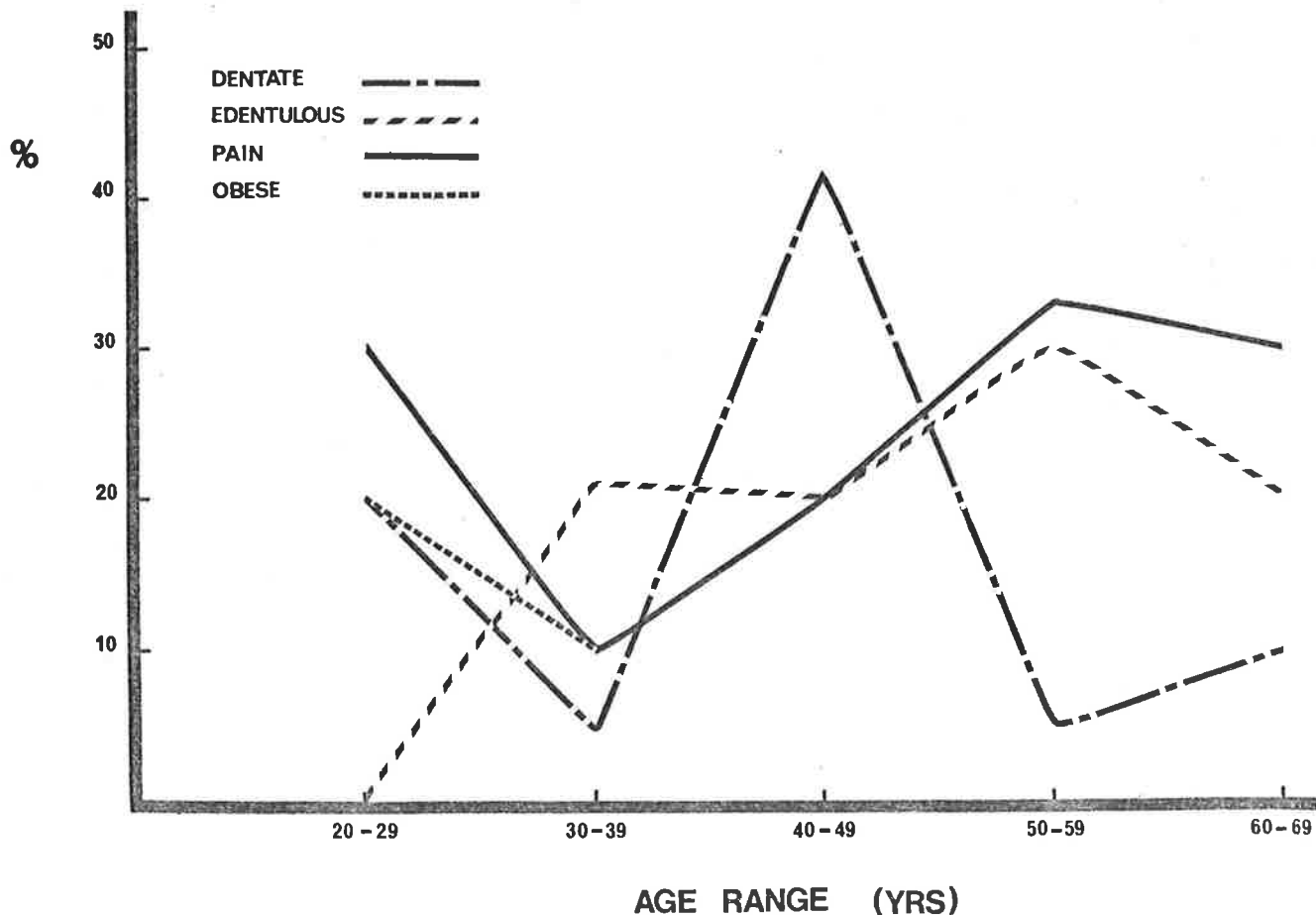


FIG. 21

FREQUENCY (%) OF 'CYSTS' - BY SUBJECTS

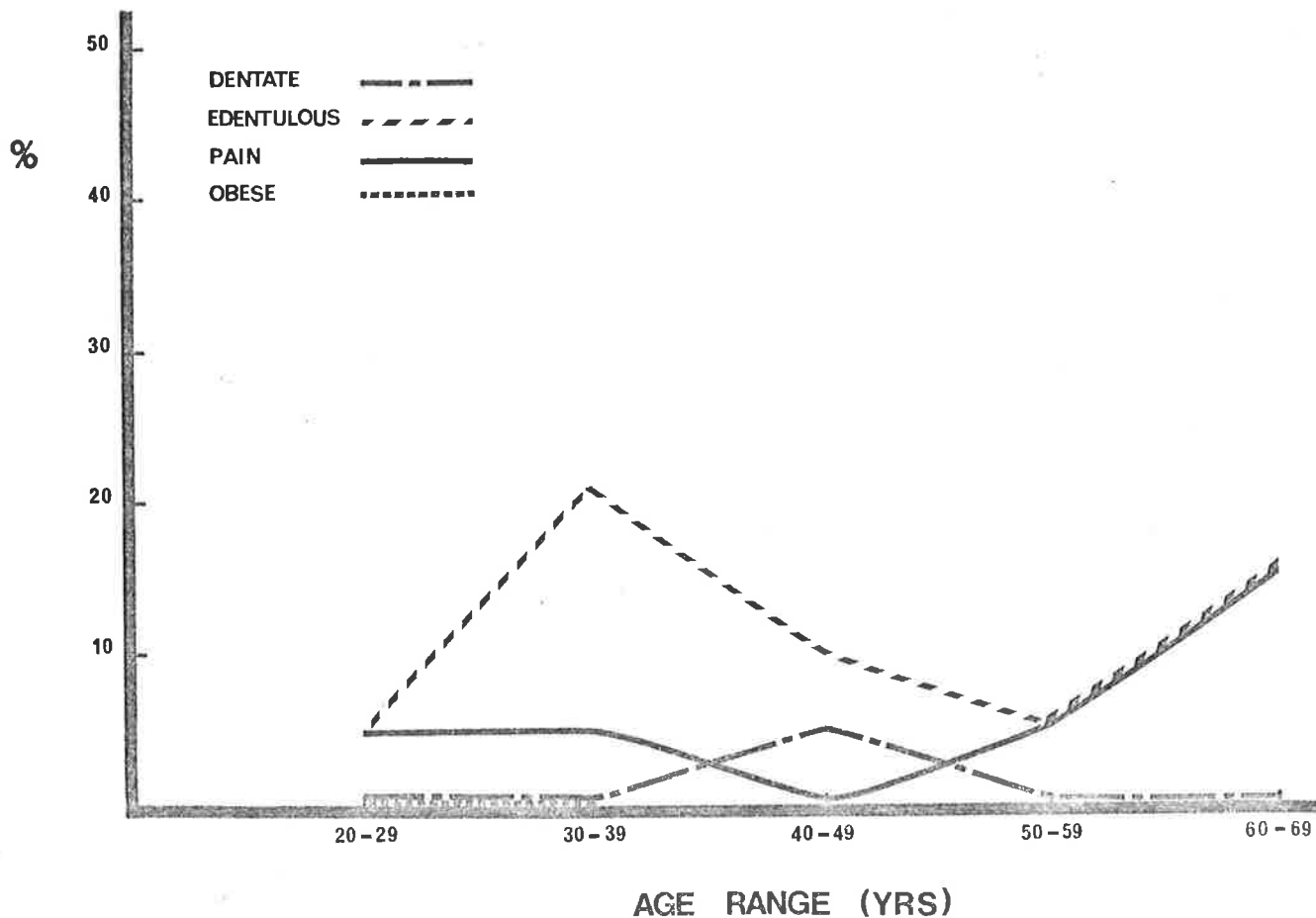


TABLE 10

FREQUENCY ANALYSIS OF RADIOLOGICAL FEATURES, BY JOINT SURFACES - DENTATE GROUP

RADIOLOGICAL FEATURE		NO. OF SURFACES EXHIBITING FEATURE					Total ³
		AGE RANGE (YRS)					
		20-29 ¹	30-39 ²	40-49 ²	50-59 ¹	60-69 ²	
OSTEOPHYTE	C	9(22)	2(5)	4(10)	6(15)	7(18)	28(14)
	E	0(0)	0(0)	1(3)	0(0)	0(0)	1(1)
	Total	9(11)	2(3)	5(7)	6(7)	7(9)	29(7)
EROSION	C	1(2)	0(0)	1(3)	2(5)	0(0)	4(2)
	E	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
	Total	1(1)	0(0)	1(1)	2(2)	0(0)	4(1)
FLATTENING	C	16(40)	18(47)	20(53)	26(65)	22(58)	102(53)
	E	11(27)	5(13)	10(26)	8(20)	13(34)	47(24)
	Total	27(34)	23(30)	30(39)	34(42)	35(46)	149(38)
SCLEROSIS	C	19(47)	20(53)	32(84)	33(82)	32(84)	136(70)
	E	14(35)	4(10)	11(29)	19(48)	15(39)	63(32)
	Total	33(41)	24(32)	43(57)	52(65)	47(62)	199(51)
CONCAVITY	C	4(10)	0(0)	7(18)	2(5)	3(8)	16(8)
	E	0(0)	1(3)	2(5)	0(0)	0(0)	3(2)
	Total	4(5)	1(1)	9(12)	2(2)	3(4)	19(5)
CYST	C	0(0)	0(0)	1(3)	0(0)	0(0)	1(1)
	E	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
	Total	0(0)	0(0)	1(1)	0(0)	0(0)	1(.25)

C = Condyle E = Eminence % frequencies in parentheses

¹ N (No. of joints) = 40 ² N = 38 ³ N = 194

TABLE 11

FREQUENCY ANALYSIS OF RADIOLOGICAL FEATURES, BY JOINT SURFACES -EDENTULOUS

GROUP

RADIOLOGICAL FEATURE		NO. OF SUBJECTS EXHIBITING FEATURE					Total ⁴
		AGE RANGE (YRS)					
		20-29 ³	30-39 ²	40-49 ¹	50-59 ¹	60-69 ¹	
OSTEOPHYTE	C	6(17)	6(16)	5(12)	9(22)	11(27)	37(19)
	E	1(3)	0(0)	0(0)	0(0)	1(2)	2(1)
	Total	7(10)	6(8)	5(6)	9(11)	12(5)	39(10)
EROSION	C	1(3)	1(3)	1(2)	1(2)	1(2)	5(3)
	E	0(0)	0(0)	0(0)	1(2)	0(0)	1(1)
	Total	1(1)	1(1)	1(1)	2(2)	1(1)	6(2)
FLATTENING	C	14(39)	19(50)	23(57)	27(67)	22(55)	105(54)
	E	5(14)	1(3)	14(35)	11(27)	9(22)	40(21)
	Total	19(26)	20(26)	37(46)	38(47)	31(39)	145(37)
SCLEROSIS	C	10(28)	19(50)	24(60)	21(52)	30(75)	104(54)
	E	12(33)	8(21)	18(45)	16(40)	11(27)	65(34)
	Total	22(31)	27(36)	42(52)	37(46)	41(51)	169(44)
CONCAVITY	C	0(0)	4(10)	5(12)	6(15)	6(15)	21(11)
	E	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
	Total	0(0)	4(5)	5(6)	6(7)	6(7)	21(5)
CYST	C	1(3)	4(10)	2(5)	1(2)	3(7)	11(6)
	E	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
	Total	1(1)	4(5)	2(2)	1(1)	3(4)	11(3)

C = Condyle E = Eminence % frequencies in parentheses

¹ N (No. of joints) = 40 ² N = 38 ³ N = 36 ⁴ N = 194

TABLE 12

FREQUENCY ANALYSIS OF RADIOLOGICAL FEATURES, BY JOINT SURFACES - PAIN GROUP

RADIOLOGICAL FEATURE		NO. OF SUBJECTS EXHIBITING FEATURE					Total ⁴
		AGE RANGE (YRS)					
		20-29 ¹	30-39 ²	40-49 ¹	50-59 ³	60-69 ¹	
OSTEOPHYTE	C	8(20)	8(21)	11(27)	9(25)	12(30)	48(25)
	E	0(0)	0(0)	0(0)	1(3)	1(2)	2(1)
	Total	8(10)	8(10)	11(14)	10(14)	13(16)	50(13)
EROSION	C	4(10)	1(3)	1(2)	3(8)	7(17)	16(8)
	E	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
	Total	4(5)	1(1)	1(1)	3(4)	7(9)	16(4)
FLATTENING	C	27(67)	26(68)	31(77)	26(72)	32(80)	142(73)
	E	9(22)	12(32)	5(12)	8(22)	14(35)	48(25)
	Total	36(45)	38(50)	36(45)	34(47)	46(57)	190(49)
SCLEROSIS	C	21(52)	24(63)	31(77)	28(78)	27(67)	131(68)
	E	14(35)	14(37)	16(40)	16(44)	23(57)	83(43)
	Total	35(44)	38(50)	47(59)	44(61)	50(62)	214(55)
CONCAVITY	C	6(15)	2(5)	4(10)	7(19)	7(17)	26(13)
	E	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
	Total	6(7)	2(3)	4(5)	7(10)	7(9)	26(7)
CYST	C	1(2)	1(3)	0(0)	1(3)	5(12)	8(4)
	E	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)
	Total	1(1)	1(1)	0(0)	1(1)	5(6)	8(2)

