A contemporary and evidence-based view of canine protected occlusion

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Dentists and orthodontists at one time or another have been exposed to the gnathological concept of occlusion. Certainly, a well-known and advocated precept of gnathology is that of "canine (mutually) protected occlusion (CPO)." The basic premise of CPO is that, on laterotrusive movements of the mandible, only the canines (possibly first premolars) contact and therefore protect the remaining dentition from adverse occlusal torsion forces on contacts to and from centric occlusion (and/or centric relation). Furthermore, it is contended that CPO is the optimal (ideal) type of functional occlusion for the natural dentition vis-à-vis dentures and is the functional occlusion type toward which restorative and orthodontic treatments should be directed.[1,2] It is also argued that orthodontists who do not obtain canine protected functional occlusions are doing a disservice to their patients and possibly not practicing state-of-the-art orthodontics.[3-10] That is, gnathologists maintain that orthodontists who do not establish a gnathologic finish, including CPO, potentially predispose patients to temporomandibular disorders (TMD) and orthodontic tooth relapse.[3-7,11]

However, many past notions in dentistry and orthodontics, particularly related to gnathology, have not withstood the test of time or the rigors of science.[12,13] And, with the recent emphasis for dentists to practice evidence-based decision making,[14-16] it makes sense to fully investigate and evaluate the concept of CPO. The purpose of this article is to discuss past and present knowledge and information on the general topic of functional occlusion (particularly regarding CPO and orthodontics) and relate it to logical considerations

relevant to science and evidence-based decision making. We provide a provocative and insightful perspective on what constitutes the optimal functional occlusion type for orthodontic treatment. Our goal was to call orthodontists to reconsider their views on functional occlusion—particularly CPO—in light of current knowledge and evidence. Many peripheral topics, issues, and controversies about functional occlusion, such as centric relation,[17] articulators in orthodontics,[18] and the general topic of occlusion, TMD, and orthodontics[19-23] was addressed in previous studies.

Classic studies by Angle[24,25] and a later study by Andrews[26] established criteria for the optimal (ideal) morphologic relationship of the human dentition (although there is little evidence of a biological relationship associated with these criteria). However, the optimal functional occlusion type has not been so easily identified and has essentially eluded the dental profession. Ash and Ramjford[27] wrote: "Orthodontic classifications are related more to anatomic and esthetic standards than to neuromuscular harmony and functional stability. It has not been possible to develop a consensus on a numerical index or system of values that applies both to form and function of the masticatory system." Based primarily on laterotrusive movements from centric occlusion, several functional occlusion types were recognized or advocated: balanced occlusion,[28,29] CPO,[1,30-36] group function occlusion,[37-41] mixed canine-protected and group function,[42] flat plane (attrition) teeth occlusion,[43,44] and biologic (multivaried, physiologic) occlusion.[45]

No single type of functional occlusion has been found to predominate in nature. For example, D'Amico,[1] Ismail and Guevara,[46] and Scaife and Holt[2] all found that CPO predominated, whereas Beyron[41] and MacMillan[37] found predominance of group function occlusion. In addition, the natural occurrence of balanced occlusion (ie, with nonworking contacts) was found in populations studied by Weinberg,[47] Yuodelis and Mann,[48] Ingervall,[49] Gazit and Lieberman,[50] Sadowsky and BeGole,[51] Sadowsky and Polson,[52] Rinchuse and Sassouni,[53] Shefter and McFall,[54] deLaat and van Steenberghe,[55] Ahlgren and Posselt,[56] Egermark-Eriksson

et al,[57] and Weinberg and Chastain.[58] Although balanced occlusion for the natural dentition is considered injurious and contravening by gnathologists, it is perhaps the norm rather than the exception with regard to prevalence. Woda et al[29] stated, "Pure canine protection or pure group function rarely exists and balancing contacts seem to be the general rule in the populations of contemporary civilization."

LITERATURE REVIEW
Brief history of CPO

Well over a century ago, Bonwill and Gysi recommended balanced occlusion (bilateral balanced and 3-point contact) for denture construction.[27] The thinking was that, to prevent dislodgement, the denture must have at least 3 points of contact during all possible mandibular movements: "Bilateral balanced and three-point contact has been sponsored chiefly by prosthetists in order to secure a supposed mechanical advantage in stabilization of dentures."[37] In the 1930s, McLean[59] contended that this concept also applied to the natural dentition. He based his conjecture on his examinations of animals and humans. He further believed that periodontal bone resorption would result from excessive occlusal forces if teeth were not bilaterally balanced. About the same time, MacMillan[37] took a different view and recommended a shift from balanced occlusion (ie, bilateral balanced) to unilateral balanced occlusion for both natural and prosthetically restored dentitions. He believed that bilateral balanced occlusion never existed in nature, either in animals or man. His evidence was based on the evaluation of "various types of masticatory excursions of lower animals." Arguing in favor of unilateral balanced occlusion over bilateral balanced occlusion, MacMillan[37] stated: "Unilateral balance in molar mastication is beautifully illustrated in comparative anatomy." He also contended that the analysis of the masticatory process in humans via cinematography demonstrated that the nonworking-side teeth do not come in contact during mastication: "The buccal cusp of the mandibular molar of the idle side never comes in contact with the lingual cusp of the maxillary molar."

Once balanced occlusion was considered obsolete, with general agreement that nonworking-side contacts (balancing) were to be precluded (this is debated today), the next issue that needed to be addressed was what type of working-side lateral functional occlusion is preferred. Two working-side schemes took precedence—CPO and group function occlusion (unilateral balanced). The requisites for CPO are that only the canines contact (an alternate scheme includes the first premolars) on the working side during eccentric lateral mandibular movements, whereas, on the nonworking side, there are no balancing-side contacts.[27] The advocates of CPO argued that humans innately possess the long and dominant canine that is evident in carnivorous animals.[1,60,61] They further argued that the canine is the strongest human tooth type and has the most sensitive proprioceptive fibers. They therefore concluded that the canines are the best teeth to protect the occlusion from eccentric forces that occur on movements to and from centric occlusion (and/or centric occlusion). The CPO enthusiasts also argued that population studies confirm the prevalence of CPO over group function occlusion. They further maintained that the only reason some modern humans do not have CPO is that they eat coarse and abrasive diets that adversely wear down their canines.[1,61,62] In a telemetry study (miniaturized radio transmitters placed in temporary bridges and gold inlays), Butler and Zander[62] found that, when the functional occlusions of 2 subjects were periodically changed from CPO to group function occlusion, there were fewer lateral contacts when each subject worn canine protected occlusion (perhaps CPO restricts lateral excursive movements). Also, Ash and Ramjford[27] believed that a steep canine rise on the so-called Michigan occlusal splint can reduce the electromyography (EMG) activities of the masseter and anterior temporalis muscles.

On the other hand, the group function occlusionists[37-41] argued that equivalent population studies confirm the prevalence of group function occlusion, not CPO. They also indicated that Australian aborigines had group function occlusions.[41] Furthermore, they reasoned that the canine is not necessarily the strongest human tooth (molars have at least 4 roots and offer great support for the dentition). Furthermore, the canines are not necessarily the last teeth lost with age and do not necessarily have more sensitive proprioceptive systems than any other teeth.[39] In addition, Ash and Ramjford[27] argued that prominent canines can adversely "restrain normal lateral movements and the patient may develop chewing motions with a steep path of closure into centric occlusion." As previously discussed, in addition to CPO and group function occlusion, several other less popular functional occlusion types have also been advocated.[43-45]

The issue of balanced occlusion

Nine studies published from 1972 to 1991 that included a total of 959 subjects reported the occurrence of balancing contacts ranging from 34% to 89%.[20,49,51-55,57] Ingervall[49] found that approximately 85% of 100 subjects with normal static occlusions had balanced occlusions. Rinchuse and Sassouni[53] found that 85% of 27

normal static occlusion subjects had balanced occlusions. Sadowsky and BeGole[51] reported that 89% of 75 subjects with various types of Angle malocclusions had balancing contacts. Furthermore, de Laat and van Steenberghe[55] found that 61% of 121 Belgian dental students with various Angle malocclusions had balancing contacts. Shefter and McFall[54] reported that 56% of 66 subjects with Angle malocclusions had balancing contacts. Also, Sadowsky and Polson[52] found that 45% of 111 subjects with Angle malocclusions had balancing contacts. Egermark-Eriksson et al[57] reported that 34.5% of 238 subjects with Angle malocclusions had balancing contacts. In addition, Ahlgren and Posselt[56] found that 34% of l20 subjects with Angle malocclusions had balancing contacts. Finally, Tipton and Rinchuse[20] found that 75% of 101 subjects (52 of 101, or 51.1%, with normal static occlusions) had balanced occlusions.

