Craniomandibular Disorders in the Geriatric Patient

This paper represents a general review of basic age-related changes that take place in the craniomandibular apparatus and the most frequently presenting conditions associated with craniomandibular disorders (CMD) in the elderly. The evaluation of geriatric patients with signs or symptoms of CMD must consider (1) normal age-related changes in the craniomandibular apparatus and their impact on both normal function and responses to stress; (2) the role of dentition status and dental prostheses in CMD; and (3) the contribution of malignant disease, psoriasis, arthritic conditions, pseudogout, granulomatous vascular conditions, and metaplastic involvement of tissue to the pathosis of CMD. The clinician must also be aware of various effects of psychologic, sociologic, and biologic aspects of aging on the development of headache and atypical facial pain as components of CMD in the geriatric patient.
J OROFACIAL PAIN 1993;7:38–53

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Recent increasing knowledge and interest in craniomandibular disorders (CMD) is evidenced by extensive publicity in both the professional and popular press. It is estimated that pain and dysfunction of the craniomandibular apparatus afflict up to 48% of the population in Western countries.[1–3] Although complaints among patients over the age of 65 decrease by 35% to 50% in most studies, the incidence of geriatric CMD is still highly significant.[2] Craniomandibular disorders present complex and frustrating diagnostic challenges to medicine and dentistry because they can mimic many different conditions, they may be part of a much larger systemic and behavioral complex, and they usually have multiple contributing etiologic factors. Some patients present with obvious and fundamental problems such as an inability to open or close the mouth, but others present with localized or diffuse pain in the face, head, neck, or shoulders. Still others may report vertigo, tinnitus, hearing loss, paresthesias, nausea, excessive lacrimation or salivation, dryness or flushing of the skin, and visual disturbances.

This wide array of sensory, motor, and autonomic possibilities makes diagnosis difficult and misdiagnosis common. Many CMD patients go unsuccessfully from doctor to doctor seeking relief from their symptoms. Many eventually wonder whether their condition may be psychosomatic (especially if a frustrated practitioner suggests it). The fact is, however, that in chronic pain disorders it is usually the pain that leads to mental symptoms, not the reverse.[4]

It is common for a patient's mental state to progress from anxiety to deepening depression as the search for relief proves fruitless. During this relief-seeking process, many patients subject themselves to major occlusal or surgical interventions that are not appropriate and often exacerbate the problems. Hostility toward health professionals often results. In addition, patients may have become dependent on medications prescribed by doc-

tors unaware of the complexities of managing chronic pain.

The current approach to diagnosis and treatment of CMD recognizes a multifactorial etiology and is multidisciplinary in nature. There is an increasing emphasis on conservative and behavioral treatments rather than irreversible surgical or occlusal intervention. The problems and challenges of providing an accurate diagnosis and effective treatment for these disorders are compounded in the geriatric patient by the physiology and biochemistry of normal age-related changes in the elderly. Practitioners must evaluate how these changes can predispose elderly patients to CMD and how they affect pre-existing conditions. Treating geriatric patients demands that the clinician thoroughly review the medical history, perform a physical examination, obtain a radiographic evaluation, and seek proper consultations to rule out any contributing systemic problems. Older individuals are less tolerant of noxious insults (having reduced inflammatory and immune responses, healing response, and reserve capacities) and at a much greater risk of debilitating or life-threatening diseases (infectious, neoplastic, and genetic), and a misdiagnosis may lead to long-term, expensive therapy without resolution of the problem and may have grave consequences for the patient. There are many disturbing reports of geriatric patients who were referred to multiple practitioners and treated for CMD for whom the final diagnosis turned out to be malignant lesions, endocrine disorders, metabolic disorders, or infections.[5–14] Misdiagnosis of CMD can result in improper treatment and prolongation of disease states with potentially fatal results.

This paper highlights the effects of aging on the craniomandibular apparatus and reviews those conditions that present with particular frequency in the geriatric population. Practitioners must consider these effects and conditions when formulating a differential diagnosis and providing treatment for elderly individuals. This approach helps ensure competent diagnosis and management for all patients.

Animal Models of Aging

The effects of age on the craniomandibular apparatus have been studied most comprehensively in animal models such as the rat[15] and the senescence-accelerated mouse.[16] The majority of these studies have focused on the most publicized part of this apparatus — the temporomandibular joint (TMJ).

Physiologic degeneration of the TMJ occurs with a definite progression that is related to chronologic age.[17] Remodeling of joint tissue is a physical phenomenon that accommodates functional changes of the joint during aging, but remodeling can also act in a destructive manner leading to joint deformation and degeneration.[18–21] Histopathologically, age-related changes in the TMJ have been staged as follows. In health, articular surfaces are intact and smooth. Two to four rows of flattened fibroblasts underlie the surface, a cartilage layer exhibits strong metachromasia with toluidine blue, and the deepest parts of the cartilage are calcified and in close apposition to bone marrow. At the onset of degeneration, articular surfaces become wavy fibroblasts spread sporadically and unevenly, cartilage exhibits weak metachromasia, and subchondral bone is found between calcified cartilage and bone marrow. Later, fissures extend from the articular surface to the cartilage layer, and areas around fissures are cell-free and nonmetachromatic. Next, articular surfaces become ulcerated and degeneration progresses into calcified cartilage. Horizontal clefts then appear between articular and calcified cartilage, and many areas of denuded calcified cartilage are evident. Sclerosis of subchondral bone begins. Finally, there is marked proliferation of cartilage at the joint margins (lipping). The majority of these changes have been observed in autopsied TMJ tissues from elderly individuals.[22]

