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Biological Adaptation and Normative Values

Barry J. Sessle, BDS, MDS, BSc, PhD, DSc(hc), FRSC

What Do We Know?

Biologic Determinants

The occlusal interface is often considered in terms of a structural entity, and to reflect several structural components of the masticatory system: alveolar and cranial bone, the temporomandibular joint (TMJ) including its disc and associated ligaments, the dentition (both pulp and periodontium), and the masticatory muscles (including ligaments and tendons and other connective tissues as well as contractile skeletal muscle fibers). However, a number of intrinsic physiologic processes act upon the masticatory system and collectively determine its parameters of function and thereby the occlusal interface. As shown in the working schema (Fig 1) provided for discussions at the symposium, these physiologic determinants include cardiovascular, endocrine, immunologic, metabolic, and neural (both peripheral [eg, receptors, nerve fibers] and central nervous system [CNS]) influences.[1–6]

These intrinsic determinants are themselves acted upon by an array of environmental and genetic factors that thereby influence the function of the masticatory system. Environmental influences include mechanical stress and trauma, disease, nutrition, and "life events" associated with occupational loading of tissues, psychologic stress, socioeconomic conditions, etc. Genetic factors encompass gene-based determinants of development and aging, gender, and homeostatic processes. As noted in Fig 1, some of these factors (eg, mechanical stress, trauma) can act directly on components of the masticatory system. Most times, however, these determinants act indirectly through their influence on the intrinsic processes; they can also influence each other (eg, genetic abnormalities resulting from environmental insults).[1–3,5,7–9]

Adaptive Potential

The masticatory system has a remarkable capacity to adapt to these various influences, and it has considerable functional reserve. Figure 1 outlines some examples of such "positive" responses to mechanical loading, infection, or trauma of craniofacial tissues; they include TMJ remodeling, reparative dentin, a host of protective reflexes (eg, nociceptive jaw opening, airway maintenance, tongue-protrusion reflexes), and acute pain and associated reversible nociceptive changes in the CNS. But several factors have been demonstrated or invoked to be risk or predisposing factors that can compromise these adaptive responses, and as a consequence, the functional reserve of the tissues may be exceeded. The level of evidence, however, varies in the extent to which these factors do compromise the masticatory system. Nonetheless, such influences identified in the literature include aging, disease, genetic abnormalities, gender, parafunctions, psychologic stress and neurologic dysfunction, nutritional inadequacies, and macro- or microtrauma and overloading of tissues.

The threshold of the adaptive potential of the tissues may be exceeded and be associated with a number of "negative" responses (Fig 1). These negative outcomes would include such pathophysiologic manifestations as chronic pain (eg, temporomandibular disorders [TMD]) that is thought to reflect neuroplastic changes manifesting exaggerated nociceptive responses and behavior, damage or disease of TMJ tissues (eg, disc tear or perforation, osteoarthritis) or the teeth (eg, dental caries, periodontal disease), maladaptive behavior (eg, bruxism), and neurologic disorders (eg, tardive dyskinesia, Parkinsonism).[1–5,7,9,10] All these outcomes can be associated with serious impairment of the function of the masticatory system and thereby influence the occlusal interface.

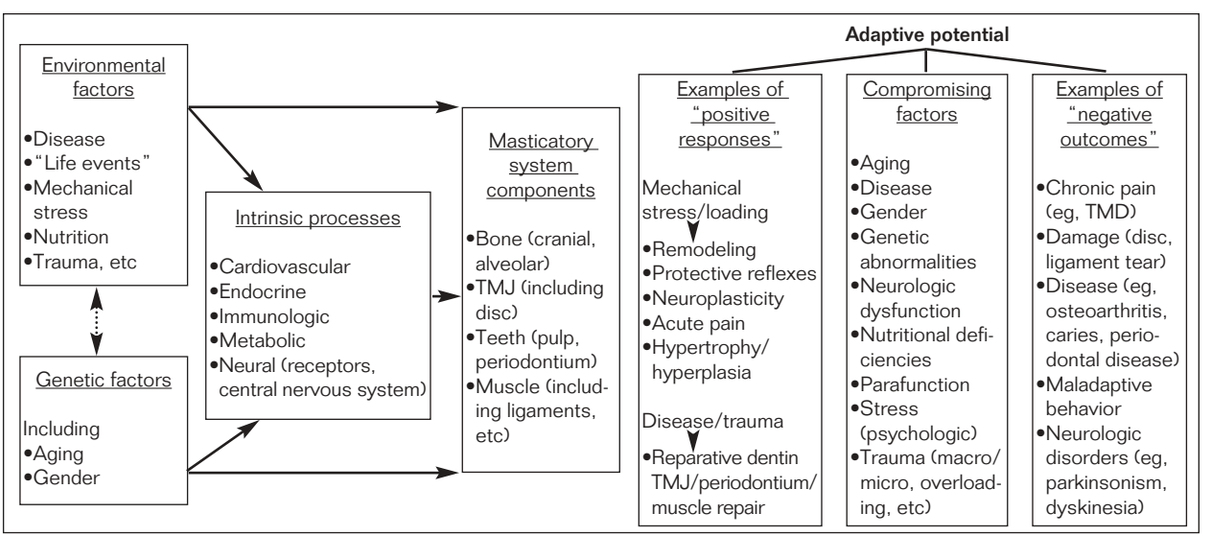

Fig 1 Biologic determinants and adaptive potential.

What Do We Not Know?

While the various components of the masticatory system are well-known and the environmental and genetic factors acting directly or indirectly (through "intrinsic" processes) on them have been broadly identified, the mechanisms by which many of these factors operate have only partly been elucidated. Furthermore, many of the influences noted above that have been invoked as possible compromising factors on adaptive capacity have a limited scientific basis.

Examples of "what we do not know" in a comprehensive manner include:

- Biologic reactions to mechanical stress and trauma
- Immunologic influences and neuroimmune interactions
- Molecular, physiologic, and genetic basis of chronic pain
- Molecular, physiologic, and genetic basis of the aging process
- Molecular, physiologic, and genetic basis of gender differences
- Neural basis of maladaptive behavior and neurologic dysfunction
- Importance of these factors in compromising the adaptive capacity of the masticatory system

What Research Strategies Are Needed?

Some examples of research directions that might be followed to address these deficiencies in knowledge related to biologic adaptation and normative values for the occlusal interface and other components of the masticatory system are outlined below.

- Determine the molecular mechanisms used by cells of the masticatory system to synthesize, maintain, and degrade the extracellular matrix and tissues; these processes encompass genetic regulation and the effects of hormones, mediators, pharmacologic agents, mechanical loads, aging, and developmental changes.
- Define the molecular and cellular composition of each tissue (eg, expression of genes, level of proteins) in normal and dysfunctional states.
- Clarify the mediators and inhibitors of inflammation; this would include processes of immunologic modulation of normal tissue function as well as those involved in injury, degeneration, and repair.
- Define the structural (eg, dimensions, anatomy), biomechanical (eg, displacement, force, stress, strain, plasticity, cycles), and physical (eg, density, modulus, conductivity) parameters for normal and dysfunctional conditions.
- Determine the neural mechanisms involved in the detection and regulation of the functional and dysfunctional states of the masticatory system; this encompasses the processes involving nociceptors, other somatosensory receptors, the autonomic nervous system, and the influences of the peripheral nervous system on tissue repair. It also includes the role of the central as well as peripheral nervous systems in regulating jaw function in both health and disease, since both joint loading and joint and occlusal stabilization depend on neural feedback.
- Define the role of cellular, humoral, genetic, neural, and neuroendocrine/hormonal factors associated with biologic responses to materials used in masticatory and TMJ repair and restoration of function.
- Elucidate neuroplasticity at molecular, cellular, physiologic, and pharmacologic levels in normal conditions and in response to injury or placement of appliances in the craniofacial region; this would include consideration of various neurotransmitters, neuromodulators, transcellular and intracellular messengers involved in nociception and plasticity both in peripheral tissues and within the CNS, and factors involved in the transition from acute to chronic pain.
- Clarify the effects of systemic, genetic, aging, and hereditary diseases on structure and function of the TMJ, muscle, and periodontium and the processes underlying associated developmental aberrations.
- Delineate those factors related to sex or gender differences that predispose women to develop TMD and related craniofacial pain conditions and other dysfunctional states.
- Develop indicators (markers) for disease or dysfunctional processes affecting the periodontium, muscle, and TMJ. These indicators should include markers of early pathology (eg, molecular markers and other diagnostic markers such as imaging) and differentiate between adaptive processes and pathologic states.

What Needs Highlighting in Educational Programs?

All topics listed in the first section should be presented in educational programs for dentistry students. A more in-depth outline of these topics should be provided for prosthodontic students, including an emphasis on the limitations of current evidence and gaps in knowledge and some possible research strategies to address them (as discussed above).

Satisfactory Occlusal Relations for the Individual with a Craniofacial Anomaly

Bruce Ross, DDS, MSc

Introduction

It is possible to describe in a biologic and mechanical way the elements of a "perfect" occlusion. This is a valuable concept for diagnosis and planning the correction of variations from the ideal. However, since perfect occlusions are relatively rare in normal populations, it would seem that nature does not require such perfection. The clinical problem is to know what compromises will still provide a fully functional, healthy, and esthetic dentition for a lifetime.

What Do We Know?

Providers of dental services often apply unrealistic criteria as treatment goals. Surgeons and speech pathologists know they cannot achieve perfection and accept excellence or worse, depending on the original condition. When the malocclusion is mild, it is reasonable for the clinician to disdain treatment that would produce a less-than-ideal result. For many clinicians, this occurs for reasons of personal pride, and not necessarily with the patient's best interests in mind. Yet an analysis of the dentitions of a large group of orthodontists or prosthodontists would reveal a large percentage with untreated, imperfect occlusions.

The individual with a congenital anomaly of the craniofacial complex, whether it is a deficiency, excess, or deformation, has problems that are somewhat unusual. Treating the extremes of malocclusion, however, provides great insight into the nature of a satisfactory occlusion and the minimum requirements for achieving it. Actual problems, not deviations from normal, should be treated. In hemifacial microsomia, for example, the condition is usually unilateral, often with a mandibular condyle and ramus that are severely dysplastic or even absent, no temporomandibular joint, no temporal fossa, and severely hypoplastic or virtually absent muscles of mastication. Yet these patients routinely have an occlusion that has developed well in this distorted environment, have no associated pain or discomfort, can chew and swallow efficiently, and can speak with normal articulation. Apart from the loss of hearing on the affected side, there may be no dysfunction except in the narrowest of definitions: that the temporomandibular joint is absent or grossly dysplastic and mandibular function is not "normal." The primary goal of treatment is to establish optimum facial esthetics, in the course of which there is usually very favorable surgical repositioning of the jaw(s) that permits the orthodontist to realign the teeth in the new jaw position and achieve an excellent occlusion. Treatment is usually for sociologic rather than biologic indications.

The patient's priorities must be given primary importance. In cleft lip and palate, for example, the top priorities are good speech and a healthy self-esteem. With these, the child can grow to enjoy a normal life. The next priority is the appearance, which is a function of the extent of the original nose and lip deformity and the skill of the reconstructive surgeon. Further down the list is the need for a healthy, reasonably functional, and esthetic occlusion. While the dentition contributes to self-esteem, appearance, and speech, it is mainly the maxillary anterior teeth that contribute to those goals.

The most satisfactory approach to these complex problems is the multidisciplinary team. Interdisciplinary communication and treatment planning are essential—not only learning what the other specialists are doing, but when they will do it, what their treatment will do for our treatment, and what our treatment will do to assist them. One of the major benefits of the team approach is discovering the capabilities and limitations of each specialty so the clinician can incorporate or seek assistance from surgical, medical, and paramedical specialists and avoid tunnel vision.

In cleft lip and palate, for example, there are several ways to manage the dental problems arising from a congenital cleft of the anterior maxilla and alveolus with the usual absence of the lateral incisor in the cleft region. The surgeon's first commitment is to repair the oronasal fistula. An inexperienced prosthodontist might feel that a fixed or removable prosthesis would then give the best possible result. The instability of the maxillary segments, however, would preclude long-term success, and the surgeon would therefore be induced to include an autogenous bone graft to the repair to unify the segments. The prosthodontist could then proceed, or an osseointegrated implant might be used to replace the lateral incisor. The orthodontist, however, might feel that the space is amenable to closure by orthodontic tooth movement through the graft, thus negating the need for any prosthesis but leaving the canine to act as a lateral incisor. If the space is very wide, the orthodontist and surgeon might plan on orthognathic surgery to advance the posterior segment so that the canine contacts the central incisor and then unite the two segments with a bone graft. This would result in an intact, stable maxilla and intact natural dentition in excellent occlusion with no prosthesis required. The canine-central situation is not "perfect," but it is excellent with minor adjustments.

Treatment is often undertaken too soon. A good initial result may be obtained, but very often further growth results in relapse and treatment must be redone later. The total time and effort expended on attempting to maintain an excellent occlusion throughout childhood cannot be justified in terms of extra expense, squandering of patient and parent cooperation, and trauma and inconvenience to the child. Compliance is a serious problem with severe malocclusions. After a childhood of almost constant treatment from a wide variety of specialists, rebellion sometimes occurs in adolescence. Orthodontists and prosthodontists are limited to the existing jaw relations, and if a patient refuses orthognathic surgery, it is impossible to treat without great compromise.

What Do We Not Know?

