Gnathology and the Myoprotected Occlusion, A Hypothesis

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The term *Gnathology* is defined from gnathos, meaning jawed, and from stomato —stomat, meaning mouth. Therefore, it means to define the jawed mouth complex.[1] It includes jaws, teeth, their investing tissues, and all of the neuromuscular system of the head and neck. It is this interconnected region of the human body in which we are interested.

The concept of mutually-protected occlusion versus a non-protective occlusion has become meaningful in modern dentistry. The terms pertain to the occlusion and disclusion of the teeth during mandibular motion, how the position of teeth can influence jaw movement, and how the movements in turn can be destructive or protective to the gnathostomatic system.

When the jaw closes in its most posterior border in "terminal hinge" position and all teeth meet at the same time, we say centric relation and centric occlusion are the same. Relation pertains to the jaw position relative to the joints, occlusion refers to upper-to-lower tooth contact.

During movements of the mandible, teeth articulate. In some areas, upper and lower teeth disengage completely. In others, they slide upon each other. The manner in which this occurs may be destructive or protective to teeth and other parts of the system.

A mutually-protected occlusion may be described as follows: During the protrusive movement of the mandible, the mesial incline of the buccal cusps of the lower first premolars should slide upon the distal incline of the maxillary cuspids. The lower incisor edges slide upon the lingual surfaces of the upper incisors into incisive position. This guiding discludes all posterior teeth, thereby preventing the bumping and scraping of cusps between upper and lower buccal teeth. It is therefore said in protrusive jaw relation, anterior teeth are protecting posterior segments.

In lateral motion upper and lower cuspids on the excursion side should function upon each other disengaging all other teeth on both sides, until the incisal edges of the centrals and laterals (on this side) also glide upon each other. Here the cuspids protect all other teeth from being rocked against each other and the tips of the cuspids are in turn protected from wear by the incisal edges of the anterior segment.

The posterior teeth, in their cusp-fossa relation, resist the vertical force of mandibular closure in centric closure. The anterior teeth do not have tight contact in centric. The upper incisors are protected from the closing force of the lower incisors, upper incisor flaring or wedging is prevented.

Since the posterior segments protect anterior units and anterior teeth protect the distal segments and since the cuspids protect anterior as well as posterior segments, but incisors in turn protect the tip of the cuspids, it is clear there is a trifold mutual protection in this occlusal arrangement; consequently, this scheme of arrangement has been called "a mutually-protected occlusion."[2]

The cusps and inclines of teeth must also be in harmony with centric closure. If they are not, they are then introduced into the occlusal scheme as irritation or fulcrum.[3] In this case, various deleterious responses of the neuromuscular system can be precipitated, exciting the muscles in an effort to eliminate or mask the irritation. For instance, the mandible when moved in terminal hinge closure may contact teeth in centric prematurity. It is then guided by the deflecting inclines into the most functional intercuspal position. The mandible, thereby, will be shifted straight forward, to the side, or both.

If such a discrepancy between centric relation and centric occlusion exists, due to the patient's neuromuscular response, mandibular positioning can adjust intercuspal contact to accommodate the occlusion, thereby protecting the teeth and periodontium from being stressed.

The muscles will be programmed by the teeth to guide the condyles in a position so that closure automatically occurs into the most functional intercuspal relation. The patient may be completely unaware of any prematurity or deflection. This ability of the system is proprioceptively adjust muscle function is known as occlusal programming.

Yet such a mandibular accommodation, if severe enough, could, in turn, introduce its own stresses.

