The Curious History of Occlusion in Dentistry

Drs. Barry Glassman and Don Malizia discuss some myths about occlusion and bites, and challenge restorative dentists to rethink "what they know."

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Empirical evidence guides old maps, which prevents exploration of new frontiers

by Drs. Barry Glassman and Don Malizia

The tendency in dentistry is to think of occlusion as a static relationship that describes how maxillary and mandibular teeth fit together when the elevators contract and maintain contraction—in other words, maximum intercuspation. The concept is simple, but it’s fascinating how the concept of occlusion can evoke such significant disagreement and controversy. Occlusal concepts are at the heart of every dental visit every day in every dentist’s office, and yet there’s no greater cause of disagreement amongst dentists.

Competing concepts of “occlusion” have been at the center of the conflicting TMD camps over the years. The existing controversies are one of the contributing factors that has led to limited teaching of occlusal concepts at the undergraduate level in dental schools worldwide. At the heart of the problem: Many “facts” about occlusion have been introduced into dentistry empirically and passed down as truths.

By examining the history of “evidence” of occlusion, we can reveal how several assumptions about occlusion came into existence (and why they continue to be taught as “science”). Exposing these potential myths and beginning the discussion about their origin and validity is critical to help all dentists more thoroughly and
accurately understand the role of occlusion in their patient’s health and, consequently, improve their daily dentistry.

The history of the "science" of occlusion
An attempt to trace the history of the study of occlusion takes us back to 19th-century orthodontists, who sought to decipher nature’s grand plan for the arrangement of the dentition. At a meeting of the Philadelphia Academy of Stomatology in 1898, Edward H. Angle proposed that orthodontics be based on the science of dental occlusion and offered a definition of normal occlusion as the ideal to be attained in the treatment of malocclusion.¹

Angle’s concepts of an ideal occlusion became widely accepted as a goal of orthodontic treatment "and the basis of normal dental function and health." No serious attempts to examine this hypothesis were made until 75 years later, when the National Institute of Dental Research and the National Research Council of the National Academy of Sciences "assembled three independent panels of orthodontic experts to evaluate research related to malocclusion, variations in dental occlusion and disabling orthodontic conditions."²

The concepts of ideal occlusion developed by Angle, and the assumptions of the dysfunction and pain associated with malocclusions remained untested and unchallenged in dentistry.¹

In 1901, Karolyi’s paper³ discussing the role of occlusion in bruxism is among the first found, but contains no references. Without any evidence, Karolyi theorized that "abnormality of occlusal structure was a basis for abnormal temporomandibular joint function, abnormal masticatory muscle function, periodontal disease and bruxism."³ Ackerman says, referring to occlusion, that "there was even some quasi-religious beliefs about the nature and perfection of form that were tied to it." This theory was continually taught and passed down through generations. Dr. Peter Dawson, in his third edition, refers to the fact that "Karolyi got it right."⁴

In fact, the concepts became further reinforced by Costen in 1934.⁵ Costen, an otolaryngologist, used 11 case reports of patients with decreased vertical dimension who reported having ear symptoms that included altered hearing ability and dizziness. Costen referenced Goodfriend, whose work demonstrated joint changes in cadavers, but had no correlation to assumed symptoms, simply stating that there are changes in the joint structures, which occurred over time. He made the assumption that these changes were related to "occlusion."

Goodfriend dissected cadaver heads in the 1920s with attempts to relate the dental occlusion with joint anatomy.⁶ There was no control group, and he had no clinical or social histories on these cadavers. Nevertheless, he drew the conclusion that "muscle spasms, external injuries, deleterious habits, and stressful life situations unquestionably play an important role in the predisposition and exacerbation of craniofacial disorders." He said that therapy should consist of "re-establishment of harmonious balanced dental occlusion that supports adequate maxillomandibular relationships and positions the closed-jaw condyle relationship in the forecenter of the temporal fossa."⁷