Surprisingly, one serious problem with multispecialist treatment in severe cases is excessive treatment. First, we would like to know the most efficacious method giving the best

long-term results. Unfortunately, most treatment is the result of trial and error. Only fairly recently has a major incentive for quality outcomes research arisen. Such research is sparse at present, and sometimes of poor quality. Many clinicians seem inclined to accept only results that confirm their own beliefs. Second, we would like to know the most efficient method providing the best results with the simplest technique and fewest interventions. With severe cases, there is a tendency to micromanage treatment, which is wasteful of personnel and facilities without improving results.

What standards are essential as opposed to merely desirable? Normal teeth and jaws should satisfy certain criteria: *(1)* a well-functioning occlusion (not necessarily perfect) where food is masticated sufficiently (whatever that is) and swallowed; *(2)* unimpeded breathing and speech; *(3)* dentition and supporting structures free from pathology and maintainable in comfort for life; and *(4)* an appearance that is good enough to the person and to the society in which he or she lives. "Good enough" is not necessarily a derogatory term.

What are the long-term factors for success? What predictions of relapse related to the soft tissue effects, especially lips, cheeks, and tongue, can be made? These are known in general, but individual responses are difficult to predict. Relapse tendencies of implants for adolescents are also not fully known.

What Research Strategies Are Needed?

- Expose dental specialists to multidiscipline teams during their training.
- Encourage treatment outcome studies and improve the quality.
- Especially encourage studies on long-term results.

The Rationale for Management of Morphologic Variations and Nonphysiologic Occlusion in the Young Dentition

S. Ross Bryant, BSc, DDS, MSc, PhD, FRCD(C)

What Do We Know?

Assessment

Nonphysiologic. Signs or symptoms of abnormal function.[1]

Nonphysiologic occlusion. Lack of functional adaptation in masticatory system that is difficult to define based on abnormal form/morphologic deficit.[2]

- Signs or symptoms of occlusal instability or lack of adaptation in teeth, periodontium, joints, or muscles.[2]
- Typically involves missing, malformed, or otherwise deficient dental, osseous, or neuromuscular structures, and may involve a lack of patient satisfaction with function or esthetics.[2]

Malocclusion. A dental occlusion typified by variation from ideal form/morphology.[2,3]

- Not necessarily associated with lack of functional adaptation, but often impairs facial appearance.[3]
- Originally, Angle[4] classification—Class I, II, and III corresponding to anteroposterior position of first molar and alignment of teeth around the arch perimeter.
- Now, variation about one or more of the three planes of space—orbital (sagittal or anteroposterior variation), midsagittal (transverse or lateral variation), and Frankfurt (vertical variation).[5–7]

Etiology. Genetic/environmental factors mediated through dental, osseous, or neuromuscular sites.[3]

- Specific genetic and environmental agents cannot usually be identified.[5]
- Environmental factors, eg, mandibular trauma, parafunctional habits, early loss of primary teeth.[5]
- Genetic factors, eg, congentially missing teeth, jaw growth direction, specific genes not known.

Distribution

Normative data. Available for anteroposterior and vertical relationships from lateral cephalometric studies.[8]

- Also available for transverse relationships and tooth size prediction and comparison.[9,10]
- Appearance and physiologic occlusion are complex concepts not lending to normative study.

Malocclusion. Ideal occlusion is unusual; nonetheless, functional adaptation is the norm.

- Approximately 70% of North American youths have malocclusion.[11,12]
- Malocclusion Angle Class I (50% to 55%), Class II (15% to 20%), and Class III (less than 1%).[11,12]
- The most common type of malocclusion is malalignment (crowding) with Angle Class I.[5]

Missing teeth. Some children have missing or malformed dental, osseous, or neuromuscular structures.

- 2% to 10% of children have congenitally missing permanent teeth.[13,14]
- Usually, up to two teeth are congenitally missing; premolars more commonly missing than lateral incisors.[13]
- Oligodontia with six or more congenitally missing teeth occurs in fewer than 1 in 1,000 children.[15]
- Teeth missing because of trauma may be even less common in youth, but typically involve incisors.[16]

Impact

Psychosocial impact. Malocclusion is thought to manifest a negative psychosocial impact, mainly through perceptions of facial appearance as influenced by personal and societal values.[17,18]

- Variations in tooth position correlate well with facial attractiveness among American youths, and malaligned or protruding teeth can yield negative social status.[18]
- Similar handicap experiences have been found in other developed countries[19]; 7% of Welsh children are teased at least once a week about tooth appearance.[20]
- Psychosocial debilitation of malaligned teeth may be disproportionately worse than anticipated.[21]
- Impact of congenitally missing teeth may not be obvious; only 15% of afflicted children have esthetic complaints,[22] a finding that likely relates to the typical location and extent of deficit.

Functional impact. Severe malocclusion, although relatively uncommon, can compromise chewing function in the young dentition.[23]

- Negative functional impact of malalignment and other mild malocclusion has not been established.
- Depending on location and extent of the deficit, the functional impact of congenitally missing teeth may be minimal; 40% of children with congenitally missing teeth have no complaints.[24]
- More extensive childhood deficiencies in teeth and other oral structures, often associated with rare syndromes such as ectodermal dysplasia, variously yield both esthetic and functional problems.[14]

Future oral deterioration and disease. Malocclusion has been thought to increase the likelihood of future oral disease or deterioration; however, studies have not borne this out as a major problem.

- There is an elevated 1 in 3 risk that a child with a Class II malocclusion and protruding maxillary teeth will traumatize maxillary incisors enough to cause fracture or devitalization.[24]
- A severe Class II malocclusion with an impinging overbite, although relatively uncommon, may also predispose the maxillary incisors to lingual tissue trauma and attachment loss.[5]
- It has been hypothesized that malalignment of teeth predisposes to both caries and periodontal disease; however, evidence does not defend the supposition.[25,26]
- Risks of tooth wear and temporomandibular disorders (TMD) have also been attributed to malocclusion; however, scientific evidence questions any essential role that malocclusion has in the pathophysiology of either condition.[27–31]

Management

Treatment strategies. Abnormal occlusal form and function can be managed as needed with orthodontic, surgical, operative, and/or prosthodontic treatment.[2,5]

- In developed countries, childhood malocclusion is often treated orthodontically to improve dentofacial esthetics and the distribution of occlusal stress in the masticatory system, and occasionally to align teeth and edentulous segments in preparation for prosthodontic rehabilitation.[2,5]
- Prosthodontic treatment is often implemented as a form of salvage for the functional and esthetic compromises that can result from missing or damaged teeth and oral structures.[2]
- Morphologic criteria do not appear to satisfactorily identify when intervention is needed.
- The masticatory system normally demonstrates substantial adaptive capacity, particularly in youth.
- A major bioethical obligation in any treatment intervention is "above all do no harm."
- A potential biologic price is inherent in all treatment strategies.
- We lack long-term studies comparing treatment and no treatment for abnormal occlusion.
- Occlusal treatment should be minimized where occlusal stability/functional adaptation are evident.
- Functional adaptation following treatment is an inadequate justification for it.

Gnathologic approach. Highly adjustable articulators have been argued to better enable location of the condyles in a predetermined centric relation position in precise harmony with centric and eccentric occlusal contacts.[32]

- Based in early prosthodontic literature (Table 1),[33,34] this approach has recently been promoted such that orthodontics is about doing a "full mouth reconstruction in enamel."[35]
- Nearly ubiquitous "malarticulation," and the failure to adhere to gnathology, is said to lead to oral deterioration including tooth wear, TMD, pulpitis, periodontal disease, and orthodontic relapse.
- It does appear that the gnathologic approach can provide a physiologic endpoint; however, it does not appear to be necessary for achieving an esthetic and functional therapeutic occlusion.

Functionalist approach. Treatment involving occlusal changes—orthodontic, prosthodontic, or otherwise—should optimize function and appearance in keeping with a physiologic occlusion.[2,36]

- Based in early prosthodontic literature (Table 1),[37] this approach took on a practical tone with Beyron's proposals[38,39] for the optimal morphologic objectives of a therapeutic occlusion as follows: *(1)* acceptable interocclusal distance; *(2)* stable tooth-to-tooth contact with axially directed forces on posterior teeth; *(3)* bilateral

Table 1 Theories on Managing Abnormal Occlusal Function and Form

Study	Year	Subject
Prosthodontic theory		
McCollum[33]	1938	Gnathology, malarticulation, balanced occlusion
Schuyler[37]	1929	Balanced occlusion may not suffice for natural dentition
Beyron[38]	1954	Characteristics of functionally optimal occlusion
Mohl et al[2]	1988	Physiologic occlusion
Hobo and Takayama[32]	1997	Gnathologic occlusion
Orthodontic theory		
Angle[4]	1900	Malocclusion in A-P plane, alignment
Simon[6]	1922	Malocclusion in three planes
Ackerman and Proffit[7]	1969	Esthetics, alignment, symmetry, malocclusion in three planes
Roth[35]	1995	Gnathologic complete-mouth rehabilitation in enamel
Rinchuse[36]	1995	Esthetics, functional occlusion

centric contacts; *(4)* freedom in retrusive range with intercuspal position at or anterior to retruded contact position; and *(5)* multidirectional freedom of occlusal contact.

- It does appear that Beyron's approach can provide variable scope for achieving an esthetic, functional, and physiologic therapeutic occlusion across disciplines; however, sound evidence to support this is lacking.

What Do We Not Know?

Theories abound, but evidence is short. The lack of evidence certainly pervades clear and unequivocal morphologic, functional, and psychosocial criteria for defining physiologic and nonphysiologic occlusions in the young dentition, especially in the context of biologic and psychosocial adaptation to variations in form and function.

In the absence of intervention, we do not know if the various proposed criteria of occlusion are either necessary or sufficient for the maintenance of a physiologic occlusion in the young dentition, and we do not know what variations in occlusal form and function can be adapted to in an individual and why. We do not know the relative importance of the various proposed features for therapeutic occlusal interventions in the young dentition in relation to their effect on long-term outcomes, including the relationship between the intervention and its biologic price.

What Research Strategies Are Needed?

Additional normative studies of the form, function, and disability related to young and aging dentitions in various human societies are required. Continued research is needed into the features and mechanisms underlying biologic and psychosocial coping and adaptation with variations in human occlusal form and function. Also required are additional efficacy and effectiveness studies (both randomized controlled trial and cohort studies including community-based cohort studies) of the biologic and psychosocial outcomes of various therapeutic occlusal interventions (including nonintervention strategies) used to manage variations in occlusal form and function in the young dentition in various human cultures and societies.

What Needs Highlighting in Educational Programs?

We need to continue to improve undergraduate and graduate programs in their mandate to promote evidence-based decision making for diagnosing and treating problems in the young masticatory system (or indeed any dentition), even when the evidence is not definitive. We need to maintain constant vigilance in maintaining at least an equal priority on the questions of why and when to intervene and when not to intervene, compared to the more popular and commercial question of how to intervene.

Determinants of a Healthy Aging Dentition: Maximum Number of Bilateral Centric Stops and Optimum Vertical Dimension of Occlusion

Winfried Walther, Dr Med Dent, PhD

Introduction

In 1969 Beyron stated that the determinants of a healthy aging dentition are a maximum number of bilateral centric stops and an optimum vertical dimension of occlusion.[1] This approach should be analyzed considering contemporary results of dental research.

What Do We Know?

Occlusal Philosophies and Knowledge About Occlusal Contacts

Teachers of occlusal philosophies emphasized the location, size, distribution, and number of occlusal contacts, starting from the concept that an ideal occlusion can be found.

Clinical investigations have established knowledge about the means of recording occlusal stops and the variations in occlusal contacts.

Occlusal indicators vary, and their markings may not be reproducible. A gold standard for recording occlusal contacts has yet not been established.[2–5] Occlusal contacts change throughout the day and over longer intervals and depend on the pressure and physical state of the masticatory system.[3,6,7] The location of occlusal contacts found in clinical studies differs from theoretic considerations, ie, from the concept of tripodism.[8]

Number of Occlusal Contacts

In an elderly population, approximately five contacts on each side of the posterior region of the tooth arch are to be found. Short-span fixed appliances exhibit more occlusal contacts than do longer span prostheses.[9] In young adults, the number of occlusal contacts relates with masticatory muscle activity.[10]

Maximum Number of Bilateral Centric Stops

The concept of providing the patient with the maximum possible number of bilateral centric stops has been challenged by the concept of the shortened arch. There is evidence that the shortened dental arch consisting of anterior and premolar teeth can provide adequate functional rehabilitation.[11,12]

Occlusion and the Development of Situations Leading to Symptoms

Changes of the occlusal contact pattern toward a traumatizing contact may lead to periodontal alterations. The effect of trauma from occlusion has been evaluated in animal models. A traumatic occlusion leads to increased mobility and reversible alterations of the periodontal apparatus.[13] There is support for the assumption that a stable occlusion in the intercuspal position is an essential prerequisite for the maintenance of extended fixed partial dentures on periodontally compromised abutment teeth.[14]

In rare instances, patients complain about continuous discomfort after restorative dental treatment because of the lack of familiarity of their own bite (phantom bite syndrome). Treatment success is rarely, if ever, obtained.[15]

Optimum Vertical Dimension

The difference between vertical dimension of occlusion and rest position (clinical freeway space) has been stated to be of decisive value for diagnostic and restorative procedures. Postural jaw position varies within the same person and is influenced by body posture, speech, and emotional tension.[16] Measures of clinical freeway space depend on the method used.[17] Electromyographic monitoring of the jaw muscles has not been proven to allow diagnostic decisions.[18] The patient has a good chance of adapting to an increase in vertical dimension.[19]

What Do We Not Know?

The variability of the results described does not support the assumption that the clinical reality of teeth and jaw relations can be described using mechanistic models.