In the 1970s, orthodontic gnathologists argued that orthodontic patients' functional occlusions should be finished to CPO.[3-5] They then alleged that, when orthodontists ignore patients' functional occlusions and rely on hand-held models rather than articulators, patients would predictably finish with balancing contacts and eventual TMD. These orthodontic gnathologists were partially accurate in their assessment of nongnathologically treated postorthodontic patients; they did have balanced occlusions. However, comparison groups consisting of subjects with ideal static occlusions and Angle malocclusions also had balanced occlusions and to an equivalent extent.[12,13,18-21,42,51,52] In addition, there was no difference in the TMD signs and symptoms between orthodontically treated and untreated subjects.[12,13,18-21,51,52,63] Also, TMD increases with age irrespective of orthodontic treatment.[57,64]

Several points need to be clarified regarding nonworking-side functional tooth contacts. Two terms are often used synonymously when describing when and where teeth touch, ie, tooth "contacts" vs tooth "interferences." Although both terms indicate that the teeth touch, there is a semantic difference between an occlusal "contact" and an occlusal "interference." Occlusal contacts are considered benign compared with occlusal interferences. Ash and Ramfjord[27] wrote: "A balancing side contact is not a balancing side interference if it does not interfere with function nor cause dysfunction . . . or . . . injury to any of the components of the masticatory system." Furthermore, Ash and Ramfjord[27] argued against the claim that all lateral forces and stresses on the teeth from balancing contacts are problematic and undesirable: "Lateral stress on the teeth is desirable within physiologic limits; it stimulates the development of a strong fibrous periodontal attach-

ment around the neck of the teeth and lessens the potential for traumatic periodontal injury from incidental or accidental occlusal forces."[27] Thus, the term occlusal balancing "contact" refers to a condition when the teeth come together usually without incident; ie, without tooth mobility, deflection of the mandible, or effect on the temporomandibular joints.

On the other hand, an occlusal "interference" is generally considered destructive and a harsher condition. Ash and Ramfjord[27] stated: "the term occlusal interference refers to an occlusal contact that interferes in a meaningful way with function or parafunction." The sixth edition of *Glossary of Prosthodontic Terms* defines an occlusal interference as any tooth contact that inhibits the remaining occluding surfaces from achieving stable and harmonious contacts.[65] That is, an occlusal interference is a contact that can force the mandible to deviate from a normal pattern of movement. Based mostly on empirical data, gnathologists claimed that occlusal interferences can cause tooth mobility, trauma from occlusion, deflection of the mandible, bruxism, relapse of tooth position, and TMD.[66-68] The type of balancing side occlusal contacts typically found in young postorthodontic subjects and their matched comparison groups are "contacts" and generally not "interferences."[69]

The criterion for the acceptance of a functional occlusion type as the optimal or preferred was based on 2 notions: a single functional occlusion type predominates in nature and provides subjects (or is found associated) with the fewest TMD signs and symptoms.[20] As previously discussed, a single type of functional occlusion has not been demonstrated to predominate in nature. Furthermore, electromyographic[70] and intraoral telemetry[62] studies (miniature radio implants embedded in dental prostheses and occlusal contacts monitored outside the mouth similar to the telemetry used for monitoring space flight) of functional occlusions that used prosthetically restored or replaced teeth are equivocal. In addition, the findings from provocation studies, in which functional occlusal interferences ("high crowns") were produced in subjects, were also inconclusive (ie, symptoms other than TMD found; samples biased because of dental nurses or students).[71-76]

The superiority of 1 type of static or functional occlusion to ameliorate TMD has not been demonstrated. Data from epidemiologic studies led to the conclusion that morphological or functional occlusion variables play a minor role, or no role, in the multifactorial etiology of TMD.[20,22,23,42,51-53,57,62,77-87] In this regard, Dolwich[88] stated: "Although proposed occlusal factors appear to be mechanically logical, they are based

upon empirical, clinical observations and have not been proven by controlled studies." A cause-and-effect relationship has not been established between occlusion (ie, morphologic or functional) and TMD.[12,13,19,22,58,89-92] At best, there might be an association between these 2 variables. Also, confusion has sometimes arisen because correlation or association research was wrongly interpreted as causality. There are several anecdotal and correlational reports of relationships between TMD signs or symptoms and Angle malocclusions in general,[93,94] overbite,[57,82,95-99] overjet,[57,82,100,101] Angle Class II,[57,79,89,102-105] Angle Class III,[57] crossbite,[56,84,106,107] "tilted teeth,"[108] loss of molars,[109-112] functional occlusal interferences,[88,113-122] and centric slides greater than 4 mm.[63,111] Interestingly, many of these conditions were speculated to be a result of TMD, rather then the cause of TMD.[63]

There is the belief that treating to a "functionally optimal occlusion," including the attainment of CPO, might be of greater relevance to orthodontic alignment stability than to TMD.[68] In this regard, Roth[3-5] and Cordray[6] believed this to be particularly true in regard to establishing a correct centric relation position. However, Luther[123] believed that, even if a particular functional occlusion is achieved, it will not be stable. Furthermore, Lopez-Gavito et al[124] found no difference in the long-term stability of mandibular arches between patients with anterior tooth contacts and those with anterior open-bite malocclusions with no canine contact in centric occlusion (and in functional excursions). This leads to the contention that the final assessment of functional occlusion contacts for orthodontic patients is never quite finished because human occlusion is in flux (unless, of course, there is lifetime retention).[125]

Although some EMG data suggest that CPO elicits a better EMG recording than any other type of functional occlusion, other studies contradict or minimize these findings. Some EMG studies that support the superiority of CPO follow. For instance, Williamson and Lundquist[126] found that the EMG activity from the temporalis and masseter muscles was less (better) during lateral excursions in subjects with CPO vs group function occlusion. Their findings were supported by those of McDonald and Hannam[127] and Shupe et al.[128] Belser and Hannam[129] reported that, although CPOs do not significantly alter muscle activity during mastication, they significantly reduce muscle activity during parafunctional clenching. Boero[130] believed that CPO (vs group function) produced the least EMG activity and would therefore have the least occlusal loading. Interestingly, several authors surmised that the canines perhaps have some special proprioceptive function that reduces muscle activity.[131,132]

Conversely, the validity of EMG analyses was recently questioned: "quantitative electromyography of the masticatory muscles seems to have limited value in diagnostics and in evaluation of individual treatment results. . . . Despite EMG findings . . . it has yet to be shown in clinical, prospective trials that canine protected occlusion has a therapeutic effect—prevents or cures TMD."[133] A descriptive study of 300 dental students by Bush[134] showed that bilateral canine guidance does not offer protection from facial muscle tenderness. Furthermore, Clark and Evans[125] argued that EMG functional occlusion studies are seriously flawed. That is, there is no proper description of what constitutes normal EMG activity in the masticatory muscles, and the studies lack appropriate control or comparison groups.