When there are degenerative changes on the articular surface, new tissue is deposited to compensate for the loss of bearing area in the middle portion. As degenerative changes progress, continuous deposition of newly formed connective tissue on the circumferential area finally leads to deformation of the condyle. This compromised TMJ is now susceptible to local etiologic factors which can perpetuate the degeneration. Mechanical overloading of the TMJ tissue is thought to be the major contributing factor.[23]

Experimental animal studies have shown that mechanical loads are vital for maintaining normal growth, morphology, and function of secondary cartilage within the TMJ. Decreased loads on the TMJ condylar cartilage reduce cell proliferation and matrix synthesis[24,25] resulting in a small condyle with a thin cartilaginous articular covering.[26,27] There is also a drop in enzyme activity associated with bone mineralization within the hypertrophic layer of the condylar cartilage, as well as a reduction in the amount of subcondylar trabecular bone.[28,29] A return to normal loading usually reverses all of these changes.[27,28] In vitro

studies confirm that normal mechanical loading stimulates cell division, matrix synthesis, and enzyme activity in the tissues of the TMJ.[25,30]

With age, TMJ tissues lose most of their ability to adapt to changing functional loads. This is thought to play a major role in age-related TMJ degeneration.[31,32] Changes in functional load in the aged are usually due to loss of posterior tooth support and dietary alterations (softer diets). Animal studies have shown that a soft diet contributes to a reduction in proliferation and maturation of articular cartilage and porosity in the subcondylar trabecular bone.[15] This finding is critical because senescent animals lack the ability to reverse these degenerative changes even after the restoration of normal functional loads.[15,27,28,33] Clinically, many elderly patients with a history of TMJ problems remain symptomatic even after their dentitions have been restored.[2] Therefore, a fully functioning dentition throughout life is more than a dentist's ideal — it is necessary for a quality life in later years.

Dentate Individuals

The relationship between the occlusal state and the development of functional disorders is unclear and controversial. Some studies report that the highest incidence of subjective symptoms occurs in those patients who possess the largest numbers of occluding teeth,[34] while other studies report that a decrease in the number of occluding teeth results in a higher degree of mandibular dysfunction.[35–37] There is documentation that adequate oral function can be maintained in shortened dental arches provided they include at least four posterior occlusal contacts.[38] Still other studies have demonstrated no relationship between the number of remaining teeth and the frequency of signs or symptoms of mandibular dysfunction.[39,40]

Occlusion as an etiologic factor in mandibular dysfunction and TMJ sound production has been investigated. Histopathologic studies on rats and human cadavers suggest a correlation between loss of posterior teeth and histologic changes in the TMJ, perhaps due to changes in functional loading.[41–43] TMJ degeneration and disorders have been reported after loss of posterior teeth,[44,45] and the frequency of TMJ clicking and pain are significantly higher in patients with missing posterior teeth compared to patients with full dentitions.[36,46,47] The clicking and pain occur much more frequently on the side of the missing teeth than on the side supported by dentition.

Two mechanisms have been proposed to explain why TMJ clicks increase with decreasing tooth support. One proposes that with loss of posterior support, the condyle has increased mobility in cranial and radial directions. This mobility allows increased contraction of supporting musculature, causing a peak loading point of friction abnormal to the joint (excess pressure) and resulting in a decreased spreading or production of synovial fluid. This frictional sticking is then released as audible energy (the click), and the joint resumes normal function on the downward slope of the loading point.[48] A second theory suggests that clicking is caused by anterior displacement of the disc and posterior displacement of the condyle: clicking occurs when the condyle snaps over the posterior band of the disc.[49,50] It is unknown whether loss of posterior teeth causes anterior disc displacement and posterior condylar displacement because of overclosure of the joint on the side of the missing teeth.[51]

Another point to consider, especially in elderly patients, is posterior tooth wear. This wear may take the form of parafunction (bruxism), abrasion (diet or oral-habit related), or occlusal breakdown (caries). As the posterior teeth wear, the anterior teeth develop facets as a result of tooth-to-tooth contact (attrition) and become locked against each other. The uneven distribution of load and incisal interferences caused by posterior tooth wear and attrition of the anterior teeth may be factors which are responsible for the development of dysfunction. This may help to explain the clinical success of bite-plane types of appliances in these cases. The objectives of the appliances (elimination of occlusal interferences, repositioning of the mandible into a stable intercuspal position, and increasing the vertical dimension of occlusion) may be secondary in importance to the fact that the appliances disocclude the anterior teeth and restore posterior tooth contact.

Edentulous Individuals

A common belief among dentists is that complete-denture wearers do not suffer from TMJ dysfunction. There are several reasons why this misconception persists. One is that denture wearers are usually adapted to an impaired oral function and will accept mandibular dysfunction without complaint. Secondly, most practitioners do not perform a functional examination of the masticatory system for these patients, thus many symptoms of TMJ dysfunction remain concealed. Finally, there

are very few studies concerning mandibular dysfunction in complete-denture wearers. However, several epidemiologic investigations have demonstrated the existence of TMJ dysfunction in complete-denture wearers.[52–56]

Approximately 15% to 20% of complete-denture wearers have symptoms consistent with functional disorders of the masticatory system.[36,57–59] These symptoms most commonly involve but are not limited to (1) difficulty or pain during mandibular movements, (2) pain in the TMJ region, (3) deviation of the mandible during opening, and (4) tenderness of the masticatory muscles to palpation. It was thought that the most common causes of this symptomatology were incorrect vertical dimension and centric relation.[54,58,60] Recently, however, it has been suggested that posterior occlusal wear producing incisal interference may be an etiologic factor.[61] This hypothesis is based on the fact that a majority of complete-denture wearers (particularly elderly patients who have been wearing their dentures for a long time) exhibit a markedly reduced vertical height of occlusion as a result of denture tooth wear and alveolar bone loss. For most of these patients, loss of vertical dimension (and subsequent "abnormal" repositioning of the condyle in the TMJ) does not have a noticeable effect on their masticatory apparatus. These individuals are comfortable and functional.[62] Perhaps the lower limit of what is considered normal for a vertical dimension of occlusion should be reassessed.