To develop the rules of an "optimal occlusion," the authors of occlusal philosophies interpreted anatomic findings. They assumed that these needed no further substantiation.[20,21] Such rules were taught and applied without prior scientific investigation. As a consequence, the scientific thinking of the dental profession appears to be retarded with regard to occlusal treatment and diagnosis. Furthermore, there is a gap between the clinician who performs restorative treatment as a matter of routine using (mostly) reliable conventional techniques and the scientist who is preoccupied with problems that do not occur in the dental office.

To close this gap, prosthodontists should try to find scientific questions that help to resolve clinical problems. The aim should be to compile information that helps clinicians to organize their restorative treatment in a more efficient way. Dental scientists need indicators, ie, quantitative measures, for monitoring clinical care to validate the appropriateness of clinical procedures. The clinician demands criteria to assess the individual case with regard to restorative treatment needs and methods.

Focusing research efforts on relevant care issues may achieve quality promotion of dental treatment. For this reason, it is important to know:

- What are the clinical indicators of the outcome of occlusal treatment with regard to: *(1)* preservation of oral tissues; and *(2)* prevention of complications?
- How can these indicators be used to create rules for decision making and treatment performance?
- Which determinants of decision making and treatment performance affect the outcome indicators?

What Research Strategies Are Needed?

Defining Indicators of Treatment Outcome: Strategies of Communication

A multidimensional approach for defining indicators should be elaborated.[22] Using appropriate consensus techniques may be useful.[23] Dental practitioners should be involved in the discussion. The dental office should be regarded as a resource of clinical knowledge. New ways of communicating between those who perform dental care and those who perform dental research should be introduced.

Defining and Validating Criteria for Treatment Planning: Strategies of Health Service Research

A rational approach to the way of recording and assessing the initial findings is the basis of clinical reasoning. To evaluate treatment needs associated with occlusal corrections, the methodology of health service research could be applied. Groups of clinicians should reach consensus on guidelines that subsequently can be put into practice. The

clinical outcome can then be evaluated and can finally be used for the reassessment of the management of treatment.

Randomized Studies

It is widely accepted that there is a need for more randomized studies. There is one severe problem with randomizing studies on prosthodontic treatment, as patients like to make their own decisions as to treatment alternative. Therefore, this type of study will be restricted to university settings.

There is no doubt that controlled clinical studies will become increasingly important for the validation of the treatment of temporomandibular disorders. Studies concerned with restorative procedures should take the patient's preference into account.[24] Controlled studies comparing prosthodontic devices should provide information about adverse clincal effects of occlusal treatment and psychometric documentation of patient comfort to assess the benefit of the treatments examined.[25] The usefulness of clinical trials exclusively dedicated to occlusal items of restorations should be considered.

What Needs Highlighting In Educational Programs?

It can be expected that in the future, dental schools will continue to deliver their particular set of occlusal rules to their students. Additionally, educational programs for postgraduate students and clinicians should be established. These should create a clinical learning environment that enables clinicians to discover the preconditions of successful treatment based on mutual efforts for quality promotion.

Determinants of a Healthy Aging Dentition: Freedom in the Retrusive Range of Occlusal Contacts and Multidimensional Freedom for Functional Tooth Contact

Per Alstergren, DDS, PhD

Introduction

Valid and clinically relevant determinants for a healthy aging dentition would indeed be of great value in prosthodontics, with benefits for patients and clinicians. Such determinants could, for example, be based on functional parameters, esthetic variables, and patient expectations. Possible determinants also include occlusal factors, although no conclusive evidence for the contribution of occlusal factors to oral health, disease, or function seems to exist today.

The large biologic variation, for example, in individual morphology and disease, as well as variations in reconstruction designs, makes studies of possible determinants very complicated. Placebo treatment, double blinding, and control of all other variables than that studied are very difficult, if not impossible in many cases. On the other hand, an absence of any relationship seems unlikely, since the occlusion is a fundamental part of the masticatory system.

What Do We Know?

An extensive search of the MEDLINE database regarding freedom in the retrusion range or multidimensional freedom as determinants for the healthy aging dentition did not reveal any study specifically dealing with these issues. However, a small number of other studies provide a few, indirect indications of different strengths regarding the influence of these factors on craniomandibular musculoskeletal disease or patient satisfaction. These indirect facts are mentioned below, but they have to be further investigated before any conclusion may be drawn regarding use of these factors as determinants for the healthy aging dentition.

Occlusal Factors, Prosthodontic Treatment Outcome, and Disease

In patients with mandibular implant-supported fixed prostheses opposing maxillary complete dentures, occlusal factors are of minor and limited importance for patient satisfaction as well as clinical and radiographic treatment outcome.[1]

Pullinger et al[2] showed that while the contribution of occlusal factors to various forms of temporomandibular disorders (TMD) is not zero, most of the variation in each disease population was not explained by occlusal factors. Indeed, certain features such as anterior open bite were considered to be a consequence of rather than an etiologic factor for disease. For example, the longer slides between the retruded position (RP) and the intercuspal position (IP) found in patients with disc displacement or osteoarthrosis are probably a result of the disease.[3] The only qualitative systematic review of TMD treatment with splints or occlusal adjustment so far published found that only 18 of several hundred studies fulfilled the criteria for inclusion. The mean quality scores for these were below the general level for studies in medicine, although they were higher than in a similar review regarding periodontal studies (!). Accordingly, one of the conclusions was that better designed studies are needed in this area.

Prevention

A prospective placebo-controlled study of 123 orthodontically treated healthy adolescents compared TMD signs and symptoms before active or placebo occlusal adjustment, including reduction of RP-IP slides in the active adjustment; the status 3 years later suggested that occlusal adjustment

prevents future masticatory muscle tenderness and stabilizes the occlusion.[5] However, this study included adolescents, and a larger part of the treatment group showed masticatory muscle tenderness before treatment than the control group.

Experimental Malocclusion

Experimentally induced RP-IP interferences in healthy individuals have been reported to elicit pain similar to that reported by patients with TMD.[6] This and similar studies induced acute forms of nonideal occlusion, and this particular study was performed on a small number of healthy, young male subjects with complete dentitions. In addition, no control groups, neither healthy individuals nor TMD patients, were included, and, except for two subjects, the degree of masticatory muscle tenderness after the interference was induced has to be considered within the normal range.[7]

Conclusion

No study has specifically examined the value of freedom in the retrusion range or multidimensional freedom as determinants for a healthy aging dentition after prosthodontic treatment regarding craniomandibular musculoskeletal disorders, masticatory function, esthetics and patient satisfaction.

What Do We Not Know?

The following list comprises important topics that could progress clinical research aimed to eventually improve clinical outcome of prosthodontic therapies. One suggestion is that the coordination of the research for the two first topics be carried out by an international prosthodontic association. The topics are as follows.

- No standardized outcome measure(s) for prosthodontic treatment—regarding overall outcome, craniomandibular musculoskeletal disorders, masticatory function, esthetics, or patient satisfaction—seem to exist.
- What also seems to be missing is a generally accepted and validated diagnostic classification system for specific diseases and conditions possibly initiating prosthodontic treatment. Many studies regarding occlusal factors and prosthodontic treatment or disease have used the very unspecific diagnosis "TMD." However, more specific diagnoses are most probably needed to be able to identify relevant biologic mechanisms behind specific conditions.
- Are there any associations between specific occlusal factors and outcome of prosthodontic treatment in different diagnostic subgroups?
- Which are the biologic mechanisms behind a possible influence of occlusal factors on treatment outcome?
- With the knowledge of such mechanisms, what can we do to modulate them to improve the treatment outcome?
- Do these factors have any prognostic value in determining prosthodontic treatment outcome?
- By which factors, if any, can patients be identified in whom certain occlusal factors might be of importance for treatment outcome?

What Research Strategies Are Needed?

Define, test, and validate clinically relevant outcome measures for overall outcome or function, esthetics, disease, and patient satisfaction separately. Use an available and validated diagnostic classification, or develop and validate a novel diagnostic classification system, for example, to replace the TMD diagnosis with a more appropriate system. With the combination of validated diagnoses and outcome measures, perform adequately designed studies to investigate determining factors as well as mechanisms and prognostic values for acceptable outcomes of prosthodontic treatment.

What Needs Highlighting in Educational Programs?

Modern and evidence-based international prosthodontic curricula, including training in clinical decision making for undergraduate and postgraduate educations, and integration of these curricula into the main curriculum for each faculty should be undertaken. Continuous learning programs should be developed for teachers in evidence-based clinical prosthodontics.

Usual and Unusual Orofacial Motor Activities Associated with Tooth Wear

Gilles Lavigne, DMD, MSc, FRCD(C)
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What Do We Know?

Definition

Usual orofacial motor activities include chewing, swallowing, breathing, and speaking. In dentistry, any unusual activity is called a parafunction, a general term describing nonfunctional oromandibular and lingual activities such as jaw clenching; bruxism (tooth grinding); tooth tapping; cheek, lip, or tongue biting; nail biting; tongue thrusting; lip licking; tongue protrusion; gum chewing; object biting (eg, cigarette, pipe, pencil, candy); hypersalivation/swallowing; and backward, forward, or lateral head or jaw posture (eg, phone on shoulder, computer work). A parafunction may occur while the individual is awake or asleep, or during both states. Parafunction may be voluntarily induced (eg,

gum chewing), be repeated as a habit or tic (eg, clenching), or be involuntary (eg, sleep bruxism). It is not rare to observe several parafunctions in a patient.

Differential

Parafunctions must be distinguished from: *(1)* the usual orofacial activities of chewing, swallowing, breathing, and speaking; and *(2)* unusual (eg, too frequent, too intense/powerful, in unusual timing/sequence) activities such as "involuntary" chewing, oromandibular myoclonus (tooth tapping during sleep that could be associated with epilepsy or a familial condition), excessive swallowing, oral tardive or idiopathic dyskinesia (eg, tongue protrusion, air expiration, lip suckling), Parkinsonian tremor (chin, lip, tongue), Gilles de la Tourette syndrome, REM behavior disorders (muscle atonia/paralysis during this stage of sleep), snoring, or sleep apnea.

Causes

The parafunction could: *(1)* result from psychosocial factors (eg, stress or anxiety, a child mimicking parents' oral habits such as nail biting or clenching) or reactivity associated with anger or frustration; *(2)* be associated with a medical or drug-related condition (eg, oral tardive dyskinesia, Parkinson disease), sleep parasomnia (eg, bruxism/tooth grinding, REM behavior disorder, oromandibular myoclonus), or sleep disorders (eg, sleep apnea); or *(3)* be concomitant with intraoral conditions (eg, pain; occlusion; xerostomia; hypersalivation; oral lesions; discomfort with restorations, dentures, or orthodontic or snoring–sleep apnea devices).

Consequences

If the parafunction is too loud (eg, air expiration, snoring), too apparent (eg, severe tooth loss, involuntary tongue protrusion while eating), or induces handicap (eg, interferes with speech or eating), it may cause social reclusion, anxiety, and poor self-esteem. Although there is some controversy in the literature concerning the effects of parafunctions, they can also result in oral mucosa or dental damage, restoration breakdown, occlusal discomfort (eg, fixed partial denture against implant rehabilitation), pain, jaw movement limitation, and temporomandibular joint sounds. In the most extreme cases, parafunctions may be risk factors for health, or even cause life-threatening conditions (eg, food aspiration in the airway of a Parkinsonian or oral tardive dyskinesia patient; hypertension and vehicle accident in a patient with sleep apnea).

What Research Strategies Are Needed?

Recognition of Problem and Consequence

A valid questionnaire needs to be developed to survey prevalence and psychosocial factors in relation to functional consequences in nonrehabilitated and rehabilitated populations (eg, denture, implants), in temporomandibular disorder (TMD) and non-TMD patients, and in those with and without bruxism; population-based study is needed. Reliable tools for quantifying usual and unusual parafunctions need to be validated with the use of ambulatory or laboratory tools. The observations collected using the above methodology need to be validated with regard to associations of cause and effect; experimental studies may be designed to further explore these connections.

Examples of research questions include:

1. Is excessive tooth wear caused by the following factors: poor enamel quality (eg, enamel biopsy), lack of oral lubrication (eg, xerostomia), diet, habit (eg, smoking, alcohol), anxiety/lifestyle, aging, clenching or tooth grinding, occlusal discomfort, sleep arousal mechanism (eg, polysomnography), or medical condition (eg, cardiovascular hyper-responsiveness, medication, neurologic disorders, gastroesophageal reflux)?
2. Is the pattern of bruxism/tooth grinding related to types of jaw movements (eg, protrusive, lateral)? Is wear localized on one tooth, or is a group of teeth associated with the same type of parafunction?
3. Could xerostomia, lack of oral lubrication, or gastroesophageal reflux explain patients' tooth wear, discomfort, or parafunction?
4. Is poor enamel quality associated with higher wear and tear in normal function, or is it an increased risk factor in the presence of parafunction?
5. Are concomitant sleep disorders (eg, bruxism/tooth grinding, apnea, REM behavior disorder) risk factors for parafunction? If concomitant, should they influence our treatment planning?
6. An examination of the influence of gender, ethnicity, and cultural factors on parafunction is needed.

Assessment of Management Strategies: Efficacy, Cost Benefit, Risk

- Oral rehabilitation (eg, splint) with cognitive-behavioral strategies to reduce parafunction
- Use of medication and/or physical therapy to manage crises (short-term or recurrent ones)
- Design of an oral splint used with different types of parafunction and/or concomitant sleep apnea or bruxism or following implant rehabilitation
- The relation between occlusal comfort and adaptation and extensive treatment planning concerning the selection of dental material to be used (eg, implants against fixed partial denture)

What Needs Highlighting in Educational Programs?