Relationship of static occlusion to functional occlusion

Few studies have examined the possible relationship between static occlusion and functional occlusion. Scaife and Holt[2] studied the dentitions of 1200 US military trainees and found that 940 had Angle Class I occlusions. CPO was found to be associated with Angle Class II and then with Angle Class I occlusions and was the least associated with Angle Class III malocclusions. That study was limited in that it did not differentiate between Class I malocclusions and normal (ideal) occlusions and did not identify or describe other functional occlusion types besides CPO. Sadowsky and BeGole[51] examined 75 subjects with various types of Angle malocclusions and found that 91% had balanced occlusions. Tipton and Rinchuse[20] found a trend for 101 subjects to have balanced occlusions more often associated with normal (ideal) static occlusion (or Class I occlusions). It appears that balanced occlusion exists to a far greater extent than gnathologists maintain and that balanced occlusion appears to be more predominant in subjects with normal (ideal) static occlusions (or Class I occlusions) vs Angle malocclusions.

Clarification of balancing contacts

Some clarification and qualification of the balanced occlusions in postorthodontic subjects and their comparison groups need to be addressed. The balancing contacts, for the most part, were contacts and not interferences. Next, most balancing-side contacts (interferences were not found) were on the distal sides of the posterior molars.[42,53] The exact order of frequency of teeth found to have balancing contacts were the distal aspect of the second molars, the distal aspect of the first molars, and the mesial aspect of the second molars.[42,53] Of note, all teeth (tooth types in addition to

molars—eg, incisors, canines, and premolars) were prone to have balancing-side contacts, although the occurrence was very limited. Furthermore, statistical analysis of the data confirmed that, when there was canine contact alone on the working side, there was a greater probably for a lack of balancing-side contacts on the nonworking side.[42,53] This is apparently the justification of some orthodontists for the extrusion of canines past their normal contact points or the addition of resin buildups to the worn incisal edges of these teeth to attain CPO. However, one must be mindful of the negative esthetic effect of canine extrusion on the smile arc. A contemporary esthetic treatment objective involves attaining a consonant smile arc whereby the incisal edges of the maxillary incisors and canines should be parallel to the curvature of the lower lip upon smiling.[135] It is argued that the unjustified extrusion of the maxillary canines to obtain CPO creates a nonconsonant smile arc—ie, flattening of the maxillary incisal curvature relative to the curvature of the lower lip.

If one considers the aforementioned and reflects on the relationship between static and functional occlusion, some provocative thoughts come to mind. Notably, there can be little doubt that the typical and normal type of functional occlusions in postorthodontic subjects and their nonorthodontically treated "ideal" static occlusion counterparts is balanced occlusion and not CPO (or even group function occlusion).[42,51-53,56,57] Importantly, however, not all balanced occlusions are identical. With this in mind and for any who consider it heretical to consider any version of balanced occlusion as normal, it might be just as logical and correct to consider an alternate term, "modified canine protected/ group function occlusion," rather than balanced occlusion. The balancing-side contacts (not interferences) in these groups were generally minor and for the most part on the distal aspects of the most posterior teeth, and, from the perspective of the gnathologist or occlusionist, easily amendable to occlusal equilibration.[42,53] Therefore, when discussing the predominance of balanced occlusion in orthodontically treated subjects and their matched counterparts, the nature of balanced occlusion must be made clear.

Masticatory chewing patterns

When jaw motions are examined from the frontal plane, 7 patterns have been identified that appear to be sex-specific and related to craniofacial morphology and the interdigitation of teeth.[136] Subjects with normal occlusions tend to have more simple, uncrossed, and elliptical movements than do subjects with malocclusions.[137] The characteristics of the shape of the masticatory cycle are finalized in the second year of life when the deciduous dentition reaches full occlusion[138] and do not vary much throughout life. When malocclusions are corrected with orthodontic treatment, the chewing cycles that were characteristic of the malocclusion generally remain, even with a new normalized occlusion in place.[139] Parenthetically, it is possible that subjects with more vertical chewing patterns (shapes) would best fit a canine protected functional occlusion scheme and those with horizontal chewing patterns would prefer more lateral freedom that would be consistent with balanced or group function occlusions.

Chewing efficiency is closely related to the amount of tooth surface used during the maceration of food.[140] Subjects with normal occlusions have more efficient chewing than those with malocclusions,[141] but no specific masticatory pattern has been identified as the most efficient.[142] The relative efficiency of how the occlusal interdigitations of teeth interact with the different masticatory patterns of jaw movement is still largely unanswered.

Views and concepts of functional occlusion must consider and account for the current knowledge of human mastication and chewing pattern type to establish efficacious guidelines concerning the optimal functional occlusion to achieve for each patient. The potential relationships between chewing pattern type, craniofacial morphology, static occlusion type, and functional occlusion type should be studied and evaluated to ascertain appropriate compatibility matches.

Questionable validity of the functional occlusion data

The validity of the functional data from research studies, as well as those from traditional gnathological functional occlusion recordings, is subject to question. The contrary research findings that show very different occlusal patterns during lateral excursions might reflect more a difference in methodology than the actual results from the studies.[143] Although many functional occlusion recordings can be demonstrated to be reliable, are they valid? For the most part, the recordings and measurements are static and not dynamic. Subjects are not typically asked to chew, swallow, or exercise any parafunctional movements. Subjects are usually asked to move or place their teeth or mandibles in a certain test position, and this static position is then recorded.[20,43,53] For instance, to record lateral eccentric jaw movements, a subject is typically asked to slide his or her mandible to a cusp-to-cusp end position (some 3-5 mm laterally), and this stationary border position is then recorded. Whether the subject actually functions in this position appears to be irrelevant. For instance, 1 person with a

more vertical chewing pattern might only function laterally 1 mm or so from centric occlusion, whereas another with a more horizontal chewing pattern can actually function in the more extreme lateral cusp-to-cusp border position (some 3-5 mm from centric occlusion). Because the extent of the lateral mandibular movements can vary from person to person, the question is how does a researcher or practitioner know a priori the extent of the lateral mandibular movement for each subject? (The lateral border movement might be 1 mm for 1 subject and 4 mm for another.) The laterotrusive records in research studies assume that all subjects naturally move and exercise mandibular movements similarly. Is this logical?

Another type of recording scheme used in research studies is to have each subject slide his or her mandible so many millimeters (maybe 3-5) to the right or left, rather than to the cusp-to-cusp border end position. Parenthetically, the range of lateral tooth contacts can vary up to 5 mm[62,144-150]; this represents half of the lateral width of an average tooth or the width of a cusp. However, Ingervall et al[151] stated that lateral excursive movements greater than 3 mm are probably rare, and tooth contacts closer to centric occlusion are more relevant.

A further variation is to record several different lateral eccentric movements at several progressive millimeter test positions to the right and then to the left.[20] Also, at times, the entire slide from centric to the laterotrusive cusp-to-cusp position is recorded rather than any particular millimeter movement to the right or left. In addition, studies and findings differ on how the occlusal contacts were recorded based on whether they were directly viewed or aided by an intermediate material such as impression compound, wax, articulating paper, or dental floss.[152] The location and severity of the occlusal contacts in any subject vary throughout the day.[153] A further consideration is whether functional recordings (static) are doctor manipulated or patient governed. In this regard, doctor-manipulated records are considered more reliable, but less valid and physiologic, than patient guided and generated records, and vice versa.[154-156]

An exception to the above were the recommendations many years ago of Masserman[157] and Masserman et al.[158] Masserman[157] recommended a technique for recording functional occlusion that involved the placement of 30-gauge green wax over half of the occlusal surfaces of the mandibular arch (and repeated for the other side). Then the subject was asked to eat an apple, and the cusp contacts were evaluated by observing the extent of the perforations in the occlusal wax on the nonworking side (opposite the side on which the apple was chewed). Masserman[157] explained: "While in conversation, the patient is asked to chew a section of an apple on the side opposite the wax only. This is done very casually and as the patient chews reflexly, he produces a functional recording of tooth contact in the wax." He believed that this method was far superior to using an articulator: "In the diagnosis or treatment, an occlusion should be proved on a functional level. . . . [R]egardless of the instrument employed or the technique used, every occlusion must be functionally validated in the mouth." He further argued that humans can never exactly duplicate on a conscious level functions that are naturally performed on a preconscious level. He stated:[157]