Experienced clinicians treating complete-denture wearers who suffer from TMJ disorders know that wear of acrylic teeth and alveolar resorption are predictable events in these individuals. Wear is concentrated on posterior teeth because of food abrasion. These practitioners realize a great measure of success with the addition of self-curing acrylic resin to restore posterior tooth contact.[63]

The issue of parafunctional activities and their sequellae is very important for elderly persons wearing complete dentures. The etiology of parafunctions is most likely multifactorial, and many different therapeutic mechanisms have been advocated.[64–66] It has been demonstrated that complete-denture wearers most commonly exhibit clenching as a form of parafunction and that there is a direct correlation between the frequency and duration of clenching and the frequency and severity of headache, degenerative changes in the components of the TMJ, and mandibular dysfunction.[67–69] It is postulated that old, poorly fitting, and unstable dentures are the main cause for clenching in these patients. It is obvious in the aged patient (in whom many of the components of the craniomandibular apparatus may already by compromised) that the functional chain is only as strong as its weakest link. When the limit of tolerance for one or more of the components has been exceeded, the parafunction may cause asymmetric muscle hypertrophy, tooth wear, damage to the denture base, damage to supporting tissues, TMJ pain, headache, mandibular dysfunction, or any combination of these signs and symptoms, depending on which components have the least resistance.

The issue of compromised normal masticatory function is also important for complete-denture wearers. Many studies have shown that chewing ability and efficiency are reduced in these patients even when properly constructed prostheses are worn.[70–73] Chewing ability and efficiency were correlated with mandibular dysfunction. The greater the number of symptoms of mandibular dysfunction, the greater the reduction in these parameters. These parameters were also correlated with many single symptoms of dysfunction, such as unilateral chewing and clenching. Of even greater significance is the fact that 50% of those patients with reduced chewing ability and efficiency had impaired general health (most likely due to the exclusion of essential foods from their diets). It is obvious that unacceptable dentures and impaired general health can interact, and, through excessive parafunctional activity, initiate various symptoms of CMD when physiologic limits are exceeded.

There are conflicting views regarding the most appropriate ways of correcting these problems. Some practitioners feel that simply inserting a new pair of dentures will not significantly improve masticatory function in these patients.[74] They advocate replacing the dentures with fixed partial restorations resting on osseointegrated oral implants (at least in one arch). Others, however, believe that insertion of meticulously constructed new dentures accompanied by proper patient education can reduce or eliminate clinical signs of CMD, eg, headache, muscle pain, and mandibular parafunction.[67,75]

Those edentulous individuals who do not wear dentures display an extremely low incidence (approximately 5%) of temporomandibular dysfunction (TMD).[61] This is somewhat surprising because it is found that most of these patients have well-preserved alveolar ridges and actually masticate by achieving opposing alveolar ridge contact. They chew most foods reasonably well but function at an extremely reduced vertical dimension. Radiographic examination has shown that the

condyle is posteriorly repositioned during this masticatory activity. Those patients that do exhibit signs and symptoms of TMD usually never progress beyond mild discomfort.

Malignant Disease

The importance of obtaining a thorough medical history and comprehensive physical examination as well as adequate TMJ radiographs/imaging cannot be overemphasized. For elderly patients presenting with symptoms of CMD, this is the most significant part of the data collection phase. Because of the complex symptoms and few overt abnormal physical signs, diagnosing CMD is challenging. On occasion, a patient diagnosed with CMD may actually have a rapidly progressing malignant process.

The various malignant tumors with symptoms that may mimic CMD can arise from three sites: (1) tissue intrinsic to the joint (bone, cartilage, and synovia), (2) direct extension of neighboring tissues (parotid gland, skin, and nerve), and (3) metastases. It has been estimated that fewer than 1% of all tumors metastasize to the maxillofacial area.[76] However, the true incidence of metastases could be significantly higher.[77,78] It is probable that many metastatic conditions are missed because of infrequent radiologic search for disseminated disease in the craniomandibular area, exclusion of the mandible in postmortem examinations, and poor radiographic assessment of disease. Most metastases to the mandible are found in the region of the molars and premolars. Adenocarcinoma is the most common of all metastatic tumors in the jaws, comprising 70% of these abnormalities, and usually originate in the breast, kidney, or lung.[78–80] The rarity of condylar metastases is probably due to the isolated nature of its blood supply and small amount of red marrow (limited to the third molar region, where most metastases are usually found).[81–83]

In approximately 50% of reported cases of metastases involving the condyle, the presenting symptoms were TMJ related.[84] There are several ominous signs and symptoms that should alert the clinician to consider malignant disease in the differential diagnosis: These include (1) unexplained swelling, (2) paresthesia and paresis, (3) abnormalities related to the eighth cranial nerve (auditory changes, tinnitus, vertigo), (4) trismus (rapidly occurring), (5) eustachian tube involvement, (6) sudden occlusal changes, (7) pathologic fracture (condyle), (8) a known malignant lesion elsewhere in the body, and (9) sudden onset of TMJ problems in an elderly patient who was previously asymptomatic.[85,86]

Neoplastic lesions occurring in the condyle are most often either benign tumors or local primary malignancies. Osseous and cartilaginous tumors are the most common benign neoplasms to arise in the condylar area. Malignant tumors involving the TMJ are usually a result of direct extension from neoplasms of the skin, parotid, ear, or nasopharynx.[84] If there is any doubt as to the possible contribution of a neoplasm to the patient presentation, more extensive evaluation and specialist referral should be considered.