We must develop a better understanding of causes, pathophysiology, consequences, and management of parafunction and associated health conditions. Revision in the following areas of practical science and review of current research should be undertaken:

- Cognitive-behavioral factors in relation to management strategies

- Oral physiology in relation to pain (eg, nerve damage, arthritis), sleep, respiration, taste, salivation (oral lubrication), occlusion, and quality of life
- Oral diagnosis/medicine including pharmacology/differential diagnosis (including mood alteration/psychiatric-psychologic condition, sleep, and neurologic disorders)
- Global assessment of patient condition to improve treatment planning
- Learning how to reassess the validity of techniques used on a regular basis in daily practice; how to remain critical and up to date on evidence-based clinical sciences

Local Factors Associated with Parafunction and Prosthodontics

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What Do We Know and Not Know?

Methods to Assess Parafunction

There are several ways to assess parafunctional activity. Questionnaires are the most commonly used method. Clinical examination and observations of tooth wear are also widely used in clinical settings. The reliability of these methods is questionable. In addition, the wear on occlusal splints[1] and interarch contacts and force[2] are measured. Portable electromyographic recording of the masticatory muscles during sleep[3] is a more objective method to assess bruxism. Polysomnography in a sleep laboratory[4] is currently considered the most specific method of analysis. The former recording method has limitations in data size and in the number of channels for sampling compared with the latter. However, the latter is expensive, and the subject's parafunctional activity might be affected by the unfamiliar conditions of the sleep laboratory.

Parafunction and Tooth Wear

A number of systems to classify and assess tooth wear have been reported. Many of these use a five-point scale based on the severity of tooth wear, as determined from study casts.[5,6] Intra- and interexaminer reliability are keys to the usefulness of these methods, and the reliability of these approaches has been confirmed in many studies. However, no method is universally accepted. In addition to these rating systems, sophisticated methods, such as digitization of the amount of wear[7] and observation by scanning electron microscopy, have been introduced. However, the usefulness of such methods in a clinical setting or for large samples is questionable.

Over a 2-year observation period, bruxers developed more tooth wear than did nonbruxers.[7] Tooth wear is associated with many factors, and parafunctional activity can cause tooth wear.[7] However, the presence of attrition cannot be used as a criterion to define the bruxer group.[6] In general, the incidence and severity of tooth wear are thought to increase with age. However, the prevalence of nocturnal grinding decreases with age after 50 years of age.[8]

Parafunction and Occlusion

The mean amplitude and duration of bruxism events were 22.5 kgf and 28.5% of the maximum conscious clench, respectively. The maximum bruxing force (15.6 to 81.2 kgf) generally does not exceed the maximum conscious clench, although it can exceed the maximum conscious bite force in some individuals.[2] The occlusal force during nocturnal bruxism can easily exceed the level during normal function such as chewing.

Although occlusal interference has historically been regarded as a cause of bruxism, evidence countering this historic concept has been reported: Experimental occlusal interference reduces muscle activity during sleep[3]; occlusal adjustment does not stop bruxism, although it might be a cause of bruxism; no significant difference in occlusion is seen in bruxism and control groups; moreover, there is no difference in the effect on bruxism by an occlusal splint covering the occlusal surface of the dentition or not.[9]

Parafunction and Myofascial Pain/Temporomandibular Disorders

Bruxism is thought to cause or be a risk factor for myofascial pain/temporomandibular disorders (TMD). Although there are many reports associating bruxism and myofascial pain/TMD, there is no strong evidence for a cause-and-effect relationship between bruxism and myofascial pain/TMD.[10,11]

There are many reports on the prevalence of TMD, and most agree that TMD is more prevalent in women than in men. By contrast, the studies on gender differences in bruxism do not show a constant result—while some studies reported no gender difference in the incidence of bruxism,[12] others reported diverse gender differences.[13] A twin study with a large sample size reported that more women have bruxism than men, and that the incidence of bruxism increases with age from 30 to 50 years.[14] It is also controversial whether there is a gender difference in jaw muscle pain experimentally induced by clenching.[15]

Parafunction and Prosthodontic Treatment

Parafunction and factors such as restorative materials, restoration design, implant design and location, occlusal

vertical dimension, and periodontically compromised dentition are thought to be important in prosthodontic treatment. Few data are available on these topics. Some studies report that bruxism may not be a primary factor, but it contributes to the wear of restorative materials,[16] tooth survival in periodontitis,[17] cracks in posterior teeth,[18] implant failure,[19] and complications with fixed partial dentures on implants.[20] Most of the studies in this field define a bruxer according to the subject's reports or tooth wear. However, such definitions are unreliable.

What Research Strategies Are Needed?

First, we need to establish valid criteria and a method of defining a bruxer that will be used universally. An objective measurement of bruxism, which can be used in clinics, should also be devised.

Studies on the role of bruxism in the etiology of myofascial pain/TMD from the perspectives of cause-and-effect relationships and the role of bruxism in perpetuating or aggravating TMD are needed.

The following questions should be answered. Is there a gender difference in the prevalence of bruxism? Is there a typical natural course for bruxism over a lifetime? Why does the incidence of bruxism decrease after middle age? Is there a gender difference in experimentally induced muscle pain?

The effects of bruxism on prosthodontic treatment, especially on prosthesis longevity, need to be studied. The following studies are suggested.

1. Wear of teeth and restorative materials and surface characteristics of materials
2. Wear/destruction of restorative materials and bruxism
3. Restoration design and bruxism
4. Implant design and location and bruxism
5. Periodontically compromised dentition and bruxism
6. Occlusal vertical dimension and bruxism

To conduct such studies, bruxism must be defined using a reliable, possibly quantitative, method.

What Needs Highlighting in Educational Programs?

Better understanding of the definition, causes, pathophysiology, consequences, and management of parafunction and associated health conditions should be included in dental education. Students should be taught how to recognize bruxism in clinical assessment. The effects of bruxism on pain, dysfunction, and prosthodontic treatment need to be emphasized in prosthodontic education.

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Musculoskeletal Disorders and the Occlusal Interface

Thuan Dao, DMD, MSc, PhD

Introduction

Temporomandibular disorders (TMD) are the musculoskeletal disorder most prevalent in the orofacial area. Several aspects of occlusion have been scrutinized in an effort to establish a link with TMD. These beliefs are mostly based on data that suggest a positive relationship between TMD and the occlusal parameter being studied, and on reports that signs and symptoms of the disorders improve with occlusal rehabilitation. A positive association between the signs/symptoms and the disease is a necessary, but not sufficient, provision for a causal relationship. In this article, the evidence for causality linking malocclusion and TMD will be assessed using the criteria suggested by Fletcher et al.[1]

What Do We Know?

Malocclusion in its various aspects is highly prevalent in both sexes, and in all age groups. TMD and related symptoms are primarily a condition of young and middle-aged adults, mainly female, and their prevalence tends to diminish in the older age groups. Thus, the association between occlusion and TMD does not appear to make epidemiologic sense. Occlusal interferences are highly prevalent in both TMD patients and control subjects. Therefore, they lack sensitivity and specificity for identifying a TMD or control population.

The association between occlusion and TMD does not satisfy any of the criteria that are considered essential in establishing causal relationship. Pain and degenerative joint diseases can induce changes perceived at the occlusal level. There are emerging data on peripheral and central pain-processing mechanisms and their modulation by the reproductive hormones.

What Do We Not Know?

- Why do some patients tolerate acute changes in their occlusion while others do not?
- What are the musculoskeletal responses and adaptations to sensory inputs resulting from changes in occlusion?
- What is the influence of various occlusal schemes on function (eg, masticatory efficiency)?

What Research Strategies Are Needed to Link Occlusion and TMD?

A temporality relationship between cause and effect must be established. A test of temporality is positive if a consistent

exposure to the cause is followed by the occurrence of the disease, ie, causes should precede effects. This fundamental principle can only be assessed with well-designed cohort studies and randomized controlled trials (RCT). The association between occlusal factors and TMD has been typically reported within cross-sectional (either case-control or case series) studies. With those study designs, the temporal relationship cannot be evaluated.

The strength of the association between the suspected cause (occlusion) and effect (TMD) must be determined. It is usually expressed in terms of the magnitude of the relative risk (in RCT and cohort studies) or relative odds (in either cohort or case-control studies) of developing the effect when exposed to the suspected cause. Although numerous papers have reported the association between various occlusal factors and TMD, only one cross-sectional population-based study that reports on the odds ratio[2] and one case-control study with discussion on odds ratios (ie, the amount that the independent variables, occlusal factors, can differentiate the dependent variables, disease versus health) were identified.[3]

A dose-response relationship must be established. This is present when variable amounts of the suspected cause are associated with increasing risk or severity of the effect. While there is no consensus about the definition of occlusal interferences, about which occlusal factor or a combination of these would play a determinant role in the development of TMD and should be appraised, most studies either do not include an assessment of risk or report the various degrees of TMD using measuring instruments with questionable diagnostic validity.

Reversible associations would have to be shown. A factor is more likely to be the cause of disease if its removal results in a decreased risk of disease.[1] Attempts to show reversible associations are highlighted in RCTs that assess the efficacy of oral splints and occlusal adjustments in the management of TMD.[4] The three RCTs that obtained the highest quality scores between 1961 and 2001 compared occlusal to palatal splints (0.60 to 0.78)[5–7] and found no between-group differences if pain reports (rather than perceived relief) were used as the primary outcome variable. These results cast doubt on the therapeutic effect of splints and question the rationale of using splints to remove occlusal interferences in the management of TMD. In spite of their low quality scores (from 0.24 to 0.57), the RCTs on occlusal interferences reported a significant improvement of the TMD signs and symptoms over time, but no differences were found between the treatment and control groups. Removal of occlusal interferences does not appear to be any better than nonocclusal therapies or placebo intervention in the management of TMD.

Consistency must be established. Causation is particularly supported when studies using several different research designs all lead to the same results.[1] However, if more weight is given to case-control[3,8] and population-based epidemiologic studies[2,9–13] rather than case series or anecdotal reports, the consistent lack of association between various occlusal parameters and TMD becomes evident.

The concept of biologic plausibility is satisfied if the cause and effect is consistent with knowledge of the mechanisms of disease as they are currently understood. Biologic plausibility is not supported in the current context for at least three reasons:

1. Malocclusion in its various aspects is highly prevalent in both sexes and in all age groups. Yet an appraisal of the epidemiologic literature on TMD and related symptoms from different population-based studies reveals consistently that it is primarily a condition of young and middle-aged adults, mainly female, and that its prevalence tends to diminish in the older age groups.
2. Occlusal interferences are highly prevalent in both TMD patients and control subjects. Therefore, they lack sensitivity and specificity for identifying a TMD or control population.
3. The current knowledge of pain mechanisms does not substantiate the association between occlusion and TMD.

What Needs Highlighting in Educational Programs?

- Clinical epidemiology on orofacial pain, identification of risk factors
- Misbeliefs about the causal role of occlusion in orofacial pain
- Differential diagnosis and management of orofacial pain, including behavioral and cognitive therapies
- Clinical applications of basic science knowledge about peripheral and central pain mechanisms

Conclusion

While a causal relationship often cannot yet be established, its strength increases if it satisfies a combination of the rules of evidence for causation. As reviewed above, the association between occlusal factors and TMD does not appear to satisfy any of the criteria that are considered essential in establishing causation. Furthermore, there is evidence that suggests that perceived changes in occlusion may be the consequence of pain rather that its cause. As a result, occlusal therapies cannot be justified, and prosthodontic treatment should aim at achieving improvement of the patient's orofacial comfort and function with optimal prostheses, but not as a specific therapy for TMD. This is consistent with the goals of managing TMD, which include palliation of the condition, pain control, and reestablishment of motor function.

The mechanistic occlusal view of the pathophysiology of TMD is being eclipsed by the evolving knowledge of the peripheral and central pain-processing mechanisms and their modulation. Future investigations on the pathophysiology of TMD need to mirror current developments in research on pain and its interaction with movement, rather than focusing on old concepts linking nonspecific dental structures to multiple disorders of the masticatory apparatus.

Musculoskeletal Disorders and the Occlusal Interface

Maria Nilner, DDS, Odont Dr

Introduction

In 1973, Beyron[1] presented proposals for description of a functionally optimal occlusion and principles for occlusal rehabilitation; according to him, optimal occlusion is characterized by:

- Stable jaw relationship with bilateral contact in retrusive closure
- Freedom in retrusive range, with maximum intercuspation slightly (1 mm or less) and straight in front of the retruded contact position
- Stable tooth-to-tooth relationships providing axially directed forces on the posterior teeth in complete occlusal closure
- Smooth bilateral function, facilitated by group contact on the laterotrusive side close to maximum intercuspation and by no contact on the mediotrusive side; in symmetric protrusion, early anterior group contact
- Acceptable interocclusal distance

Available evidence for Beyron's proposals, including references of studies with different evidence levels,[2] is presented in the following text and summarized in Table 1.