C = Condyle E = Eminence % frequencies in parentheses

¹ N (No. of joints) = 40 ² N = 38 ³ N = 36 ⁴ N = 194

TABLE 13

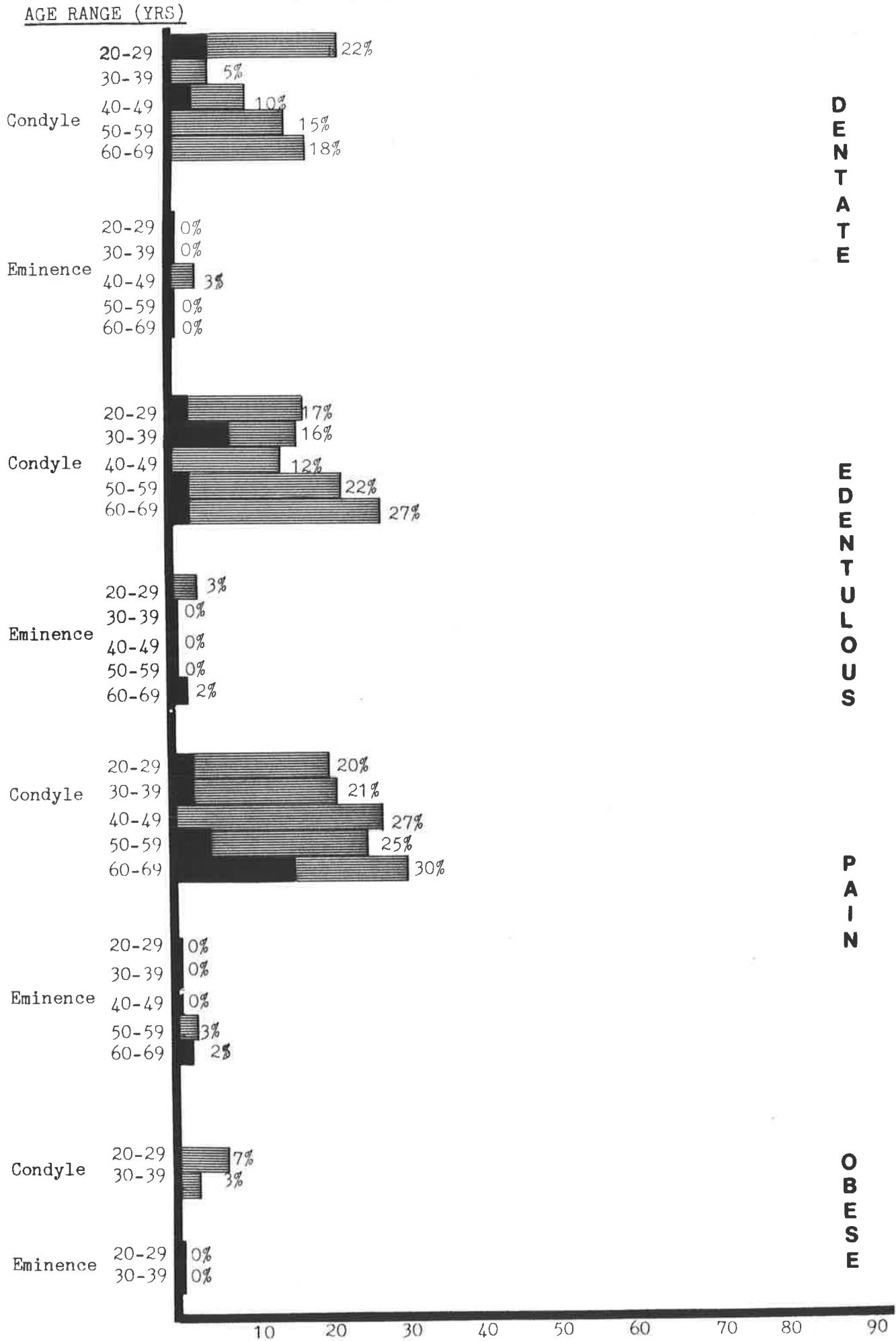
FREQUENCY ANALYSIS OF RADIOLOGICAL FEATURES, BY JOINT SURFACES - OBESE GROUP

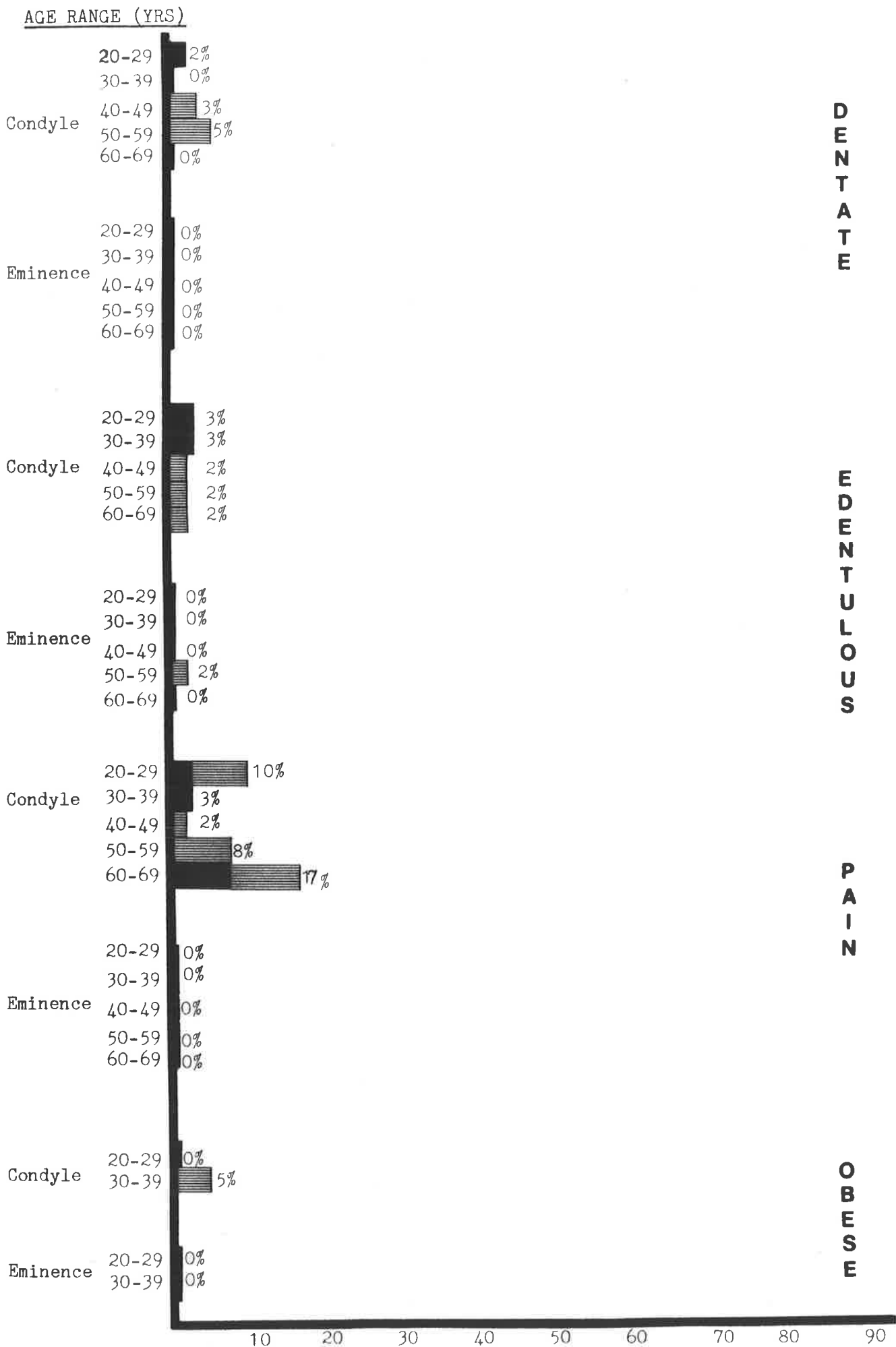
RADIOLOGICAL FEATURE		NO. OF SUBJECTS EXHIBITING FEATURE		
		AGE RANGE (YRS)		
		20-29 ¹	30-39 ²	Total ³
OSTEOPHYTE	C	3(7)	1(3)	4(5)
	E	0(0)	0(0)	0(0)
	Total	3(4)	1(1)	4(3)
EROSION	C	0(0)	2(5)	2(3)
	E	0(0)	0(0)	0(0)
	Total	0(0)	2(3)	2(1)
FLATTENING	C	25(62)	18(47)	43(55)
	E	12(30)	8(21)	20(26)
	Total	37(46)	26(34)	63(40)
SCLEROSIS	C	20(50)	24(63)	44(56)
	E	8(20)	3(8)	11(14)
	Total	28(35)	27(35)	55(35)
CONCAVITY	C	6(15)	1(3)	7(9)
	E	0(0)	1(3)	1(1)
	Total	6(7)	2(3)	8(5)
CYST	C	0(0)	0(0)	0(0)
	E	0(0)	0(0)	0(0)
	Total	0(0)	0(0)	0(0)

C = condyle E = eminence % frequencies in parentheses

¹ N (No. of joints) = 40 ² N = 38 ³ N = 78

FIGURE 22





Frequency (per cent) of erosions - by surfaces.
 Solid areas indicate % frequency of total surfaces which
 were scored as grade 2 (gross).

FIGURE 24

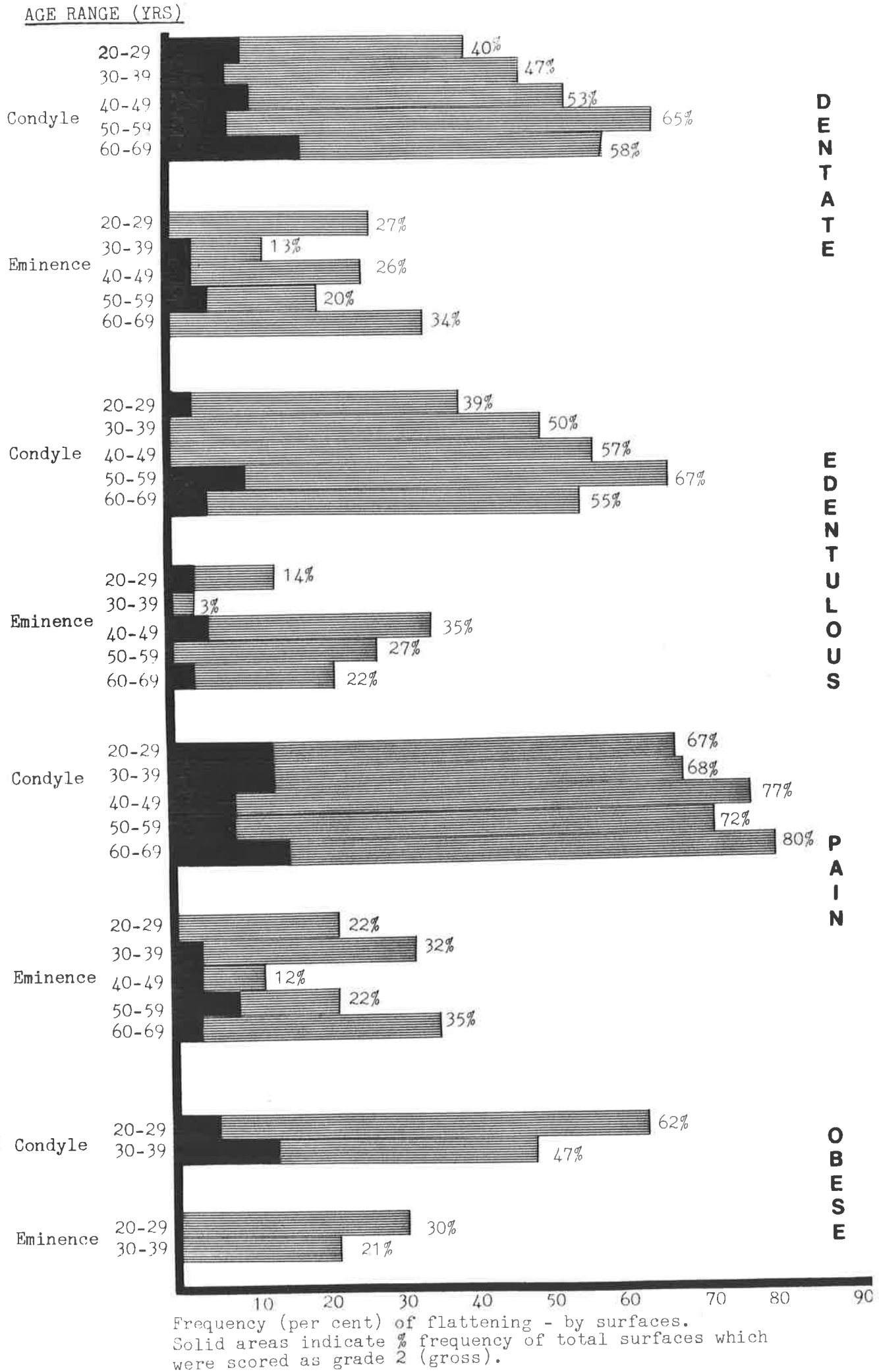
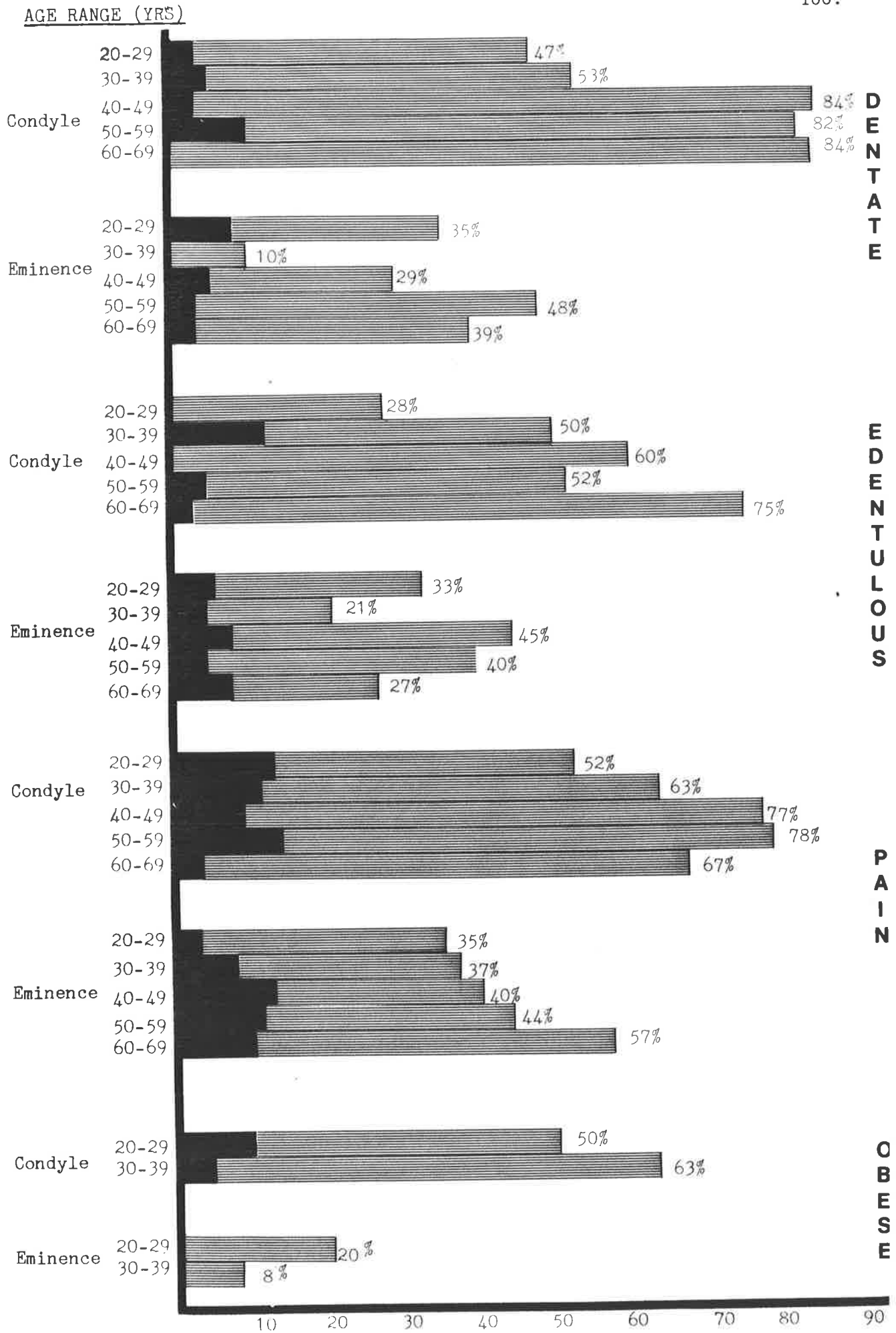
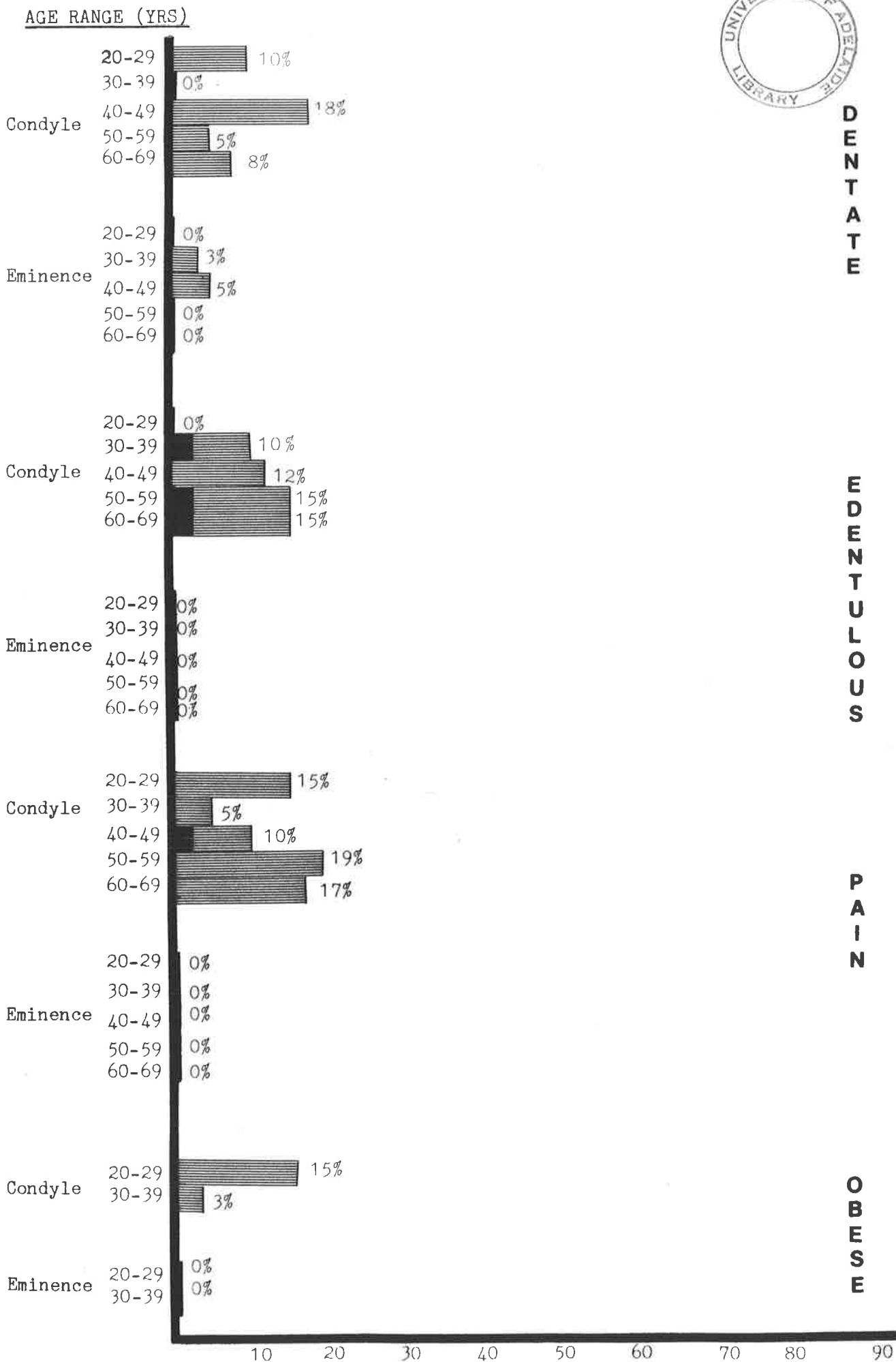