Costen reports changes in symptoms with increased vertical dimension with an assumption that the altered pressures on the joint were responsible for those changes. "The mechanics of occlusion and dental problems are not included here"—as he proceeds to make the assumption that "occlusion," including vertical dimension, is directly related to TMJ pathology. He suggested that this altered pressure in the joint served as an etiology for glossopharyngeal neuralgia and altered Eustachian tube function. This, then, is the birth of the concept of the need for posterior joint support and what we have termed the "occlusion/pain and dysfunction connection"—a concept that continues to be taught today. Costen, referring to the work of Goodfriend, stated that either pathological or adaptive changes in the joints of cadavers occurred using a mechanical model of force distribution. They thus created an "anatomic explanation of pain connected with disturbed joint function" that, according to Costen, was "fairly simple."⁸

More recent work has delved deeply into force vectors in terms of direction and magnitude, and it’s now recognized that the forces are related to the direction of the muscle contraction as well as the details of the dental contact—including the observation that the more anterior the posterior point of dental contact is, the less force on the joint itself.⁹,¹⁰ It has also been shown that there is no relationship between "posterior support" and the development of degenerative changes in the joint.¹¹

Goodfriend, whom Costen references for the "dental science," needed to support his clinical findings and assumptions of mechanism. Both can truly be called pioneers, but their work was performed and written about without the use of the scientific method. The map they created is now outdated, yet its concepts are still taught.

Relying on faulty "evidence"
In 1956, Sears published a paper that further cements the relationship between occlusion and temporomandibular joint "disturbances," reporting that the use of pivot appliances had been so predictably effective for resolving both dental and nondental related symptoms that there had been a hesitation to publish the findings. Sears quotes McPhee, who writes, "In the evaluation of this survey, there appears to be a close relation between malocclusion and insanity."¹¹ The mechanism provided includes a report that the condyles are "forced into strained relations in the fossa" by the teeth meshing and that anterior contact creates a Class III lever, and suggests that the cause of these dental and nondental symptoms is the occlusion. Sears states, "The tooth occlusion out of harmony with jaw relation causes mechanical stress of the temporomandibular joint."¹¹ This paper continues to be referenced and is on the reading list for many prosthetic graduate programs to this
The work of Travell and Ramjford both appear in the early 1960s. Ramjford’s 1961 study involved occlusal adjustments on 32 patients with pre- and post-EMG studies with no control group. They reported complete relief of all patients with both intracapsular and extracapsular disorders by occlusal adjustments. Conversely to Costen’s work, they suggested that vertical dimension was related to symptoms, but that “any type of occlusal interference was found to trigger muscle spasms and pain and the “most significant interference was a discrepancy between centric relation and centric occlusion.” They reported bruxism in all 32 patients, and related the cause of bruxism to both psychic and occlusal factors.

Empirical versus evidence-based studies
The first area of concern is, of course, the fact that the definition of centric relation itself has undergone many changes, and the definition itself remains an area of controversy in dentistry. “It could be argued that the progressive modifications in the definition of CR have done more to eliminate centric slides than 20 years of grudging acquiescence of the precepts of gnathology.”

In addition is the predominance of evidence that has led to the occlusion and pain “disconnect.” Pullinger states, “Belief in and rejection of a relationship of occlusion and temporomandibular joint (TMJ) condyle-fossa position with normal and abnormal function are still contentious issues. Clinical opinions can be strong, but support in most published data (mostly univariate) is problematic.”

In her study in 2006, Michelotti introduced interferences into patients without causing increased symptoms, and in her review article in 2010 she listed a series of well-done studies that demonstrated no evidence between malocclusion and “TMD.”

Mock equilibrations have been done and have resulted in similar results to actual equilibrations. It is generally accepted that nocturnal bruxism is a centrally mediated disorder, and yet “old maps” continue to dominate dental education.

At about the same time that Ramjord’s study was released, Janet Travell’s theory of muscle spasm—particularly of the lateral pterygoid—related to the interferences Ramford was referencing began to surface. Travell references Morgan’s text, which had an entire chapter on muscle spasm, as well as a chapter by Goodfriend. Travell’s vicious-cycle theory suggests that dental interferences cause hyperactivity and muscle spasms, most notably in the lateral pterygoid. However, that spasm is very rare and occurs only in rare cases of dystonia. Furthermore, Lund demonstrated that interferences do not cause hyperactivity but results in hypoactivity. His evidence-based pain-adaptation theory replaced the vicious-cycle theory, but the latter continues to be taught in dentistry. Travell’s muscularily oriented theory suggesting a direct relationship to dental occlusion is critically important to those who depend on the role of interferences in occlusal theories—so the empirical evidence and anecdotal reports that support this theory continue to dominate claims, and are used to refute more recent science. The fact is that very little is understood about the physiology of muscle pain, and it is realized that trigger points are more likely a central sensitization issue, as opposed to a true peripheral physiological phenomenon.

