Temporomandibular Joint and Occlusion

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The orthodontic concept of occlusion is bound by the same physiologic parameters and facts that bind all segments of dentistry. However, the orthodontist, per se, may have opinions which seem to be fragmented from the generally accepted knowledge of occlusion that exists in periodontics, restorative dentistry and prosthetics. This divergence is very likely spawned by the changes created or noted in the development of our growing patients. The majority of orthodontic patients are in a vital growing age period. The presence of deciduous teeth with possible mandibular parafunctions such as mandibular thrusting and bruxism, the irregular loss of deciduous teeth and the functional changes associated with supraeruption, deflection of permanent teeth and probable premature contacts are ever changing features frequently noted in the growing child. Often these near pathologies of occlusion create no apparent symptoms or disturbance. We rarely note those rude sounds of crepitus or clicking in a child's temporomandibular joint despite the presence of the mechanical and functional agents that in the adult are responsible for these pathognomic disturbances.

The existence of severe malocclusion, either dental or structural, in the growing child does not create functional problems as frequently or severely as the same occlusions may produce in adults. This observation on the part of the pedodontist and orthodontist has perhaps aided in their concept of occlusion which is accepted and practiced by these two divisions of children's dentistry. We acknowledge that the growing child has a developing muscle, skeletal and nervous system which is labile and very adaptive. We place stainless steel crowns on teeth for space maintenance and note mandibular shift, tooth depression and trauma without the occurrence of temporomandibular joint disturbances. We move teeth into extraction sites, alter the vertical dimension of the denture, and pit inclined planes against cusps in the most traumatic fashion, and only in a certain few instances do we see immediate evidence of functional sequelae. Thus, is it not conceivable that the orthodontist and pedodontist are more difficult to impress with the consequences of functional occlusion concepts that are accepted by other dental practitioners?

Recently, at a specialty meeting, I presented a paper on the pathophysiology of occlusion as noted in an orthodontic practice. At its conclusion one prominent orthodontic educator stated that he had never seen an orthodontic patient before, during or after orthodontic treatment who had a "clicking" temporomandibular joint. A few weeks later he wrote and stated that since my paper he had seen two children with this problem and that he apparently had not been looking for what he had previously missed. I believe, regrettably, that this has been the orthodontic perspective. Many of us see the symptoms of occlusal dysfunction but without a patient's complaint of pain we ignore what we know is present—whether it be idiopathic or pathogenic.

Orthodontists are very closely associated with the improvement of oral health, esthetics and, hopefully, function in the young patient. Fortunately we are dealing with a system which expresses itself in a wide range of adaptability, probably due to the maturation of the nervous system at this

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age and certainly contingent upon the copious blood supply to this vital region.

Our concern with centric occlusion and centric relation revolves about the use of elastics, cervical traction and functional space maintainers, all mechanistic factors. We do not, of necessity, establish these positions as frequently nor as accurately as our colleagues who deal with the adult dentition. I personally believe that many orthodontically retained patients could be more stable if our assessment of the finished occlusion were more critical and we relied upon the parameters of judgment useful in periodontics, prosthetics and restorative dentistry. I would therefore emphasize that all of our cases must be evaluated dynamically as well as esthetically before, during and after orthodontic therapy.

Despite the seeming peripheral relation of orthodontics to the main stream of dental occlusion concepts, we should be consciously and conspicuously engaged in a better understanding of the functioning dentition. For it is the orthodontist who establishes the basis of the child's occlusion with which that individual must function and hopefully live with until old age. Errors in our judgment or ability can easily jeopardize a child's future oral health and unfortunately commit him to the status of a dental cripple. At this juncture I cannot heap all the abuse upon my fellow orthodontists for we often see and treat, in our clinic temporomandibular joint program, adults who have been crippled by improper and injudicious dental procedures.

How can we, as dental practitioners in the main channel or in the side stream of a specialty, improve the quality of our care to our patients? I believe we must become more cognizant of the total functional features of the stomatognathic system. To treat a child's malocclusion solely for esthetic improvement relegates the orthodontist to existence purely as an oral beautician. For the dental practitioner to fill a single tooth without regard to its occlusal relation, functional contacts and physiologic position relegates that practitioner to the role of a pure technician or mechanic. We must become more informed and more appreciative of the functioning dentition both in the normal instances and the so-called abnormal or adapted situations.

