

Dentistry's Conundrum

Gene McCoy*

Dentist in San Francisco, California, USA.

*Correspondence:

Gene McCoy, Dentist in San Francisco, California, USA.

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SUMMARY

The dental profession's understanding of occlusion and temporomandibular disorders remains clouded by fundamental terminological and conceptual misalignments. The conflation of "occlusion" with masticatory function represents a persistent red herring in dental discourse. This semantic misdirection distracts from evaluating the masticatory system's biological complexity. To resolve this, occlusion should be strictly defined as dental closure, while masticatory function must be assessed independently—specifically, distinguishing between normal function and pathological dysfunction.

Awaiting definitive studies to explain TMDs parallels waiting indefinitely at an abandoned depot. A more productive framework emerges through analogy: Just as carpal tunnel syndrome is causally linked to typing without requiring exhaustive evidence, TMD etiology logically centers on parafunctional vertical clenching. This mechanism targets the temporomandibular joint menisci with forces reaching 1,600 Newtons, positioning it as the primary etiological driver.

The prevailing question of occlusion's relationship to TMDs should be replaced with two targeted inquiries: What is the liability of parafunction (clenching) in TMD development and does malocclusion initiate the parafunction?

Keywords

Dentistry, Oral, Parafunctional behaviors.

Dentistry serves as the gatekeeper for oral and maxillofacial health, tasked with identifying and managing disease and parafunctional behaviors that impact this critical biological system. Yet, the global epidemic of temporomandibular disorders (TMDs) reveals a glaring failure in fulfilling this responsibility. Despite decades of research and clinical advancements the dental profession has struggled to adopt effective diagnostic and treatment paradigms for TMDs. This paper aims to review the circumstances that created this conundrum.

The Human Mastication System

is the simplest of all our biological systems. Its primary purpose is to break down food as it begins its journey through the digestive system from mouth to anus. It consists of a movable mandible embedded with a u-shaped row of teeth opposing another u-shaped row of teeth in the maxilla. Cradled in a muscular sling, the

mandible functions vertically in a slight elliptical motion toward the maxilla where opposing overlapping teeth mechanically break down boluses of food to begin the digestive process. The mandible's condyles are articulated to the glenoid fossae of the maxilla insulated by cartilaginous discs, menisci. When full closure (occlusion) occurs, the condyles are comfortably seated in the anterior-superior position of their respective fossae: centric relation (CR). Other incomplete occlusions may occur due to swallowing anterior to CR depending on the posture of the body: centric occlusion (CO). When the mandible is not functioning and free from dysfunction, it rests with the teeth slightly apart.

An Acceptable Occlusion

is any closure that ensures CR; however, one has to consider the force behind it. If it is a diminished force such as a swallowing CO, it is of little consequence even if it is anterior to CR. If it is an empowered clenching force capable of forces up to 1640 Newtons [1], it can result in dire consequences to the TMJ in both CO and CR.

Why has Occlusion Gained such a Controversial Reputation?

The word “occlusion” is defined as closure, whether it be a door, heart valve, or teeth. In dentistry, according to Dorland’s Medical Dictionary, it is a static definition focused on tooth contact or closure. As dental science evolved, there was a need to understand the complexity of the entire mastication system, not just static closure. So, by the late 20th century, the definition of occlusion was broadened to include the dynamic and functional relationships among all the components of the mastication system. The term “functional occlusion” then emerged during this period of conceptual expansion along with a recognized growing confusion. Creating two different interpretations of the term introduced ambiguity that shifted focus away from the holistic evaluation of the system itself.

Not only was the term inherently contradictory; it implied that there was a specific occlusal scheme that was prerequisite for proper function. There were questions as to what constituted “good” or “bad” occlusion, how to define it, and how to achieve it. And most importantly, what did the way teeth touched each other in closure have to do with the system? The role of any biological system’s gatekeeper remains constant: to understand normal function and to identify and manage dysfunction or disease. This should have been the guiding principle for the dental profession, rather than complicating the terminology.

The American Dental Association (ADA) seeking clarity, initiated three conferences to interpret the general concept of occlusion to fit comfortably into a dental school’s curriculum. The first conference (1952) was chaotic with only one point of agreement: that any concept of occlusion should fit into one of two categories, “harmonious medium occlusion” (functional) and “disharmonious peripheral occlusion” (parafunctional). Unresolved, the 1975 conference again sought to define end goals without success. However, one important point of clarity did emerge from that meeting: Dr. Parker Mahan stated that a preferred goal was “to understand normal stomatognathic system function, to recognize occlusal dysfunction to determine its etiology, and how to treat it”. Dr. Mahan was spot-on. It is the goal of the restorative dentist to manage the system free from parafunction; referring to the system as “functional occlusion” somehow implied that it could be achieved by the occlusal anatomy of the dentition, but there are many factors in the etiology of parafunction aside from how the teeth touch.