Initiation of therapy directed at specific causes of CMD in the elderly is indicated only after a comprehensive workup has excluded all other possible causes of dysfunction. A patient initially diagnosed with CMD whose symptoms progressively worsen despite appropriate therapy should be reassessed. It is mandatory that clinicians systematically evaluate the success of their treatment. This allows earlier diagnosis and treatment, thus improving the prognosis.

Infection

Infection should be considered in the differential diagnosis of CMD in the geriatric patient. This patient population is at an increased risk for infectious processes because of compromises in general health (debilitation, malnutrition, impaired immune system, decreased healing capacity, inadequate inflammatory response, etc). Organisms may enter the TMJ via a penetrating wound; ruptured capsule; direct invasion from an adjacent infection in the middle ear, parotid gland, or tooth; and hematogenous spread from a distant source. Patients with concurrent illnesses like diabetes, systemic lupus, enteritis, and immunosupressed states are at particular risk.[87,88]

Rheumatoid patients most commonly present with a septic arthritis of the TMJ as a complication of their systemic condition (they have an impaired immune response in addition to damaged joints). Any microorganism can cause the condition, but most cases (95%) show Neisseria gonorrhoeae and Staphylococcus aureus as the infecting organisms.[89,90] Clinical features include an extremely painful, red, hot, swollen joint which is tender and slightly fluctuant. There is usually an associated limitation of movement, fever, and leukocytosis. Early diagnosis and treatment are very important since the condition has a significant

mortality.[91] Furthermore, left untreated, the resultant destroyed, fibrotic, or fused TMJ can leave the patient with devastating morbidity.

If septic arthritis is suspected, a detailed examination must be carried out to exclude both local and distant sources of infection. Aspiration and examination of TMJ fluid may prove useful for definitive bacterial culture. There is unanimous agreement that systemic antibiotics should be given as soon as a provisional diagnosis is made. In cases that fail to respond rapidly, drastic intervention is indicated (surgical drainage or irrigation with antibiotics or excision of the condyle)[92,93] because of the proximity of the TMJ to the temporal lobe and potential intracranial spread of infection.

Otitis media (an infection of the middle ear) is a condition frequently seen in aged, diabetic patients. Often the infection will spread to involve the TMJ. Anatomically, the anterior bony canal wall is related to the joint (the joint capsule is attached to the squamotympanic suture). Congenital dehiscences of the cartilaginous canal (fissures of Santorini) in this suture may account for the spread of infection into the TMJ.[94] The most common pathogen involved in this infectious process is Pseudomonas aeruginosa.[95] Clinically, features of this condition typically include preauricular swelling, TMJ tenderness, and painful mandibular movement.[96]

The practitioner must be careful not to confuse TMJ infection with trismus. Trismus from inferior alveolar or posterior superior alveolar blocks is not uncommon. However, it is usually immediate in onset and self-limiting. Reflex trismus from hemorrhage or trauma to the medial pterygoid muscle should abate quickly. In the case of infection, the onset of pain and the limitation of mandibular movement are usually more progressive. Computerized tomography is a helpful diagnostic aid in the evaluation of infection.[10] It can help to define the anatomic extent of the pathosis because it will illustrate diffuse inflammation of muscles and obliteration of normal tissue planes.[96]

Psoriasis

Various types of arthritis may be found in patients with psoriasis, a fairly common, chronic, erythematous, scaling lesion of the skin. Psoriasis tends to appear over the elbows, knees, and scalp but the silver-scaled lesions may cover any cutaneous surface. The nails of the patient may show changes such as pitting, hyperkeratosis, brownish discoloration, or total destruction. It is common to see a sausage-like swelling of the fingers and toes. The cause of psoriasis is generally not known, but a hereditary predisposition is suspected.[97]

Psoriasis is a chronic, often puritic, skin disease. It is generally considered a "minor" disease limited to the skin. It may, however, progress to more severe forms that become systemic in nature (more often in men than in women).[97] The disease may affect the quality of life through psychosocial stress factors. It may be associated with arthritis,[98] evoke muscular hyperactivity in the masticatory system,[99] and directly affect the components of the TMJ[100] when it becomes a systemic symptom complex. At this stage, the presentation of CMD (headache, facial pain, pain in the TMJ, arthritic involvement of the TMJ, occlusal parafunction, and impaired chewing ability) become part of the presentation of poor general health.[101,102]

When the TMJ in these patients becomes involved in an arthritic process, it is difficult to distinguish between various forms of arthritis (rheumatoid, osteoarthritis, etc). Radiographic findings are not specific: erosion of the condylar surface, flattening of the condyle, and proliferative changes.[100] Patients have symptoms similar to those of rheumatoid arthritis of the TMJ, but the pain is almost always unilateral. Diagnosis is based on the arthritis occurring in a patient who has psoriasis and a negative rheumatoid factor. Successful management has been achieved using conservative modalities (ultrasound, physical therapy, exercise, and salicylates).[102]