Literature Review

A considerable mass of information has accumulated over the years relating to the human temporomandibular joints and their associated structures, nerve, muscle, teeth, tongue and mandible. The accumulation of material in all sciences has rendered the single scientist of today impotent to comprehend even the research findings of his intimate microcosm of interest. An illustration in point would be the exhaustive 1925 monograph of Muller[1] which was directly primarily toward European literature on mandibular movement. At that date he cited six hundred works by over three hundred authors. Now, fifty years later, we are totally baffled by the scope, content, diversity of method and conflict of conclusions in the literature. To adequately assess this mass of information (and misinformation) is totally beyond my ability, interest and intelligence. However, throughout time there have been a few studies and findings which seem valid and important to our discussion today.

In any research study the quality of the work is a reflection of the individual investigator's intelligence, project design and, to some degree, the sample size. Over the years some of the early works, which today are still cited, had a paucity of subjects but a potency for perpetuation.

Luce[2] employed only two subjects, Morey[3] three, Walker[4] "a large number," Ulrich[5] twelve, Campion[6] ten subjects, Bennett,[7] Zsigmondy,[8] Jeppener-Haltenhoff[9] used themselves, Thouren[10] employed five cases, and Sicher[11] a grown man. It should be cited that the latter study by Sicher was the first recorded evidence of a roentgenographic examination of the temporomandibular joint.

Hildebrand[12] did an extensive and masterful study of mandibular movement which no doubt provided germination material for the later works of others.[13-21] These later investigators employed cineradiographic, electromyographic, roentgenographic as well as peripheral and central nervous system electrophysiologic techniques. The sophistication of this armamentarium and the subsequent investigative conclusions still point to one salient and never contradicted fact: There is a general reproducibility of patterns for a specific group but there continues to exist a degree of variability in the intrasubject response. To put this into a more common context, all Class II, Division 1 malocclusions do not respond equally well to a common treatment rationale but require continual monitoring and control throughout therapy. Thus the response of the neuromuscular system, supporting tissues, joints, teeth and jaws have an infinite range of change to treatment or nontreatment from complete health and excellent function to extreme degeneration and dysfunction. What is the common denominator? Is there one or are there many? How best can we, as practitioners, observe, guide and control our treatment procedures to provide the maximum of functional health and stability to all of our patients? Which of our present health-function parameters, recognized today, indicate no treatment or treatment with a guarded prognosis? In this presentation I will not consider the implications of periodontic involvement, root resorption, tooth mobility, tooth devitalization, alveolar and cortical bone loss or impacted teeth, but concentrate upon the temporomandibular joints and their relation to the dental occlusion and, conversely, the relation of the dental occlusion to the temporomandibular joints.

Prenatal Development

The prenatal development of the human mandible and associated temporomandibular joints interested anatomists at a much earlier date than jaw movement had interested dentists. Serres[22] was first to report the two cartilaginous bars comprising the first branchial arch in the early fetus. Although he termed these structures as "maxillare inferieure temporaire," a more descriptive analysis was given by Meckel.[23] Thus the first branchial arch, that anlage of the mandible and the associated temporomandibular joints, became known as Meckel's cartilage. The final disposition of Meckel's cartilage was the subject of long controversy with several believing that all but the tympanic portion disappeared in early fetal development (Serres,[22] Meckel,[23] Magetot and Robin,[24] Strelzoff,[25] and Stieda[26]). Opposed to this were Reichert,[27], Callender,[28] and Kolliker[29] who believed that the anterior portion entered into formation of the mandibular ossification while the remainder disappeared.

Other views have existed and may be summarized as follows: 1) The mandible forms from five ossification centers, all of which fuse (Macalister[30]). 2) Cruveilhier[31] stated that each half has a main center of ossification but the angular, condyloid and coronoid develop independently to join the mandible later. 3) Bland and Sutton[32] postulated that each half ossifies from six centers which unite in late fetal development.

Certain highlights of human fetal mandibular development may be noteworthy.[33,34]

At approximately the 14 mm c.r. (6 wk.) stage there occurs a great increase in cellular activity in the ossicle—Meckel's cartilage area.