FIGURE 25

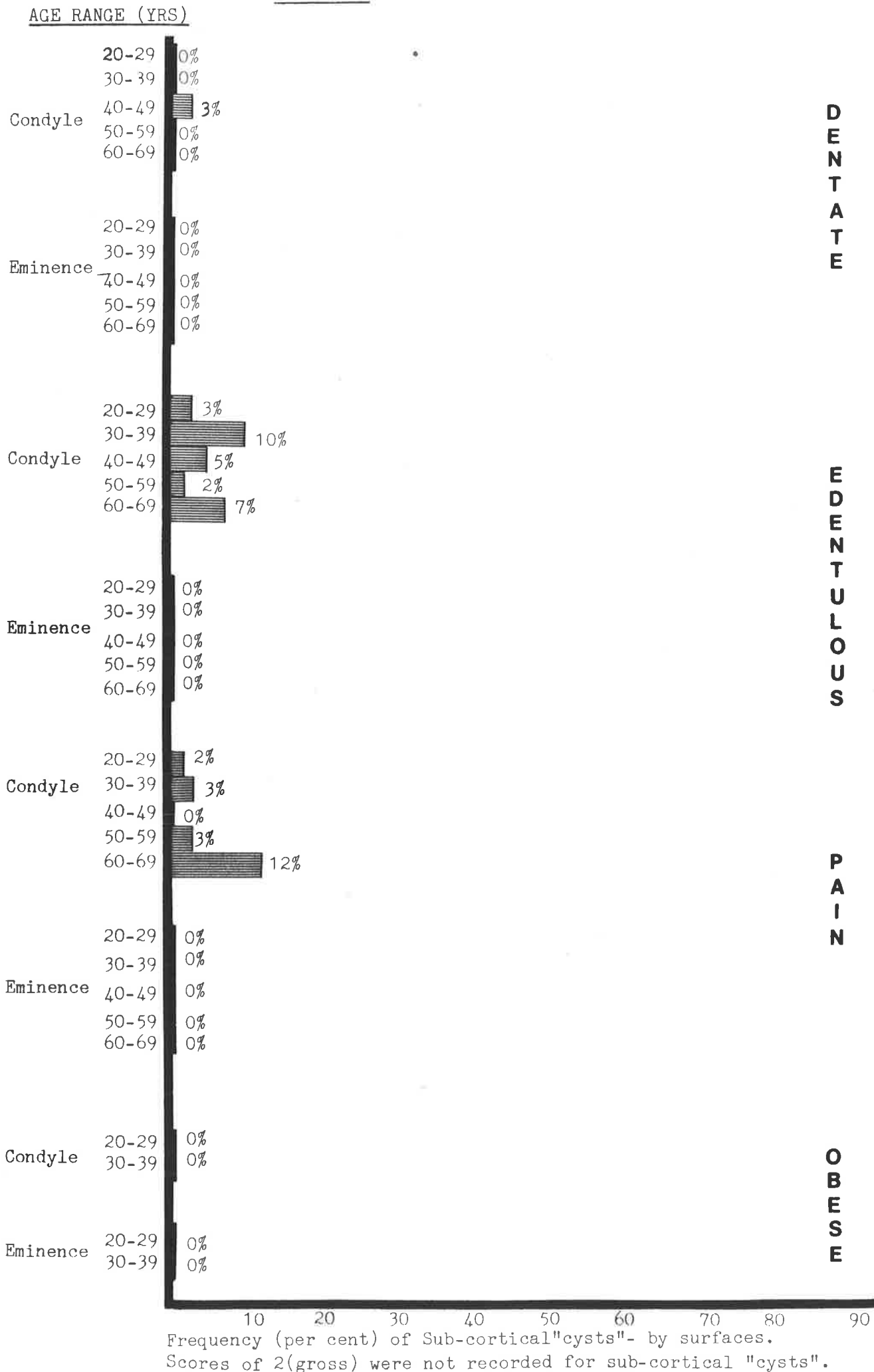


Frequency (per cent) of Sclerosis - by surfaces.
 Solid areas indicate % frequency of total surfaces which were scored as grade 2 (gross).

FIGURE 26



Frequency (per cent) of concavities - by surfaces.
 Solid areas indicate % frequency of total surfaces which were scored as grade 2 (gross)



Frequency (per cent) of Sub-cortical "cysts"- by surfaces.
 Scores of 2(gross) were not recorded for sub-cortical "cysts".

In each study group and across all age groups the most common features were flattening and cortical sclerosis, and except for sclerosis in the 20-29 years age group of edentulous subjects, both these alterations were more common, often markedly so, in the condyle than in the eminence.

A higher frequency of observations in the condyle as compared to the eminence was also evident with each of the other four radiological features. There were no instances in which these were found to be more common in the eminence than the condyle, and the overall frequency of osteophytes, erosions and concavities of the articular eminence was low. Only one subject was found to have an erosion of the articular eminence and there was not one instance of a sub-cortical "cyst" affecting the eminence.

The most common findings after flattening and sclerosis, in all groups, were osteophytes followed by concavities. In most instances it was the condyle in which these features were noted. The frequency of erosions was very low generally, reaching a maximum in the older age groups of the pain subjects, and being almost entirely restricted to condylar surfaces. Sub-cortical "cysts" were also rare, there being only one recorded in the dentate group and none in the obese group, while in the pain and edentulous groups there were a total of eight (six subjects) and eleven (eleven subjects) respectively. These findings were restricted entirely to the condyles.

Number of Joint Surfaces exhibiting Radiological Changes

Flattening and sclerosis were the features observed most often on more than one joint surface and in more than one joint, in one subject. Osteophytes and concavities also had a small tendency to occur bilaterally on the condyles, but this was never observed on the articular eminences.

There were only three instances in which erosions were recorded on both condyles in one individual (all pain subjects), and two instances of bilateral condylar sub-cortical "cysts" (also pain subjects). As with osteophytes, erosions and sub-cortical "cysts" were never seen bilaterally on the articular eminences.

Frequency of Gross Changes

The frequency of radiological features allotted a score of 2 (gross change) varied between the study groups and with different radiological features, and was less for the articular eminences than for the condyles. There was no instance of a 2 score for sub-cortical "cysts" in any group. The only features scored as 2 in the obese group were flattening and sclerosis of the condyles and these were a relatively small proportion in each instance. Scores of 2 were recorded for osteophytes, erosions and concavities in the condyles of the pain, edentulous and dentate groups, and for osteophytes in the articular eminences of the pain and edentulous groups. There were no scores of 2 for erosions or concavities on the eminence in any study group.

Scores of 2 for flattening and sclerosis in both condyles and eminences were allotted to subjects in the pain, edentulous and dentate groups but constituted a relatively small number compared to the total for each group.

8.4 AGE AND JOINT CHANGE

An association between aging and an increased incidence of each radiological feature was examined, using the chi-square test, for each study group. For the pain, dentate and edentulous groups, subjects over the age of 40 years were compared with those under 40 years, and in the obese group the 30-39 year old subjects were compared with those in the

20-29 years age range. The results of these tests are given in Tables 14, 15, 16 and 17.

In all groups most radiological features were more common in older individuals, however, a significant ($p < .05$) association with age was seen only for flattening and sclerosis in the edentulous group and sclerosis alone in the dentate group. Chi-square values for all remaining features in each group are well below the level of significance.

In view of these findings, it was considered justifiable to pool the numbers of affected individuals from each group for each radiological finding, other than flattening and sclerosis, when inter-group comparisons were made. For inter-group comparisons of the frequencies of flattening and sclerosis, the 20-39 years and 40-69 years age ranges were considered separately.

8.5 STUDY GROUP AND JOINT CHANGE

Associations between frequency of radiological features and the various study groups were tested using the chi-square analysis.

Edentulous and dentate subjects

Chi-square values for the association between the frequency of radiological features and the edentulous state are given in Table 18. The only feature which is significantly more common in edentulous subjects ($p < .01$) is the sub-cortical "cyst". The incidence of cortical sclerosis in the 40-69 years age range of dentate subjects, although considerably higher than in the edentulous group, does not reach the $P < .05$ level of significance.

TABLE 14

FREQUENCIES OF RADIOLOGICAL FEATURES IN SUBJECTS IN THE 20-39 YEARS AND 40-69 YEARS AGE RANGES - DENTATE GROUP.

RADIOLOGICAL FEATURE	NO. OF SUBJECTS EXHIBITING FEATURE			
	20-39 ¹ yrs.	40-69 ² yrs.	χ^2 (df=1)	P
OSTEOPHYTE	9(23)	11(19)	0.05	.80 < P < .90
EROSION	1(3)	3(5)	0.01	.90 < P < .95
FLATTENING	27(69)	50(86)	3.13	.05 < P < .10
SCLEROSIS	28(72)	58(100)	15.75	0.0 < P < .001**
CONCAVITY	5(13)	11(19)	0.27	.50 < P < .70
CYST	0(0)	1(2)	0.04	.80 < P < .90

% frequencies in parentheses

¹ N = 39 ² N = 58

** significant (P < .001)

TABLE 15

FREQUENCIES OF RADIOLOGICAL FEATURES IN SUBJECTS IN THE 20-39 YEARS AND 40-69 YEARS AGE RANGES - EDENTULOUS GROUP

RADIOLOGICAL FEATURE	NO. OF SUBJECTS EXHIBITING FEATURE			
	20-39 ¹ yrs.	40-69 ² yrs.	χ^2 (df=1)	P
OSTEOPHYTE	11(30)	19(32)	0.001	.975 < P < .98
EROSION	2(5)	4(7)	0.03	.80 < P < .90
FLATTENING	27(73)	55(92)	4.77	.025 < P < .05*
SCLEROSIS	27(73)	55(92)	4.77	.025 < P < .05*
CONCAVITY	4(11)	14(23)	1.62	.20 < P < .25
CYST	5(14)	6(10)	0.04	.80 < P < .90

% frequencies in parentheses

¹ N = 37 ² N = 60 * significant (p < .05)

TABLE 16

FREQUENCIES OF RADIOLOGICAL FEATURES IN SUBJECTS IN THE 20-29 YEARS AND
30-39 YEARS AGE RANGES - OBESE GROUP

RADIOLOGICAL FEATURE	NO. OF SUBJECTS EXHIBITING FEATURE			
	20-39 ¹ yrs.	40-69 ² yrs.	χ^2 (df=1)	P
OSTEOPHYTE	3(15)	1(5)	0.22	.50 < P < .70
EROSION	0(0)	2(11)	0.58	.40 < P < .50
FLATTENING	17(85)	16(84)	0.14	.70 < P < .75
SCLEROSIS	15(75)	15(79)	0.008	.90 < P < .95
CONCAVITY	4(20)	2(11)	0.14	.70 < P < .75
CYST	0(0)	0(0)	-	-

% frequencies in parentheses

¹ N = 20 ² N = 19

TABLE 17

FREQUENCIES OF RADIOLOGICAL FEATURES IN SUBJECTS IN THE 20-39 YEARS AND
40-69 YEARS AGE RANGES - PAIN GROUP

RADIOLOGICAL FEATURE	NO. OF SUBJECTS EXHIBITING FEATURE			
	20-39 ¹ yrs.	40-69 ² yrs.	χ^2 (df=1)	P
OSTEOPHYTE	12(31)	27(47)	1.80	.10 < P < .20
EROSION	4(10)	9(16)	0.19	.50 < P < .70
FLATTENING	35(90)	54(95)	0.04	.80 < P < .90
SCLEROSIS	35(90)	56(97)	0.87	.30 < P < .40
CONCAVITY	8(21)	16(28)	0.30	.50 < P < .70
CYST	2(5)	4(7)	0.005	.90 < P < .95

% frequencies in parentheses

¹ N = 20 ² N = 50

TABLE 18

FREQUENCIES OF RADIOLOGICAL FEATURES IN THE EDENTULOUS AND DENTATE GROUPS

RADIOLOGICAL FEATURES	NO. OF SUBJECTS EXHIBITING FEATURE				
	Edentulous ¹	Dentate ¹	χ^2 (df=1)	P	
OSTEOPHYTE	30(31)	20(22)	2.18	.10 < P < .20	
EROSION	6(6)	4(4)	0.11	.70 < P < .75	
FLATTENING	20-29 yrs.	27(73)	27(69)	0.01	.90 < P < .95
	40-69 yrs.	55(92)	50(86)	0.43	.50 < P < .70
SCLEROSIS	20-39 yrs.	27(73)	28(72)	0.02	.80 < P < .90
	40-69 yrs.	55(92)	58(100)	3.2	.05 < P < .10
CONCAVITY	18(19)	16(16)	0.04	.80 < P < .90	
CYST	11(11)	1(1)	7.2	.005 < P < .01*	

% frequencies in parentheses

¹ N = 97

* significant (P < .05)

TABLE 19

FREQUENCIES OF RADIOLOGICAL FEATURES IN THE OBESE AND DENTATE (20-39yrs) GROUPS

RADIOLOGICAL FEATURES	NO. OF SUBJECTS EXHIBITING FEATURE			
	Obese ¹	Dentate ¹	χ^2 (df=1)	P
OSTEOPHYTE	4(10)	9(23)	1.48	.20 < P < .25
EROSION	2(5)	1(3)	0.00	.995 < P < 1.0
FLATTENING	33(87)	27(69)	1.81	.10 < P < .20
SCLEROSIS	30(77)	28(72)	0.07	.75 < P < .80
CONCAVITY	6(15)	5(13)	0.00	.995 < P < 1.0
CYST	0(0)	0(0)	-	-

% frequencies in parentheses

¹ N = 39

Obese and Non-Obese Dentate Subjects

Chi-square values for the association between the frequency of radiological features and obesity are given in Table 19. There are no significant differences between obese and non-obese subjects for any of the features examined.