Maximum Number of Bilateral Centric Stops

The Oral Health Impact Profile (OHIP), implying an evaluation of the function of the jaws, has shown social and psychologic factors to be of importance for the oral health.[3]

Hatch et al[4] showed that tooth units, together with bite force, were determinants of masticatory performance. In young girls (11 to 15 years), as well as in 19-year-olds, the number of centric stops varied.[5,6] No sex difference has been reported. The masticatory efficiency index in that study was higher in the group with normal occlusion than in the Class II malocclusion group; when the girls estimated their masticatory ability, the normal group reported their ability to be better than did the Class II group. When pooling the Class II malocclusion and normal groups in a logistic regression analysis, the number of occlusal contacts was found to be the most influential predictor for the variation of masticatory efficiency index. In a 2-year longitudinal study, young girls with normal occlusion continuously had more occlusal contacts than did girls with Class II malocclusion.[7]

It is obvious that the methods used to record contacts, for example, the thickness of the recording medium and the biting procedure, are of importance. The number of occlusal contacts in light biting is only about half that in hard biting in young girls with both normal and Class II malocclusion, as well as in young adults and adults.[7,8]

In adults, the masticatory ability is affected to a lesser degree than masticatory performance when occlusal units are missing, and the capacity of the stomatognathic system to adapt to loss of molar support is great. This statement relies on the studies of the shortened dental arch (SDA) concept.[9,10]

Table 1 Literature, in Alphabetic Order, Lending Support to Evidence for Beyron's Proposed Criteria for an Optimal Occlusion

Study	Year	Level of evidence*
Allen et al[3]	2001	2c
Beyron[1]	1973	5
De Boever et al[12]	2000	2a
De Boever et al[11]	2000	2a
Hakestam et al[13]	1997	2c
Hatch et al[4]	2000	2c
Henriksson et al[6]	1998	2b
Henriksson et al[7]	2000	2b
Marklund and Wänman[15]	2000	2a
Riise[8]	1982	ES
Rivera-Morales and Mohl[16]	1991	3a
Vallon and Nilner[14]	1997	2b
Wänman and Agerberg[5]	1991	ES
Witter et al[9]	1988	2b
Witter et al[10]	1990	2b

ES = epidemiologic study.
*1a = Systematic review of RCTs; 1b = individual RCT; 2a = systematic review of cohort studies; 2b = individual cohort studies; 2c = "outcomes" research; 3a = systematic review of case-control studies; 3b = individual case-control study; 4 = case series; 5 = expert opinion.

The masticatory ability and oral comfort in adults were presented to be sufficient as long as 20 "well-distributed teeth" or 10 occluding pairs remained, such as in premolar dental arches. The number of patients in that study was sufficient, making it possible to estimate the minimum number of teeth needed to have an acceptable masticatory function and oral comfort. The reason for the difference found regarding reported pain in and around the temporomandibular joint in the SDA and control groups was not further analyzed. Sex differences in patients with SDA have not been reported. A review regarding tooth loss and prosthodontic treatment[11] found no proof for prosthodontic restoration as prevention for or treatment of temporomandibular disorders (TMD).

Edentulous occlusal gaps most often are recommended to be replaced for esthetic function, but also to avoid possible negative consequences such as tipping of neighboring teeth or elongation of an opposing tooth. The frequency and severity of such changes after loss of single teeth without replacing them are not well known, but no or only minor overeruption has been presented, even in a long-term perspective. Tilting or elongation of teeth can give rise to interferences, which in turn can disturb the function and possibly create musculoskeletal disorders. The risk for this is, however, not known. In controlled studies in which interferences have been created, symptoms of TMD have been reported to occur, although the studies included small samples only.[12] When and why such disorders occur in connection with artificial interferences are not fully known. There is no linear relationship between tooth loss and musculoskeletal disorders. This is at least understood from a study of edentulous non–denture wearing individuals in whom signs and symptoms of musculoskeletal disorders were practically absent.[11,12]

A better esthetic and chewing function, as well as an enhancement of the general well-being, were important factors reported by patients who were to undergo extensive prosthodontic treatment. Different personality traits seemed to play a role in patient expectations. Patient satisfaction with prosthodontic treatment has been reported to be multidimensional.[13]

Rheumatoid arthritis is one example of an inflammatory joint disease in which continuous changes of the maximum centric stops may occur because of destruction of joint tissue. What impact this has on the individual patient's function or dysfunction is not fully understood.[11]

Freedom in Retrusive Range of Occlusal Contact

The study of freedom in the retrusive range of occlusal contact will be enabled by a sagittally oriented rotation of the mandibular jaw upward, leading to an initial intermaxillary occlusal contact, where the mandible is situated in the retruded contact position (RCP) or centric relation. It is an established fact that in most individuals this mandibular position, RCP, lies slightly posterior to the intercuspal position (IP). In fewer than 10% of a large sample do these two positions coincide, but normally there is a distance between RCP and IP of up to 1 mm in the incisal region. The distance can be asymmetric, and there seems to be no difference with different ages or between sexes. Epidemiologic studies have presented associations between asymmetric slide and dysfunctional symptoms, but the associations have not identified patients with musculoskeletal disorders. Slides of more than 2 mm are seldom found in asymptomatic populations, and a distance of 2 mm or more has been proposed to be more common in patients complaining of musculoskeletal disorders than in patients without these disorders. Lateral slide has also been alleged to be one type of occlusal disturbance found more commonly among patients suffering from musculoskeletal disorders.[5,12] In a randomized controlled study of patients with musculoskeletal disorders treated with occlusal adjustment, the treated patients reported themselves to be better in an up to 3-month perspective, but not in a longer one.[14]

Multidimensional Freedom of Contact Movements

Multidimensional freedom of contact movements has been examined in population-based studies showing that the natural dentition exhibits various contact patterns under protrusive as well as lateral excursions of the mandible, but without any clear-cut correlations to musculoskeletal disorders, dysfunction, or better function. Mediotrusion interferences, for example, have been found to correlate to single symptoms of musculoskeletal disorders but have not alone been able to identify such disorders.[15] Findings in different studies have, however, been contradictory. Gender differences have not been proven.[12] When prosthodontic treatment is indicated, there are various occlusal philosophies or principles for different reconstructive measures.

Interocclusal Distance

An acceptable interocclusal distance can be judged by measuring the space between the maxillary and mandibular teeth when the mandible is in the rest position, known as the interocclusal distance, or freeway space. The width of the space, ie, the vertical distance, between the resting and closed positions is recommended to be in the range from 1 to 3 mm. Clinical studies with electromyographic recordings have, however, presented the rest position to be up to 8 mm.

There is no precise method available to measure the interocclusal distance, but a potential for adaptation to altered vertical dimensions of occlusion has been reported, and no report has claimed great difficulties with patient acceptance of a new freeway space. The experimental studies, however, included only a small number of individuals, and all the increased occlusal vertical dimensions were tested by use of splints, not fixed restorations.[16]

What Do We Know?

- The occlusion is not the only etiologic factor in patients suffering from musculoskeletal disorders.
- There is a great variation in the number of centric stops at light and hard biting in young ages and adults.
- There is great belief in the benefit of filled occlusal gaps for function and improvement of musculoskeletal disorders.
- Inflammatory joint diseases can influence the occlusal interface.
- There are individual contact patterns at jaw movements both with and without symptoms of musculoskeletal disorders.
- In most individuals, the RCP lies slightly posterior to the intercuspal position.
- Depleted dentition (fewer than 20 teeth) could lead to functional problems.

What Do We Not Know?

- The importance of the number of centric stops for an individual's masticatory function and dysfunction, or for general health and psychosocial function
- Why and when patients rehabilitated with an SDA will develop musculoskeletal disorders
- The efficacy of restored molar support in musculoskeletal disorder patients with SDA
- If and in what individual cases the occlusion is important for evolving musculoskeletal disorders
- When tilting and elongation do occur, and the time span for adaptation to occlusal gaps in different regions of the mouth
- How, when, and why inflammatory joint diseases influence the occlusal interface
- The importance of occlusal design for function and dysfunction or evolution of musculoskeletal disorders

What Research Strategies Are Needed?

- Clinical research regarding function, dysfunction, and musculoskeletal disorders, where a holistic view of the patients is central.
- Clinical studies in both short- and long-term perspectives to achieve knowledge about the influence of tooth loss on psychosocial factors and quality of life.
- Clinical studies of patients rehabilitated with different treatment modalities to understand their acceptance of reconstructions in both short- and long-term perspectives.
- Clinical studies on patients suffering from inflammatory joint diseases to achieve knowledge of the development of the diseases in the stomatognathic system and about the importance of different kinds of occlusal treatments.
- For a better understanding of the effect on function, dysfunction, and musculoskeletal disorders, randomized controlled trials are needed in the evaluation of different kinds of reconstructions or reconstructions made according to different therapeutic principles for the mandibular position, contact pattern of mandibular excursions, and occlusal designs.

What Needs Highlighting in Educational Programs?

An integrated clinic for students to meet patients—allowing an opportunity to examine, discuss, diagnose, prognosticate, treat, and eventually rehabilitate together with clinicians trained in research—would probably give students a good opportunity to understand evidence-based dentistry.[17] It has been shown that the visual appearance of patients can provide valuable clues to the nature of the underlying disorder.[18] Because of the rapid development in dentistry, students need to be trained in reading research articles to become critical readers of the literature. Training students to critically appraise medical information will increase the number of clinicians who are capable of self-directed lifelong learning. An educational curriculum should focus on learning, not teaching.[19]

Erosion and Tooth Surface Loss

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What Do We Know?

Tooth wear is a universal problem, but severe cases justifying treatment may only approach 7% of the population.[1] Prevalence studies suggest that low to moderate levels of erosion are common in children.[2,3] Different countries recognize the terminology differently.[4]

The titratable acidity of a drink is more important than the pH, and some foods, especially citrus fruits, have a higher erosive potential.[5] The presence of certain drinking habits, such as holding or swilling drinks in the mouth before swallowing, prolongs the erosive attack and may be more important than the drink itself.[6] Conditions associated with movement of gastric juice into the mouth, such as anorexia and bulimia nervosa, rumination, chronic alcoholism, and reflux disease, are all associated with dental erosion.[7] Gastroesophageal reflux is a very common condition, with about 60% of adults suffering from its effects at some point in their lives; high reflux, past the upper esophageal sphincter, is associated with dental erosion.[8]

Tooth wear indices are useful to assess prevalence but are inaccurate and insensitive for the management of tooth wear in individual cases. Indices limited to a particular cause are generally unhelpful in the clinical management of patients. Measuring techniques using mathematic calculation of reference points from superimposition of data points[9] or the use of acid-resistant metal disks[10] have attempted to overcome the difficulty of consistent and stable reference points. Salivary flow, buffering capacity, and the role of oral tissues, especially the tongue, may influence tooth wear.[11-13]

Clinical guidelines to assess treatment need are helpful in the planning process. As teeth wear, compensatory alveolar eruption reacts to produce short clinical crowns with no obvious change in the vertical dimension.[14,15] Conventional management of crown preparations may not be relevant for tooth wear because of the effects of alveolar compensation. Vertical space can be gained by reversing this process, often called the "Dahl effect."[16]

What Do We Not Know?

There is no evidence to suggest that the high levels of wear observed in children will inevitably lead to longitudinal progression. Do early signs of tooth wear inevitably lead to further deterioration, or is the process phasic, with episodes of activity and inactivity? Teeth wear normally, so what is an acceptable rate of wear?

The susceptibility to erosion may alter with tooth position and between patients' teeth.[17] Why does erosion occur when no obvious cause can be found from the clinical history and the use of special investigations? Does saliva have a role? Is the diet more important than gastric causes?

What is the relative importance of erosion and attrition? Both factors are commonly present in susceptible patients. Are some of the signs, such as interferences, important in the progression of erosive and attritional wear? Interferences are commonly observed in the worn dentition. This is not remarkable, since the wear generally produces flattened occlusal surfaces. Therefore, are these interferences a cause or consequence?

What Research Strategies Are Needed?

Prevalence of Tooth Wear and Erosion in North America

The role of attrition has been well-investigated in North America, but there appears to be a paucity of information

on erosion, apart from Xhonga and Sognnaes[18] and a few other studies. Why is erosion considered to be more important in Europe than in North America?

Role of Diet in Erosion

Why do some patients develop tooth wear when they have a mildly erosive diet and yet others do not when they have a very highly erosive one? Dietary causes are commonly blamed for much of the increase in the prevalence of tooth wear. Is this a true increase or a perceived one? There are few objective data to support this assumption. Questionnaires used to assess dietary habits are notoriously inaccurate in large population studies but often remain the most convenient way for assessment.

Tooth Surface Changes

Ultrastructural studies are needed to observe what happens at the tooth surface after contact with acids. Is the wear seen on noncontacting surfaces a result of erosion, or is it combined with abrasion, possibly from the soft tissues or the tongue?

Role of Gastric Juice

Despite the evidence that reflux and other sources of gastric acid are important in the development of tooth wear, some patients present with severe tooth wear and, despite careful investigations, there is no obvious cause. What, then, is happening? In the United Kingdom, many clinicians suspect that gastric causes are more important than dietary ones. Is this true? Certainly the potential damage caused by gastric acid, with a very low pH, seems to be more severe than that caused by dietary acids. Another area to investigate is the susceptibility of teeth to erosion, within the same patient and between patients.

Saliva

There has been considerable work on saliva, but it is difficult to measure accurately. Which is more important in erosion—resting or stimulated saliva? Both are difficult to measure. Clinical evidence from xerostomic patients has not produced definitive results, but further investigation is required.

Measurement

To date, many approaches to the measurement of tooth wear have assumed attrition as the primary cause of wear and used surface features as reference points. If acid has a role in the tooth wear, then by definition no surface reference point is stable. If a method to measure wear was designed to be convenient, accurate, and quick, preventive regimens could be assessed. With the development of accurate measurement techniques, the progression of wear—either attritional or erosive—could be assessed in small or large samples. Can the use of proton pump inhibitors or other antireflux mechanisms reduce the activity of regurgitation erosion? Prevention of erosion with fluoride or sealing tooth surfaces with dentin bonding agents needs further evaluation.