With the 17 mm c.r. (4 wk.) fetus we note differentiation to precartilage in the Meckel's and Reichert's cartilage zones. The mandibular symphysis is marked by a wide separation of the right and left Meckel's bars. Meckel's bar is one of the earliest sites of the body to show cartilage formation. Already a membranous ossification is occurring lateral to the mandibular symphysis. The mandible and clavicle are the earliest skeletal elements to be formed in bone. In the area of the mental foramen a classical pattern of membrane bone formation takes place. The connective tissue of the mesoderm has a preosseous change as osteoblasts form, which then produces an osteoid tissue followed by bone deposition. This original mental foramen ossification center extends along the lateral wall of Meckel's cartilage to form the anterior half of the outer and inner walls of the future alveolar trough. The foramen of the mental nerve at this stage is well-formed. From this point in time the mandible develops parallel but free from the parabolic arc of the two Meckel's bars. Meckel's cartilage has three main contributions other than a strut for mandibular development: 1) the distal portion becomes incorporated in the symphyseal area as the genio tubercles; 2) the middle portion gives rise to the sphenomandibular ligament and the fibrous periosteum which lines the mylohyoid groove; and 3) the proximal differentiates into the malleus and possibly the incus.

The condyloid and coronoid processes ossify lateral and independent to Meckel's cartilage. By 44 mm c.r. (9 wk.) their adult form is determined, not to demean postnatal function but to express possible prenatal deformity. The growing condyle continues to be formed in cartilage until adult size is attained.

The lateral and medial troughs of bone which are external to Meckel's cartilage provide the grooves for the dental lamina to send down the invaginations representing the deciduous and permanent toothbuds. The lateral pterygoid muscle extends its ligament medial, superior and distal to the developing condyle to form the posterior attachment of the capsule and the disc of the joint. In a brief intrauterine span of seven weeks [from 17 mm c.r. (7 wk.) to 103 mm c.r. (14 wk.)] the first branchial arch has developed from a primitive anlage to a miniature adult mandible. All that remains now is for increase in size. Function has occurred with intrauterine mandibular movements of sucking, protrusion, opening and closing.[35] By the time the fetus has reached term the trigeminal and facial nerve systems are functional in preparation of birth.

Postnatal Development

The postnatal development of the human mandible, temporomandibular joints or, perhaps, the entire stomatognathic system has been of particular interest to this group. The studies of Brodie and his legions of graduates have focused upon normal growth and development of the skeletal and soft tissue structures of the area. Thompson has long shown all of us the basic importance of early treatment for functional malocclusions and our responsibility to the functional aspect of our treatment procedures.

Of particular interest to me has been the role of the neuromuscular system in adaptation to malocclusion or in the

achievement of normal occlusion. Our research indicates a fundamental continuum of coordination and synchrony with normal form-function relation to harmony, stability and health of the stomatognathic system.[36]

At an early age many children will exhibit a bruxing habit about which some parents seem quite concerned. My personal observation is that if the child at four, five, six or seven is not bruxing at night there may be need for concern. In these instances of bruxing the proprioceptors of the mandibular musculature are being developed and reinforced. The occurrence of cusped teeth may stop or slow the habit since lateral excursions are limited. In many children with Class II, Division 1 deep-bite malocclusion the possibility of bruxing and elevator muscle development is inhibited. In these cases the early deciduous dentition musculature is not fully developed for all mandibular excursions.

The same pattern may be seen later in electromyographic records of deep-bite subjects. They are primarily "choppers"[37] and do not possess the lateral eccentric to centric glide so essential to proper bolus trituration.

The occurrence of unilateral or bilateral buccal crossbites, anterior crossbites, supraerupted teeth, extreme deep overbite (excessive incisal guidance) are all functional factors which have a greater traumatic potential to the joint components than all of the disagreeable esthetic features of excessive maxillary protrusion or mandibular retrusion. During treatment at any instant in time at which we permit or create vertical violations of the "free-way" space, we are jeopardizing the proprioceptive protection of the teeth and jaws. Fortunately for us and our patients some do seem to adapt to or accept dysfunction and avoid pathology.

Functional Aspects

Earlier reference was made to John Thompson and his work in defining functional and structural malocclusion. Hixon once said to me that Thompson was the one who was ". . . waving a flashlight in our face to bring our attention as orthodontists for our responsibility to function." Yes, Jack did wave and "holler" and now twenty-five years later some of us are seeing the light. What I have to present is a reaffirmation or extension of his thoughts.

For many years our specialty would demand that each of us put the "plaster on the table" to prove our clinical ability. I do not devalue this method of appraisal but I strongly urge you to consider more than that static assessment of our success. Let us also be cognizant of the functioning relation of all cusps and inclines as well as condyles and fossae. The functional integrity or ability of this stomatognathic system is a responsibility of its neuromuscular component. This component acts in deference to mechanical tooth position and to the health of the bilateral temporomandibular joints. There is, therefore, a chain of response that is a closed circle. Muscles contract, jaws move to tooth contact, tooth contact proprioceptively brings muscle control, a true biofeedback system. Homeostasis is then the basis of health and function of this system, not in Claude Bernard's original concept of balance in blood and tissue fluid composition, but more in Cannon's relative constancy of the "internal environment." It is a functional homeostasis and not one of hormones or metabolites.