Occlusion vs occluding: The stipulation of occlusion
It may seem, then, that there’s an argument for the hypothesis that there’s no relationship between occlusion and pain. From a clinical standpoint, this would be a very difficult argument to make, because every dentist has adjusted an occlusion and noted a significant change in a reported pain pattern. Every dentist has created a high spot in a restorative technique that led to a patient complaint that was then resolved with an occlusal adjustment. The issue, then, isn’t if occlusion matters, but when and how it matters. Looking specifically at the mechanisms that cause occlusion to be a factor in pain and dysfunction becomes the real issue.

As dentists, our education leads us to the tendency to “stipulate” occlusion: We tend to think of the maxillary and mandibular teeth as units that “fit” together. When given maxillary and mandibular stone models, the first thing dentists tend to do is put them together and evaluate how they “fit” in a goal to determine the quality of the “occlusion.” In reality, maximum intercuspation rarely occurs during function. There is some form of dental contact on an average about 20 minutes total in a 24-hour period. Occlusion” actually rarely occurs, as evaluated by articulating models. Occlusion as noted by articulating models occurs only in patients when their elevators go into contraction and stay contracted. This occurs when we ask a patient to voluntarily “close,” but not commonly in function. Dentists often check lateral movements on an articulator as though lateral excursions occur in function. Again, this is a result of the stipulation of occlusion being an essential part of the dental education.

As a result, when thinking about occlusion, we have suggested that we consider not just the occlusion itself, but when this “occlusion” occurs. We refer to the noun of occlusion as becoming a factor when it becomes the verb of occluding. In addition, we have suggested that maximum intercuspation is actually, in some ways, pathological.

This concept has often been misinterpreted as though it’s being suggested that occlusion doesn’t matter. Of course it does! What’s being proposed is that the stipulation of occlusion is at the heart of the empirical science of the 20th century, and a contributing factor that prevents the evidenced-based information from becoming part of the dental knowledge base. This “stipulation” makes the role of occlusion and the anecdotal changes we observe easy to explain, and many of the myths associated with occlusion are dependent on such stipulation.

Clearly, then, the occlusal changes can be related to the symptomatic changes, but the mechanism of that...
change needs to be reconsidered.

**Why are the old maps followed, rather than being rewritten?**

Alvin Toffler has written: "The illiterate of the 21st century will not be those who cannot read and write, but those who cannot learn, unlearn, and relearn."

It's a generally accepted fact that temporomandibular joint dysfunctions, as well as nonodontogenic pain patterns, are not points of emphasis in the undergraduate dental setting. Without exception, our general dental residents confirm this. Many of the "old maps" created the basis for various TMD philosophies taught by pioneers. These old maps became the guidelines for the concepts of legendary pioneers that either still teach or have surrogates who teach, resisting the newer information based on more powerful evidence than the empirical evidence of many of the 20th-century studies.

When the National Institute of Dental Research and the National Research Council of the National Academy of Sciences conducted their studies in the early to mid-1970s, they concluded that there was the lack of a clinically meaningful definition of malocclusion, and that there was no interference in function (or aesthetics) that could be related to malocclusion.\(^{24, 25}\)

Ackerman states, "The 'science of occlusion' emerged from a pseudoscientific tradition already characterized in the 19th century as 'composed merely of so-called facts connected together by a misapprehension under the guise of principles' and that, from the beginning, there were strong overtones of religious belief in the concept of occlusion."

The "TMD" camps and restorative gurus, thus, continue to teach many of these myths, and the myths are taught as facts, leading to controversy ... often with religious fervor.