Pain and Pain-free Subjects

Because of the heterogeneous composition of the pain group with regard to dentition, 3x2 chi-square tables were developed to test for possible associations between the frequency occurrence of each radiological feature and the dentate, partially dentate and edentulous sub-groups. These chi-square values are given in Table 20 and show no significant association between any group and the frequency of radiological changes. Consequently, the pain subjects were considered to be homogeneous when associations were tested between the frequency of radiological features and the presence of pain in the TMJ region.

Table 21 presents the results of the chi-square analysis of differences between pain subjects and dentate pain-free subjects. The higher frequencies in pain subjects of osteophytes and erosions, and of flattening in the 20-39 years age range, are all significant. There are no significant differences for other features.

There were no significant differences between pain subjects and edentulous pain-free subjects (Table 22), although the values for flattening and sclerosis in the younger age group did approach significance with a higher frequency of these features in the pain subjects.

TABLE 20

FREQUENCIES OF RADIOLOGICAL FEATURES IN THE DENTATE, PARTIALLY DENTATE
AND EDENTULOUS SUBJECTS OF THE PAIN GROUP

RADIOLOGICAL FEATURE	NO. OF SUBJECTS EXHIBITING FEATURE				P
	Dentate ¹	Part. Dentate ²	Edentulous ³	χ^2 (df=2)	
OSTEOPHYTE	11(31)	14(48)	14(44)	2.31	.30 < P < .40
EROSION	4(11)	2(7)	7(22)	3.2	.20 < P < .25
FLATTENING	33(92)	29(100)	28(88)	3.68	.10 < P < .20
SCLEROSIS	33(92)	28(97)	30(94)	0.68	.70 < P < .75
CONCAVITY	9(25)	7(24)	8(25)	0.01	.99 < P < .995
CYST	2(6)	1(3)	3(9)	0.93	.50 < P < .70

% frequencies in parentheses

¹ N = 36 ² N = 29 ³ N = 32

TABLE 21

FREQUENCIES OF RADIOLOGICAL FEATURES IN THE PAIN AND DENTATE PAIN-FREE GROUPS

RADIOLOGICAL FEATURE	NO. OF SUBJECTS EXHIBITING FEATURE			P	
	Pain ¹	Dentate ¹	χ^2 (df=1)		
OSTEOPHYTE	39(40)	20(21)	7.89	.001 < P* < .005	
EROSION	13(13)	4(4)	4.13	.025 < P* < .05	
FLATTENING	20-39 yrs.	35(90)	27(69)	3.85	.025 < P* < .05
	40-69 yrs.	54(95)	50(86)	0.84	.30 < P < .40
SCLEROSIS	20-39 yrs.	35(90)	28(72)	2.97	.05 < P < .10
	40-69 yrs.	56(97)	58(100)	0.51	.40 < P < .50
CONCAVITY	24(25)	16(16)	1.54	.20 < P < .25	
CYST	6(6)	1(1)	2.37	.10 < P < .20	

% frequencies in parentheses

¹ N = 97

* significant (P < .05)

FREQUENCIES OF RADIOLOGICAL FEATURES IN THE PAIN AND EDENTULOUS PAIN-FREE GROUPS

RADIOLOGICAL FEATURE	NO. OF SUBJECTS EXHIBITING FEATURE			
	Pain ¹	Edentulous ¹	χ^2 (df=1)	P
OSTEOPHYTE	39(40)	30(31)	1.44	.20 < P < .25
EROSION	13(13)	6(6)	2.10	.10 < P < .20
FLATTENING	20-39 yrs.	27(73)	2.53	.10 < P < .20
	40-69 yrs.	55(92)	0.003	.95 < P < .975
SCLEROSIS	20-39 yrs.	27(73)	2.53	.10 < P < .20
	40-69 yrs.	55(92)	0.54	.40 < P < .50
CONCAVITY	24(25)	18(19)	0.76	.30 < P < .40
CYST	6(6)	11(11)	1.03	.30 < P < .40

% frequency in parentheses

¹ N = 97

TABLE 23

FREQUENCIES OF RADIOLOGICAL FEATURES IN THE PAIN AND COMBINED DENTATE AND EDENTULOUS PAIN-FREE GROUPS

RADIOLOGICAL FEATURE	NO. OF SUBJECTS EXHIBITING FEATURE.			
	Pain ¹	Pain-free ²	χ^2 (df=1)	P
OSTEOPHYTE	39(40)	50(26)	5.68	.01 < P* < .02
EROSION	13(13)	10(5)	4.96	.025 < P* < .05
FLATTENING	20-39 yrs.	54(71)	4.13	.025 < P* < .05
	40-69 yrs.	105(89)	0.36	.50 < P < .70
SCLEROSIS	20-39 yrs.	55(72)	4.0	.025 < P* < .05
	40-69 yrs.	113(96)	0.03	.80 < P < .90
CONCAVITY	24(25)	34(18)	1.68	.10 < P < .20
CYST	6(6)	12(6)	0.07	.75 < P < .80

% frequencies in parentheses

¹ N = 97 ² N = 194

* significant (P < .05)

Because of the heterogeneity of dental status in the pain group, and the absence of significant differences between dentate and edentulous pain-free subjects, apart from an increased frequency of sub-cortical "cysts" in the edentulous group, a chi-square comparison between pain subjects and all dentate and edentulous pain-free subjects was carried out. The results are presented in Table 23 and, except for the additional significantly higher frequency of sclerosis in the 20-39 year age group of pain subjects, are similar to those obtained in comparing the pain group and dentate pain-free group.

A chi-square analysis was performed to test the association between age and the increased frequency of osteophytes and erosions in pain subjects as compared with pain-free subjects. These results are shown in Table 24 and indicate that the main contribution to the higher frequency of osteophytes comes from subjects over the age of 40 years. The higher frequency of erosions is, however, spread fairly evenly across all age groups in the pain subjects.

8.6 ASSOCIATION BETWEEN SIDE OF SYMPTOMS AND RADIOLOGICAL FINDINGS IN THE PAIN SUBJECTS

Table 25 shows the distribution of side of symptoms for each age group in the pain subjects, and the total number of subjects who experienced pain on the left, right or both sides. In most subjects the pain was sited unilaterally, with a slight prevalence for the right side. Only nine subjects (9%) complained of equal pain in both joint regions.

No association was apparent between the side on which pain occurred and the presence of a particular radiological feature. For most features the testing of such an association was impractical

TABLE 24

FREQUENCIES OF OSTEOPHYTES AND EROSIONS IN THE 20-39 YEARS AND 40-69 YEARS AGE RANGES OF THE PAIN AND COMBINED PAIN-FREE GROUPS.

RADIOLOGICAL FEATURE		NO. OF SUBJECTS EXHIBITING FEATURE			
		Pain ¹	Pain-free ²	χ^2 (df=1)	P
OSTEOPHYTE	20-39 yrs.	12(31)	20(26)	0.08	.75 < P < .80
	40-69 yrs.	27(47)	30(25)	6.99	.005 < P* < .01
EROSION	20-39 yrs.	4(10)	3(4)	0.85	.30 < P < .40
	40-69 yrs.	9(16)	7(6)	3.24	.05 < P < .10

% frequencies in parentheses

¹ N = 97 ² N = 194

* significant (P < .05)

TABLE 25

SYMPTOMATIC SIDE IN PAIN GROUP

AGE RANGE (YRS.)	NO. OF SUBJECTS EXPERIENCING PAIN		
	Left	Right	Both
20-29	8	11	1
30-39	5	12	2
40-49	7	10	3
50-59	9	6	3
60-69	12	8	0
TOTAL	41	47	9

TABLE 26

SIDE WITH THE GREATER RADIOLOGICAL INDEX SCORE IN PAIN GROUP

AGE RANGE (YRS).	NO. OF SUBJECTS WITH GREATER RADIOLOGICAL INDEX		
	Left	Right	Equal
20-29	7	4	9
30-39	8	7	4
40-49	10	7	3
50-59	8	7	3
60-69	5	8	7
TOTAL	38	33	26

TABLE 27

ASSOCIATION BETWEEN SIDE WITH PAIN AND SIDE WITH THE GREATER RADIOLOGICAL INDEX SCORE¹.

GREATER INDEX	PAIN		
	Left	Right	Total
Left	14	16	30
Right	14	21	35
TOTAL	28	37	65

¹ Subjects with bilateral pain and equal index scores for both joints excluded.

$$x^2 = .2928 \quad .50 < P < .70$$

because of the relatively low frequency with which the feature occurred. Therefore, an association was sought between the side with the symptoms and the side on which the greater degree of significant radiological joint change was observed. To do this, an index of the significant radiological changes, i.e. osteophytes, erosions, flattening and sclerosis, was developed for each joint in a similar fashion to that described by Kopp and Rockler (1979). For this, a score of 1 was allotted for the presence of any of these radiological features in both the condyle and articular eminence of a given joint. Thus each joint could be allotted from 0-8 points. The joint (left or right) allotted the greater index score in each subject is shown in Table 26.

In Table 27 an association is sought between the side with pain and that with the higher index score, using only those subjects with unilateral pain whose radiological index is greater on one side than the other. From Table 27, and a comparison of Tables 25 and 26, it is apparent that in these subjects there is no relationship between the side on which pain was experienced and the joint with the greater significant radiological change.

Although only 9 subjects complained of pain in both joints, there were 26 subjects allotted an equal radiologic index for both joints, and despite a slight prevalence of symptoms on the right side, there was a slight but opposite tendency as regards the side with the greater radiological joint index.

CHAPTER IXDISCUSSION9.1 SUBJECTS

The study was limited to females because of the difficulty in finding a sufficient number of matching male subjects in the pain and obesity groups. The great majority of patients presenting to the Dental Department of the Royal Adelaide Hospital with complaints of pain in the region of the temporomandibular joint (TMJ) are female. This is in accord with the 70%-90% female preponderance observed in nearly all clinical series concerning the temporomandibular joint myofascial pain dysfunction group of disorders (Helkimo, 1979). Approximately 95% of patients being treated for morbid obesity by jaw-wiring are also female.

A minimum age of 20 years was selected because this is the approximate time at which calcification of a uniform sub-articular cortical plate is complete in the TMJ. In addition, there is general agreement from studies of skeletal and autopsy material (Oberg et al., 1971; Mongini, 1972, 1975; Hansson and Oberg, 1977; Wedel et al., 1978) that remodelling changes are exceedingly rare in the TMJ before the age of 20 years. There are also comparatively few patients below this age presenting for management of obesity or with complaints of pain in the TMJ region, and it is now rare to find edentulous patients under the age of 20 years, and relatively unusual below 40 years of age.

The upper age limit of 69 years delineated the upper limits of patient availability in the dentate and pain groups, and in fact, the accumulation of sufficient dentate subjects over the age of 40 years proved difficult in the Dental Department patient population.

There were very few patients over the age of 39 years treated for morbid obesity by jaw wiring, and consequently this was selected as the upper age limit for this group. A 20-69 years age range was also generally in keeping with previous studies on morphological joint change (Ericson and Lundberg, 1968; Oberg et al., 1971; Hansson and Petersson, 1978).

The exclusion of subjects suffering from a systemic disease known to influence the joints was necessary in order to limit the number of variables which could be associated with the aetiology of radiological joint changes. Subjects with a history of systemic steroid intake were excluded following the work of Silbermann (1976), who suggested the possibility that degenerative joint changes may be caused by such medication.

It was considered necessary to obtain both dentate and edentulous pain-free control groups because of the considerable variation in the dentition of the pain subjects, and the suggestion by a number of workers, including Moffett et al. (1964), Oberg et al. (1971), and Mongini (1972), that the extent of bony remodelling change in the TMJ shows a positive association with the number of missing teeth.

Because of the extreme difficulty in finding sufficient numbers of fully dentate subjects over the age of 40 years, a compromise was made in accepting individuals missing no more than one molar or premolar tooth in each quadrant. It was felt that posterior occlusal support was not jeopardized by such minor losses and consequently the amount of joint remodelling would not be significantly affected.

By excluding edentulous subjects whose radiographs showed

any evidence of tooth sockets a minimum period of edentulousness of approximately one year was assured (Tyas, 1974). In general, the older the subject, the longer had been the period of edentulousness and thus the longer the period over which remodelling changes, if any, initiated by this condition had acted.

The rather general requirement for inclusion in the pain group, i.e. pain in the region of the TMJ which had necessitated the patient's seeking treatment, undoubtedly allowed the inclusion of a number of subjects with TMJ osteoarthritis (TMJ-OA), although most subjects were classified clinically and radiologically as suffering from the myofascial pain dysfunction syndrome. However, because of recent studies which have shown gross bony changes in the joint in the absence of articular soft tissue damage, an essential feature of osteoarthritis (Oberg et al., 1971; Bean et al., 1977), and Kopp's (1977^{a,b}) inability to distinguish TMJ-OA from MPD on symptomatic or clinical grounds, it seems that a definitive diagnosis of TMJ-OA is difficult, if not impossible, in the absence of corroborative histologic or direct visual evidence. Therefore, no attempt was made to separate these two conditions in the pain group.

The recent suggestions by Toller (1977), Ogus (1979) and Laskin (1979) that MPD and TMJ-OA are closely related also supported this decision.

The inclusion of a morbidly obese group in this study arose for two reasons. Firstly, it has been suggested (Chapter VI) that obesity may predispose to osteoarthritis (OA) in joints not required to bear body weight and therefore it was thought to be of interest to examine the effects of obesity on the TMJ. Secondly, because of a large study into the

efficacy of "jaw-wiring" in the management of morbid obesity, being carried out in the Royal Adelaide Hospital, there was a plentiful supply of morbidly obese subjects available for comparison with an otherwise normal group.

9.2 RADIOLOGY

As discussed in the literature review, the standard orthopantomograph (OPG) is accepted by a number of authorities as providing a satisfactory depiction of the TMJ in an oblique lateral plane. Uotila (1964) used the OPG in a major study of rheumatoid arthritis of the temporomandibular joints and stated that "the roentgenograms of the temporomandibular joints obtained.....must be regarded as at least equally good as those obtained by the oblique-lateral (i.e. transcranial) techniques". Similarly, in a comparative study of circular tomography, lateral transcranial plain radiography, and standard orthopantomography of the TMJ, Blair and Chalmers (1972) concluded that all three techniques were equally effective in demonstrating features on the joint surfaces, provided the joint could be seen on the radiograph.

The majority of standard orthopantomographs in this study, when examined in a darkened room with a masked illuminator, were found to provide satisfactory diagnostic detail of the condyle and articular eminence. This supports the opinions of the aforementioned investigators who have attested to the value of the OPG for TMJ radiology.

The inability to visualize the glenoid fossa on the majority of radiographs in the present study indicates an inherent defect in the OPG. This was also noted by Uotila (1964) and Blaschke and

White (1979). However, detail of the fossa is also poorly depicted on transpharyngeal (Hansson and Petersson, 1978) and transcranial (Lindvall et al., 1976) TMJ radiographs and even tomography may produce erroneous images of this region (Lindvall et al, 1976; Eckerdal and Ahlqvist, 1979). It therefore appears that, given the state of current radiographic technology, we must accept a low level of diagnostic capability for this joint component.

The general use of the OPG as a screening film was also a factor in favour of its use in this study. The simultaneous presentation of both joints, with the entire dentition and alveolus, on one film, was of considerable benefit as the state of the dentition was a variable in the study. Valid comparisons of changes in both joints in subjects were facilitated by the radiography of both joints under identical conditions in the OPG.

Although accepting the necessity of additional plain films and tomograms for a complete radiographic examination of the TMJ, the radiation exposure occasioned by such examinations is considerable (Stanson and Baker, 1976). All patients included in this study had already been radiographed and thus no additional radiation exposure was required.