In a recent paper titled "Masticatory Dysfunction in the Pre- and Postorthodontic Patient"[39] six categories were presented that could and do upset the balance in this system. They are:

1. Congenital and birth-related traumas
2. Dental-facial growth abnormalities
3. Microtrauma to teeth and supportive structures
4. Macrotrauma to face and jaws
5. Infection and disease involvement of joints
6. Iatrogenic insults to teeth and jaws.

For your immediate attention I will limit my material to the last category which is an area where we have a major role in improving function and avoiding pathology: iatrogenic insults to teeth and jaws.

Normal TMJ Function

The masticatory pattern of an individual is usually as characteristic as his walking gait. The mammalian synovial joints have four varieties of articular nerve receptors.[40] Type I corpuscles are widely distributed throughout the fibrous capsule but with increased concentrations at the anterior and posterior regions. Type II have a similar but less populous distribution as Type I with some in the bilaminar zone of Rees. Type III are distinct in that they are located only in the lateral ligament. Type IV are not of a corpuscle type but unmyelinated and free nerve endings (pain) in the joint capsule and ligament. These have different response rates and levels. Type I is low threshold, slow adapting mechanoreceptors; Type II are low threshold, rapid adapting; Type III have a higher threshold and slow adapting, and Type IV are concerned with the pain response system of the joint. Clark and Wyke[41] have stated that derangements of this reflex system ". . . as by trauma, disease or malocclusion might result in changes of the normal pattern of mandibular muscle activity that may be significant in clinical circumstances."

Previous electromyographic studies have conclusively illustrated that there exists a deranged pattern of elevator function in severe malocclusions.[42] This pattern of muscle function improves and approaches the "normal" functional pattern with orthodontic treatment. Thus we see a neurogenic basis for dysfunction of the musculature from within the joint and the tooth relations in function. I propose that the first evidence of our concern for dysfunction occurs in the dental occlusion and interdigitation which is reflected reflexly in the muscles of the mandible and, from there, the capsular neural elements come into play. Without correction of the occlusal factors which contributed to the musculature patterns we precipitate joint (capsule and disc) involvement and in time we witness the evidence of joint breakdown and dysfunction.[43]

Abnormal Function

Jaw deflecting tooth interferences, whether lateral, posterior, anterior or inferior, are all suspect in initiating reflexive muscle patterns of avoidance. The contraction of these mandibular muscles do have a bearing upon jaw, condyle and face development. The earlier we can institute correction of the functional malocclusion, the earlier we assure balanced structural development. The growing child illustrates the dynamic responsiveness of this system which is so notably lacking in our adult dysfunction patients.

During our orthodontic procedures we are constantly responsible to avoid or eliminate occlusal trauma factors. Usually our first knowledge of dysfunction will be joint sounds in the form of clicking with open-close movements. Crepitus, if it should occur, is indicative of pathologic degeneration and is usually subsequent to the occurrence of the "click." At this juncture our knowledge of mandible-to-maxilla relation is

important. It is wise at each appointment to check the open-close, right and left noncontact movements. In this we are assessing the joint movements and health in absence of tooth contact. At each appointment I will also check the patient's centric relation and centric occlusion and, in addition, check eccentric movements from centric occlusion to end-to-end cuspal relation. In this short one-to-two minute segment of the appointment I seek the following: cuspal interferences with brackets and tubes; lack of vertical control with mandibular second molars, excessive incisal guidance, and irregular eccentric movement associated with poor cusp position control and tooth interdigitation. I assure you that you can save a lot of mechanical effort, diagnostic frustration, as well as patient discomfort and possible dysfunction, by using your eyes, hands, ears and logic.

Subsequent to orthodontic treatment I will often do selective grinding based upon an occlusal alginate wafer of centric occlusion. This is done within four to six weeks of retention and is directed primarily to the presence of artificial (filling) tooth material which may have an altered vertical relation in the corrected malocclusion. Later, in retention and after all retention I will do eccentric checks with wax and either improve group-function patterns or canine-guidance function dependent upon my original treatment objectives and the facial musculature pattern. Occlusal equilibration per se does not eliminate, circumvent or avoid dysfunction and in no way should it be employed to compensate for mechanical treatment objectives.