Rheumatoid Arthritis

The cause of rheumatoid arthritis (RA) is unknown, but it is generally accepted that it is of multifactorial etiology (most likely an interplay between infectious, genetic, endocrine, and immune factors).[103] The highest incidence of RA occurs after age 60 and is much more common in women than in men. In some cases, emotional stress seems to predispose to the condition. A current theory proposes that a genetically predisposed host subjected to a foreign, offensive agent (infection or trauma) undergoes unique immunologic changes that result in a perpetuating inflammatory process within the joints. Abnormal antibodies are eventually produced that, in turn, trigger the production of other antibodies (rheumatoid factors) to destroy them. The complexes of these "self" and "nonself" antibodies are deposited in the joints, where they act as chronic immuno-

genic/inflammatory stimuli (eg, in the activation of complement cascade, attraction of polymorphonuclear leukocytes which release hydrolytic enzymes and mediators of inflammation, and creation of oxygen free-radicals).[104] The end result is the classic chronic rheumatoid granulomatous response of pannus, which leads to destructive changes in the joint. Successful management of the condition lies in a comprehensive, multidisplinary approach. The comprehensive treatment plan should include systemically administered drugs (anti-inflammatory or antirheumatic), local injections of corticosteriods, physical therapy, occupational therapy, psychosocial support, patient education, family involvement, surgical intervention, and vocational rehabilitation.[105]

Involvement of the TMJ in RA results from granulomatous involvement of the articular surface of the synovial membrane, leading to destruction of the underlying bone. Most patients with RA will have TMJ involvement during the course of their disease[106]; common symptoms include stiffness, crepitus, tenderness, swelling, masticatory pain, mandibular hypomobility, and occasional mandibular fixation. Radiographic findings usually demonstrate narrowed joint spaces, flattened condyles, erosions, subchondral cysts, and osteoporosis.[107] Recently, it has been shown that pain and tenderness in the TMJ and associated masticatory muscles in RA is correlated with hypothermia of the TMJ.[108] It is postulated that this decrease in intra-articular and skin surface temperature of the TMJ area in RA is due to decreases in local blood circulation (a result of disuse atrophy, which can be a sequela to local damage and muscle pain, or vasoconstriction caused by sympathetic nerve activation in the area of pain).

Of particular concern to the practitioner is the high incidence (70%) of upper airway obstruction in patients with severe arthritic destruction of the TMJ.[109] The cause of such obstruction is believed to be destruction of the condylar head and neck which severely reduces ramus height. This leads to "recession of the chin" and retraction of the tongue (especially during sleep). Thus, the patient with TMJ destruction seems to live under the constant threat of upper airway obstruction. It is advocated that these patients be given nighttime occlusal protheses similar to those that are used to alleviate the same problem in children with hypertrophic adenoids or in comatose individuals.

In general, treatment depends on the severity of the signs and symptoms observed. Because patients with RA rarely have involvement of the TMJ alone, they are usually given anti-inflammatory drugs which effectively treat all involved joints. During acute exacerbations, the patient should be restricted to a soft diet. (In the past, intermaxillary fixation was used, but this should be avoided because of the risk of fibrous ankylosis.)[110] Intra-articular steroids have been used successfully to relieve severe pain but should be used sparingly (localized osteoporosis and articular erosion may result). Surgical intervention should only be considered for those patients who do not respond to conservative therapy or who have drastic loss of function. Recently, a physical training program was developed for the stomatognathic system, and it appears to have positive results.[111] The program improves mandibular mobility and decreases clinical dysfunction in these patients.

Osteoarthritis

Osteoarthritis (OA) is the most prevalent type of arthritis: almost all people over 60 years of age display at least radiographic evidence of the disease. It is also known as degenerative joint disease or DJD. Unlike RA, it is generally accepted that OA is of a noninflammatory nature.[112] Although the incidence of OA increases with age, it is not a natural consequence of aging. OA is classified as either primary or secondary. Primary OA shows no apparent predisposing factors (tissue breakdown is assumed to be caused by undefined intrinsic factors), while secondary OA has an underlying cause (trauma, surgery, or previous infection).

The pathogenesis of OA has not been clearly defined. There may be genetic predisposition determined by a single autosomal gene (dominant in women and recessive in men), and obesity may lead to development of the condition. The development of OA is probably best considered as a physiologic imbalance between the amount of stress applied to a joint and the ability of the joint tissue to deal with that stress.[113] Clinical findings in OA include the classic dull ache of the involved joint which worsens after use, pain at rest or at night in involved joints, morning stiffness and swelling which are not prolonged, limitation of motion secondary to chronic muscle spasm, capsular fibrosis, intra-articular loose bodies, large osteophytes, accumulation of synovial fluid that limits joint movement, and bony enlargement of the joint.[114] The radiograph is the most useful diagnostic tool available. Typically, it demonstrates narrowing of joint spaces, new bone (spur) formation, and bony sclerosis.

Osteoarthritis is the most common intracapsular disorder of the TMJ. However, only a small number of these patients complain of symptoms.[115] Usually, patients with generalized OA will exhibit degenerative changes in the TMJ. Major symptoms of OA of the TMJ include unilateral pain directly over the condyle, mandibular hypomobility, crepitus, stiffness (especially after periods of inactivity), and deviation of the mandible to the affected side. Radiographic findings include loss of lamina dura from the condyle, narrowing/irregularities of joint spaces, flattening of the articular surface, osteophyte formation, marginal lipping, and Ely's cysts.[116] Management is conservative in most cases (for up to 1 year before surgery is considered). This includes a soft diet, treatment of secondary myofascial pain dysfunction syndrome, and use of anti-inflammatory drugs. In cases where ankylosis has occurred or where there is no response to conservative therapy, surgical intervention is indicated. Shaving of the condyle may help relieve the symptoms, and, in severe cases, a condylotomy may be performed.[117]

Pseudogout

Pseudogout is a crystal-induced disease characterized by acute arthritis that mimics gout caused by urate crystals (uric acid). It has been referred to as chondrocalcinosis, pyrophosphate arthropathy, and calcium pyrophosphate crystal disease. Chronic joint inflammation can develop, mimicking rheumatoid arthritis or osteoarthritis. There seems to be an increased incidence of this condition in Filipinos, Chinese, and Japanese populations.[118]