9.3 METHOD

Errors in this study may be of two types, systematic and accidental. Systematic errors occur as a result of limitations in the technology employed and accidental errors may result from variations in observer consistency and mistakes in the recording of data.

As there is no radiographic technique, or combination of techniques, which is totally accurate in depicting all TMJ surfaces, any radiological study of this joint will be imperfect. The utilization of as rigidly standardized a radiographic technique as orthopantomography, however, should ensure a more consistent distribution of errors than a technique whose results are highly dependent on the skill of the individual radiographer.

It was hoped to minimize the degree of observer subjectivity, and thereby increase observer consistency, by the use, while scoring, of a series of standard radiographs of the radiological changes under investigation. These standards were selected after a careful study of illustrations of such features in work by Worth (1979), and Hansson and Petersson (1978). However, it should be emphasized that ultimately a subjective decision has to be made by the observer, and especially in borderline cases discrepancies may occur. This has been stressed by Kopp and Rockler (1978), who showed that despite preceding training, observers differed substantially in their interpretation of the common radiological changes in radiographs of the TMJ.

The accurate evaluation of radiographs was particularly facilitated by the conditions in which this was carried out. It was very obvious that the use of a darkened room and a masked illuminator considerably increased the visible detail in the radiographs when compared to those seen on the same film under standard clinical conditions with an open illuminator. The conditions of examination must be regarded as of prime importance when evaluating TMJ changes on the OPG.

Accidental errors, especially in the recording of data, were minimized by limiting the length of each evaluating session to one hour and thus minimizing observer fatigue. The magnitude of accidental errors was analysed using a replicability procedure in which a double determination scoring of 40 randomly selected radiographs (80 joints) was made. These results showed an acceptable level of consistency for the joints scored on two separate occasions, with a minimum concordance of 89%.

9.4 RESULTS

The results of the present radiological study of adult temporomandibular joints indicate a high frequency of bony remodelling changes. This finding is consistent with the almost ubiquitous occurrence of such changes seen in studies of autopsy and skeletal material (Macalister, 1954; Blackwood, 1963; Moffett et al., 1964; Oberg et al., 1971; Mongini, 1972, 1975; Hansson and Oberg, 1977; Wedel et al., 1978).

The greatest number of unaffected joints occurred in dentate, pain free subjects (Table 5), a tendency which was observed by Blackwood (1963), Moffett et al. (1964), Ericson and Lundberg (1968) and Oberg et al. (1971). In all groups, joints without radiological changes were more common in subjects under the age of 40 years than in older subjects, a finding in keeping with the observation of Oberg et al. (1971) that macroscopic joint changes were more frequent in their older subjects.

The most common radiological changes noted in this study were flattening and cortical sclerosis. Between 79% and 92% of subjects in the different groups exhibited flattening in one or more joint components and between 77% and 94% exhibited sclerosis. (Tables 6-9). In his review of the

radiology of the TMJ, Worth (1979) also noted flattening or faceting to be one of the most common changes seen in the joint and that this was often accompanied by cortical sclerosis.

There were no studies found in the literature, on autopsy material, which examined specifically flattening or sclerosis. Most of the major studies of clinical material have concentrated on changes in the soft tissue contours of the joint rather than bony contours, the one not necessarily reflecting the other (Bean et al., 1977; Carlsson and Oberg, 1979). Mongini (1972) did, however, examine the frequency of flattening in the condyles of 100 dry mandibles and found this feature to be very common, especially in individuals over the age of 25 years, with the overall frequency of a flattened condyle varying between 77% and 88%. Mongini also observed that, in most instances, the condylar flattening was due to progressive remodelling with consequent thickening of the cortical plate.

In the present study, the frequency of condylar flattening (per se) was between 53% and 73% in the different groups, and the frequency of condylar sclerosis was between 54% and 70% (Tables 10-13). These results are not dissimilar to those reported by Mongini (1972) in his study of skeletal material. The slightly lower frequency in the present study probably reflects the inability of joint radiographs to depict subtle morphologic changes.

In contrast to flattening and sclerosis, the frequency of the other radiological features was low. Osteophytes, were exceedingly rare on the articular eminence and had a frequency on the condyles of between 5% and 25% in the different groups (Tables 10-13). Similarly, concavities were seen mainly on the condyles, their frequency on this joint component varying between 8% and 13% (Tables 10-13).

In only one subject was an erosion observed on the articular eminence. There were no sub-cortical "cysts" seen in the eminence. The frequency of condylar erosions varied between 2% and 8%, and the frequency of "cysts" between 0% and 6% (Tables 10-13). Thus in this study, as in that of Hansson and Petersson (1978), the most common changes noted in the articular eminence were flattening and sclerosis.

Site of Radiological Changes

The tendency for the majority of radiological changes to occur in the condyle is a feature of all reported radiological studies in which both the condylar and temporal joint components have been examined, (Carlsson et al., 1968; Toller, 1973; Petersson and Nanthaviroj, 1975; Lindvall et al., 1976; Hansson and Petersson, 1978). This reflects the difficulty in accurately visualizing the temporal joint component on radiographs, as it has been shown that remodelling of the temporal component is as common as that of the condyle (Oberg et al., 1971; Hansson and Oberg, 1977), and that osteoarthritic lesions are more common in the temporal component than the condyle (Oberg et al., 1971; Hansson and Oberg, 1977). The difficulty in visualizing the temporal component was further evidenced in the low frequency of gross flattening, sclerosis and osteophytic changes, and total absence of gross erosions and concavities seen in the eminence (Figs. 22-27).

The common finding in this study of flattening and sclerosis on more than one surface of a joint, and especially in more than one joint in a particular subject, was not surprising in view of the high overall frequency of these changes. That remodelling changes are rare before the third decade of life has been shown by Oberg et al. (1971) and Mongini (1972). The widespread appearance of flattening and sclerosis

thereafter is best explained as a common reaction to functional stimuli in a joint which has ceased to grow, but retained the ability to adapt to such stimuli. Consequently a fairly even distribution of these features is to be expected in any one individual.

Information concerning the frequency of bilateral osteoarthritic lesions in the temporomandibular joints of an individual is sparse, as most autopsy studies have used only one joint from each subject. Toller (1973) found no instances of bilateral disease in 130 patients whom he considered to be affected by TMJ-OA, the diagnosis being dependent on the presence of typical condylar erosions in radiographs of the joints. In the present study only three instances of bilateral condylar erosions were noted, and in no case was the eminence involved bilaterally by erosions. However, as Bean et al. (1977) have shown, sub-chondral erosion is not always associated with osteoarthritic destruction of the articular soft tissues and thus may merely represent advanced regressive remodelling.

Osteophytes and concavities manifested a somewhat greater tendency than erosions to occur bilaterally in the condyles. As with erosions, osteophytes and concavities were not seen bilaterally on the articular eminence. The significance of this finding is not clear but could be interpreted as evidence for these features being representative of functional remodelling changes rather than evidence of an active osteoarthritic lesion if Toller (1973) is correct in his belief that bilateral TMJ-OA is uncommon.

Extent of Radiological Change

The significance which can be attached to the occurrence of gross morphologic TMJ changes is uncertain. In general the frequency of gross changes was low in com-

parison to the overall frequency of any given radiological feature and in one case, that of the sub-cortical "cyst", no joint component was scored as exhibiting gross change. (Figures 22-27). An association between the frequency of gross changes and the different age and study groups was not found.

The lack of any gross scores for sub-cortical "cysts" may be significant as Uotila (1964) stated that completely normal condyles could exhibit "cyst-like" areas on an OPG, and consequently in his study such areas were recorded as positive only if they were pea-sized or larger. Had this stipulation been made for the present study, the frequency of sub-cortical "cysts" would have been zero, an interesting finding in view of the importance of this feature as a radiological sign of OA in other joints (Murray and Jacobson, 1971; Moskowitz, 1979).

Frequency Results in this Study as Compared with those of Previous Radiological Studies

A comparison of the frequency results in the present study with those of previously reported radiological studies is difficult for a number of reasons. First, the majority of similar studies have employed radiographic techniques other than the OPG, the most common being the transpharyngeal, transcranial, and transmaxillary projections or combinations of these. As a number of investigators have pointed out, the diagnostic yield may vary considerably in different radiographic projections depending on the feature being examined for and the joint component being evaluated (Petersson and Nanthaviroj, 1975; Omnell and Petersson, 1976; Hansson and Petersson, 1978). Consequently a comparison of the results obtained using the OPG with results from another radiologic projection or combination of projections would be expected to show differences, possibly of a

considerable degree.

The second variable in reported studies is the probable lack of uniformity between authors as to criteria for the diagnosis of a particular radiological change. For example, in the study by Uotila (1964) it is apparent that a condyle was scored as flattened only when gross destructive levelling was evident, whereas illustrations by Hansson and Petersson, (1978) and Worth (1979) indicate that these authors consider comparatively minor facets with consequent loss of an evenly rounded surface to constitute flattening. Similarly, a number of workers (Uotila, 1964; Lindvall et al., 1976; Hansson and Petersson, 1978; Kopp and Rockler, 1978) have referred to the difficulty in diagnosing cortical sclerosis because of the topography of the joint with its many curving surfaces and the lack of knowledge as to the limits of normal variation for this feature.

The third factor making comparison difficult is the variation in sex, age, dentition, and status as regards systemic joint disease, between the subjects in different studies. The majority of reported studies include both males and females. Many do not give a detailed breakdown of results for the two sexes or for varying dentitions. Since a higher frequency or greater degree of joint change has been found, in some studies, in females (Macalister, 1954; Ericson and Lundberg, 1968; Oberg et al., 1971; Toller, 1973) and in subjects with a greater number of missing teeth (Blackwood, 1963; Moffett et al., 1964; Oberg et al., 1971; Mongini, 1975) these are obviously important variables in a comparison of results. Similarly, age has been associated with increased joint change (Blackwood, 1963; Ericson and Lundberg, 1968; Oberg et al., 1971) and must therefore be considered in any comparison.

Knowledge of the presence of systemic joint disease, especially rheumatoid arthritis, is also essential in any radiological study of joints. Unfortunately the only studies found which have utilized the OPG to systematically evaluate TMJ changes (Uotila 1964; Blair and Chalmers 1972) have been limited to subjects with rheumatoid arthritis. Therefore comparisons with the present study are of no value.

The final difficulty confronting such a comparison of results stems from the known variation in assessment of radiological changes of the TMJ between observers, even when scoring the same radiographs and even when two observers have been previously trained together (Kopp and Rockler 1978). Consequently, differences in the results, possibly of some magnitude, could be expected in different studies reported in the literature and indeed this is the case,

In view of these problems, no detailed comparisons were made between the results of the present study and those of other similar studies previously reported. However, general comparisons between the present results and those of Ericson and Lundberg (1968) from 80 asymptomatic subjects, and Hansson and Petersson (1978) from 78 subjects with TMJ symptoms, show that there are generally similar frequencies of osteophytes, erosions and concavities.

The frequencies of both flattening and sclerosis recorded in the present study are, however, considerably higher than those reported previously. This may be explained in terms of a more liberal criterion for diagnosis of flattening (i.e. any loss of an even convexity or concavity in the joint outline), and the inability to accurately standardize

a diagnosis of sclerosis, as previously discussed. It is also possible that these features are shown more clearly on an OPG than in the radiographs used in the other studies cited. In addition it is possible that flattening and sclerosis occur more frequently in females with a consequent increase in the respective frequencies reported in this study as compared with those studies of mixed sex samples.

Age and Joint Change

Considering the reasonably high concordance values within the present study, and the presence of a number of areas of similarity with other studies, it seemed reasonable to make comparisons within and between the groups. The first of these concerned the association between age and joint change (Tables 14-17).

There was a higher frequency of subjects over the age of 40 years exhibiting radiological changes compared to those below this age. The difference was significant ($p < .05$), however, only for flattening and sclerosis in edentulous subjects and sclerosis alone in dentate subjects. There were no significant age related differences in the pain or obese groups. However, the obese group consisted entirely of persons below the age of 40 years.

A consensus concerning an association between aging and TMJ changes in the adult is not evident in the literature. Mongini (1972) reported an incidence of remodelling changes approaching 100% in condyles from subjects over the age of 22 years but did not demonstrate a relationship between aging and remodelling changes. In their study of temporomandibular joints, from cadavers aged 45-81 years, Moffett et al. (1964) found no association between aging and joint change. Wedel et al. (1978) did notice some increase in joint changes with increasing age in a study of medieval adult skulls but the difference was not significant.

Oberg et al. (1971) noted an increase in macroscopic joint remodelling changes and osteoarthritic lesions in individuals over the age of 40 years, although a much higher incidence of microscopic change was evident in adult joints of all ages. Blackwood (1963) reported TMJ-OA lesions to be rare before the fourth decade and relatively common thereafter.

In radiological studies, Toller (1973) considered that TMJ-OA was not a rare finding in young persons, although he did not analyse any possible statistical significance in age differences. Ericson and Lundberg (1968) found considerable differences in the frequency of radiological changes above and below the age of 50 years in their study of asymptomatic individuals but they were not statistically significant. Kopp and Rockler (1979) also noted an increase in the frequency of individual radiological changes with increasing age in a group of patients with mandibular pain or dysfunction but these differences were not significant.

Thus the results of the present study, in which only two of the six radiological changes looked for showed significant increases in frequency with age in the non-pain groups, are not dissimilar to those results previously cited. The lack of significant age differences in the pain group (Table 16) is interesting and although it may be accounted for in terms of heterogeneity of dentition, it could also be that joint overloading in this group overrides the influence of ageing to produce a relatively homogenous group as regards joint change.

Edentulous and Dentate Pain-Free Subjects

In comparing the two pain-free groups, all radiological features other than sclerosis occurred more frequently in edentulous subjects (Table 18). However, the only feature for which this difference was significant was the sub-cortical "cyst". As discussed previously, the significance of the occurrence of sub-cortical "cysts" in the present study is unknown, although their occurrence in other synovial joints has been linked with osteoarthritic change.

The increased frequency of the other radiological changes, apart from the sclerosis, in edentulous subjects is consistent with frequencies reported in studies of autopsy and skeletal material (Blackwood, 1963; Moffett et al., 1964; Oberg et al., 1971; Mongini, 1972). In a radiographic study Ericson and Lundberg (1968) also observed TMJ changes to be more common in full denture wearers than in fully dentate individuals and more common in both these groups than in partially dentate subjects. The differences however, were not significant. In his study of patients whom he believed to be suffering from TMJ-OA, Toller (1973) found no significant differences related to their dentition.

It is interesting to speculate that the increased incidence of sclerosis in dentate subjects, especially in those over the age of 40 years, may be a result of the ability to generate greater masticatory force with a natural dentition and consequently greater articular loading than is the case with an edentulous state. An explanation for the opposite trend in incidence for other changes would then require the postulation that this was due to abnormal joint function rather than a difference in the extent of normally directed joint loading.

Obese and Non-Obese Dentate Subjects

There is a common belief that obesity is an important aetiological factor in osteoarthritis of weight-bearing joints. An association has also been noted between obesity and osteoarthritis in non-weight-bearing articulations such as the sternoclavicular and distal interphalangeal joints (Kellgren and Lawrence 1958; Silberberg and Silberberg 1964). This phenomenon has yet to be fully explained and would seem to imply the existence of a systemic influence associated with obesity, as opposed to the local effect of increased joint loading which could occur in weight-bearing joints.

The temporomandibular and sternoclavicular joints are lined not by hyaline articular cartilage, as are other joints, but by articular fibrous tissue. Thus, if a systemic factor is involved in osteoarthritis in the obese, the TMJ could also be implicated. If this is so, a comparison of radiological changes in obese dentate persons under 40 years of age with pain-free dentate individuals of the same age, the group theoretically least likely to have TMJ-OA, could provide valuable information on the radiological diagnosis of TMJ-OA.