Although I have found a lesser need for occlusal grinding in cases which are retained with a rubber finishing appliance, I have sometimes been guilty of creating dysfunction problems with this appliance.

Other postretention problems which create dysfunction patterns are too numerous to mention; however, the more prominent are noteworthy.

1. Improper retainer design and/or instruction in wear
2. Supraerupted third molars
3. Adverse postpuberal growth resulting in jaw-to-jaw and tooth-to-tooth discordance.

Perhaps the time test of our clinical endeavor is the functional integrity of the entire stomatognathic system twenty to forty years after our treatment. Orthodontics has not had the wide base for appraisal of these cases at the present time. First of all there were relatively few orthodontists practicing forty years ago. Secondly, the loss of teeth, relapse of dentitions, degeneration of condyles and bone may be attributed to other systemic factors and *not* the cause-effect equation of orthodontic treatment. Thirdly, very few of us conscientiously follow our patients into their thirties or even twenties. I believe we must make a united effort to look at our record and evaluate the blessings of our services far beyond that juncture in time when we remove the mandibular retainer.

I have seen far too many adults at our clinic for temporomandibular joint problems who rightfully or wrongfully blame early orthodontic treatment for their problems. I personally believe we can factually disprove their contentions. If we have full documentation of our correctly treated cases whom we fully acknowledge to have functional stability and health at retention, we should be able to demonstrate continued and natural wear and adjustment. However, in those instances where we have ignored function we have directed our patients into a potentially painful and disruptive dental future.

Summary

Throughout the development of our specialty there have been individuals who have urged us to seek the total benefits for our patients that orthodontics is capable of providing. Too often their pleas have been drowned by the arguments of those interested in esthetics and static analysis. More frequently today we are "feeling the heat" of our generalists, colleagues and the other specialties who believe we have disavowed our responsibility to function and longterm stomatognathic health. Perhaps these latter groups are partially correct, for rarely have I seen in our literature reports of our clinical failures until within the past few years. We have been remiss in our educational processes and our editorial efforts when we do not admit and recognize our limitations and even our frank failures. The ideal time in treatment planning to point out our own human frailty is at the time of our diagnosis. If we firmly believe, from training and experience, that we have a limitation in our treatment procedure, we must first decide whether our treatment will do more harm than good. If so, should we treat? If we do treat, in these extreme cases, we are obligated to inform our patients and their dentists of our mechanical and thus functional limitations. I personally believe one of our present-day concerns of who is doing or should do orthodontic treatment is self-created and self-perpetuated by the orthodontist. Many of our professional colleagues believe there are few pitfalls to moving teeth and there are no limits to that movement. Why? Have we ever really documented the same in our literature or lectures?

Currently we are seeing a great emergence of mechanistic occlusion concepts being presented to the orthodontist. The orthodontist is eager to accept, learn and practice the step-by-step rules of occlusal function presented; the gnathological concepts are emerging in a wholesale fashion at our meetings. In the main, this is a good sign. We have, as a group, seen the light that Thompson and others so long have tried to present to us. However, there should be a few restraints to our total acceptance. First of all, the myriad of cusp shapes, jaw forms, facial patterns, ranges of muscle contractions, condyle-fossae contours and tooth contact times and range would indicate that a single concept is too stringent and mechanistic to fit all of our patients. Second, and perhaps most important, is the fact that the rules and concepts for restorative and full denture articulation, for reasons earlier cited, should be different for our adolescent to postadolescent patients. If we could eclectically utilize the significant proposals where applicable to our orthodontic population, all well and good. I fear, though, that the vast majority of us are "all or none" clinicians who desire to treat and retain by the "cook book" method. That is the rub. We can not universally apply any set of dogmatic rules to our total patient population. Therefore, let us apply common sense, a very uncommon commodity, and in recognition of the many different patterns of function and form,[44,45] seek to eliminate those salient traumatic features that are generally accepted to contribute to dysfunction and degeneration of the masticatory apparatus.

Conclusion

The specialty of orthodontics has progressed a great distance in the past fifty years. We are now on a new threshold which beckons us with promise. Our procedures are being modified and streamlined. Our appliances are directed more to force-delivery control and oral hygiene. Our diagnostic information is being advanced with the

aide of computers. Our patient loads and office efficiency are increased by competent management of ancillary personnel. Let us also then give more than lip service to the desire that our goal of treatment includes stability, health and function.

This group, perhaps more than any other similar group of our specialty, owes so much to that goal because of common interests and respect to the memory of Dr. Angle's teachings and his firm belief in the importance and dominance of masticatory function.

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