> [M]astication is a preconscious act. When patients are asked to record jaw movements on articulation paper, typewriter ribbon, wax, etc., the patient becomes confused in conflict between cortex (conscious) and brain stem (preconscious) function. . . . Stop a man walking, and ask him to show you how he walks. The resulting demonstration will be an awkward imitation of his natural gait. Accordingly, mandibular movements are at best a pantomime or mimicry of true functional movements. The recording is erroneous and results only in fallacious treatment.[157]

In addition, it is claimed that the type of occlusion (static or functional) is not as important as how the subject uses (or misuses) his or her occlusion.[27,123] Some people have the most perfect occlusions (both static and functional), and yet they have significant TMD,[159] whereas others have the most horrendous static and functional occlusions but no TMD. Furthermore, it is well known that the most destructive of all occlusal forces is that from parafunction (bruxing and clenching). Interestingly, this type of tooth contact is not evaluated in any functional occlusion study dealing with CPO or any gnathologic record used in contemporary clinical practice. Parenthetically, it was estimated that, under normal circumstances (swallowing and mastication), the teeth come in contact as little as 2 to 7 minutes per day in 1 study[160] and 15 to 40 minutes per day in another (possibly 2-6 hours with added parafunction).[27] Considering this, gnathologists can be criticized because they presumably do not record any relevant and meaningful dynamic aspects of mandibular movement—most importantly, patient parafunction.

DISCUSSION
Consideration of other functional occlusion types

If one accepts the rationale and validity of CPO (and this is highly debatable), there are still many other arguments against the general recommendation of this

type of functional occlusion for all orthodontic patients. First, individual functional occlusion schemes (eg, CPO) at best describe only partial and incomplete aspects of the true functional occlusion. That is, no functional occlusion type singularly describes the full and complete essence of human mandibular lateral eccentric movements. Even all the various functional occlusion schemes considered collectively do not approach the actual dynamic aspects of human mandibular motion. Thus, it does not appear to be too great a leap of faith that no 1 functional occlusion type is ideal for every patient. Ascribing to only 1 of the many functional occlusion types (ie, CPO) as superior and preferred for all patients might be fallacious.

The argument here is not so much against CPO per se as it is a call for consideration of other functional occlusion types that might be just as physiologic and healthy as CPO depending on each patient's unique stomatognathic characteristics. Therefore, it is possible that CPO is merely 1 of several optimal laterotrusive functional occlusion schemes. Isn't the "normal" in biology and physiology usually a range and not a single point or entity? Ash and Ramjford[27] stated: "Normal implies a situation commonly found in the absence of disease, and normal values in a biologic system are given with an adaptive physiologic range."

The recommendation of Isaacson[45] for a "biological concept of occlusion" some 3 decades ago seems prophetic today. His eclectic view was based on the premise that many functional occlusion types, besides CPO, could be biologic and physiologic for individual patients. That is, no single type of functional occlusion will be physiologic for every patient. For instance, he argued that, for a patient with periodontal bone loss involving the anterior teeth and who also bruxes, perhaps occlusal forces and stresses should be removed from these teeth and more force placed on the posterior teeth. The notion of a "biologic and physiologic concept of occlusion" might best be in keeping with the recommendation of the 1996 National Institutes of Health conference on TMD in which only a "gross" analysis of the occlusion (vs a detailed, microscopic analysis) was recommended.[161]

Also, irrespective of the type of functional occlusion established in a patient, how stable will it be in the future, especially CPO, when attrition of these teeth is inevitable with advanced age?[68] Will a CPO produced during orthodontics eventually evolve into a group function occlusion and then a balanced occlusion with posttreatment occlusal settling, wear, and continued facial growth and aging? Storey[162] wrote that "CPO will tend to become Group Function in time due to the wear of the maxillary canines."

Furthermore, the axiomatic notion that, to achieve a "physiologic occlusion" during orthodontics or prosthodontics, a practitioner must merely reestablish a previously healthy occlusal scheme (whether it be CPO, group function, or even perhaps balanced occlusion) is not logical and correct. The fallacy related to this type of dialectics is: when in time can a practitioner really be certain that the previous (or existing) functional occlusion is "physiologic" and healthy and worthy of "retracing" or "re-establishing"? That is, orthodontic treatment might have begun in the late mixed dentition when the deciduous canines (often with much incisal wear and attrition) are still present or during the very early stages of the development of the permanent dentition, and a final functional occlusion scheme has not yet been established or identified. Also, even if the preorthodontically treated functional occlusion is healthy and free of diseases or disorders, how can a clinician predict the future oral health of an occlusal scheme and determine it is worthy of copy?

Functional occlusion, gnathology, and TMD

It appears that some gnathologists are confident and dogmatic in their knowledge of the optimal type of functional occlusion to direct orthodontic patient treatment: "the goal of an excellent functional occlusion would be met by achieving Andrews' Six Keys, along with a seated condyle position and a mutually protected occlusion."[163] However, the evidence for this declaration and myopic view of functional occlusion has yet to be proven. Furthermore, this type of rhetoric is both naive and dangerous, particularly the general recommendation of this functional occlusion scheme for all patients. The self-serving notion that excellence in orthodontics can be accomplished only by achieving CPO is condescending to those who have a different viewpoint. The dogmatic, indiscriminant, and universal recommendation of CPO, and other gnathologic principles, has made orthodontists prisoners to the whims of this litigious society.

Perhaps orthodontists who have overly focused on the minute details of occlusion, including the need to establish CPO for all patients, should take a few steps back and thoroughly reflect on what they are doing. Do some long-held beliefs and techniques involved in gnathology seem logical today in light of evidence-based knowledge on occlusion and TMD? TMD is now considered a collection of disorders embracing many clinical problems that involve the masticatory muscles, joints, and associated structures.[19,63,88,90,92,161,164] The role of occlusion has been demonstrated to have less importance than once thought.[13,63,88,90,92,161,164] Studies in the 1960s and 1970s that placed inordinate

emphases on occlusion as causing TMD were found to lack control or comparison groups (poor diagnostic specificity). That is, there is poor diagnostic sensitivity and specificity of occlusal factors related to TMD.[165,166] In addition, condyle position has not been causally related to TMD. The centricity of the condyles in the glenoid fossa involves a range, and eccentricity does not necessarily indicate TMD.[13,63,90,161,164,167-171]

The evidence-based view on occlusion and TMD does not argue or conclude that occlusion (or condyle position) has no relevance to TMD or that orthodontists should ignore it.[63,90,161,164] What can be gleaned from the evidence-based paradigm is that occlusion is no longer considered the primary and only factor in the multifactorial nature of TMD. The gross evaluation and analysis of occlusion are still important in the diagnosis and treatment of TMD: "assessment of occlusion is necessary as part of the initial oral examination to identify and eliminate gross occlusal discrepancies such as those that may inadvertently occur as a result of restorative procedures."[161] McNamara et al[63] estimated the percentage contribution of occlusal factors to the multifactorial characterization of TMD at about 10% to 20% (and this might only be in an associational context, not cause and effect). They further stated: "Although a stable occlusion is a reasonable orthodontic treatment goal, not achieving a specific gnathologic ideal does not result in TMD signs and symptoms."[63] The evidence-based view on occlusion and TMD would include the amelioration of gross occlusal interferences that cause tooth mobility, fremitus, and deviation or deflection of mandibular closure and movement.[13]

A first step for experience-based orthodontists, who find themselves indoctrinated into many unproven gnathological precepts, would be to take a candid look at the evidence-based literature and then evaluate what they are doing that is different from the information in this body of knowledge. For instance, at present, there are 8 systematic reviews of literature (evidence-based model number 3—highest level, most compelling evidence) on the subject of TMD.[14,172] These reviews deal with TMD etiology, including the roles of occlusion and orthodontic treatment in relation to TMD, diagnostic imaging, and TMD treatments. From an evaluation of these 8 reviews, it can be concluded that occlusion and orthodontic treatment do not cause TMD, and occlusal adjustments are not recommended for the initial treatment of TMD.[172] This information certainly concurs with the views of the 2 national American ental Association conferences published in 1983[190] and 1990,[164] and the 1996 National Institutes of Health[161] conference on TMD.