Treatment Strategies

Is treatment always necessary? In most patients, tooth wear is a slow process, and treatment with crowns is not always needed, especially when the appearance of the teeth is not a major factor for the patient. Are mechanistic approaches to managing the occlusion relevant to the treatment of tooth wear? If not, is this also true of patients without tooth wear? Is the mouth more adaptive to change than we generally accept? The use of adhesive techniques to restore worn teeth is a significant area for investigation. Nonpreparation techniques using adhesive bonding techniques conserve tooth tissue, eliminate the need for elective root treatment, and consequently reduce the potential for failure from excessive parafunctional forces in patients with a combined etiology.[19,20] More information on the use of different materials for the management of tooth wear is needed. Is surgical crown lengthening to reposition the gingival tissues an appropriate treatment after the development of adhesive bonding techniques and techniques that reverse the effect of alveolar compensation to produce vertical space?

What Needs Highlighting in Educational Programs?

The importance of tooth wear is acknowledged by UK deans, and time for instruction is provided within the curricula of most UK dental schools. Much more important is the steadily declining time for costly clinical activity in preference for cheaper academic studies. The experience gained by undergraduates in basic prosthodontics is being reduced to levels that may not provide them with sufficient experience for independent practice. Vocational training to some extent may alleviate some of these deficiencies, but the scheme cannot provide teaching to the levels needed within the confines of a busy general practice.

Study Group Report and Discussion

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Christian S. Stohler, DMD, Dr Med Dent

Introduction

The occlusal interface is pivotal to successful prosthodontic treatment. However, our understanding of the implications of the occlusion for the individual, and what constitutes an acceptable range of variability for the natural and restored dentition for optimal function, is not defined. Further, the validity of a possible link between the occlusion and temporomandibular disorders remains controversial. In an attempt to address these issues, the occlusal interface was considered within the framework of four interdigitating aspects:

- Biologic adaptation and normative values as defining the biologic elements and their interrelationships in considering: normal determinants for the healthy young dentition with its adaptive potential; the biomechanical rationale for providing therapeutic changes in response to morphologic variations; and orthodontic and surgical management of developmental anomalies
- Beyron's[1] determinants of a healthy aging dentition as a foundation for prosthodontic treatment (maximum number of bilateral centric stops, adequate vertical dimension of occlusion, freedom in the retrusive range of occlusal contact, and multidimensional freedom of contact movement), and whether alternative or additional determinants might be considered to replace or modify Beyron's guidelines for treatment efficacy and effectiveness in the context of the biology of the system
- Musculoskeletal disorders and the relationship of the occlusal interface for individual patient's needs, which were appraised on the strength of research evidence
- Parafunction and the occlusal interface as defined by local and systemic factors and their implications in the short and long term for prosthodontic treatment planning and long-term treatment outcomes

The presentations reviewed the clinical and biologic research evidence and considered the strength of evidence for each topic and the implications for clinical prosthodontics. It became apparent that: *(1)* the strength of evidence as to why specific treatment philosophies and/or concepts are advocated is weak; *(2)* the basis of current treatment philosophies in prosthodontics has arisen through progressive clinical knowledge and clinical convenience; and *(3)* this has centered on clinical experience (or "it works in my hands"), rather than on the systematic testing of hypotheses through controlled clinical trials and long-term follow-up of operationally defined treatment outcomes and valid assessment measures and/or techniques.

The plenary discussion summary highlighted three broad areas of relevance for prosthodontic treatment:

- The population that requires prosthodontic services, recognizing the changing community demographics and increasing sociocultural diversity
- The occlusal interface, as defined within the biologic framework, acknowledging adaptation with time but recognizing that there are limits to this adaptive potential
- Tooth surface loss, considered on the basis of attrition and erosion, recognizing the context of the maturing dentition and the intrinsic and extrinsic factors that require consideration in clinical treatment

Research topics were proposed to encourage preclinical and clinical research in specific areas. These should address the current lack of knowledge of cause-and-effect relationships and provide, over time, a scientific basis to justify clinical decision making in prosthodontics. Educational issues were proposed to be linked with research data. The limited but emerging evidence of clinical outcomes based on long-term follow-up of specific treatment procedures was acknowledged. Undergraduate and graduate programs are now beginning to embrace evidence-based procedures for clinical decision making. This will be a stimulus for questioning much of what in the past has been the basis of prosthodontic education.

Population for Prosthodontic Services

All community groups, irrespective of age or social circumstances, have a justifiable expectation for prosthodontic care. The following issues are a responsibility of the prosthodontist and graduate prosthodontic programs.

- *Increasing life expectancy.* The encouraging development of the "graying" of the community in industrialized economies, through improved affluence, lifestyle, diet, and nutrition brings new challenges and demands. Education, prosthodontic care, and research must address social and medical consequences that will influence management strategies and the environment within which such care is offered.
- *Special-needs groups.* The varying complexity of prosthodontic care includes developmental anomalies, genetic defects, and socioeconomic influences.
- *Lifestyle and cultural needs.* Different expectations in developing economies need to be recognized and reflected in training programs that are designed to meet regional requirements.

Research issues include access to care, recognizing the unique situation (cultural, environmental, socioeconomic) of the target population.

What is the Occlusal Interface?

The following acknowledges the working scheme of biologic determinants and adaptive potential of the stomatognathic system as proposed in Dr Sessle's contribution.

Occlusion in the Natural Dentition

Functional determinants are based on several factors:

- Sensory-motor interaction and phonetics are the basis of optimal orofacial function.
- Esthetics is a major focus of psychosocial well-being.
- Psychologic and psychosocial influences are crucial for optimum function and general health.

There is a range of variability in both form and function. In general, these variations meet the comfort, physiologic, and esthetic needs of the individual. There is little evidence that the variations, or even the extremes, lead to bad outcomes or do not meet individual (comfort, physiologic, or esthetic) needs. Prosthodontic treatment within this framework needs to be emphasized in clinical practice.

Occlusion in the Prosthodontic Context

Management of the occlusion aims to restore form and function, recognizing the following pre-eminent concerns: *(1)* the broad range of variability that exists in dentitions; *(2)* adaptation to an altered dental environment takes time; and *(3)* the demands of restoring an occlusion, which vary with the complexity of the case, including complete-mouth reconstruction and periodontally and medically compromised conditions.

The framework for restoring the occlusion requires a prescription to increase the probability of a successful long-term outcome. In the context of restoring the occlusion, it is recommended that the design characteristics take into account several factors. Those features for which there is clinical research evidence, such as the need for a bilateral distribution of posterior tooth contacts in intercuspal contact positions have implications for individual tooth stability and each patient's perceived comfort. In the absence of clinical research evidence, accepted best practice suggests that the restorative design meets individual patient priorities and expectations for self-esteem, phonetics, and esthetics with consideration of, among other things, arch form (proximal tooth relationships), anterior tooth arrangement, and lower face height and occlusal vertical dimension.

Research issues include the basic, translational, and clinical research questions derived from the working scheme presented in Dr Sessle's contribution. Clinical research strategies should focus on treatment outcome issues, recognizing the need for appropriate study design and assessment measures.[2–4]

Attrition and Erosion

The resiliency of the jaw muscle system is demonstrated by its accommodation to the continual changes in the stomatognathic system as a result of attrition and erosion. The graying of the community increases long-term demands on the system.

Parafunctions are orofacial motor behaviors (aside from function) that are not well-understood and may occur during day and night. With respect to the occlusal interface, the tooth wear component is difficult to define and quantify reliably.[5] It is recognized that the individual adaptive capacity varies and may lead to undesirable sequelae, including unwanted wear of tooth structure, increasing tooth mobility, pulpal involvement, and failure of restorative work.

Tooth surface loss may also be attributed to erosion (accepting that dental caries is a major cause of tooth surface loss). It is recognized that: *(1)* the extent to which attrition and erosion influence dentitions is linked with factors such as diet, gastric disorders, oral dryness, medication, and enamel structure; and *(2)* habits, lifestyle, and occlusal morphologies vary greatly and are perceived differently among people of varying ethnic and racial backgrounds.

In future research, descriptions of parafunction and processes that lead to tooth erosion, including the identification of populations that show increased vulnerability, should be undertaken. The degree of parafunction that constitutes a risk for prosthodontic treatment (including implants) and the functional state of the stomatognathic system should be identified.

Conclusion

It was recognized that the clinical and biologic research evidence that supports our fundamental understanding of the occlusion and best practice prescription for occlusal management is not strong. This was a major focus of the discussion that needs to be acknowledged. It was accepted that the variation in treatment philosophies proposed for optimal restoration of the occlusion evolved from clinical treatment experience and, with few exceptions,[6] has not been systematically studied to determine long-term outcomes.

The opening paper reviewed the biologic determinants and adaptive potential of the complex stomatognathic system. This desirably focused the discussion on the pivotal importance of functional adaptation and the range of variability of form and function that should be recognized as acceptable for individuals. Stylized diagnostic and treatment prescriptions, often advocated as an "all or none" essential requirement, lack scientific validity and clinical justification.

It is strongly recommended that the discipline of prosthodontics, through a commitment to prosthodontic education and best practice, takes the opportunity to plan for the future. It needs to be acknowledged that prosthodontics evolved within a mechanical era when biologic factors were poorly understood. There is a need to critically review core values of prosthodontics as a biologically based discipline with variable but profoundly significant psychosocial, functional, and esthetic implications for each patient.

There is a range of normative values of anatomic (form) and physiologic (function) and esthetic variations, in addition to the impact of parafunction and aging. It is within this framework that clinical prosthodontic restoration must be blended, in recognition of its unique role for maintenance of functional integrity. In the absence of long-term outcomes data on clinical procedures, the preparation of a therapeutic occlusal scheme should acknowledge the determinants of a healthy aging dentition as proposed by Beyron[1] as the basis for occlusal scheme design. Prosthodontic education for undergraduate and graduate programs must be given appropriate recognition and time in curricula. Educational programs need to focus on the importance of this discipline (as defined above), for a contemporary understanding of treatment responsibility, to optimize outcomes for the benefit of the community.

References/Suggested Reading

Sessle

1. Hargreaves KM, Goodis HE. Seltzer and Bender's Dental Pulp. Chicago: Quintessence, 2002.
2. Kopp S. Topical review: Neuroendocrine, immune, and local responses related to temporomandibular disorders. J Orofac Pain 2001;15:9–28.
3. Milam SB. Articular disk displacements and degenerative temporomandibular joint disease. In: Sessle BJ, Bryant PS, Dionne RA (eds). Temporomandibular Disorders and Related Pain Conditions. Seattle: IASP, 1995:89–112.
4. Sessle BJ. Acute and chronic craniofacial pain: Brainstem mechanisms of nociceptive transmission and neuroplasticity, and their clinical correlates. Crit Rev Oral Biol Med 2000;11:57–91.
5. Stegenga B, de Bont LGM, Boering G, van Willigen JD. Tissue responses to degenerative changes in the temporomandibular joint: A review. J Oral Maxillofac Surg 1991;49:1079–1088.
6. Storey AT. Biomechanical and anatomical aspects of the temporomandibular joint. In: Sessle BJ, Bryant PS, Dionne RA (eds). Temporomandibular Disorders and Related Pain Conditions. Seattle: IASP, 1995:257–272.
7. Jahangiri L, Devlin H, Ting K, Nishimura I. Current perspectives in residual ridge remodeling and its clinical implications: A review. J Prosthet Dent 1998;80:224–237.
8. Mogil J. The genetic mediation of individual differences in sensitivity to pain and its inhibition. Proc Natl Acad Sci U S A 1999;96:7744–7751.
9. Zarb GA, Carlsson GE. Osteoarthrosis/osteoarthritis. In: Zarb GA, Carlsson GE, Sessle BJ, Mohl MD (eds). Temporomandibular Joint and Masticatory Muscle Disorders. Copenhagen: Munksgaard, 1994:298–314.
10. Lavigne GJ, Kato T, Kolta A, Sessle BJ. Neurobiological mechanisms involved in sleep bruxism. Crit Rev Oral Biol Med 2003;14:30–46.