The deposition of crystalline calcium pyrophosphate dihydrate in articular cartilage is a common and predominantly age-related phenomenon. These deposits are found most frequently in intra-articular fibrocartilage (usually in the knee or wrist).[118] The TMJ is anatomically unique in that fibrocartilage rather than hyaline cartilage predominates (it also contains a fibrocartilaginous meniscus). Thus, it is not surprising that it develops pseudogout (chondrocalcinosis).[119–122]

It is very likely that in the aged population, pseudogout of the TMJ is underdiagnosed. The clinical assessment of facial pain arising from the TMJ is difficult. Most cases are attributed to "pain dysfunction syndrome," in which the diagnosis is made by exclusion because there are no identifiable pathologic features. Characteristically, this syndrome affects younger age groups, but in the elderly this diagnosis may be mistakenly made for pseudogout.[123] An awareness of the fact that calcium pyrophosphate deposition may involve the TMJ should help the clinician with the differential diagnosis of acute facial pain in the geriatric patient. The diagnosis is confirmed by aspirating calcium pryrophosphate crystals from the synovial fluid of the TMJ.

Temporal Arteritis and Polymyalgia Rheumatica

Temporal arteritis and polymyalgia rheumatica are common rheumatic diseases of the elderly. Temporal arteritis is a systemic granulomatous arteritis which predominantly affects branches of the carotid artery after the age of 50. It is often referred to as giant cell arteritis.[124] Polymyalgia rheumatica is a clinical syndrome in patients over 50 years of age and is characterized by aching or morning stiffness in muscles usually associated with an elevated erythrocyte sedimentation rate. The diagnosis is made by exclusion.[124] Some groups believe that arteritis and polymyalgia rheumatica are separate but overlapping conditions because approximately 50% of patients with temporal arteritis have symptoms of polymyalgia rheumatica.[125] Other groups, however, believe that they are merely different manifestations of the same underlying systemic disease, which they refer to as a granulomatous vasculitis.[126] It has been shown that 50% of the patients who present with polymyalgia rheumatica will develop temporal arteritis within 1 year.[125]

The incidence of these diseases clearly increases with age (it is 30 times higher in the eighth decade than in the fifth decade).[127,128] The diseases are more common in colder climates (northern latitudes), and women are affected more often than men (by a factor of 5).[129,130] It is interesting to note that these diseases show a preference for Caucasian populations. Histopathologically, temporal arteritis is an inflammatory reaction of the vasculature consisting of lymphocytes, plasma cells, macrophages, eosinophils, and giant cells. The vascular intima proliferates, while the media exhibits smooth-muscle necrosis and interruption of the elastic membrane. Polymyalgia rheumatica does not demonstrate any of these inflammatory findings.[131] However, this entity is associated with very high levels of circulating immune complexes.[132]

Clinically, symptoms of temporal arteritis are predominantly associated with headache and facial pain. Patients with polymyalgia rheumatica com-

plain of aching pains, muscle stiffness or soreness (face, neck, shoulders, and buttocks), and decreased range of motion in the neck and shoulders. These symptoms are generally symmetrical and usually worse in the morning or after activity.[124] Constitutional symptoms such as fever, anorexia, weight loss, fatigue, and malaise may accompany these conditions.[133,134]

These two disease entities may present as CMD with no other associated signs or symptoms. The clinician may provide an erroneous diagnosis (usually neuralgia, tension headache, or myofascial pain dysfunction syndrome [MPD]). Headache is the most common symptom of temporal arteritis. It may be nonspecific, resembling a muscle tension headache, or it may be more localized, with pain reported in one or both temporal areas; the forehead; or occiput. Discomfort is usually described as superficial or burning, with paroxysms of lancinating pain, and can be associated with extreme scalp tenderness. There may also be varying degrees of facial pain and ocular pain with accompanying visual disturbances.[135] Polymyalgia rheumatica classically presents with pain or claudication of masticatory muscles, pharyngeal muscles, and the tongue. Pain or fatigue are brought on by activities such as chewing, swallowing, or talking. Symptoms can usually be relieved by rest. Pain in the throat or neck can be accompanied by a cough or by hoarseness, and it is not uncommon for the ears to become involved, with decreased acuity, vertigo, or ear pain.[136]

Temporal arteritis and polymyalgia rheumatica should be suspected in any patient beyond 50 years of age with new headache, sudden or transient visual disturbances, unexplained fever or weight loss, neurologic symptoms, or claudication. The clinician should check the temporal arteries (classically, upon palpation, they are swollen, tender, nodular, and exhibit decreased pulsations). The next step is to obtain an ESR and evaluate it for elevation. These conditions have been treated successfully with long-term corticosteroid therapy (temporal arteritis requires much higher doses than polymyalgia rheumatica).[137,138]

Synovial Chondromatosis

Synovial chondromatosis is a benign condition of unknown etiology which affects articular joints (usually the knee, ankle, and hip). Foci of cartilage develop through metaplasia in the underlying connective tissue of the synovial membrane. These foci enlarge and form cartilage fragments which detach from the affected synovium and are released into the joint space. The fragments then undergo calcification and are called "loose bodies."[139] The condition is also known as synovial metaplasia, articular chondrosis, and osteochondromatosis. Malignant transformation can occur but is considered to be very rare.[140]

This disease is more common in middle-aged and elderly adults, especially men.[141,142] However, the condition can affect the TMJ, and in these cases the disease is more common in women, predominantly on the right side.[143] When the TMJ is involved, there is often a preauricular swelling accompanied by pain and limitation of joint movements. There is often a marked crepitus and deviation to the affected side. Radiographically, multiple radiopaque bodies may be observed surrounding the TMJ or in the TMJ space. These loose bodies are usually of variable size.[144] In addition, sclerosis of the glenoid fossa and mandibular condyle as well as enlargement of the joint space and an irregular joint surface may be observed.