Furthermore, the astute recommendation of Ash

and Ramjford[27] regarding what constitutes normal occlusion should be considered: "Normal occlusion . . . should imply more than a range of anatomically acceptable values; it should also indicate physiologic adaptability and the absence of recognizable pathologic manifestations . . . and the capability of the masticatory system to adapt to or compensate for some deviations within the range of tolerance of the system."

CONLUSIONS

As judged by the popularity of CPO, it appears that it is perceived as proven fact rather than 1 concept of functional occlusion. Nonetheless, Clark and Evans[143] emphatically stated: "The criteria that denote an 'ideal' functional occlusion have not been conclusively established." In addition, the terminology, nomenclature, and concept of CPO, as well as group function and balanced occlusions, can be challenged based on its questionable validity. Not all subjects actually function in the laterotrusive extreme border positions represented by this functional occlusion paradigm. Those who make, or accept, the claims of the superiority of the CPO paradigm over other worthy functional occlusion types have the burden of proof, and not vice versa.

The arbitrary selection of CPO for all patients ignores the value and importance of each person's unique stomatognathic and neuromuscular functional status. Other functional occlusion types and patterns might be just as acceptable as CPO. The important point here is that, irrespective of how you define patient's functional occlusion type, there should not be any occlusal interferences (vs contact). Perhaps patients with different craniofacial structures or chewing patterns might adapt better to 1 type of functional occlusion vs another. Furthermore, a person with parafunctional bruxing habits that have much side-to-side lateral excursive movements might welcome the lateral excursive freedom of group function or balanced occlusion. There is little evidence of benefit for establishing CPO in all orthodontic patients. The "at all cost" goal of attaining CPO and the deliberate elongation of the canines through orthodontic extrusion or resin buildup is unwarranted and possibly iatrogenic.

Ackerman[15] appropriately stated: "The challenge facing orthodontists in the 21st century is the need to integrate the accrued scientific evidence into clinical orthodontic practice." With this in mind, it is time for dentistry and orthodontics to take a fresh look at what is being taught and advocated in clinical practice in regard to functional occlusion. With evidence-based dentistry at the forefront of clinical practice, some old experience-based perfunctory approaches to functional

occlusion must be revisited and perhaps abandoned in favor of more valid evidence-based information.

A first step in accomplishing this goal would be to develop more appropriate and valid research in the area of functional occlusion. In this respect, there is a need to develop more sophisticated methods for recording functional occlusion including parafunction that are dynamic, rather than static. Next, practitioners must acquire model level 3 evidence-based (systematic review) information about occlusion and TMD and put it into practice. The periodic dental and orthodontic journals are the vehicles for the dissemination of these materials (some new journals focus entirely on this issue). The old-guard notion and practice of acquiescence to experience-based "gurus," who have little or no understanding of research, experimental design, and statistics, will be yesteryear. The hope of the new millennium is that the use of unproven tests, devices, and techniques in orthodontics will become extinct and replaced by scientifically verified concepts and procedures.

- A single type of functional occlusion has not been demonstrated to predominate in nature.
- CPO, as the optimal type of functional occlusion to establish in orthodontic patients, is equivocal and unsupported by the evidence-based literature.
- CPO might be merely 1 of several possible optimal functional occlusion types toward which to direct orthodontic patients' treatments.
- Group function occlusion and balanced occlusion (with no interferences) appear to be acceptable functional occlusion schemes depending on the patient's characteristics.
- The stability and longevity of CPO is questionable.
- Reestablishing a functional occlusion through orthondontic treatment back to the type of functional occlusion that existed before treatment is problematic.
- Consideration of a patient's chewing pattern shape, craniofacial morphology, static occlusion type, current oral health status, and parafunctional habit might provide important and relevant information about the most suitable functional occlusion type for each patient.

REFERENCES

1. D'Amico A. The canine teeth: normal functional relation of the natural teeth of man. J S Calif Dent Assoc 1958;26:6-23.
2. Scaife RR, Holt JE. Natural occurrence of cuspid guidance. J Prosthet Dent 1969;22:225-9.
3. Roth RH. The maintenance system and occlusal dynamics. Dent Clin North Am 1976;20:761-88.
4. Roth RH. Temporomandibular pain-dysfunction and occlusal relationship. Angle Orthod 1973;43:136-53.
5. Roth RH. Functional occlusion for the orthodontist. II. J Clin Orthod 1981;25:100-23.
6. Cordray FE. Centric relation treatment and articulator mountings in orthodontics. Angle Orthod 1996;66:153-8.
7. Chiappone RC. A gnathologic approach to orthodontic finishing. J Clin Orthod 1975;9:405-17.
8. Slavicek R. Interview on clinical and instrumental functional analysis for diagnosis and treatment planning. Part 1. J Clin Orthod 1988;22:358-70.
9. Slavicek R. Interview on clinical and instrumental functional analysis for diagnosis and treatment planning. Part 2. J Clin Orthod 1988;22:430-3.
10. Slavicek R. Instrumental analysis of mandibular casts using the mandibular position indicator. Part 4. J Clin Orthod 1988;22:566-75.
11. Crawford SD. Condylar axis position, as determined by the occlusion and measured by the CPI instrument, and signs and symptoms of temporomandibular dysfunction. Angle Orthod 1999;69:103-16.
12. Rinchuse DJ, Sweitzer EM, Rinchuse DJ, Rinchuse DL. Understanding science and evidence-based decision making in orthodontics. Am J Orthod Dentofacial Orthop 2005;127:618-24.
13. Rinchuse DJ, Rinchuse DJ, Kandasamy S. Evidence-based vs experience-based views on occlusion and TMD. Am J Orthod Dentofacial Orthop 2005;127:245-54.
14. Ismail AI, Bader JD. Evidence-based dentistry in clinical practice. J Am Dent Assoc 2004;135:78-83.
15. Ackerman M. Evidence-based orthodontics for the 21st century. J Am Dent Assoc 2004;135:162-7.
16. Ackerman M. The myth of Janus: orthodontic progress faces orthodontic history. Am J Orthod Dentofacial Orthop 2003;123:594-6.
17. Rinchuse DJ, Kandasamy S. Centric relation: a historical and contemporary orthodontic perspective. J Am Dent Assoc 2006;137:494-501.
18. Rinchuse DJ, Kandasamy S. Articulators in orthodontics: an evidence-based perspective. Am J Orthod Dentofacial Orthop 2006;129:299-308.
19. Rinchuse DJ, Rinchuse DJ. The impact of the American Dental Association's guidelines for the management of temporomandibular disorders in orthodontic practice. Am J Orthod 1983;83:518-22.
20. Tipton RT, Rinchuse DJ. The relationship between static occlusion and functional occlusion in a dental school population. Angle Orthod 1991;61:57-66.
21. O'Reilly MT, Rinchuse DJ, Close J. Class II elastics and extractions and temporomandibular disorders: a longitudinal prospective study. Am J Orthod Dentofacial Orthop 1993;103:459-63.
22. Rinchuse DJ. Counterpoint: prevention of adverse effects on the TMJ through orthodontic treatment. Am J Orthod Dentofacial Orthop 1987;91:500-6.
23. Rinchuse DJ. Counterpoint: a three-dimensional comparison of condylar position changes between centric relation and centric occlusion using the mandibular position indicator. Am J Orthod Dentofacial Orthop 1995;107:319-28.
24. Angle EH. Classification of malocclusion. Dent Cosmos 1899;41:246-64.
25. Angle EH. Malocclusion of teeth. 7th ed. Philadelphia: SS White Dental Mfg Co.; 1907.

26. Andrews LF. The six keys to normal occlusion. Am J Orthod 1972;62:296-309.

27. Ash MM, Ramjford S. Occlusion. 4th ed. Philadelphia: Saunders; 1996.

28. McLean DW. Physiologic vs pathologic occlusion. J Am Dent Assoc 1938;25:1583-94.

29. Woda A, Vigneron P, Kay D. Non-functional and functional occlusal contacts: a review of the literature. J Prosthet Dent 1979;42:335-41.