Bryant

1. Dorlands Illustrated Medical Dictionary, ed 29. Philadelphia: Saunders, 2000.
2. Mohl ND, Zarb GA, Carlsson GE. A Textbook of Occlusion. Chicago: Quintessence, 1988.
3. Moyers RE. Handbook of Orthodontics, ed 3. Chicago: Year Book Medical, 1973.
4. Angle EH. Treatment of Malocclusion of the Teeth and Fractures of the Maxillae: Angle System, ed 6. Philadelphia: White, 1900.
5. Proffit WR, Fields HW. Contemporary Orthodontics, ed 3. St Louis: Mosby, 2000.
6. Simon PW. Grundzuge Einer Systematischen Diagnostik der Gebiss-Anomalien. Berlin: Meusser, 1922.
7. Ackerman JL, Proffit WR. The characteristics of malocclusion: A modern approach to classification and diagnosis. Am J Orthod 1969;56:443–454.
8. Steiner CC. The use of cephalometrics as an aid to planning and assessing orthodontic treatment. Am J Orthod 1962;46:721–735.
9. Bolton WA. The application of tooth-size analysis. Am J Orthod 1962;48:504–529.
10. Tanaka MM, Johnson LE. The prediction of the size of unerupted canines and premolars in a contemporary orthodontic population. J Am Dent Assoc 1974;88:798–801.
11. Popovich F, Grainger RM. Burlington Orthodontic Research Centre Progress Report. 1957–1959, Series 2:5–42. Toronto: Toronto University, 1959.
12. Kelly J, Harvey C. An Assessment of the Teeth of Youths 12–17 years, DHEW Publication No. (HRA) 77-1644. Washington, DC: National Center for Health Statistics, 1977.
13. Rolling S. Hypodontia of permanent teeth in Danish schoolchildren. Scand J Dent Res 1980;88:365–369.
14. Bergendal B, Olgart K. Congenitally missing teeth. In: Kock G (ed). Oral Implants in Young Patients: State of the Art. Jönköping, Sweden: Institute for Postgraduate Dental Research, 1996:16–27.
15. Schalk van der Weide Y. Oligodontia: A Clinical, Radiographic and Genetic Evaluation [thesis]. Utrecht, The Netherlands: University of Utrecht, 1992.
16. Andreasen JO, Ravn JJ. Epidemiology of traumatic dental injuries to primary and permanent teeth in a Danish population sample. Int J Oral Surg 1972;1:235–239.
17. Jenny J. A social perspective on need and demand for orthodontic treatment. Int Dent J 1975;25:248–256.
18. Shaw WC, Rees G, Dawe M, Charles CR. The influence of dentofacial appearance on the social attractiveness of young adults. Am J Orthod 1985;87:21–26.
19. Cons NC, Jenny J, Kohout FJ. Perceptions of occlusal conditions in Australia, the German Democratic Republic, and the United States. Int Dent J 1983;33:200–206.
20. Shaw WC. Orthodontics and occlusal management. Br Dent J 1994;177:120–121.
21. Macgregor FC. Social and psychological implications of dentofacial disfigurement. Angle Orthod 1979;40:231–233.
22. Hobkirk JA, Goodman JR, Jones SP. Presenting complaints and findings in a group of patients attending a hypodontia clinic. Br Dent J 1994;177:337–339.
23. Ostler S, Kiyak HA. Treatment expectations vs outcomes in orthognathic surgery patients. Int J Adult Orthod Orthognath Surg 1991;6:247–256.
24. O'Mullane DM. Some factors predisposing to injuries of permanent incisors in school children. Br Dent J 1973;134:328–332.
25. Sadowsky C, BeGole EA. Long-term effects of orthodontic treatment on periodontal health. Am J Orthod 1981;80:156–172.
26. Helm S, Petersen PE. Causal relation between malocclusion and caries. Acta Odontol Scand 1989;47:217–221.
27. Sadowsky C, Polson AM. TMD and functional occlusion after orthodontic treatment: Results of two long-term studies. Am J Orthod 1984;86:386–390.
28. Riolo ML, Brandt D, TenHoeve TR. Associations between occlusal characteristics and signs and symptoms of TMJ dysfunction in children and young adults. Am J Orthod 1987;92:467–477.
29. Egermark-Eriksson I, Carlsson GE, Magnusson T. A long-term epidemiological study of the relationship between occlusal factors and mandibular dysfunction in children and adolescents. J Dent Res 1987;66:67–71.
30. Smith BGN, Knight JK. Tooth wear with aetiological factors: A comparison of patterns. Oral Health 1985;75:11–14.
31. Seligman DA, Pullinger AG, Solberg WK. The prevalence of dental attrition and its association with factors of age, gender, occlusion and TMJ symptomatology. J Dent Res 1988;67:1323–1333.
32. Hobo S, Takayama H. Oral Rehabilitation: Clinical Determination of Occlusion. Tokyo: Quintessence, 1997.
33. McCollum BB. Considering the mouth as a functioning unit as a basis of a dental diagnosis. J South Calif Dent Assoc 1938;5:268–276.
34. Stallard H, Stuart CE. Concepts of occlusion. Dent Clin North Am 1963;7:591–606.
35. Roth RH. Point/counterpoint. Am J Orthod 1995;107:315–318.
36. Rinchuse DJ. Counterpoint. Am J Orthod 1995;107:319–328.
37. Schuyler CH. Principles employed in full denture prosthesis which may be applied in other fields of dentistry. J Am Dent Assoc 1929;16:2045–2054.
38. Beyron HL. Characteristics of functionally optimal occlusion and principles of occlusal rehabilitation. J Am Dent Assoc 1954;48:648–656.
39. Beyron HL. Occlusion: Point of significance in planning restorative procedures. J Prosthet Dent 1973;30:641–652.

Walther

1. Beyron H. Optimal occlusion. Dent Clin North Am 1969;13:537–652.
2. Battistuzzi PGFCM, Eschen S, Peer PGM. Contacts in maximal occlusion. J Oral Rehabil 1982;9:499–507.
3. Gazit E, Fitzig S, Lieberman MA. Reproducibility of occlusal marking techniques. J Prosthet Dent 1986;55:505–509.
4. Harper KA, Setchell DJ. The use of shimstock to assess occlusal contacts: A laboratory study. Int J Prosthodont 2002;15:347–352.
5. Millstein P, Maya A. An evaluation of occlusal contact marking indicators. A descriptive quantitative method. J Am Dent Assoc 2001;132:1280–1286.
6. Berry DC, Singh BP. Daily variations in occlusal contacts. J Prosthet Dent 1983;50:386–391.
7. Riise C. A clinical study of the number of occlusal tooth contacts in the intercuspal position at light and hard pressure in adults. J Oral Rehabil 1982;9:469–477.
8. Reiber T, Müller F. Klinische Untersuchungen zur statischen Okklusion. Dtsch Zahnarztl Z 1994;49:363–366.
9. Stipetic J, Celebic A, Baucic I, et al. Analysis of occlusal contacts in different types of prosthodontic appliances, Eichner classifications, presence RCP-ICP slide and the type of occlusion. Coll Antropol 2001;25:311–316.
10. Ferrario VF, Serrao G, Dellavia C, Caruso E, Sforza C. Relationship between the number of occlusal contacts and masticatory muscle activity in healthy young adults. Cranio 2002;20:91–98.
11. Witter DJ, Creugers NH, Kreulen CM, de Haan AF. Occlusal stability in shortened dental arches. J Dent Res 2001;80:432–436.
12. Witter DJ, van Palenstein Heldermann WH, Creugers NHJ, Käyser AE. The shortened dental arch concept and its implications for oral health care. Community Dent Oral Epidemiol 1999;27:249–258.
13. Ericsson I, Lindhe J. Lack of effect of trauma from occlusion on the recurrence of experimental periodontitis. J Clin Periodontol 1977;4:115–127.
14. Nyman S, Lindhe J, Lundgren D. The role of occlusion for the stability of fixed bridges in patients with reduced periodontal tissue support. J Clin Periodontol 1975;2:53–66.
15. Marbach JJ. Orofacial phantom pain: Theory and phenomenology. J Am Dent Assoc 1996;127:221–229.
16. Wessberg GA, Washburn MC, Epker BN, Kent OD. Evaluation of mandibular rest position in subjects with diverse dentofacial morphology. J Prosthet Dent 1982;48:451–460.
17. Miralles R, Dodds C, Palazzi C, et al. Vertical dimension. Part 1: Comparison of clinical freeway space. Cranio 2001;19:230–236.
18. Clark GT, Tsukujama Y, Baba K, Simmons M. The validity and utility of disease detection methods and of occlusal therapy for temporomandibular disorders. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1997;83:101–106.
19. Rivera-Morales WC, Mohl ND. Relationship of vertical dimension to the health of the masticatory system. J Prosthet Dent 1991;65:547–553.
20. Reynolds JM. The organization of occlusion for natural teeth. J Prosthet Dent 1971;26:56–67.
21. Stuart CE. The contributions of gnathology to prosthodontics. J Prosthet Dent 1973;30:607–608.
22. Anderson JD. The need for criteria on reporting treatment outcomes. J Prosthet Dent 1998;79:49–55.
23. Svedstrom-Oristo AL, Pietila T, Pietila I, Helenius H, Peitzfeldt P, Varrela J. Selection of criteria for assessment of occlusal acceptibility. Acta Odontol Scand 2002;60:160–166.
24. Awad AA, Shapiro SH, Lund JP, Feine JS. Determinants of patients' treatment preferences in a clinical trial. Community Dent Oral Epidemiol 2000;28:119–125.
25. de Grandmont P, Feine JS, Taché R, et al. Within subject comparisons of implant-supported mandibular prostheses—Psychometric evaluation. J Dent Res 1994;73:1096–1104.

Alstergren

1. Wennerberg A, Carlsson GE, Jemt T. Influence of occlusal factors on treatment outcome: A study of 109 consecutive patients with mandibular implant-supported fixed prostheses opposing maxillary complete dentures. Int J Prosthodont 2001;14:550–555.
2. Pullinger AG, Seligman DA, Gornbein JA. A multiple logistic regression analysis of the risk and relative odds of temporomandibular disorders as a function of common occlusal features. J Dent Res 1993;72:968–979.
3. Pullinger AG, Seligman DA. Quantification and validation of predictive values of occlusal variables in temporomandibular disorders using a multifactorial analysis. J Prosthet Dent 2000;83:66–75.
4. Forssell H, Kalso E, Koskela P, Vehmanen R, Puukka P, Alanen P. Occlusal treatments in temporomandibular disorders: A qualitative systematic review of randomized controlled trials. Pain 1999;83:549–560.
5. Karjalainen M, Le Bell Y, Jamsa T, Karjalainen S. Prevention of temporomandibular disorder-related signs and symptoms in orthodontically treated adolescents. A 3-year follow-up of a prospective randomized trial. Acta Odontol Scand 1997;55:319–324.
6. Randow K, Carlsson K, Edlund J, Öberg T. The effect of an occlusal interference on the masticatory system. An experimental investigation. Odontol Revy 1976;27:245–256.
7. Ernberg M, Lundeberg T, Kopp S. Pain and allodynia/hyperalgesia induced by intramuscular injection of serotonin in patients with fibromyalgia and healthy individuals. Pain 2000;85:31–39.

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American Academy of Orofacial Pain. Orofacial Pain. Guidelines for Assessment, Diagnosis, and Management. Chicago: Quintessence, 1996.

Carlsson GE, Egermark I, Magnusson T. Predictors of signs and symptoms of temporomandibular disorders: A 20-year follow-up study from childhood to adulthood. Acta Odontol Scand 2002;60:180–185.

Carlsson GE, Johansson A, Lundqvist S. Occlusal wear. Acta Odontol Scand 1985;43:83–90.

Ciancaglini R, Gherlone EF, Radaelli G. The relationship of bruxism with craniofacial pain and symptoms from the masticatory system in the adult population. J Oral Rehabil 2002;28:842–848.

De Boever JA, Carlsson GE. Etiology and differential diagnosis. In: Zarb GA, Carlsson GE, Sessle BJ, Mohl ND (eds). Temporomandibular Joint and Masticatory Muscle Disorders, ed 2. Copenhagen: Munksgaard, 1994:171–187.

Eckert SE, Meraw SJ, Weaver AL, Lohse CM. Early experience with Wide-Platform Mk II implants. Part I: Implant survival. Part II: Evaluation of risk factors involving implant survival. Int J Oral Maxillofac Implants 2001; 16:208–216.

Ekfeldt A, Christiansson U, Eriksson T, et al. A retrospective analysis of factors associated with multiple implant failures in maxillae. Clin Oral Implants Res 2001;12:462–467.

Huang GJ, Leresche L, Critchlow CW, Martin MD, Drangholt MT. Risk factors for diagnostic subgroups of painful temporomandibular disorders (TMD). J Dent Res 2002;81:284–288.

Hublin C, Kaprio J, Partinen M, Koskenvuo M. Sleep bruxism based on self-report in a nationwide twin cohort. J Sleep Res 1998;7:61–67.

Kamisaka M, Yatani H, Kuboki T, Matsuka Y, Minakuchi H. Four-year longitudinal course of TMD symptoms in an adult population and the estimation of risk factors in relation to symptoms. J Orofac Pain 2000;14:224–232.

Kato T, Thie NM, Montplaisir JY, Lavigne GJ. Bruxism and orofacial movements during sleep. Dent Clin North Am 2001;45:657–684.

Lobbezoo F, Lavigne GJ. Do bruxism and temporomandibular disorders have a cause-and-effect relationship? J Orofac Pain 1997;11:15–23.

Lynch CD, McConnell RJ. The cracked tooth syndrome. J Can Dent Assoc 2002;68:470–475.

Misch CE. Clenching and its effect on implant treatment plans. Oral Health 2002;Aug:11–21.

Mohl ND, Ohrback R. Clinical decision-making. In: Zarb GA, Carlsson GE, Sessle BJ, Mohl ND (eds). Temporomandibular Joint and Masticatory Muscle Disorders, ed 2. Copenhagen: Munksgaard, 1994:563–583.

Molina OF, dos Santos J, Mazzetto M, Nelson S, Nowlin T, Mainieri ET. Oral jaw behaviors in TMD and bruxism: A comparison study by severity of bruxism. J Craniomandib Pract 2001;19:114–122.

Ohayon MM, Li KK, Guilleminault C. Risk factors for sleep bruxism in the general population. Chest 2001;119:53–61.

Spilker B. Guide to Clinical Trials. New York: Raven Press, 1991.

Thie N, Kato T, Bader G, Montplaisir JY, Lavigne GJ. The significance of saliva during sleep and the relevance of oromotor movements. Sleep Med Rev 2002;6:213–227.

Koyano et al

1. Pierce CJ, Gale EN. Methodological considerations concerning the use of Bruxcore Plates to evaluate nocturnal bruxism. J Dent Res 1989;68: 1110–1114.
2. Nishigawa K, Bando E, Nakano M. Quantitative study of bite force during sleep associated bruxism. J Oral Rehabil 2001;28:485–491.