The histologic feature of synovial chondromatosis of the TMJ mimics a malignant mesenchymal tumor. Because the disease process is one of cellular multiplication rather than accretion areas of mitosis, binucleate forms, hyperchromatism, and pleomorphism all can occur.[145] The critical feature for confirming the diagnosis is the presence of chondrometaplasia.[146] Chondrometaplasia evolves through a process of intrasynovial metaplasia in the subintimal layer of the synovium. The chondrocytes initially receive their nourishment from this intrasynovial environment, but after breaking free as loose bodies they are able to receive nourishment from the synovial fluid independent of any blood supply.[147]

Clinical features of the disease resemble those associated with a slowly growing neoplasm. Practitioners have confused this condition with parotid tumors.[148] There is a progressive alteration of function accompanied by a subtle swelling and progressive pain, tenderness, and limitation. Palpation of the TMJ reveals a broad-based nonmovable swelling without fixation to the overlying skin. Onset of cracking and crepitation are variable features proportional to the amount of joint destruction and degree of calcification within the metaplastic tissues. Despite extension into soft tissues both medial and lateral to the TMJ, there have been no reports of fifth- or seventh-nerve dysfunction.[149]

Surgical intervention is the treatment of choice for this condition and is limited to excision of the involved tissues. This usually requires extirpation

of the disc and the synovial tissue. Great care must be taken during excision because any remaining synovium left behind will serve as a new source of metaplastic tissue and will lead to recurrence.[150]

Headache / Atypical Facial Pain

Approximately 75% of geriatric patients seeking primary health care complain of pain, and about 50% of those patients have no detectable organic basis for their complaints.[151,152] This fairly consistent finding reinforces the traditional tendency to categorize pain as either organic or psychogenic in origin. The ability to localize pain greatly facilitates the definition and often its etiology, while pain that is vague, poorly localized, or anatomically "atypical" creates uneasiness, frustration, and even annoyance in both patient and practitioner.

In the elderly population, the significance of the neck, face, and head (CMD apparatus) cannot be overemphasized relative to the meanings of pain (organic and psychogenic).[153] Society probably spends more money on and pays more attention to this area of the body than any other. It is the most exposed part of the body (besides the hands) and prominently advertises our identity. Further, in the head are situated four of the five sense organs, which permit us to navigate our way through the world; it also contains the primary "switching station" responsible for all communication inside and outside of the body (the brain).

The neck, face, and head possess emotional, cognitive, and communicative capacities, and have a very powerful influence on both the psyche and the soma, all of which underscore the disproportionate significance of this area in relation to the remainder of the body. Thus, the psychologic significance of pain in the CMD apparatus would be expected to carry much greater symbolic meaning than pain in other parts of the body.[153]

When dealing with the elderly population, the clinician must be cognizant of the impact of emotional status, living conditions, and life history on the overall presentation of these patients. There are a great variety of pain complaints — chronic dental pain, discomfort in structures of the oral cavity and oropharynx, TMJ syndrome, MPD, sinus problems, headaches, eye pain, various neuralgias, neck pain, ear pain, etc — and these may represent a meaningful part of some larger psychiatric syndrome.[154] For example, pain sometimes serves as an expression of guilt (as in the elderly individual who worries about being a financial or emotional burden on other family members), and pain in an exposed body part may serve an unconscious purpose (eg, wincing or contorting from pain in the face can evoke a negative response from others that may serve as punishment). Pain in the neck, face, and head may also evolve from intense anxieties over control (loss of bladder control, financial independence, and social interactions), sanity (deficits in memory, communication skills, and comprehension), and appearance (loss of youth and appearance of age-related deformities). These pains may also serve as expressions of grief (loss of loved ones), depression (loneliness), and emotional frustration (loss of sexual activity).

Research has demonstrated the linkages between guilt, grief, emotional, and depressive "stress syndromes" to neuroendocrine and immunologic status.[155,156] There have been reductions of serum serotonin levels in these conditions as well as alterations in other neurotransmitters/neuropeptides in cerebral-spinal fluid. In recent years, the hypothesis that many atypical facial pains are related to biochemical imbalances caused by depressive illnesses has gained acceptance, especially with the demonstrated response of many symptoms to psychopharmacologic intervention.[157] It has also been shown that various forms of facial causalgia (cutaneous hyperesthesias) are manifestations of delusions or hallucinations directly related to some types of organic brain syndromes.[158]

Some interesting recent studies have been completed concerning headaches in the elderly.[159–163] It is generally accepted that there is a decreased frequency of headache among these individuals.[159,160] Those headaches that do occur, however, predominantly affect women.[161–163] This was attributed to such factors as neurotic disorders coincident with menopause. In both men and women, a positive cardiovascular history, physical changes such as cold and barometric pressure, diets high in protein and glucides, and sleep disorders were all implicated as significant contributing factors in the development of headaches.[164–167] The most interesting findings of these studies are the implication of tyramine in many types of headaches,[164] a possible common origin for headaches and sleep disorders in the aged (probably associated with an alteration in serotonin metabolism),[168] and a very high correlation between dissatisfaction with living conditions (poor housing, depression, and permanent stress) and the development of psychogenic and organic headaches.