30. Gysi A. Masticating efficiency in natural and artificial teeth. Dent Digest 1915;21:74-8.

31. Kaplan RL. Concepts of occlusion. Gnathology as a basis for a concept of occlusion. Dent Clin North Am 1963;7:577-90.

32. Stuart CH, Stallard CE. Concepts of occlusion—what kind of occlusion should recusped teeth be given? Dent Clin North Am 1963;7:591-600.

33. Reynolds JM. The organization of occlusion for natural teeth. J Prosthet Dent 1971;26:56-67.

34. McAdam DB. Tooth loading and cuspal guidance in canine and group function occlusions. J Prosthet Dent 1976;35:283-90.

35. Lucia VO. Modern gnathological concepts up-dated. Chicago: Quintessence; 1983.

36. Schwartz H. Occlusal variations for reconstructing the natural dentition. J Prosthet Dent 1986;55:101-5.

37. MacMillan HW. Unilateral vs bilateral balanced occlusion. J Am Dent Assoc 1930;17:1207-20.

38. Schuyler CH. Factors contributing to traumatic occlusion. J Prosthet Dent 1961;11:708-16.

39. Alexander PC. Analysis of the cuspid protected occlusion. J Prosthet Dent 1963;13:309-17.

40. Mann AW, Pankey LD. Concepts of occlusion. The PM philosophy of occlusal rehabilitation. Dent Clin North Am 1963;7:621-36.

41. Beyron H. Occlusal relation and mastication in Australian aborigines. Acta Odontol Scand 1964;22:597-608.

42. Rinchuse DJ, Sassouni V. An evaluation of eccentric occlusal contacts in orthodontically treated subjects. Am J Orthod 1982;82:251-6.

43. Begg PR. Stone Age man's dentition. Am J Orthod 1954;40: 298-312.

44. DeShields RW. Gnathological considerations of a controversial nature. Dent Surv 1978;54:12-8.

45. Isaacson D. A biologic concept of occlusion. J Prevent Dent 1976;3:12-6.

46. Ismail J, Guevara P. Personal communications of unpublished data; 1974.

47. Weinberg LA. The prevalence of tooth contact in eccentric movements of the jaws. J Am Dent Assoc 1961;62:402-6.

48. Yuodelis RA, Mann WV Jr. The prevalence and possible role of nonworking contacts in periodontal disease. Periodontics 1965; 3:219-23.

49. Ingervall B. Tooth contacts of the functional and non-functional side in children and young adults. Arch Oral Biol 1972;17:191-200.

50. Gazit E, Liebermann MA. Occlusal contacts following orthodontic treatment. Angle Orthod 1985;55:316-20.

51. Sadowsky C, BeGole EA. Temporomandibular joint function and functional occlusion after orthodontic treatment. Am J Orthod 1980;18:201-12.

52. Sadowsky C, Polson AM. Long-term status of temporomandibular disorders and functional occlusion after orthodontic treatment: results of two long-term studies. Am J Orthod 1984;86: 386-90.

53. Rinchuse DJ, Sassouni V. An evaluation of functional occlusal interferences in orthodontically treated subjects. Angle Orthod 1983;53:122-30.

54. Shefter GJ, McFall WT Jr. Occlusal relationships and periodontal status in human adults. J Periodontol 1984;55:368-74.

55. de Laat A, van Steenberghe D. Occlusal relationships and temporomandibular joint dysfunction. Part I: epidemiologic findings. J Prosthet Dent 1985;54:835-42.

56. Ahlgren J, Posselt V. Need of functional analysis and selective grinding in orthodontics. Acta Odontol Scand 1963;25:3-13.

57. Egermark-Eriksson I, Carlsson GE, Magnusson T. A long-term epidemiologic study of the relationship between occlusal factors and mandibular dysfunction in children and adolescents. J Dent Res 1987;66:67-71.

58. Weinberg LA, Chastain JK. New TMJ clinical data and the implication on diagnosis and treatment. J Am Dent Assoc 1990;120:305-11.

59. McLean DW. Physiologic vs. pathologic occlusion. J Am Dent Assoc 1938;25:1583-94.

60. Nagao M. Comparative studies on the curve of Spee in mammals, with a discussion of its relation to the form of the fossa mandibularis. J Dent Res 1919;1:159-202.

61. Shaw DM. Form and function in teeth and a rational unifying principle applied to interpretation. Int J Orthod 1924;10:703-18.

62. Butler JH, Zander HA. Evaluation of two occlusal concepts. Parodontol Acad Rev 1968;2:5-19.

63. McNamara JA, Seligman DA, Okeson JA. Occlusion, orthodontic treatment, temporomandibular disorders: a review. J Orofac Pain 1995;9:73-90.

64. Heikinheimo K, Salmi K, Myllarniemi S, Kirveskari P. A longitudinal study of occlusal disorders at the ages 12 and 15. Eur J Orthod 1990;12:190-7.

65. VanBlarcom CW. The glossary of prosthodontic terms. J Prosthet Dent 1994;71:43-112.

66. Faulkner KD. Bruxism: a review of literature. Aust Dent J 1990;35:266-76,355-61.

67. Sved A. Equilibration as a post-retention measure. Dent Clin North Am 1960:815-20.

68. Weiland FJ. The role of occlusal discrepancies in the long-term stability of the mandibular arch. Eur J Orthod 1994;16:521-9.

69. Rinchuse DJ, Sassouni V. Functional occlusion evaluation of orthodontically treated and untreated subjects. Angle Orthod 1983;53:21-3.

70. Schaerer P, Stallard R, Zander H. Occlusal interferences and mastication: an electromyographic study. J Prosthet Dent 1967; 17:438-49.

71. Riise C, Sheikholeslam A. The influence of experimental interfering occlusal contacts on the postural activity of the anterior temporal and masseter muscle in young adults. J Oral Rehabil 1982;9:419-25.

72. Randow K, Carlsson K, Edlund J, Oberry T. The effect of an occlusal interference on the masticatory system. An experimental investigation. Odontol Rev 1976;27:245-56.

73. Magnusson T, Enbom L. Signs and symptoms of mandibular dysfunction after introduction of experimental balancing-side interferences. Acta Odontol Scand 1984;42:129-35.

74. Rugh JD, Barghi N, Drago CJ. Experimental occlusal discrepancies and nocturnal bruxism. J Prosthet Dent 1984;51:548-53.

75. Plata M, Barghi N, Rey R. Clinical evaluation of induced occlusal disharmonies. J Dent Res 1982;61:203-8.

76. DeBoever J. Experimental occlusal balancing-contact interference and muscle activity. An electromyography study with permanently applied electrodes. Parodontologie 1969;23:59-69.

77. Mohlin B, Ingervall B, Thilander B. Relationship between malocclusion and mandibular dysfunction in Swedish men. Eur J Orthod 1980;2:229-37.

78. Graham MM, Buxbaum J, Staling LM. A study of occlusal relationships and the incidence of myofacial pain. J Prosthet Dent 1982;47:549-55.

79. Bush FM. Malocclusion, masticatory muscle, and temporomandibular joint tenderness. J Dent Res 1985;64:129-33.

80. Droukas B, Lindee C, Carlsson GE. Occlusion and mandibular dysfunction: a clinical study of patients referred for functional disturbances of the masticatory system. J Prosthet Dent 1985;53:401-6.

81. Carlsson GE, Droukas BC. Dental occlusion and the health of the masticatory system. Cranio 1984;2:141-7.

82. Budtz-Jorgensen E, Luan W, Holm-Pedersen P, Fejerskov O. Mandibular dysfunction related to dental, occlusal and prosthetic conditions in a selected elderly population. Gerodontics 1985;1:28-33.

83. Ramfjord SR. Current concepts on the development of TMJ dysfunction. In: Carlson DS, McNamara JA, Ribbens KA, editors. Developmental aspects of temporomandibular joint disorders. Monograph no. 16. Craniofacial Growth Series. Ann Arbor: Center for Human Growth and Development; University of Michigan; 1985. p. 81-97.