When such a comparison was made, however, negligible differences were found between the two groups in this study (Table 19). Although this does not entirely rule out the possibility that there is a higher incidence of TMJ-OA in the obese subjects, it must be regarded as unlikely as many investigators (Blackwood, 1963; Moffett et al., 1964; Hansson and Oberg, 1977; Bean et al., 1977) believe that osteoarthritic lesions are generally preceded by bony remodelling. This is further supported by the complete absence of clinical symptoms related to the TMJ in any of the obese subjects, even though this was not a criterion for

for selection in this group.

Pain and Pain-Free Subjects

The absence of significant differences between the dentate, partially dentate, and edentulous pain subjects (Table 20) as regards the frequency of radiological changes may be due to the overriding effect of joint overloading when compared with the effect of loss of supporting dentition, - a similar explanation to that proposed for the lack of age differences in this group. However, as previously mentioned, Ericson and Lundberg (1968) were also unable to establish significant differences between dentate, partially dentate, and edentulous subjects who were all symptom free.

Significant differences were noted when the pain group was compared with the pain-free dentate group (Table 21) and with a pooled group of all dentate and edentulous pain-free subjects (Table 23). There were no significant differences between the pain group and the edentulous pain-free group (Table 22). However the frequency differences of flattening and sclerosis in individuals under 39 years of age in the pain and edentulous pain-free groups were considerable, with a higher frequency of features in the pain group.

It was difficult to devise a completely satisfactory control group for comparison with the pain group in view of the variety of dentitions included in the latter. Because the pain group comprised approximately equal numbers of dentate, partially dentate and edentulous persons, a pooled group of equal numbers of dentate and edentulous pain-free subjects was thought to constitute the most satisfactory control available from the individuals in this study. When this pooled group was compared with the pain group, all radiological changes,

except for sub-cortical "cysts" which had an equal frequency in both groups, were found to occur more commonly in the pain group. Statistically significant differences between the pain and pain-free groups were observed for osteophytes in subjects over the age of 40 years, erosions in all groups, and for flattening and sclerosis in individuals under the age of 40 years (Tables 23, 24). When the dentate pain-free group was compared with the pain group, the results were the same as for comparison with the pooled group except that the difference in frequency of sclerosis in the 20-39 years age groups, although considerable, was not significant.

When a relationship was sought between the radiological changes which occurred significantly more often in pain subjects and the side on which the pain occurred, it was apparent that none existed. Although only nine patients complained of bilateral pain, (Table 25) there were 26 for whom the radiological index of significant findings was equal on both sides (Table 26). In patients who complained of unilateral symptoms, and who had a greater radiological index for one joint than the other, there was no association between the painful side and that with the greater index score (Table 27). The use of a radiological index in testing this association is open to several criticisms. The index does not take into account the extent of each radiological change and presumes that each of the four features included is of equal importance. However, there was no method which could be used to improve the sensitivity of the index in these areas. Because of this, the infrequent occurrence of several of the features included in the index, and the previous use of a similar index by other investigators, a radiological index was employed in this analysis.

The absence of an association between radiological change and symptoms was not unexpected in view of Yemm's (1971) observation that, under experimental stress, muscle hyperactivity in patients with MPD was symmetrical on both sides, even though the muscles on one side only were painful and tender. It seems, therefore, that although the condition which causes the pain may indeed give rise to a typical pattern of bony remodelling changes in the temporomandibular joints of an affected individual, such alterations are probably not themselves painful. Thus, these observations provide further support for the hypothesis that the MPD syndrome results from hyperactivity in the masticatory muscles and that this is bilateral even though symptoms are generally unilateral.

A number of investigators (Moffett et al., 1964; Hansson and Oberg, 1977; Bean et al., 1977) believe that most osteoarthritic lesions are preceded by bony remodelling alterations. The finding that the pain group had significantly more of some radiological alterations than the asymptomatic groups therefore lends support to the proposal (Laskin, 1979; Ogus, 1979) that MPD can ultimately lead to osteoarthritis in the TMJ.

Although there were statistical differences (Tables 21, 23) between symptomatic and asymptomatic persons, there were no particular features which stood out in this regard. Thus, the extrapolation of these general findings to the clinical situation of a specific patient is of limited value. However the restriction of the diagnosis of MPD to patients with no "radiographic....evidence of organic changes in the TMJ" (Laskin, 1979) seems questionable in view of the present findings and those of previously cited autopsy studies of temporomandibular joints for example that of Bean et al. (1977).

If a clinical implication is sought from these observations, it must be that we should be cautious about overestimating the significance of radiological findings in symptomatic temporomandibular joints especially if they are used to justify aggressive treatment procedures. The main value of radiology for patients with symptomatic complaints related to the temporomandibular joint would seem still to be in the diagnosis of gross joint pathology such as trauma or tumours. Increasing conservatism in the management of TMJ osteoarthritis, with the recommendation of virtually identical treatments for both TMJ-OA and MPD (Carlsson, Kopp and Oberg, 1979; Sarnat and Laskin, 1979), further supports the above statement.

Maximum accuracy in the depiction of all joint surfaces can only be produced by a combination of tomography and plain radiography at the expense of a considerably increased radiation dose. If the results of such a high accuracy examination of joint surfaces have little effect on subsequent diagnosis and management, this increased radiation does not seem warranted. The use of a good quality panoramic tomograph such as the orthopantomograph, which will satisfactorily show both temporomandibular joints as well as the entire dentition, alveolus and contiguous structures, in return for a relatively low radiation dose (Wall et al., 1979) is therefore recommended.

9.5 SUMMARY

An investigation has been carried out into radiological changes in the temporomandibular joints of patients with symptoms related to the joint region, obese patients, and asymptomatic dentate and edentulous persons. A standard panoramic jaw tomograph, the orthopantomograph, provided the information for each subject examined.

The present radiological study has shown:

General Radiological Findings

1. The majority of orthopantomographs examined provided a satisfactory depiction of the surfaces of the condyle and articular eminence of both temporomandibular joints. The clarity of the glenoid fossa in most radiographs was, however, poor.
2. The majority of subjects in all groups displayed the presence of at least one of the radiological features under investigation. The highest frequency of joints with no radiological changes occurred in asymptomatic fully dentate subjects.
3. The scoring of radiological features showed a fairly high level of reproducibility on two separate occasions.

Specific Radiological Findings

4. Flattening or faceting and sclerosis of the cortical plate were the most common features found.
5. All features were noted to occur much more often on the condyle than the articular eminence.

6. Gross changes generally had a relatively low frequency in comparison to the total.

Comparisons within and between Groups

7. The frequency of flattening and cortical sclerosis was positively associated with increasing age in edentulous persons, as was sclerosis alone in dentate persons. There were no significant age related changes in the pain or obese groups.

8. All radiological changes except for sclerosis were more common in edentulous rather than dentate asymptomatic persons. Only sub-cortical "cysts" showed a statistically significant increased frequency in the edentulous group.

9. There were no significant differences between obese and non-obese dentate persons.

10. Within the pain group, there were no significant differences between dentate, partially dentate, and edentulous individuals.

11. Statistically significant differences between the pain and pain-free groups were found for osteophytes in subjects over the age of 40 years, erosions in all age groups, and flattening and sclerosis in subjects under the age of 40 years.

12. There was no relationship between the side on which symptoms occurred and the side with the greater joint change in the pain group.

REFERENCES

- Acheson, R.M. and Collart, A.B. 1975.
New Haven survey of joint diseases.
XVII. Relationship between some systemic characteristics and
osteoarthrosis in a general population.
Ann. Rheum. Dis. 34: 379.
- Agerberg, G., Carlsson, G.E. and Hassler, O. 1969.
Vascularization of the temporomandibular disc.
Odont. Tidsk. 77: 451.
- Agerberg, G., Carlsson, G.E., Ericson, S., Lundberg, M. and Oberg, T. 1970.
Funktionsrubbingar i tuggapparaten. En bettfysiologisk,
roentgenologisk ock serologisk undersokning.
Sver. Tandlak. Forb. Tidn. 62: 1192.
(Cited by Helkimo, 1979).
- Agerberg, G. and Lundberg, M. 1971.
Changes in the temporomandibular joint after surgical treatment.
A radiologic follow-up study.
Oral Surg. 32: 865.
- Agerberg, G. and Carlsson, G.E. 1972.
Functional disorders of the masticatory system.
I. Distribution of symptoms according to age and sex as judged
from investigation by questionnaire.
Acta. Odont. Scand. 30: 597.

Agerberg, G. and Carlsson, G.E. 1973.

Functional disorders of the masticatory system.

II. Symptoms in relation to impaired mobility of the mandible
as judged from investigation by questionnaire.

Acta. Odont. Scand. 31: 335.

Banks, P. and Mackenzie, I. 1975.

Condylotomy. A clinical and experimental appraisal of a
surgical technique.

J. Maxillofac. Surg. 3: 170.

Barbenel, J.C. 1974.

The mechanics of the temporomandibular joint - a theoretical
and electromyographic study.

J. Oral. Rehab. 1: 19.

Baume, L.J. and Derichsweiler, H. 1961

Is the condylar growth centre responsive to orthodontic therapy?

An experimental study in *Macaca mulatta*.

Oral Surg. 14: 347.

Baume, L.J. 1962.

Ontogenesis of the human temporomandibular joint.

I. Development of the condyles.

J. Dent. Res. 41: 1327.

Bean, L.R., Omnell, K.A. and Oberg, T. 1977.

Comparison between radiologic observations and macroscopic
tissue changes in temporomandibular joints.

Dentomaxillofac. Radiol. 6: 90.

Bell, W.E. 1969.

Clinical diagnosis of the pain dysfunction syndrome.
J. Am. Dent. Assoc. 79: 154.

Berry, D. 1969.

Mandibular dysfunction, pain, and chronic minor illness.
Br. Dent. J. 127: 170.

Blackwood, H.J.J. 1963.

Arthritis of the mandibular joint.
Br. Dent. J. 115: 317.

Blackwood, H.J.J. 1966^a.

Cellular remodelling in articular tissue.
J. Dent. Res. 45: 480.

Blackwood, H.J.J. 1966^b.

Adaptive changes in the mandibular joints with function.
Dent. Clinics of Nth. America. Nov. 1966. p. 559.

Blackwood, H.J.J. 1976.

The mandibular joint: development, structure and function.
In: "Scientific Foundations of Dentistry".
Cohen, B. and Kramer, I.R.H. (Eds.)
Heinemann, London.

Blair, G.S. and Chalmers, I.M. 1972.

Radiology of the temporomandibular joint. A comparison of
circular tomography with orthopantomography and lateral trans-
cranio-oblique radiography.
J. Dent. 1: 69.

Blankenship, J.R. and Ramfjord, S.P. 1976.

Lateral displacement of the mandible in rhesus monkeys.
J. Oral. Rehab. 3: 83.

Blaschke, D.D. and White, S.C. 1979.

Radiology.

In: "The Temporomandibular Joint. A Biological basis for
Clinical Practice". (3rd. Ed.)

Sarnat, B.G. and Laskin, D.M. (Eds.)

C.C. Thomas, Springfield.

Bollett, A.J. 1969.

An essay on the biology of osteoarthritis.
Arthritis Rheum. 12: 152.

Boyne, P.J. 1967.

Osseous repair and mandibular growth after
subcondylar fractures.

J. Oral Surg. 25: 300.

Breitner, C. 1940.

Bone changes resulting from experimental orthodontic treatment.
Am. J. Orthod. 26: 521.

Breitner, C. 1941.

Further investigations of bone changes resulting from
experimental orthodontic treatment.

Am. J. Orthod. 27: 605.

Brooke, R.I. 1977.

Secondary osteoarthrosis (osteoarthritis) of the temporo-
mandibular joint.

J. Canad. Dent. Assoc. 43: 323.

Brown, T. 1965.

Physiology of the mandibular articulation.

Aust. Dent. J. 10: 126.

Byers, P.D., Pringle, J., Oztop, F., Fernley, H.N., Brown, M.A. and
Davison, W. 1977.

Observations on osteoarthrosis of the hip.

Semin. Arthritis Rheum. 6: 277.

Campbell, W. 1965.

Clinical radiological investigations of the mandibular joints.

Brit. J. Radiol. 38: 401.

Campbell, W. 1970.

The mandibular joints.

In: "Symposium Ossium".

Jellife, A.M. and Strickland, B. (Eds.)

E. and S. Livingstone, Edinburgh and London.

Carlsson, G.E. Lundberg, M., Oberg, T. and Welanders, U. 1968.

The temporomandibular joint. A comparative anatomic and radiologic
study.

Odont. Revy. 19: 171.

- Ett bettfysiologiskt patient material. En översikt av symptom bilden hos 299 patienter.
Sven. Tandlak. Tidskr. 64: 889.
(Cited by Helkimo, 1979).
- Carlsson, G.E., Kopp, S. and Oberg, T. 1979.
Arthritis and allied diseases of the temporomandibular joint.
In: "Temporomandibular Joint-function and dysfunction".
Zarb, G.A. and Carlsson, G.E. (Eds.)
Munksgaard, Copenhagen.
- Carlsson, G.E. and Oberg, T. 1979.
Remodelling of the temporomandibular joint.
In: "Temporomandibular joint-function and dysfunction".
Zarb, G.A. and Carlsson, G.E. (Eds.)
Munksgaard, Copenhagen.
- Carlsson, G.E. 1980.
Mandibular dysfunction and temporomandibular joint pathosis.
J. Pros. Dent. 43: 658.
- Carraro, J.J., Caffesse, R.G. and Albano, E.A. 1969.
Temporomandibular joint syndrome.
Oral Surg. 28: 54.
- Chalmers, I.M. and Blair, G.S. 1974.
Is the temporomandibular joint involved in primary osteoarthritis.
Oral Surg. 38: 74.
- Cimasoni, G. 1963.
Histopathology of the temporomandibular joint following bilateral extractions of molars in the rat. A preliminary report.
Oral Surg. 16: 613.

Cobb, S., Merchant, W.R. and Rubin, T. 1957.

The relation of symptoms to osteoarthritis.

J. Chronic Dis. 5: 197.

Collins, D.H. 1949.

The pathology of spinal and articular diseases.

Edward Arnold, London.

Costen, J.B. 1934.

A syndrome of ear and sinus symptoms dependent upon disturbed function of the temporomandibular joint.

Ann. Otol. Rhinol. Laryngol. 43: 1.

Davison, P.R. 1974.

Evaluation, diagnosis and treatment of occlusal problems.

Mosby, St. Louis.

De Boever, J.A. 1979.

Functional disturbances of the temporomandibular joint.

In: "Temporomandibular joint-function and dysfunction".

Zarb, G.A. and Carlsson, G.E. (Eds.)

Munksgaard, Copenhagen.

Du Brul, E.L. 1979.

Origin and adaptations of the hominid jaw joint.

In: "The temporomandibular joint. A biological basis for clinical practice." (3rd Ed.)

Sarnat, B.G. and Laskin, D.M. (Eds.)

C.C. Thomas, Springfield.

Durkin, J.F., Heeley, J.D. and Irving, J.T. 1979.

Cartilage of the mandibular condyle.

In: "Temporomandibular joint-function and dysfunction".

Zarb, G.A. and Carlsson, G.E. (Eds.)

Munksgaard, Copenhagen.

Eckerdal, O. 1973.

Tomography of the temporomandibular joint. Correlation between tomographic image and histologic sections in a three-dimensional system.

Acta Radiol. Diagnosis Suppl. 329.

Eckerdal, O. and Ahlqvist, J. 1979.

Thin bony walls of the temporomandibular joint. Morphologic properties and tomographic reproduction.

Acta Radiol. Diagnosis 20: 385.

Editorial. 1973.

Pathogenesis of osteoarthritis.

Lancet i : 1131.

Editorial. 1977.

Pathogenesis of osteoarthritis.