In lateral positioning especially one of the condyles must shift downward, forward, and inward. To perform this function the muscles responsible for the motion obviously are under tension on the shifting condyle side. The lateral pterygoid on that side is stressed in this manner 24 hours a day, seven days a week, 365 days a year. This muscle can never relax as long as the occlusal prematurity exists.[3]

It is well known that man can accommodate to occlusal irritations. Tissues of the gnathostomatic system can tolerate a certain amount of stress, yet there are individual limits as to the amount to which the system can be subjected. Excessive stressing may excite self equilibration in the form of bruxism in an effort to modify the occlusal scheme. This bruxing in turn will introduce new forces, stressing mainly the investing tissues in a direction contrary to the design of the structures to best withstand force.[3]

Stress can therefore be a causative factor. Pathology may be precipitated in the weakest link of the system. These symptoms may be described as occlusal disease. They are: pulpitis, premature occlusal wear, accelerated peridontal breakdown, temporomandibular joint disfunction syndromes and disturbances associated with hypertonicity of the muscles of the system. Such as a stiff or sore neck, headaches, ear-aches, and spasms. Of course, other etiologies can also precipitate these conditions.[3] By modifying the occlusion, dentists can either eliminate or introduce stresses into the system. I refer to procedures such as occlusal equilibration, restorative procedures, and orthodontics.[3]

The muscle adaptive mechanisms programming mandibular closure may be insufficient protection for the occlusion. In cases where the slide or shift into centric occlusion is excessive, the adaptive muscles may have become stressed beyond their

physiological limits, and or excessive "corrective bruxing" may have caused occlusal disease and pain.

The system has one additional protective device. This last neuromuscular response seeks to splint the mandibular movements, thereby preventing occlusal contact. Such a protective crutch will mask the occlusal scheme.

Since teeth occlude mainly during swallowing, the cushioning must occur at that moment. It is here and now that we observe the tongue as an interocclusal mass of muscle. Contact is made into a well padded cushion. Immediately all cuspal interferences are eliminated. Condyles can maintain their terminal hinge position allowing the stressed musculature in this region to relax. Aches and tension syndromes around the head and neck may subside. Any pulpitis caused by occlusion may recover. We have now a tongue or myoprotected occlusion.

The tongue action in turn may be assisted by various muscle groups during swallowing. Normally, the oral content is prevented from escaping into the vestibules by the occluded buccal teeth. During the "deviate swallow" however, the circumoral and buccinator groups, typically, contract to establish the confining walls.

The classic deviate swallow is now observed. To jump in and seek the help of a myofunctional therapist would be to deprive this gnathostomatic system of its last protection.

Obviously, a system with such functional problems which has defensively been thrusting, will, in time also, have superimposed all the undesirable occlusal results which are usually associated with the so-called habit.

An examination at this point in time will correctly blame the deviate swallowing for the typical but secondary occlusal problems. Yet, the original causative factor may not be recognized.

In an article *Bottle Feeding As Preventive Orthodontics,* written fifteen years ago, it was noted, "Many crossbites and some Class II malocclusions are the direct result of the malfunction of tongue and lips, initiated by a nonphysiological nipple. Many orthodontic relapses of Class II malocclusions and crossbites are due to the continuous malfunction of the tongue and lips after treatment".[4]

My attitude today toward this idea is less positive. What was written then was opinion based on clinical observations and research of the German literature. Equally, what is postulated today is opinion based upon observations of many renowned gnathologists. Both approaches are theoretical, yet both have some merit.

Accordingly, it may be further deduced that orthodontic treatment itself creates the perfect environment to invoke protective tongue habits.

However the light force principle of tooth movement fail at any moment during treatment, resulting in pain, or should intercuspation become temporarily adverse, as of necessity it must, then the gnathostomatic system could call upon this protective neuromuscular response. If fortune smiles the reflex will be of transitory nature.

Orthodontic cases upon completion may have prematurities in either centric relation or they may have inadequate disclusion of posterior teeth in lateral excursion. The first may occur when the desired neutral Class I relation-occlusion has not been reached. The second is the result of insufficient cuspid protection together with lack of lingual crown torque in the molar region.

