# **Biological Adaptation and Normative Values**

Barry J. Sessle, BDS, MDS, BSc, PhD, DSc(hc), FRSC Professor and Canada Research Chair Faculty of Dentistry, University of Toronto Ontario, Canada

#### 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.<sup>1-6</sup>

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 genebased 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).<sup>1-3,5,7-9</sup>

#### 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,

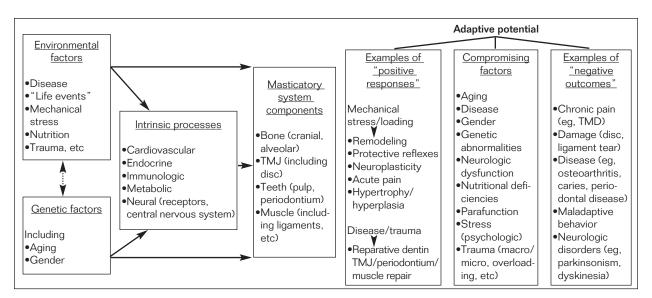


Fig 1 Biologic determinants and adaptive potential.

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).<sup>1-5,7,9,10</sup> All these outcomes can be associated with serious impairment of the function of the masticatory system and thereby influence the occlusal interface.

### 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, conduc-

tivity) 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).

# References

- 1. Hargreaves KM, Goodis HE. Seltzer and Bender's Dental Pulp. Chicago: Quintessence, 2002.
- Kopp S. Topical review: neuroendocrine, immune, and local responses related to temporomandibular disorders. J Orofac Pain 2001;15:9–28.
- 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 Press, 1995:89–112.
- 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.

- 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.
- 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 Press, 1995:257–272.
- 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.
- Seltzer Z, Dorfman R. Identifying genetic and environmental risk factors for chronic orofacial pain syndromes: human models. J Orofac Pain 2004;18:311–317.
- 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.
- Lavigne GJ, Kato T, Kolta A, Sessle BJ. Neurobiological mechanisms involved in sleep bruxism. Crit Rev Oral Biol Med, 2003;14:30–46.

# Satisfactory Occlusal Relations for the Individual with a Craniofacial Anomaly

Bruce Ross, DDS, MSc Division of Orthodontics Hospital for Sick Children Toronto, Canada

#### 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 temporo-mandibular 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 selfesteem, 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 Copyright of International Journal of Prosthodontics is the property of Quintessence Publishing Company Inc. and its content may not be copied or emailed to multiple sites or posted to a listserv without the copyright holder's express written permission. However, users may print, download, or email articles for individual use.