Br. Med. J. 2: 979.

Ericson, S. and Lundberg, M. 1968.

Structural changes in the finger, wrist and temporomandibular joints. A comparative radiologic study.

Acta Odont. Scand. 26: 111.

Findlay, I.A. and Kilpatrick, S.J. 1960.

Analysis of the sounds produced by the mandibular joint.

J. Dent. Res. 39: 1163.

Fine, E. 1971.

Psychological factors associated with non-organic temporomandibular joint pain-dysfunction syndrome.

Br. Dent. J. 131: 402.

Franks, A.S.T. 1964.

The social character of temporomandibular joint dysfunction.

Dent. Practit. 15: 94.

Franks, A.S.T. 1965.

Masticatory muscle hyperactivity and temporomandibular joint dysfunction.

J. Pros. Dent. 15: 1122.

Freeman, M.A.R. and Meachim, G. 1973.

Ageing, degeneration and remodelling of articular cartilage.

In: "Adult articular cartilage."

Pitman Medical, Alden Press, Oxford.

Furstman, L. 1965.

The effect of loss of occlusion upon the mandibular joint.

Am. J. Orthod. 51: 245.

Gelb, H., Calderone, J.P., Gross, S.M. and Kantor, M.E. 1967.

The role of the dentist and the otolaryngologist in evaluating temporomandibular joint syndromes.

J. Pros. Dent. 18: 497.

Gibbs, C.H. and Suit, S.R. 1973.

Movements of the jaw after unexpected contact with a hard object.
J. Dent. Res. 52: 810.

Goldin, R.H., McAdam, L., Louie, J.S., Gold, R. and Bluestone, R. 1976.

Clinical and radiological survey of the incidence of
osteoarthritis among obese patients.
Ann. Rheum. Dis. 35: 349.

Goldstein, I.B., Grinker, R.R., Heath, H.A., Oken, A. and Shipman, W.G. 1964.

Study in psychophysiology of muscle tension: I. Response
specificity.
Arch. Gen. Psychiat. 11: 322.

Goldstein, I.B. 1965.

The relationship of muscle tension and autonomic activity to
psychiatric disorders.
Psychosom. Med. 27: 39.

Goodman, P., Green, C.S. and Laskin, D.M. 1976.

Response of patients with myofascial pain-dysfunction
syndrome to mock equilibration.
J. Am. Dent. Assoc. 92: 755.

Gordon, T. 1968

Osteoarthritis in U.S. adults.

In: "Population studies of the rheumatic diseases".

International Congress Series No. 148.

Bennett, P.H. and Wood, P.H.N. (Eds.)

Excerpta Medica Foundation, Amsterdam.

Goss, A.N. 1974.

The myofascial pain dysfunction syndrome.

I. Aetiology and diagnosis.

N.Z. Dent. J. 70: 192.

Greene, C.S., Lerman, M.D., Satcher, H.D. and Laskin, D.M. 1969.

The TMJ pain-dysfunction syndrome: Heterogeneity of the patient population.

J. Am. Dent. Assoc. 79: 1168.

Greene, C.S. and Laskin, D.M. 1971.

Meprobamate therapy for the myofascial pain-dysfunction (MPD) syndrome: A double blind evaluation.

J. Am. Dent. Assoc. 82: 587.

Greene, C.S. and Laskin, D.M. 1972.

Splint therapy for the myofascial pain-dysfunction syndrome: A comparative study.

J. Am. Dent. Assoc. 84: 624.

Greene, C.S. 1979.

Myofascial pain-dysfunction syndrome: the evolution of concepts.

In: "The temporomandibular joint. A biological basis for clinical practice". (3rd Ed.)

Sarnat, B.G. and Laskin, D.M. (Eds.)

C.C. Thomas, Springfield.

Grieg, J.H. and Mysaph, F.W. 1973.

A method of radiological demonstration of the temporomandibular joints using the orthopantomograph. Radiology 106: 307.

Griffin, C.J. and Munro, R.R. 1971.

Electromyography of the masseter and anterior temporalis muscles in patients with temporomandibular dysfunction. Archs. Oral. Biol. 16: 929.

Griffin, C.J., Hawthorn, R. and Harris, R. 1975.

Anatomy and histology of the human temporomandibular joint. Monogr. Oral Sci. 4: 1.

Gupta, O.P., Forrest, E.J., Sassouni, V. and Mundell, R.D. 1971.

Studies on experimental malocclusion in rabbits. I. Method of induction of malocclusion and its effects on the temporomandibular joint. Am. J. Orthod. 60: 54.

Hankey, G.T. 1954.

Temporomandibular arthrosis. An analysis of 150 cases. Br. Dent. J. 97: 249.

Hannan, A.G. and Matthews, B. 1968.

Reflex jaw opening in response to stimulation of periodontal mechanoreceptors in the cat. Archs. Oral Biol. 14: 1401.

Hansson, L.G. and Petersson, A. 1978.

Radiography of the temporomandibular joint using the transpharyngeal projection. A comparison study of information obtained with different radiographic techniques.
Dentomaxillofac. Radiol. 7: 69.

Hansson, T. and Nordstrom, B. 1977.

Thickness of the soft tissue layers and articular disc in temporomandibular joints with deviations in form.
Acta. Odont. Scand. 35: 281.

Hansson, T. and Oberg, T. 1977.

Arthrosis and deviation in form in the temporomandibular joint. A macroscopic study on a human autopsy material.
Acta. Odont. Scand. 35: 167.

Hansson, T., Oberg, T., Carlsson, G.E. and Kopp, S. 1977.

Thickness of the soft tissue layers and the articular disk in the temporomandibular joint.
Acta Odont. Scand. 35: 77.

Hekneby, M. 1974.

The load of the temporomandibular joint: Physical calculations and analyses.
J. Pros. Dent. 31: 303.

Helkimo, E., Carlsson, G.E. and Carmeli, Y. 1975.

Bite force in patients with functional disturbances of the masticatory system.
J. Oral. Rehab. 2: 397.

Helkimo, M.I. 1974.

Studies on function and dysfunction of the masticatory system. I. An epidemiological investigation of symptoms of dysfunction in Lapps in the north of Finland.

Proc. Finn. Dent. Soc. 70: 37.

Helkimo, M.I. 1979.

Epidemiologic surveys of dysfunction of the masticatory system.

In: "Temporomandibular joint - function and dysfunction".

Zarb, G.A. and Carlsson, G.E. (Eds.)

Munksgaard, Copenhagen.

Hiniker, J.J. and Ramfjord, S.P. 1966.

Anterior displacement of the mandible in adult rhesus monkeys.

J. Pros. Dent. 16: 503.

Hollender, L. and Ridell, A. 1974.

Radiography of the temporomandibular joint after oblique sliding osteotomy of mandibular rami.

Scand. J. Dent. Res. 82: 466.

Ingervall, B., Carlsson, G.E. and Thilander, B. 1976.

Postnatal development of the human temporomandibular joint.

II. A microradiographic study.

Acta. Odont. Scand. 34: 133.

Janzen, E.K. and Bluher, J.A. 1965.

The cephalometric, anatomic, and histologic changes in *Macaca mulatta* after application of a continuous-acting retraction force on the mandible.

Am. J. Orthod. 51: 823.

- Johnson, D.L., Shipman, W.G. and Laskin, D.M. 1972.
Physiologic responses to stressful stimuli in patients with
myofascial pain-dysfunction syndrome.
Abstract no. 191, Chicago, I.A.D.R.
- Johnson, L.C. 1962.
Joint remodelling as the basis for osteoarthritis.
J. Am. Vet. Med. Assoc. 141: 1237.
- Katzberg, R.W., Dolwick, M.F., Helms, C.A., Hopens, T., Bales, D.J.
and Coggs, G.C. 1980.
Arthrotomography of the temporomandibular joint.
Am. J. Roentgenol. 134: 995.
- Kellgren, J. H. and Lawrence, J.S. 1958.
Osteoarthritis and disc degeneration in an urban population.
Ann. Rheum. Dis. 17: 388.
- Kellgren, J.H. 1961.
Osteoarthrosis in patients and populations.
Br. Med. J. ii: 1.
- Kellgren, J.H., Lawrence, J.S. and Bier, F. 1963.
Genetic factors in generalised osteo-arthrosis.
Ann. Rheum. Dis. 22: 237.
- Kidokoro, Y., Kubota, K., Shuto, S. and Sumino, R. 1968.
Reflex organisation of masticatory muscles in the cat.
J. Neurophysiol. 31: 695.

Klein, I.E., Blatterfein, L. and Miglino, J.C. 1970.

Comparison of the fidelity of radiographs of mandibular condyles made by different techniques.

J. Pros. Dent. 24: 419.

Kopp, S., Carlsson, G.E., Hansson, T. and Oberg, T. 1976.

Degenerative disease in the temporomandibular, metatarsophalangeal and sternoclavicular joints. An autopsy study.

Acta. Odont. Scand. 34: 23.

Kopp, S. 1977^a

Subjective symptoms in temporomandibular joint osteoarthritis.

Acta. Odont. Scand. 35: 207.

Kopp, S. 1977^b

Clinical findings in temporomandibular joint osteoarthritis.

Scand. J. Dent. Res. 85: 434.

Kopp, S. and Rockler, B. 1978.

Variation in interpretation of radiographs of temporomandibular and hand joints.

Dentomaxillofac. Radiol. 7: 95.

Kopp, S. and Rockler, B. 1979.

Relationship between clinical and radiographic findings in patients with mandibular pain or dysfunction.

Acta. Radiol. Diagnosis 20: 465.

Kovaleski, W.C., Bailey, J.O. and Ash, M.M. 1976.

Evaluation via TMJ radiography of condylar position in normal patients.

J. Dent. Res. 55B Abst. No. 1074.

Kreutziger, K.L. and Mahan, P.E. 1975.

Temporomandibular degenerative joint disease.

Part I. Anatomy, pathophysiology, and clinical description.

Oral Surg. 40: 165.

Kydd, W.L. 1959,

Psychosomatic aspects of temporomandibular joint dysfunction.

J. Am. Dent. Assoc. 59: 31.

Laskin, D.M. 1969.

Etiology of the pain-dysfunction syndrome.

J. Am. Dent. Assoc. 79: 147.

Laskin, D.M. 1979.

Myofascial pain-dysfunction syndrome: etiology.

In: "The temporomandibular joint. A biological basis for clinical practice". (3rd Ed).

Sarnat, B.G. and Laskin, D.M. (Eds.)

C. C. Thomas, Springfield.

Lawrence, J.S., Bremner, J.M. and Bier, F. 1966.

Osteoarthritis: prevalence in the population and relationship between symptoms and x-ray changes.

Ann. Rheum. Dis. 25: 1.

Levy, B.M. 1964.

Embryological development of the temporomandibular joint.

In: "The temporomandibular joint". (2nd Ed.)

Sarnat, B.G., (Ed).

C. C. Thomas, Springfield.

Lindahl, L. and Hollender, L. 1977.

Condylar fractures of the mandible.

II. A radiographic study of remodelling processes in the temporomandibular joint.

Int. J. Oral Surg. 6: 153.

Lindblom, G. 1960.

On the anatomy and function of the temporomandibular joint.

Acta. Odont. Scand. 17, Suppl. 28.

Lindqvist, B. 1974.

Bruxism in twins.

Acta. Odont. Scand. 32: 1.

Lindsay, K.N. 1977.

An autoradiographic study of cellular proliferation of the mandibular condyle after induced dental malocclusion in the mature rat.

Archs. Oral Biol. 22: 711.

Lindvall, A.M., Helkimo, E., Hollender, L. and Carlsson, G.E. 1976.

Radiographic examination of the temporomandibular joint. A comparison between radiographic findings and gross and microscopic morphologic observations.

Dentomaxillofac. Radiol. 5: 24.

Lund, K. 1974.

Mandibular growth and remodelling processes after condylar fractures. A longitudinal roentgencephalometric study.

Acta. Odont. Scand. 32 Suppl. 64.

Lupton, D.E. 1966.

A preliminary investigation of the personality of female temporomandibular joint dysfunction patients.

Psychother. Psychosom. 14: 199.

Lupton, D.E. 1969.

Psychological aspects of temporomandibular joint dysfunction.

J. Am. Dent. Assoc. 79: 131.

McNamara, J.A. 1975.

Functional adaptations in the temporomandibular joint.

Dent. Clinics of Nth. America. 19: 457.

Macalister, A.D. 1954.

A microscopic survey of the human temporomandibular joint.

N.Z. Dent. J. 50: 161.

Madsen, B. 1966.

Normal variation in anatomy, condylar movements, and arthrosis frequency of the temporomandibular joints.

Acta Radiol. Diagnosis 4: 273.

Mankin, H.J. and Lippiello, L. 1971.

The glycosaminoglycans of normal and arthritic cartilage.

J. Clin. Invest. 50: 1712.

Mankin, H.J. 1976.

Biochemical changes in articular cartilage in osteoarthritis.

In: "A.A.O.S. Symposium on osteoarthritis".

Mosby, St. Louis.

Meikle, M.C. 1979.

Remodelling.

In: "The temporomandibular joint. A biological basis for clinical practice". (3rd Ed.)

Sarnat, B.G., and Laskin, D.M. (Eds.)

C.C. Thomas, Springfield.

Mercuri, L.G., Olson, R. E. and Laskin, D.M. 1979.

The specificity of response to experimental stress in patients with myofascial pain-dysfunction syndrome.

J. Dent. Res. 58: 1866.

Moffett, B.C., Johnson, L.C., McCabe, J.B. and Askew, H.C. 1964.

Articular remodelling in the adult human temporomandibular joint.

Am. J. Anat. 115: 119.

Moffett, B.C. 1966.

The morphogenesis of the temporomandibular joint.

Am. J. Orthod. 52: 401.

Mohl, N.D. 1973.

Alterations in the temporomandibular joint.

Oral Surg. 36: 625.

Molin, C. 1972.

Vertical isometric muscle forces of the mandible. A comparative study of subjects with and without manifest mandibular pain dysfunction syndrome.

Acta. Odont. Scand. 30: 485.

Mongini, F. 1972.

Remodelling of the mandibular condyle in the adult and its relationship to the condition of the dental arches.

Acta Anat. 82: 437.

Mongini, F. 1975.

Dental abrasion as a factor in remodelling of the mandibular condyle.

Acta. Anat. 92: 292.

Mongini, F. 1977.

Anatomic and clinical evaluation of the relationship between the temporomandibular joint and occlusion.

J. Pros. Dent. 38: 539.

Moskowitz, R.W. 1979.

Clinical and laboratory findings in osteoarthritis.

In: "Arthritis and allied conditions". (9th Ed).

McCarty, D.J. (Ed).

Lea and Febiger,, Philadelphia.

Moulton, R.E. 1968.

Emotional factors in non-organic temporomandibular joint pain.

In: "Facial pain and mandibular dysfunction".

Schwartz, L.L. and Chayes, C.M. (Eds.)

Saunders, Philadelphia.

Muir, H. 1977.

Molecular approach to the understanding of osteoarthrosis.

Ann. Rheum. Dis. 36: 199.

Munro, R.R. 1972.

Electromyography of the masseter and anterior temporalis muscle in subjects with potential temporomandibular joint dysfunction.

Aust. Dent. J. 17: 209.

Murnane, T.W. and Doku, H.C. 1971.

Light and electron microscopic appearance of synovial lining tissues in a patient with temporomandibular joint dysfunction.

Oral Surg. 31: 452.

Murray, R.O. and Jacobson, H.G. 1971.

The radiology of skeletal disorders.

Churchill Livingstone, Edinburgh, London.

Ogus, H. 1975.

Rheumatoid arthritis of the temporomandibular joint.

Br. J. Oral Surg. 12: 275.

Ogus, H. 1979.

Degenerative disease in the temporomandibular joint in young persons.

Br. J. Oral Surg. 17: 17.