The system will try to eliminate these unpleasant cuspal interferences. Self equilibration may start. All stresses produced by bruxing will follow, resulting eventually in occusal disease. Again the tongue could be the last resource the system can call upon. Myoprotection in turn will cause the orthodontically treated case to relapse. Even though

the deviate swallow is secondary here, more often than not, the therapist is called upon to save the case.

Typically, bruxing on one side to eliminate maxillary molar lingual cusp interference in lateral excursion will show excessive wear of the cuspid on the opposite side. This is occurring because the mandible, as a lever, must be stabilized on the opposite side during this self equilibration. Less trained observers see this as cuspid function, when in reality it is dysfunction. Therefore, when examining a patient for tongue habits, observe whether excessive cuspid wear can be detected. It may be a sign that there is molar interference on the opposite side during lateral excursion, and that the patient is, or was, bruxing in an attempt to remove this disturbance in the occlusal scheme.[5]

To differentiate between primary and secondary thrust is very difficult indeed. Diagnostically it must be determined whether the patient has a splinting habit, protecting himself from his occlusal scheme, or from the stress induced symptoms, and/or has he occlusal disease; hence does he now have a protective thrust. Myofunctional therapy cannot be successful in these cases, unless simultaneously, occlusal interferences and hangups are also eliminated.

This review has been presented to show a portion of the other side of the coin, in order that the therapist or referring dentist may appreciate any skepticism about myofunctional therapy that may exist in the dental profession.

If at all possible the referring Dentist should relay to the therapist his diagnostic views of the type of thrust with which he will have to deal.

A history always is helpful. It should be established whether any of the parents or siblings show a similar habit; if yes, then one can almost be certain the thrust is inherited or imitated just as so many other mannerisms become family traits. One often finds look-alike family habits, a smile, mimic similarities, walk and posture all fall into this category.

A thrust may be the perpetuation of a swallowing pattern learned at the nursing bottle with a nonphysological nipple or even at the mother's breast when the rate of flow was too great. In these cases one probably does not deal with a protective thrust.

By the time the patient is referred to the myotherapist it is virtually impossible to tell from the condition of dentition what the origin of the habit was: protective, bottle induced, inherited, or imitated. A carefully taken history may be the only way to find some clues.

In theory, the deviate swallow may originate as a primary one, the dentition then in time may loose its mutual protective scheme, the habit now is primary and secondary-protective. Myotherapy, to be a success, should be accompanied by occlusal rearrangement.

When to begin myofunctional therapy may have an answer if one can establish the type of thrust present. A purely primary habit can be treated prior to the onset of orthodontic therapy. This may even in some cases, prevent orthodontic intervention. However, should the tongue action originate as a protective response, or should the primary habit now also be protective, then therapy should start at the beginning of occlusal correction.

Further, it is argued in dental circles that thrust will disappear whenever the malocclusion has been corrected.

Gnathologically oriented men will probably agree, because they feel if and when correction is complete, and only then, the protective thrust will become superfluous as soon as the occlusion is no longer in need of being cushioned. It is doubtful however, whether purely primary thrust will compatily vanish.

Often, however, cause and effect cannot properly be differentiated. The old question, "What comes first, the chicken or the egg?", is still the real problem. Both approaches are speculation. Here, therefore, is fertile ground for controlled investigations with statistically significant conclusions.

Oral myology is in its infancy. To establish credibility, theories must have scientific justification. This was made clear about ten years ago, this author called as an "expert witness' before the Food and Drug Administration in Washington to testify for a company which was to market the Nuk Sauger Nipple as a preventive orthodontic device. After arriving with 'before' and 'after' models and headfilms showing corrections of crossbites and open bites with these nipples in deciduous dentitions. The sample consisted of three cases. The F.D.A. reviewed the material and asked the author to return with records of not less than 1,000 infants who had used the Nuk Sauger, and with records of an equal control group who had gone without these devices.