Recognizing and Managing Parafunction

While developmental anomalies, osteoarthritis, and trauma play a role in TMD etiology, at the heart of this misconception lies the profession’s inability to grasp the complexity of parafunction. Falling under the umbrella of bruxism [2], clenching and grinding are two fundamentally distinct and powerful activities that differ not only in their force application to the TMJ, but in their management. Unfortunately, the dental community has completely embraced and accommodated bruxism while ignoring the vertical form “dental compression syndrome” (DCS) [3]. It was not apparent then, since bruxism occurs while sleeping, that a guard would suffice, so in

the early nineteenth century management focused on equilibrating dentitions to achieve the best occlusal scheme to tolerate these destructive forces. The first approach—termed “group function”—aimed to distribute bruxism’s forces across the occlusal surfaces of bicusps and molars through equilibration. Later, D’Amico, informed by his studies on attrition [4], reduced these forces by directing them forward to the cuspids. Accordingly, a paradigm began to develop that sought to determine the best occlusal scheme to tolerate and minimize these lateral parafunctional forces. The focus on removing enamel from the occlusal surfaces of teeth to address lateral parafunctional forces (bruxism) has led to a narrow perspective on the mastication system, neglecting other crucial aspects such as vertical parafunctional forces, clenching. DCS is up to four times as powerful, lasts twice as long, has a unique set of footprints, focuses explicitly on the TMJ, and possesses a long list of etiological factors. While horizontal parafunction results in the condyles gliding past the discs with reduced impact, vertical parafunctional forces target the menisci directly, leading to microtrauma, inflammation, and eventual displacement. When displacement did occur, symptom management prioritized preventive measures by replacing the damaged cartilage discs with Teflon-coated implants. This strategy exacerbated the condition, resulting in the recall of 25,000 implants with one implant working its way into the brain: the cause, clenching [5]. While clenching and grinding share certain central nervous system triggers, DCS has an extensive etiological portfolio that includes medications, exercise, sports, sleep apnea, pain, fear, and stretching. DCS has been referred to as a silent disease [6], working quietly within one’s subconscious. It is therefore critical for the GP to recognize the signs in order to educate the patient for self-management.

Hard Tissue and Prosthetic Deformations of Dental Compression Syndrome

GINGIVAL NCLs

The unique loss of tooth structure, known as non-carious lesions (NCLs), has been a contentious topic among dentists for nearly a century. Figures 1 and 2.

WI Ferrier once remarked [7], “Their etiology seems to be shrouded in mystery”. We now understand that these multi-shaped deformations are examples of hard tissue fatigue due to compression failure. Fatigue, a weakened state brought about by repeated stressing, is one of the most insidious causes of loss of strength in a structure [8]. Fatigue factors did not come into recognition until the introduction of rotating metal machinery in the middle of the nineteenth century. In time with the emerging science of biomechanics, dentists began to recognize their significance in the study of the mechanical behavior of living materials; minute particles of tooth structure are being shed at vulnerable sites of high stress. Biological structures, such as teeth and bone are termed viscoelastic and are subject to deformation. Engineers refer to this type of fatigue as corrosion fatigue [9].

The cuspid in figure 2 demonstrates changing sites of failure as the alveolar bone recedes.



Figure 1



Figure 2

The cuspid in Figure 2 shows multiple sites of fatigue due to receding alveolar bone.

The Coupola

The most common NCL is a perfectly rounded depression, aka; occlusal dimples, found at the tips of functional cusps. Figure 3. Nothing of significance has been written about them except that they are associated with compression. Although the coupola contrasts dramatically in geometric design with the wedge-shaped NCLs, there are two striking similarities; they are both site specific in that they are found at sites of high stress on teeth, and they exhibit a glassy sheen. Kornfeld wrote about this phenomenon in 1932 when he observed that these defects were hard, smooth, and glasslike in appearance [10]. The author suggests that the glassy effect is due to the exit of positive ions produced by the compression of appetite crystals in the dentition and alveolar bone-a piezoelectric effect.



Figure 3

Deformation of Bone – Exostosis

Articles on torus palatinus and torus mandibularis have appeared since 1814 [11]. Figures 4 and 5 Although there is no consensus as to their etiology, many associate their occurrence with TMDs and masticatory hyperfunction. Specifically, it is suggested that the negative ions generated from the compression of appetite crystals are responsible for the aggregates of new bone growth.



Figure 4



Figure 5

Deformations of Restorative Materials

Fatigue easily manifests itself in prostheses and restorative materials such as amalgam and acrylic. Termed Luder Lines or molecular slip bands, the molecules in the alloy are rearranging themselves under the influence of compressive strain. Figures 6 and 7 One can demonstrate the effect by bending a metal coat hanger back and forth and examining the stress configuration that is produced.



Figure 6

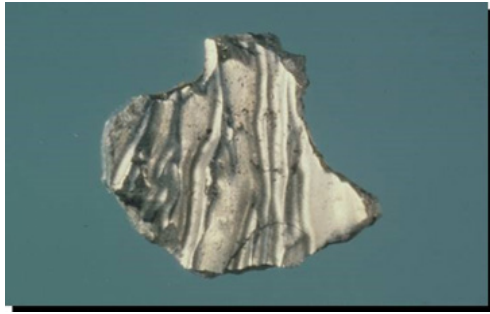


Figure 7

Fatigue in Acrylic appears as wavy lines or sets. Figures 8 and 9 The deformations in the oral environment are important diagnostic tools, but their appearance does not mean that the patient is currently affected with DCS, as it may have been from a prior stressful period in their lives.



Figure 8



Figure 9

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