84. Meng HP, Dibbets JMH, van de Weele L, Boering C. Symptoms of temporomandibular joint dysfunction and predisposing factors. J Prosthet Dent 1987;57:215-22.

85. Zarb G. Developmental aspects of temporomandibular joint disorders. In: Carlson DS, McNamara JA, Ribbens KA, editors. Developmental aspects of temporomandibular joint disorders. Monograph no. 16. Craniofacial Growth Series. Ann Arbor: Center for Human Growth and Development; University of Michigan; 1985. p.107-15.

86. Stringert HG, Worms FW. Variations in skeletal dental patterns with structural and functional alterations of the temporomandibular joint: a preliminary report. Am J Orthod 1986;89:285-97.

87. Pullinger AG, Solberg WK, Hollender L, Petersson A. Relationship of mandibular condylar position to dental occlusal factors in an asymptomatic population. Am J Orthod Dentofacial Orthop 1987;91:200-6.

88. Dolwich FW. Diagnosis and etiology of internal derangements of the temporomandibular joint. In: Griffiths RH, editor. The president's conference on the examination, diagnosis and management of temporomandibular disorders. Chicago: American Dental Association; 1983. p. 112-7.

89. McLaughlin RP. Malocclusion and the temporomandibular joint—a historical perspective. Angle Orthod 1988;85:185-91.

90. Griffiths RH. Report of the president's conference on examination, diagnosis and management of temporomandibular disorders. J Am Dent Assoc 1983;106:75-7.

91. Storey AT. Orthodontic treatment and temporomandibular function: the etiology of TM disorders. In: Carlson DS, editor. Craniofacial growth theory and orthodontic treatment. Monograph no. 23. Craniofacial Growth Series. Ann Arbor: Center for Human Growth and Development; University of Michigan; 1999. p. 106-7.

92. American Academy of Pediatric Dentistry, University of Texas Health Science Center at San Antonio Dental School. Treatment of temporomandibular disorders in children: summary statements and recommendations. J Am Dent Assoc 1990;120:265-9.

93. Solberg WK, Bibb CA, Nordstrom BB, Hanson TL. Malocclusion associated with temporomandibular joint changes in young adults at autopsy. Am J Orthod 1986;89:326-30.

94. Moyers RE. The development of occlusion and temporomandibular joint disorders. In: Carlson DS, McNamara JA, Ribbens KA, editors. Developmental aspects of temporomandibular joint disorders. Craniofacial Growth Series. Monograph no. 16. Ann Arbor: Center for Human Growth and Development; University of Michigan; 1985. p. 115-20.

95. Brandt D. Temporomandibular disorders and their association with morphologic malocclusion in children. In: Carlson DS, McNamara JA, Ribbens KA, editors. Developmental aspects of temporomandibular joint disorders. Monograph no. 16. Craniofacial Growth Series. Ann Arbor: Center for Human Growth and Development; University of Michigan; 1985. p. 296-7.

96. Williamson EH. Temporomandibular dysfunction in pretreatment adolescent patients. Am J Orthod 1977;72:429-33.

97. Mohlin B, Kopp S. A clinical study on the relationship between malocclusions, occlusal interferences and mandibular pain and dysfunction. Swed Dent J 1978;2:105-12.

98. Lieberman MA, Gazit E, Fuchs C, Lilos P. Mandibular dysfunction in 10-18 year old school children as related to morphological occlusion. J Oral Rehabil 1985;12:209-14.

99. de Laat A, van Steenberghe D, Lesaffe E. Occlusal relationship and temporomandibular joint dysfunction. Part II. Correlation between occlusal and articular parameters and symptoms of TMJ dysfunction by means of stepwise logistic regression. J Prosthet Dent 1986;55:116-21.

100. Nesbitt BA, Moyers RE, TenHave T. Adult temporomandibular joint disorder symptomatology and its association with childhood occlusal relations. In: Carlson DS, McNamara JA, Ribbens KA, editors. Developmental aspects of temporomandibular joint disorders. Monograph no. 16. Craniofacial Growth Series. Ann Arbor: Center for Human Growth and Development; University of Michigan; 1985.

101. Riolo ML, Brandt D, TenHave T. Associations between occlusal characteristics and signs and symptoms of TMJ dysfunction in children and young adults. Am J Orthod Dentofacial Orthop 1987;92:467-77.

102. Ricketts RM. Laminography in the diagnosis of temporomandibular joint disorders. J Am Dent Assoc 1953;46:620-48.

103. Perry HT Jr. Physiology of mandibular displacement. Angle Orthod 1960;30:51-60.

104. Thompson JR. Abnormal function of the stomatognathic system and its orthodontic implications. Am J Orthod 1962;48:758-65.

105. Berry DC, Watkinson AC. Mandibular dysfunction and incisor relationship. Br Dent J 1978;144:74-7.

106. Egermark-Eriksson I, Ingervall B. Anomalies of occlusion predisposing to occlusal interferences in children. Angle Orthod 1982;52:293-9.

107. Vanderos A. The relationship between craniomandibular dysfunction and malocclusion in white children with unilateral cleft lip and palate. Cranio 1989;7:200-4.

108. Agerberg A. Occlusal and temporomandibular joint relations: a comparative study. Cranio 1987;5:233-8.

109. Lubsen CC, Hansson TL, Nordstrom BB. Histomorphometric analysis of cartilage and subchondral bone in mandibular condyles in young human adults at autopsy. Arch Oral Biol 1985;30:129-36.

110. Takeda Y, Ishihara H, Kobayashi P. Influence of occlusal interferences on nocturnal sleep and masseter muscle activity (AADR abstract 561). J Dent Res 1989;68:391.

111. Lundh H, Westesson PL, Kopp S. A three-year follow-up of patients with reciprocal temporomandibular joint clicking. Oral Surg Oral Med Oral Pathol 1983;63:530-5.

112. Barghi N, Agular T, Martinez C, Woodall WS, Maaskant BA. Prevalence of types of temporomandibular joint clicking in subjects missing posterior teeth. J Prosthet Dent 1987;57:617-20.

113. Nilner M. Prevalence of functional disturbances and disorders of the stomatognathic system in 15-18 year olds. Swed Dent J 1981;5:189-97.

114. Egermark-Eriksson I. Mandibular dysfunction in children and in individuals with dual bite. Swed Dent J 1982;6:9-20.

115. Mohlin C, Carlsson B, Friling B, Hedgegard B. Frequency of symptoms of mandibular dysfunction in young Swedish men. J Oral Rehabil 1976;3:9-18.

116. Forssel H, Kirveskari T, Kangasniemi P. Response to occlusal treatment in headache patients previously treated to mock occlusal adjustment. Acta Odontol Scand 1987;45:77-80.

117. Kirveskari P, Le Bell Y, Salonen M, Forssell H, Grans L. Effect of elimination of occlusal interference on signs and symptoms of craniomandibular disorder in young adults. J Oral Rehabil 1989;16:21-6.

118. Kirveskari P, Alanen P, Jamsa T. Association between craniomandibular disorders and occlusal interferences. J Prosthet Dent 1989;62:66-9.

119. Krough-Poulsen WG, Olsson A. Occlusal disharmonies and dysfunction of the stomatognathic system. Dent Clin North Am 1966;19:627-35.

120. Egermark-Eriksson I. The dependence of mandibular dysfunction in children on functional and morphologic malocclusion. Am J Orthod 1983;83:187-94.

121. Cacchiotti D, Bianchi P, McNeil C, Plesh O. Use of the mandibular position indicator in TMJ disorder diagnosis (AADR abstract 1674). J Dent Res 1989;68:391.

122. Pullinger AG, Seligman DA, Solberg WK. Temporomandibular disorders. Part II. Occlusal factors associated with temporomandibular joint tenderness and dysfunction. J Prosthet Dent 1988;59:363-7.

123. Luther F. Orthodontics and the temporomandibular joint: where are we now? Part 2. Functional occlusion, malocclusion, and TMD. Angle Orthod 1998;68:305-18.