There seems to be a concern that our old pioneers will be proven incorrect. Under no circumstances would we suggest that those who created the early maps were wrong, and today they remain respected pioneers. Sadly, this has not been the case in dentistry.

Much has been learned and continues to be learned about the role of mandibular growth and function. Ackerman writes, "A departure from the dogma of ideal occlusion does not reduce contemporary orthodontists to 'de facto cosmetologists,' but rather frees them to enhance a patient’s dentofacial appearance and, in some cases, oral function. It opens the way to establishing a scientifically sounder model of occlusal function and oral health. Regrettably, some orthodontists, to paraphrase W.C. Fields, would on the whole rather be in 19th-century Philadelphia."

New maps would free the general dentist to understand in greater depth the role of occlusion in pain and dysfunction as a potential contributing factor, rather than making assumptions about its key role and the need to idealize occlusions based on some non-evidence-based concepts. The acceptance of the contributing role of occlusion in temporomandibular joint pathology as well as extracapsular disorders would allow other contributing factors to be appropriately considered without eliminating inappropriate dental contact as a potential contributing factor itself. Greene and Reid have carefully reviewed the key role of conservative reversible therapies and the need to have specific diagnostically driven therapies based upon well-designed risk benefit considerations in an ethical dental model.\(^{27}\)

The old maps that lead us only to the role of occlusion and interferences in pain and dysfunction often take us down the wrong road, and often lead dentists in honest attempts to help their patients with pain and dysfunction down frustrating paths.

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**Learn more about occlusion for CE credits**

Dr. Barry Glassman’s two-hour online course gives useful, common-sense advice and reviews TMD concepts so common patient reports of pain and dysfunction can be more readily diagnosed and conservatively treated. To take the course and earn 2.5 hours of CE credit.
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Interesting and important to know where we came from...so we can make better decisions in where we are going!
excellent work here Barry and Don!

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Howard Goldstein
Super Admin

Great history on an important topic... Thanks!!

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The Curious History of Occlusion in Dentistry
Jason and Howard....

THANKS....

I did not realize that this was published. I was just sent this link.

I personally believe this is a very important subject -- and it may be misleading to think that it simply reviews the "history" of occlusion.

Having a greater understanding of "occlusion" vs. "occluding" and understanding how and why "occlusion" matters is of critical importance. Understanding occlusal dyesthesias is more important for general dentistry than is generally accepted, in my humble opinion, Understanding our role in the development of the disorder is critical.

I hope this helps some who read it.

Hope you guys are well

barry

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Wow! Where to start? Where/how does reference 10 support your statement? "It has also been shown that there is no relationship between "posterior support" and the development of degenerative changes in the joint.10

Sounds like a title of a presentation that will be given at the AES next month......
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velogeek
Official Townie

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April 2017...........

because you asked

McGill

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mostofthosewhoclaimt
oknowNMdont

The author of the article wrote: "It has been shown that there is no relationship between posterior support and the development of degenerative changes in the joint." A reference was cited - which implies that the reference gives support for that statement. I'm having a hard time finding anything the the referenced article that shows that there is no relationship. I'm either missing something in the referenced article or it was a bogus citation. I'm simply asking for help determining which the answer is.

By the way, I'm sure you're well aware that if you get soap in the end of your urinary canal while washing it, and then you urinate, it hurts like heck! And you don't need any supporting literature to know that, do you?
Are you bothered that they did not specifically look at the joint?

I'm bothered that someone who who many people consider an expert in the field and listed to for advice, would write such a statement and send it to the masses along with a citation to a study that he says supports his statement. How else does internal derangement of a joint ever occur when there has been no acute traumatic blow to the jaw?
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mostofthosewhoclaimt

oknowNMdont

Official Townie
Posts: 102
Last Post: 01/14/17
Member Since: 06/04/04

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A traumatic blow is the only way an internal derangement can occur? I'm sure Okeson and Piper might disagree.

I think we're on the same page there. I didn't mean that a traumatic blow is the only way to get internal derangement, I meant it is the only other way besides the usual way, which is lack of posterior occlusal support (and then overclosing and/or retruding on the posterior hypo-occlusion). It makes no sense to me to say that there is no relationship between posterior support and degenerative changes in the joint.