3. Rugh JD, Barghi N, Drago CJ. Experimental occlusal discrepancies and nocturnal bruxis. J Prosthet Dent 1984;51:548–553.
4. Velly-Miguel AM, Montplaisir J, Rompre PH, Lund JP, Lavigne GL. Bruxism and other orofacial movements during sleep. J Craniomandib Disord Facial Oral Pain 1992;6:71–81.
5. Smith BGN, Knight JK. An index for measuring the wear of teeth. Br Dent J 1984;156:435–438.
6. Seligman DA, Pullinger AG, Solberg WK. The prevalenceof dental attrition and its association with factors of age, gender, occlusion and TMJ symptomatology. J Dent Res 1988;67:1323–1333.
7. Pintado MR, Anderson GC, DeLong R, Douglas WH. Variation in tooth wear in young adults over a two-year period. J Prosthet Dent 1997;77: 313–320.
8. Ekfeldt A, Hugoson A, Bergendal T, Helkimo M. An individual tooth wear index and an analysis of factors correlated to incisal and occlusal wear in an adult Swedish population. Acta Odontol Scand 1990;48:343–349.
9. Ichiki R, Koyano K, Tsukiyama Y. Effect of occlusal splint designs on nocturnal masticatory muscle activities [abstract 330]. J Dent Res 1999;78 (special issue):147.
10. Dao TT, Lund JP, Lavigne GJ. Comparison of pain and quality of life in bruxers and patients with myofascial pain of the masticatory muscles. J Orofac Pain 1994;8:350–356.
11. Magnusson T, Egermark I, Carlsson GE. A longitudinal epidemiologic study of signs and symptoms of temporomandibular disorders from 15 to 35 years of age. J Orofac Pain 2000;14:310–319.
12. Lavigne GJ, Montplaisir JY. Restless legs syndrome and sleep bruxism: Prevalence and association among Canadians. Sleep 1994;17:739–743.
13. Gross AJ, Rivera-Morales WC, Gale EN. A prevalence study of symptoms associated with TM disorders. J Craniomandib Disord 1988;2:191–195.
14. Hublin C, Kaprio J, Partinen M, Koskenvuo M. Sleep bruxism based on self-report in a nationwide twin cohort. J Sleep Res 1998;7:61–67.
15. Plesh O, Curtis DA, Hall LJ, Miller A. Gender difference in jaw pain induced by clenching. J Oral Rehabil 1998;25:258–263.
16. Ekfeldt A. Incisal and occlusal tooth wear and wear of some prosthodontic materials. An epidemiological and clinical study. Swed Dent J Suppl 1989;65:1–62.
17. McGuire MK, Nunn ME. Prognosis versus actual outcome. III. The effectiveness of clinical parameters in accurately predicting tooth survival. J Periodontol 1996;67:666–674.
18. Ratcliff S, Becker IM. Quinn L. Type and incidence of cracks in posterior teeth. J Prosthet Dent 2001;86:168–172.
19. Ekfeldt A, Christiansson U, Eriksson T, et al. A retrospective analysis of factors associated with multiple implant failures in maxillae. Clin Oral Implants Res 2001;12:462–467.
20. Brägger U, Aeschlimann S, Burgin W, Hämmerle CH, Lang NP. Biological and technical complications and failures with fixed partial dentures (FPD) on implants and teeth after four to five years of function. Clin Oral Implants Res 2001;12:26–34.

Dao

1. Fletcher RH, Fletcher S, Wagner EH. Clinical Epidemiology. The Essentials. Baltimore: Williams & Wilkins, 1988.
2. John MT, Hirsch C, Drangsholt MT, Mancl LA, Setz JM. Overbite and overjet are not related to self-report of temporomandibular disorder symptoms. J Dent Res 2002;81:164–169.
3. Pullinger AG, Seligman DA. Quantification and validation of predictive values of occlusal variables in temporomandibular disorders using a multifactorial analysis. J Prosthet Dent 2000;83:66–75.
4. Forssell H, Kalso E, Koskela P, Vehmanen R, Puukka P, Alanen P. Occlusal treatments in temporomandibular disorders: A qualitative systematic review of randomised controlled trials. Pain 1999;83:549–560.
5. Rubinoff MS, Gross A, McCall WD Jr. Conventional and nonoccluding splint therapy compared for patients with myofascial pain dysfunction syndrome. Gen Dent 1987;35:502–506.
6. Ekberg EC, Sabet ME, Petersson A, Nilner M. Occlusal appliance therapy in a short-term perspective in patients with temporomandibular disorders correlated to condyle position. Int J Prosthodont 1998;11:263–268.
7. Dao TTT, Lavigne GJ, Charbonneau A, Feine JS, Lund JP. The efficacy of oral splints in the treatment of myofascial pain of the jaw muscles: A controlled clinical trial. Pain 1994;56:85–94.
8. Seligman DA, Pullinger AG. Analysis of occlusal variables, dental attrition, and age for distinguishing healthy controls from female patients with intracapsular temporomandibular disorders. J Prosthet Dent 2000;83:76–82.
9. De Kanter RJ, Truin GJ, Brugersdijk RC, et al. Prevalence in the Dutch adult population and a meta-analysis of signs and symptoms of temporomandibular disorder. J Dent Res 1993;72:1509–1518.
10. Tervonen T, Knuuttila M. Prevalence of signs and symptoms of mandibular dysfunction among adults aged 25, 35, 50 and 65 years in Ostrobothnia, Finland. J Oral Rehabil 1988;15:455–463.
11. Harriman LP, Snowdon DA, Messer LB, et al. Temporomandibular joint dysfunction and selected health parameters in the elderly. Oral Surg Oral Med Oral Pathol 1990;70:406–413.
12. Bibb CA, Atchison KA, Pullinger AG, Bittar GT. Jaw function status in an elderly community sample. Community Dent Oral Epidemiol 1995;23: 303–308.
13. Sato H, Osterberg T, Ahlqwist M, Carlsson GE, Grondahl HG, Rubinstein B. Association between radiographic findings in the mandibular condyle and temporomandibular dysfunction in an elderly population. Acta Odontol Scand 1996;54:384–390.

Nilner

1. Beyron H. Occlusion: Point of significance in planning restorative procedures. J Prosthet Dent 1973;30:641–652.
2. Ball C, Sackett DL, Phillips B, Haynes D, Straus S, Daves M. Levels of evidence and grades of recommendation. Oxford Centre for Evidence-Based Medicine. (http://cebm.jr2.ox.ac.uk/levels.html) Statement of purpose and methods. J Evid Base Dent Pract 2002;2:6A–7A.
3. Allen FP, Macmillan AS, Locker D. An assessment of sensitivity to change of the oral health impact profile in a clinical trial. Community Dent Oral Epidemiol 2001;29:175–182.
4. Hatch JP, Shinkai RSA, Sakai S, Rugh JD, Paunovich ED. Determinants of masticatory performance in dentate adults. Arch Oral Biol 2000;46:641–648.
5. Wänman A, Agerberg G. Etiology of craniomandibular disorders: Evaluation of some occlusal and psychosocial factors in 19-year-olds. J Craniomandib Disord 1991;5:35–44.
6. Henriksson T, Ekberg EC, Nilner M. Masticatory efficiency and ability in relation to occlusion and mandibular dysfunction in girls. Int J Prosthodont 1998;11:125–132.
7. Henriksson T, Nilner M, Kurol J. Signs of temporomandibular disorders in girls receiving orthodontic treatment. A prospective and longitudinal comparison with untreated Class II malocclusions and normal occlusion subjects. Eur J Orthod 2000;22:271–281.
8. Riise C. A clinical study of the number of occlusal tooth contacts in the intercuspal position at light and hard pressure in adults. J Oral Rehabil 1982;9:469–477.
9. Witter DJ, van Elteren P, Käyser AF. Signs and symptoms of mandibular dysfunction in shortened dental arches. J Oral Rehabil 1988;15:413–420.
10. Witter DJ, van Elteren P, Käyser AF, van Rossum GMJM. Oral comfort in shortened dental arches. J Oral Rehabil 1990;17:137–143.
11. De Boever JA, Carlsson GE, Klineberg IJ. Need for occlusal therapy and prosthodontic treatment in the management of temporomandibular disorders. Part II. Tooth loss and prosthodontic treatment. J Oral Rehabil 2000;27:647–659.
12. De Boever JA, Carlsson GE, Klineberg IJ. Need for occlusal therapy and prosthodontic treatment in the management of temporomandibular disorders. Part I. Occlusal interferences and occlusal adjustment. J Oral Rehabil 2000;27:367–379.
13. Hakestam U, Söderfeldt B, Ryden O. Dimensions of satisfaction among prosthodontic patients. Eur J Prosthodont Restorative Dent 1997;5:111–117.
14. Vallon D, Nilner M. A longitudinal follow-up of the effect of occlusal adjustment in patients with craniomandibular disorders. Swed Dent J 1997;21:85–91.
15. Marklund S, Wänman A. A century of controversy regarding the benefit or detriment of occlusal contacts on the mediotrusive side. J Oral Rehabil 2000;27:553–562.
16. Rivera-Morales WC, Mohl ND. Relationship of occlusal vertical dimension to the health of the masticatory system. J Prosthet Dent 1991;65:547–553.
17. Jokstad A. Evidence-based medicine applied to fixed prosthodontics. In: Karlsson S, Nilner K, Dahl B (eds). A Textbook of Fixed Prosthodontics: The Scandinavian Approach. Stockholm: Gothia, 2000:330–349.

18. Norman GR, Lee R, Cunnington PW, Shali V, Marriott M, Regher G. Pattern recognition. Acad Med 1996;71:862–864.
19. Bowden J, Marton F. The University of Learning. Beyond Quality and Competence in Higher Education. London: Kogan Page, 1988.

Bartlett

1. Smith BGN, Robb ND. The prevalence of toothwear in 1007 dental patients. J Oral Rehabil 1996;23:232-239.
2. O'Brien M. Children's Dental Health in the United Kingdom. London: OPCS, 1993:1-130.
3. Bartlett DW, Coward PY, Nikkah C, Wilson RF. The prevalence of tooth wear in a cluster sample of adolescent schoolchildren and its relationship with potential explanatory factors. Br Dent J 1998;184:125-129.
4. Bartlett DW, Phillips KM, Smith BGN. A difference of perspective—The North American and European interpretations of tooth wear. Int J Prosthodont 1999;12:401-408.
5. Grenby TH, Phillips A, Desai T, Mistry M. Laboratory studies of dental properties of soft drinks. Br J Nutr 1989;62:451-464.
6. Moazzez M, Smith BGN, Bartlett DW. Oral pH and drinking habit during ingestion of a carbonated drink in a group of adolescents with dental erosion. J Dent 2000;28:395-397.
7. Clark DC. Oral complications of anorexia nervosa and/or bulimia. J Oral Med 1985;40:134-138.
8. Bartlett DW, Evans DF, Anggiansah A, Smith BGN. A study of the association between gastro-esophageal reflux and palatal dental erosion. Br Dent J 1996;181:125-132.
9. Chadwick RG, Mitchell HL, Cameron I, Hunter B, Tulley M. Development of a novel system for assessing tooth and restorations wear. J Dent 1997;25:41-47.
10. Bartlett DW, Blunt L, Smith BGN. Measurement of tooth wear in patients with palatal erosion. Br Dent J 1997;182:179-184.
11. Azzopardi A, Bartlett DW, Watson TF, Sherriff M. The measurement and prevention of erosion and abrasion. J Dent 2001;29:395-400.
12. Bashir E, Ekberg O, Lagerlof F. Salivary clearance of citric acid after an oral rinse. J Dent 1995;23:209-212.
13. Meurman JH, ten Cate JM. Pathogenesis and modifying factors of dental erosion. Eur J Oral Sci 1996;104:199-206.
14. Dahl BL, Krogstad O. The effect of partial bite raising splint on the occlusal face height. An x-ray cephalometric study in human adults. Acta Odontol Scand 1982;40:17-24.
15. Dahl BL, Krogstad O, Karlson K. An alternative treatment in cases with advanced attrition. J Oral Rehabil 1975;2:209-214.
16. Gough MB, Setchell D. A retrospective study of 50 treatments using an appliance to produce localised occlusal space by relative axial tooth movement. Br Dent J 1999;187:134-139.
17. Tucker K, Adams M, Shaw L, Smith AJ. Human enamel as a substrate for in vitro acid dissolution studies: Influence of tooth structure and morphology. Caries Res 2002;32:135-140.
18. Xhonga FA, Sognnaes RA. Dental erosion: Progress of erosion measured clinically after various fluoride applications. J Am Dent Assoc 1973;87:1223-1228.
19. Hemmings KW, Darbar UR, Vaughan S. Tooth wear treated with direct composite restorations at an increased vertical dimension: Results at 30 months. J Prosthet Dent 2000;83:287-293.
20. Burke FJ, Qualtrough AJ, Hale RW. Dentin-bonded all-ceramic crowns: Current status. J Am Dent Assoc 1998;129:455-460.

Klineberg and Stohler

1. Beyron H. Occlusion: Point of significance in planning restorative procedures. J Prosthet Dent 1973;30:641–652.
2. Sessle BJ. Dental research: Strategic advances and the issue of quality. J Dent Res 1994;73:1241–1244.
3. Bland M, Peacock J. Statistical Questions in Evidence-Based Medicine. Oxford, UK: Oxford University Press, 2001.
4. Peat JK, Mellis C, Williams K, Xuan W. Health Science Research. A Handbook of Quantitative Methods. Sydney: Allen and Unwin, 2001.
5. Marbach JJ, Raphael KG, Janal MN, Herschkorn-Roth R. Reliability of clinical judgment of bruxism. J Oral Rehabil 2003;30:113–118.
6. Walton TR. An up to 15-year longitudinal study of 515 metal-ceramic FPDs: Part 1. Outcome. Int J Prosthodont 2002;15:439–445.