Oberg, T., Carlsson, G.E. and Fajers, C.M. 1971.

The temporomandibular joint. A morphologic study on a human autopsy material.

Acta Odont. Scand. 29: 349.

Olsson, A. and Krogh-Poulson, W.G. 1966.

Occlusal disharmonies and dysfunction of the stomatognathic system.

Dental Clinics of Nth. America. Nov. 1966. p. 627.

Omnell, K.A. and Petersson, A. 1976.

Radiography of the temporomandibular joint utilizing oblique lateral transcranial projections. Comparison of information obtained with standardized technique and individualized technique. Odont. Revy. 27: 77.

Paatero, Y.V. 1961.

Pantomography and orthopantomography.

Oral Surg. 14: 947.

Perry, H.T., Lammie, G.A., Main, J. and Teuscher, G. W. 1960.

Occlusion in a stress situation.

J. Am. Dent. Assoc. 60: 626.

Perry, H.T. 1968.

The symptomatology of temporomandibular joint disturbance.

J. Pros. Dent. 19: 288.

Petersson, A. and Nanthaviroj, S. 1975.

Radiography of the temporomandibular joint utilizing the transmaxillary projection. A comparison of the information obtained with the oblique lateral transcranial projection versus the transmaxillary projection.

Dentomaxillofac. Radiol. 4: 76.

Pietrokovski, J. 1970.

Tooth drift and changes in the temporomandibular joint
following tooth extraction in the monkey.

J. Periodont. 41: 353.

Pomp, A.M. 1974.

Psychotherapy for the myofascial pain-dysfunction (MPD)
syndrome: A study of factors coinciding with symptom remission.

J. Am. Dent. Assoc. 89: 629.

Posselt, U. 1964.

Physiology of occlusion and rehabilitation.

Blackwell Scientific publications, Oxford.

Poswillo, D.E. 1970.

Experimental investigation of the effects of intra-articular
hydrocortisone and high condylectomy on the mandibular condyle.

Oral Surg. 30: 161.

Poswillo, D.E. 1972.

The late effects of mandibular condylectomy.

Oral Surg. 33: 500.

Prentiss, H.J. 1918.

Preliminary report upon the temporomandibular articulation
in the human.

Dent. Cosmos 60: 505

(Cited by Greene, 1979).

Quantrill, J.R. and Lewis, J.E.S. 1974.

The interpretation of temporomandibular joint radiographs.
Sth. Afr. Med. J. 48: 1905.

Radin, E.L., Paul, I.L. and Rose, R.M. 1972.

Role of mechanical factors in pathogenesis of primary
osteoarthritis.
Lancet i, 519.

Radin, E.L., Parke, H.G., Pugh, J.W., Steinberg, R.S., Paul, I.L.
and Rose, R.M. 1973.

Response of joints to impact loading.
III. Relationship between trabecular microfractures and
cartilage degeneration.
J. Biomech. 6: 51.

Ramfjord, S.P. 1961^a

Bruxism, a clinical and electromyographic study.
J. Am. Dent. Assoc. 62: 21.

Ramfjord, S.P. 1961^b

Dysfunctional temporomandibular joint and muscle pain.
J. Pros. Dent. 11: 353.

Ramfjord, S.P. and Hiniker, J.J. 1966.

Distal displacement of the mandible in adult rhesus monkeys.
J. Pros. Dent. 16: 491.

Ramfjord, S.P. and Ash, M.M. 1971.

Occlusion. (2nd. Ed.)

Saunders, Philadelphia.

Ramfjord, S.P., Walden, J.M. and Enlow, R.D. 1971.

Unilateral function and the temporomandibular joint in rhesus monkeys.

Oral Surg. 32: 236.

Resnick, D. 1974.

Temporomandibular joint involvement in ankylosing spondylitis. Comparison with rheumatoid arthritis and psoriasis.

Radiology. 112: 587.

Reuben, B. and Laskin, D.M. 1977.

Electromyographic analysis of masticatory muscle activity in myofascial pain-dysfunction syndrome.

J. Dent. Res. 56B: 232.

Richards, L. 1978.

Attrition and the temporomandibular joint.

Thesis, Univ. of Adelaide.

Ricketts, R.M. 1964.

Roentgenography of the temporomandibular joint.

In: "The temporomandibular joint". (2nd Ed).

Sarnat, B.G. (Ed).

C.C. Thomas, Springfield.

Ricketts, R.M. 1964.

Degenerative disease of the mandibular joint.

J. Dent. Res. 43: 819.

Robinson, M. 1946.

The temporomandibular joint: Theory of reflex controlled non-lever action of the mandible.

J. Am. Dent. Assoc. 33: 1260.

Rothwell, P.S. 1972.

Personality and temporomandibular joint dysfunction.

Oral Surg. 34: 734.

Ruben, M.P. and Mafla, E. 1971.

Effects of traumatic occlusion on the temporomandibular joint of rhesus monkeys.

J. Periodont. 42: 79.

Rugh, J.D. and Solberg, W.K. 1979.

Psychological implications in temporomandibular pain and dysfunction.

In: "Temporomandibular joint - function and dysfunction".

Zarb, G.A. and Carlsson, G.E. (Eds.)

Munksgaard, Copenhagen.

Salter, R.B. Gross, A. and Hall, J.H. 1967.

Hydrocortisone arthropathy - An experimental investigation.

Canad. Med. Assoc. J. 97: 374.

Sarnat, B.G. and Laskin, D.M. 1979.

Surgical considerations.

In: "The temporomandibular joint. A biological basis for clinical practice". (3rd. Ed.)

Sarnat, B.G. and Laskin, D.M. (Eds.)

C.C. Thomas, Springfield.

Schwartz, L.L. 1955.

Pain associated with the temporomandibular joint.

J. Am. Dent. Assoc. 51: 394.

Schwartz, L.L. and Cobin, H.P. 1957.

Symptoms associated with the temporomandibular joint.

Oral Surg. 10: 339.

Schwartz, L.L. 1959.

Disorders of the temporomandibular joint.

Saunders, Philadelphia.

Schwartz, L.L. and Marbach, J.J. 1965.

Changes in the temporomandibular joints with age.
(Degenerative joint disease).
Periodontics 3: 184.

Schwartz, R.A., Greene, C.S. and Laskin, D.M. 1979.

Personality characteristics of patients with myofascial pain-dysfunction (MPD) syndrome unresponsive to conventional therapy.
J. Dent. Res. 58: 1435

Seifert, M.H., Whiteside, C.G. and Savage, O. 1969.

A 5-year follow-up of fifty cases of idiopathic osteoarthritis of the hip.
Ann. Rheum. Dis. 28: 325.

Seward, F.S. 1976.

Tooth attrition and the temporomandibular joint.
Angle Orthod. 46: 162.

Sharpe, C.J., Gee, E.J. and Griffin, C.J. 1965.

The osteogenic potential of the human condyle.
Aust. Dent. J. 10: 287.

Shore, N.A. 1976.

Temporomandibular joint dysfunction and occlusal equilibration.
2nd. Ed.
Lippincott, Philadelphia.

Sicher, H. 1948.

Temporomandibular articulation in mandibular overclosure.
J. Am. Dent. Assoc. 36: 131.

Silberberg, R. and Silberberg, M. 1964.

Pathogenesis of osteoarthritis.
Path. Microbiol. 27: 447.

Silbermann, M. 1976.

Experimentally induced osteoarthritis in the temporo-
mandibular joint of the mouse.
Acta. Anat. 96: 9.

Skiba, T.J. and Laskin, D.M. 1976.

Masticatory muscle silent periods in patients with
MPD syndrome.
J. Dent. Res. 55B: 249.

Sokoloff, L., Mickelsen, O., Silverstein, E., Jay, G.E. and
Yamamoto, R. 1960.

Experimental obesity and osteoarthritis.
Am. J. Physiol. 198: 765.

Sokoloff, L. 1979.

The pathology and pathogenesis of osteoarthritis.
In: "Arthritis and allied conditions". (9th Ed.)
McCarty, D.J. (Ed.)
Lea and Febiger, Philadelphia.

Solberg, W.K., Flint, R.T. and Brantner, J.P. 1972.

Temporomandibular joint pain and dysfunction: A clinical study of emotional and occlusal components.

J. Pros. Dent. 28: 412.

Stanson, W. W. and Baker, H.L. 1976.

Routine tomography of the temporomandibular joint.

Radiol. Clinics of Nth. America. 14: 105.

Stecher, R.M. 1955.

Heberden's nodes: A clinical description of osteoarthritis of the finger joints.

Ann. Rheum. Dis. 14:1.

Stockli, P.W. and Willert, H.G. 1971.

Tissue reactions in the temporomandibular joint resulting from anterior displacement of the mandible in the monkey.

Am. J. Orthod. 60: 142.

Storey, E. 1975.

Growth and remodelling of bone and bones. Role of genetics and function.

Dental Clinics of Nth. America. 19: 443.

Summa, R. 1918.

The importance of the inter-articular fibrocartilage of the temporomandibular articulation.

Dent. Cosmos 60: 512.

(Cited by Greene, 1979).

Swanson, A.G. 1971.

Prominent neurologic symptoms and their management: pain.

In: "Cecil-Loeb Textbook of Medicine". (13th Ed.)

Besson, P.B. and McDermott, W. (Eds.)

Saunders, Philadelphia.

Sweet, M.B.E., Thonar, E.J.M.A., Immelman, A.R. and Solomon, L. 1977.

Biochemical changes in progressive osteoarthritis.

Ann. Rheum. Dis. 36: 387.

Takada, K. Yoshimura, Y. et al. 1971.

Clinical study of the temporomandibular joint disturbances.

I. Statistical observation of patients with temporomandibular arthrosis.

J. Osaka Univ. Dent. Sch. 11: 7.

Takiguchi, R. and Kamijo, Y. 1975.

Scanning electron microscopical study on morphological changes in surfaces of the mandibular joint because of resorption.

Bull. Tokyo Dent. Coll. 16: 163.

Tammisalo, E.H. and Mattila, K. 1963.

Kondyylien kavautuminen ortoradiaalisessa pantomografiassa.

(Condyles in orthoradial pantomography).

Suom. Hammas. Toim. 59: 247.

Tammisalo, E.H. 1964.

Orthopantomographic roentgenography of the temporomandibular joint.

Suom. Hammas. Toim. 60: 139.

- Taylor, R.C., Ware, W.H., Fowler, D. and Kobayashi, J. 1972.
A study of temporomandibular joint morphology and its relationship to the dentition.
Oral Surg. 33: 1002.
- Thilander, B. 1965.
Chin-cap treatment for Angle class III malocclusion.
Europ. Orthod. Soc. Trans. 41: 311.
- Thilander, B., Carlsson, G.E. and Ingervall, B. 1976.
Postnatal development of the human temporomandibular joint.
I. A histological study.
Acta. Odont. Scand. 34: 117.
- Thomson, H. 1959.
Mandibular joint pain. A survey of 100 treated cases.
Br. Dent. J. 107: 243.
- Thomson, H. 1971.
Mandibular dysfunction syndrome.
Br. Dent. J. 130: 187.
- Toller, P.A. 1969.
The transpharyngeal radiography for arthritis of the mandibular condyle.
Br. J. Oral Surg. 7: 47.
- Toller, P.A. 1973.
Osteoarthrosis of the mandibular condyle.
Br. Dent. J. 134: 223.

- Toller, P.A. 1974.
Temporomandibular arthropathy.
Proc. Roy. Soc. Med. 67: 153.
- Toller, P.A. and Glynn, L.E. 1976.
Degenerative disease of the mandibular joint.
In: "Scientific foundations of dentistry".
Cohen, B. and Kramer, I.R.H. (Eds.)
Heinemann, London.
- Toller, P.A. 1977.
Ultrastructure of the condylar articular surface in severe
mandibular pain-dysfunction syndrome.
Int. J. Oral Surg. 6: 297.
- Tveito, L. 1974.
Beurteilung der Gelenkspaltbreite.
Dtsch. Zahnarztl. Z. 29: 550.
(Cited by De Boever, 1979).
- Tyas, M.J. 1974.
Identification of skeletal remains from dental evidence.
Aust. Dent. J. 19: 12.
- Uotila, E. 1964.
The temporomandibular joint in adult rheumatoid arthritis.
A clinical and roentgenologic study.
Acta. Odont. Scand. 22. Suppl. 39.
- Uotila, E. 1965.
Orthopantomographic differential diagnosis of the temporo-
mandibular joint.
Suom. Hammas. Toim. 61: 34.
- Updegrave W.J. 1971.
Radiography of the temporomandibular joints.
Semin. Roetgenol. 6: 381.

Vestergaard Christensen, L. 1971.

Facial pain and internal pressure of masseter muscle in
experimental bruxism in man.

Archs. Oral Biol. 16: 1021.

Vestergaard Christensen, L. 1976.

Facial pain in negative and positive work of human jaw muscles.

Scand. J. Dent. Res. 84: 827.

Wall, B.F. Fisher, E.S., Paynter, R. Hudson, A. and Bird, P.D. 1979.

Doses to patients from pantomographic and conventional dental
radiography.

Br. J. Radiol. 52: 727.

Walton, M. 1979.

Obesity as an aetiological factor in the development of
osteoarthritis.

Gerontology. 25: 36.

Wedel, A., Carlsson, G.E. and Sagne, S. 1978.

Temporomandibular joint morphology in a medieval skull material.

Swed. Dent. J. 2: 177.

Weinberg, L.A. 1972.

Correlation of temporomandibular dysfunction with radiographic
findings.

J. Pros. Dent. 28: 519.

Weinberg, L.A. 1973.

What we really see in a TMJ radiograph.

J. Pros. Dent. 30: 898.

Weinberg, L.A. 1978.

An evaluation of asymmetry in TMJ radiographs.

J. Pros. Dent. 40: 315.

Weisengreen, H.H. 1975.

Observation of the articular disc.

Oral Surg. 40: 113

Wolff, H.G. 1972.

Headache and other head pain.

3rd Ed. (revised by Dalessio, D.J.)

Oxford University Press, New York.

Worth, H.M. 1979.

Radiology of the temporomandibular joint.

In: "Temporomandibular joint- function and dysfunction".

Zarb, G.A. and Carlsson, G.E. (Eds.)

Munksgaard, Copenhagen.

Wright, D.M. and Moffett, B.C. 1974.

The postnatal development of the human temporomandibular joint.

Am. J. Anat. 141: 235.

Yale, S.H., Allison, B.D. and Hauptfuehrer, J.D. 1966.

An epidemiological assessment of mandibular condylar morphology.

Oral Surg. 21: 169.

Yemm, R. 1969^a

Variations in the electrical activity of the human masseter muscle occurring in association with emotional stress.

Archs. Oral Biol. 14: 873.

Yemm, R. 1969^b

Masseter muscle activity in stress: adaptation of response to a repeated stimulus in man.

Archs. Oral Biol. 14: 1437.

Yemm, R. 1971.

A comparison of the electrical activity of masseter and temporal muscles of human subjects during experimental stress.

Archs. Oral Biol. 16: 269.

Yemm, R. 1979.

Neurophysiologic studies of temporomandibular joint dysfunction.

In: "Temporomandibular joint-function and dysfunction".

Zarb, G.A. and Carlsson, G.E. (Eds.)

Munksgaard, Copenhagen.

Youdelis, R.A. 1966.

Ossification of the human temporomandibular joint.

J. Dent. Res, 45: 192.

Yune, H.Y., Hall, J.R., Hutton, C.E. and Klatter, E.C. 1973.

Roentgenologic diagnosis in chronic temporomandibular joint dysfunction syndrome.

Am. J. Roentgenol. Radium. Ther. Nucl. Med. 118: 401.

Zimmermann, A.A. 1951.

Evaluation of Costen's syndrome from an anatomic point of view.

In: "The temporomandibular joint".

Sarnat, B.G. (Ed).

C.C. Thomas, Springfield.