These are the kinds of studies that are needed. For instance, take a number of thrusters presenting typical dentition, remove dental interference on one group and not in the control group. Will the thrust disappear? Further, give myofunctional therapy to a portion of the treated group, and to a number of the control group. What are the findings? Numerous combinations are open for study. Numerous difficulties will obviously present themselves during such an undertaking.

In my opinion, these are the trends and some of the problems. The International Association of Oral Myology should be ideally suited to undertake such studies. It is my suggestion that they should seriously consider embarking on such programs.

The doubt and confusion existing in this area of dentistry could be minimized.

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Literary Review

ORAL MYOFUNCTIONAL DISORDERS, Richard H. Barrett, M.Ed. and Marvin L. Hanson, Ph.D. The C. V. Mosby Company, P.O. Box 14737, St. Louis, Mo. 63178, 1974, 308 pages, with 95 figures, 24 tables, 8 forms. price $29.50

Until recently, the literature on tongue thrust was scattered in the various journals of the dental and speech professions. In 1970, Fletcher (in ASHA Reports #5) summarized this literature in a 'state of the art' which he later expanded in his book *TONGUE THRUST IN SWALLOWING AND SPEAKING* (1974). Still we had little appreciation for the subject of tongue thrust through the perspective of historical continuity. It was necessary then, that ideas and methodologies be traced from their origins, through stages of development, to our current levels of understanding. In *ORAL MYOFUNCTIONAL DISORDERS,* Barrett and Hanson have written an informative, readable account of the history of tongue thrust: its nature, controversies, and theories of etiology, diagnosis and treatment. The authors approach the subject systematically and comprehensively, assuming limited prior exposure on the part of the reader. The result is that even those with a great deal of exposure will find themselves impressed with the thoroughness and objectivity of the book as well as the clinical and research challenges it presents.

Historically, several implied dichotomies have persisted in the literature on tongue thrust. Consistent with this perspective, Barrett and Hanson compare the contributions of British versus American writers. This comparison prompts the realization that factors which have been viewed as dichotomous are actually artifacts of an historical continuum. Barrett and Hanson note, for example, that with only two exceptions, all of the literature on tongue thrust until 1960 had been written by dentists. In addition they state that "The British orientation in dentistry has historically been toward research and theoretical formulation. American dentists, meanwhile, have shown a propensity toward clinical experimentation." To elucidate, the authors have summarized separately the contributions of each of the following writers. Rix, Gywnn-Evans, Ballard, Tulley, Ardran and Kemp and Leech are cited as representative of the British literature and Angle, Lischer, Rogers, The Truesdells, Klein, Straub, Moyers, Ricketts and Harrington as representative of the American literature.

Several chapters of *ORAL MYOFUNCTIONAL DISORDERS* will help to bridge the gap between the orientations of speech and dental practitioners. For the speech clinician, Barrett and Hanson have provided an excellent overview of dental anatomy, physiology, development, malocclusion, corrective principles and clinical specialties. For the dentist, the discussion of speech development, disorders and the relationship between speech and dentistry is informative. Of theoretical and clinical interest to members of all disciplines involved in tongue thrust, are the chapters entitled: Etiologies (15 theories are discussed), Developmental physiology (of swallowing) and Controversies. The second part of the book, devoted to the Fundamentals and Procedures of treatment for oral myofunctional disorders, is of obvious clinical significance. This section (essentially a treatment manual) is highlighted by discussions of a classification system for tongue thrust types, diverse treatment approaches including appliance therapy, surgery, hypnosis and various oral myo(functional) therapy programs—from Straub to behavior modification, and finally, a detailed presentation of the authors' comprehensive approach to the diagnosis and treatment of oral myofunctional disorders.

ORAL MYOFUNCTIONAL DISORDERS is an excellent text for use in courses offered to dental, speech pathology and oral myology students. Practitioners of oral myology will be inspired by answers to bothersome questions and new insights into clinical methodologies, and although the book is primarily clinical, researchers will find it stimulates new levels of inquiry into old challenges.

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