124. Lopez-Gavito G, Wallen TR, Little RM, Joondeph DR. Anterior open-bite malocclusion: a longitudinal ten-year postretention evaluation of orthodontically treated patients. Am J Orthod 1985;87:175-86.

125. Clark JR, Evans RD. Functional occlusion: I. A review. J Orthod 2001;28:76-81.

126. Williamson EH, Lundquist DO. Anterior guidance: its effect on electromyographic activity of the temporalis and masseter muscles. J Prosthet Dent 1983;49:816-23.

127. McDonald JWC, Hannam AG. Relationship between occlusal contacts and jaw-closing muscle activity during tooth clenching, parts I and II. J Prosthet Dent 1984;52:718-28, 862-7.

128. Shupe RJ, Mohammed SE, Christensen LV, Finger IM, Weinberg R. Effects of occlusal guidance on jaw muscle activity. J Prosthet Dent 1984;51:811-8.

129. Belser UC, Hannam AG. The influence of altered working-side occlusal guidance on masticatory muscles and related jaw movements. J Prosthet Dent 1985;53:406-13.

130. Boero RP. The physiology of splint therapy: a literature review. Angle Orthod 1989;59:165-80.

131. Graham GS, Rugh JD. Maxillary splint occlusal guidance patterns and electromyographic activity of the jaw-closing muscles. J Prosthet Dent 1988;59:73-7.

132. Besler UC, Hannam AG. The influence of altered working-side occlusal guidance on masticatory muscles and related jaw movement. J Prosthet Dent 1985;53:406-13.

133. Cecere F, Ruf S, Pancherz H. Is quantitative electromyography reliable? J Orofac Pain 1996;10:38-47.

134. Bush FM. Malocclusion, masticatory muscle, and temporomandibular joint tenderness. J Dent Res 1985;64:129-33.

135. Sarver DM. The importance of incisor positioning in the esthetic smile: the smile arc. Am J Orthod Dentofacial Orthop 2001;120:98-111.

136. Ahlgren J. Mechanism of mastication. Acta Odontol Scand 1966;24 (suppl 44):1-109.

137. Gibbs CH, Messerman T, Reswick JB, Derda HJ. Functional movements of the mandible. J Prosthet Dent 1971;26:604-20.

138. Green JR, Moore CA, Ruark JL, Rodda PR, Morvee WT, VanWitzenburg MJ. Develoment of chewing in children from 12 to 48 months: a longitudinal study of EMG patterns. J Neurophysiol 1997;77:2704-16.

139. Throckmorton GS, Buschang PH, Hayasaki H, Pinto AS. Changes in the masticatory cycle following treatment of posterior unilateral crossbite in children. Am J Orthod Dentofacial Orthop 2001;120:521-9.

140. Yurkatas A, Manly RS. Measurement of occlusal contact area effective in mastication. Am J Orthod 1965;35:185-95.

141. Owens S, Buschang PH, Throckmorton GS, Palmer L, English J. Masticatory performance and areas of occlusal contact and near contact in subjects with normal occlusion and malocclusion. Am J Orthod Dentofacial Orthop 2002;121:602-9.

142. Yamashita S, Hatch JP, Rugh JD. Does chewing performance depend upon a specific masticatory pattern? J Oral Rehabil 1999;26:547-53.

143. Clark JR, Evans RD. Functional occlusion: I. A review. J Orthod 2001;28:76-81.

144. Hickey JC, Allison ML, Woebel JB, Boucher CO, Stacy RW. Mandibular movements in three dimensions. J Prosthet Dent 1963;13:72-90.

145. Graf H, Zander HA. Tooth contact patterns in mastication. J Prosthet Dent 1963;13:1055-66.

146. Adams S, Zander HA. Functional tooth contacts in lateral and in centric relation. J Am Dent Assoc 1964;69:465-73.

147. Schaerer P, Stallard RE. The use of multiple radio transmitters in studies of tooth contact patterns. J Prosthet Dent 1965;3:5-9.

148. Glickman I, Pameifer JH, Roeber FW, Brion MA. Functional occlusion as revealed by miniaturized radio transmitters. Dent Clin North Am 1969;13:667-79.

149. Butler JH, Stallard RE. Physiologic stress and tooth contact. J Perio Res 1969;4:152-8.

150. Pameijer JA, Brion W, Glickman I, Roeber FW. Intraoral occlusal telemetry. Part V. Effect of occlusal adjustment upon tooth contact during chewing and swallowing. J Prosthet Dent 1970;24:492-7.

151. Ingervall B, Hahner R, Kessi S. Pattern of tooth contacts in eccentric mandibular positions in young adults. J Prosthet Dent 1991;66:169-76.

152. Taki A, Nakano M, Bando E, Hewlett ER. Evaluation of three occlusal examination methods used to record tooth contacts in lateral excursive movements. J Prosthet Dent 1993;70:500-5.

153. Berry DC, Singh BP. Daily variation in occlusal contacts. J Prosthet Dent 1983;50:386-91.

154. Helkimo M, Ingervall B, Carlsson GF. Comparison of different methods in active and passive recordings of the retruded position of the mandible. Scand J Dent Res 1973;81:265-70.

155. Helkimo M, Ingervall B, Carlsson GE. Variation of retruded and muscular position of the mandible under different recording conditions. Acta Odontol Scand 1971;29:423-7.

156. Lundeen HC. Centric relation records: the effect of muscular action. J Prosthet Dent 1974;31:244-9.

157. Masserman T. A concept of jaw function with a related clinical application. J Prosthet Dent 1963;13:130-40.

158. Masserman T, Reswick JB, Gibbs C. Investigation of functional mandibular movements. Dent Clin North Am 1969;13:629-42.

159. Behrents R. JCO interviews Dr. Rolf Behrents on adult craniofacial growth. J Clin Orthod 1986;20:842-7.

160. Alpern MC. The ortho evolution—the science and principles behind fixed/functional/splint orthodontics. New York: GAC International; 2003.

161. National Institutes of Health Technology Assessment Conference Statement. Management of temporomandibular disorders. J Am Dent Assoc 1996;127:1595-606.

162. Storey AT. Functional stability of orthodontic treatment: occlusion as a cause of temporomandibular disorders: In: Nanda R, Burstone CJ, editors. Retention and stability in orthodontics. Philadelphia: Saunders; 1993. p. 203-15.

163. Giardot RA, Redmond WR. Management and marketing—one pathway to successful orthodontic practice. J Clin Orthod 2005;39:415-8.

164. McNeil C, Mohl ND, Rugh JD, Tanaka TT. Temporomandibular disorders: diagnosis, management, education, and research. J Am Dent Assoc 1990;120:253-60.

165. Mohl ND. Temporomandibular disorders: role of occlusion, TMJ imaging and electronic devices—a diagnostic update. J Am Coll Dent 1991;58:4-10.

166. Mohl ND, Dixon C. Current status of diagnostic procedures for temporomandibular disorders. J Am Dent Assoc 1994;125: 56-64.

167. Gianelly AA. Orthodontics, condylar position, and TMJ status. Am J Orthod Dentofacial Orthop 1989;95:521-3.

168. Gianelly AA, Hughes HM, Wohlgemuth P, Gildea C. Condylar position and extraction treatment. Am J Orthod Dentofacial Orthop 1988;93:201-5.

169. Gianelly AA. Condylar position and Class II deep bite, no overjet malocclusion. Am J Orthod Dentofacial Orthop 1989; 96:428-32.

170. Gianelly AA, Cozzani M, Boffa J. Condylar position and maxillary first premolar extraction. Am J Orthod Dentofacial Orthop 1991;99:473-6.

171. Gianelly AA, Anderson CK, Boffa J. Longitudinal evaluation of condylar position in extraction and nonextraction treatment. Am J Orthod Dentofacial Orthop 1991;100:416-20.

172. Rinchuse DJ, McMinn J. Summary of evidence-based TMD systematic reviews. Am J Orthod Dentofacial Orthop 2006 (in press).