The clinician must work with the psychiatrist in the diagnosis and management of headache and atypical facial pain in the geriatric patient. The

psychiatrist can encourage an ongoing practitioner-patient relationship in which adequate time can be provided to compile a personal history, medical history, and psychologic history, as well as to determine any relationship between pain onset and critical life events, and the contribution of depressive states to somatization. Psychiatrists can also provide conservative and effective treatment through interest, support, pharmacologic intervention, and the placebo effect of communicative interaction. For example, pain related to grief reactions will often subside in time with the interest and support of an empathetic caregiver. Depression and its accompanying pain will often respond to tricyclic antidepressants, while delusional psychoses often respond to neuroleptics (phenothiazines or haloperidol). Psychotropic drugs, even in the absence of clear-cut signs of depression or anxiety, have been found to be effective in reducing pain, especially in the context of a positive practitioner-patient relationship.[153] Thus, the psychiatrist can provide diagnostic, therapeutic, and management recommendations and, more importantly, can help the practitioner to become aware of his or her own susceptibility to frustration and sense of failure with these patients. This will help to preserve the practitioner's emotional peace of mind and protect suffering patients from needless and ineffective procedures.

The nature of the human condition determines that self, self-image, self-esteem, and emotional status have a disproportionate focus in those structures of the body that are most visible and communicative to others (the neck, face, and head). It is not surprising, therefore, that patients with or without evidence of anatomic or neurologic lesions often present with an astounding array of pains localized in these areas. Attempts to distinguish subjective pain referable to these structures as either organic or psychogenic puts patients at risk for incorrect and damaging treatment. The tunnel vision of specialists who may be inclined to perceive a patient's complaint of pain as specifically related to their area of expertise can result in an inadequate evaluation. Thus, patients whose vague pains sometimes resemble pains of dental, sinus, TMJ, or neural origin may be identified as having atypical pain only after various or traumatic and expensive dental procedures have been completed which may provide an additional basis for pain.

In addition to the usual careful physical examination, it is essential to take a thorough personal, social, medical, and psychologic history (including a history of medication use and autobiographic description of the pain). Asking the correct questions about vegetative symptoms (such as anorexia, constipation, and insomnia) and precipitating life events, suicidal thoughts, loss of interest, and decreased sexual activity can often reveal a depressive illness as a possible basis for pain in the neck, face, and head. In connection with prolonged grief and depression, it is not uncommon for pain to begin at the time of or on the anniversary of important losses, tragedies, and changes in one's life. Painful symptoms will sometimes replicate the precise symptoms experienced by a loved one during a fatal illness. It should also be kept in mind that burning sensations of the oral cavity are a very frequent symptom of depression, especially in the elderly.[169]

Conclusion

In evaluating the geriatric patient who has presented with signs or symptoms of CMD, it is imperative that the practitioner consider the following when formulating the differential diagnosis: (1) Normal age-related changes in the craniomandibular apparatus and their impact on normal function as well as on responses to stress; (2) the implications of the status of the dentition and dental prostheses to CMD; and (3) the contribution of malignant diseases, psoriasis, arthritic conditions, pseudogout, granulomatous vascular conditions, and metaplastic involvement of tissues to the pathosis of CMD. The clinician must also be aware of the various implications of the psychologic, sociologic, and biologic aspects of aging to the development of headache and atypical facial pain as components of CMD in the geriatric patient.

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Resumen

Desórdenes craneomandibulares en el paciente geriátrico

Este artículo presenta una revisión general de los cambios básicos relacionados a la edad, y que ocurren en el sistema craneomandibular del paciente geriátrico; también se revisan las condiciones que están frecuentemente asociadas a los desórdenes craneomandibulares en los ancianos. La evaluación de los pacientes geriátricos con signos/sintomas de desórdenes craneomandibulares debe incluir; (1) cambios normales relacionados a la edad, en el sistema craneomandibular y su impacto en cuanto a la función normal y las respuestas al stress; (2) el estado de la dentadura y las prótesis dentales en relación a los desórdenes craneomandibulares; y (3) la contribución de las enfermedades malignas, psoriasis, problemas artríticos, pseudogota, problemas de tipo vascular granulomatoso, y los compromisos metaplásicos del tejido a las patosis de los desórdenes craneomandibulares. El clínico también debe estar consciente de los diversos efectos de los aspectos psicológicos, sociológicos, y biológicos del envejecimiento; sobre el desarrollo de la cefalea y los dolores faciales atípicos, como componentes de los desórdenes craneomandibulares en el paciente geriátrico.

Zusammenfassung

Kraniomandibulare Störungen in geriatrischen Patienten

Diese Abhandlung is ein allgemeiner Überblick über grundlegende altersbedingte Veränderungen, welche im kraniomandibularen Apparat vorgehen, und die häufigst erscheinenden Zustände, die mit kraniomandibularen Störungen in geriatrischen Patienten verbunden sind. Die Abschätzung geriatrischer Patienten, welche Zeichen/Symptome kraniomandibularer Störungen zeigen, muss in Betracht nehmen: (1) normale altersbedingte Veränderungen im kraniomandibularen Apparat und deren Einwirkung auf normale Funktionen sowie auch ihre Reaktion zu Belastungen; (2) die Rolle der Dentitionslage und dentalen Prothesen in kraniomandibularen Störungen; und (3) die Mitwirkung bösartiger Krankheiten, Schuppenflechten, arthritische Zustände, Pseudogicht, granulomatose vaskulare Zustände, und metaplastische Verwicklung von Geweben mit der Pathologie der kraniomandibularen Störung. Der klinische Therapeut muss sich auch bewusst sein hinsichtlich der verschiedenen psychologischen, soziologischen, und biologischen Aspekte des Alterns auf die Entwicklung von Kophschmerzen und untypischem Schmerz im Gesicht als Bestandteile der kraniomandibularen Störungen im geriatrischen Patient.