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Functional assessment of the stomatognathic system. Part 1: The role of static elements of analysis

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Objectives: To review the elements of static analysis in the functional assessment of the stomatognathic system, as promoted for more than a century by gnathologists, and summarize the available scientific evidence, including anthropologic observations. **Method and materials:** A thorough search was conducted using PubMed, the Cochrane Library database, and Google Scholar. From peer-reviewed articles and other scientific literature, up-to-date information addressing three topics was identified: *(a)* the anthropologic perspective with particular consideration for the role of progressive dental wear over time, *(b)* descriptions of gnathologic principles and evidence on their scientific validity, and *(c)* the methodologic inaccuracies introduced by seeking to correlate variables directly rather than allowing for causal inference. **Results:** For decades gnathology attempted to describe a structure-function correlation within the stomatognathic system by means of a model whose principles were static and mechanistic references. No scientific validation was ever achieved, placing clinical and research consensus out of reach. **Conclusions:** A historical perspective helps to place the fundamentals of gnathology into context: They were conceived to solve technical difficulties but were then assumed to be physiologic stereotypes. This misconception led to a decades-long promotion of mechanistic theories to describe oral function, but the evidence available today supports a more flexible and adaptable approach. Gnathologic arguments have been relegated to become exclusively of technical relevance in oral rehabilitation. *(Quintessence Int 2021;##:##–##; doi: ##########)*

Key words: dental occlusion, gnathology, oral function, real-world data, temporomandibular disorder, tooth wear

Oral health is a key indicator of overall health, wellbeing, and quality of life. The World Health Organization (WHO) defines oral health as "a state of being free from chronic mouth and facial pain, oral and throat cancer, oral infection and sores, periodontal disease, tooth decay, tooth loss, and other diseases and disorders that limit an individual's capacity in biting, chewing, smiling, speaking, and psychosocial wellbeing."[1]

Oral health care targets various psychologic and physical factors influencing and being influenced by oral activities and functions. For example, mastication is a fundamental, rhythmic, and complex function, controlled by a central pattern generator located in the brainstem, requiring high levels of muscular coordination and the capacity to adapt.[2] Large individual variations exist, eg, performance, number of cycles prior to swallowing, amplitude and velocity of jaw movements, jaw muscle activity, and the chewing rate.[3] Considering the frequency of repetition, hundreds of times a day, the way individuals chew directly influences multiple morphologic aspects of growth, maturation, and aging of the stomatognathic system (SS).[4] However, the literature on the relationship between morphologic aspects and physiologic functions of the SS remains inconclusive.[5]

For over a century, gnathology attempted to identify and define this relationship with a mechanistic model. However, extensive criticism over the years highlighted multiple facets to this model resulting in a proliferation of controversy. In this critical review the fundamentals of gnathology are reviewed, pay-

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ing particular attention to static parameters of reference in assessing SS function. It summarizes the available scientific evidence, beginning with an anthropologic perspective, and provides a historical backdrop to the many controversies. It also takes up the technical side of the concepts, which remain important in full-mouth rehabilitation. The overall purpose is therefore to provide a comprehensive summary on the controversial topic of oral function assessment.

Ab origine considerations

Diet seems to have played a predominant role in the evolution of modern human occlusion.[6] The first hominins, so-called "australopiths" and species of the genus *Homo* were adapted to omnivorous diets, showing mandibles with short and deep corpora, severely worn incisors and canines, and low-cusped molars that allowed tough food to be chewed once reduced in size by anterior dentition.[7]

It is today well recognized that significant changes occurred when *Homo erectus* started to use and control fire, particularly for cooking, which became widespread some 500,000 years ago.[8] Cooked food was much easier to chew than raw food, reducing masticatory strain on the SS, leading to a reduction in tooth size, eg, canines.[9] In fact, natural selection favors the combination of characteristics most efficient in a particular environment, and any mutation leading to fewer complex structures ultimately leads the organism to conserve energy.[10]

Similar evolutionary changes usually take place over thousands of years.[11] Nevertheless, environmental pressures on the human genome increased further, and more dramatically, in the wake of two events: the advent of farming approximately 12,000 years ago, marking the beginning of the Neolithic period, and the start of the Industrial Age, about 200 years ago, characterized by the widespread availability of soft and sugar-rich foods. Today these rapid environmental variations are considered to have created a closely interrelated group of oral health problems, eg, dental caries, periodontal disease, and malocclusion.[12] The prevalence of dental caries has been assessed in less than 2% of fossilized Paleolithic human teeth.[13] The first sharp increase dates to the beginning of the Neolithic period, rising to 9% to 10% or more.[14] In the 19th and 20th centuries, a second and more dramatic increase occurred, where 50% to 90% of individuals exhibited caries.[15] Although other factors, eg, genetic propensity and developmental defects, may have played a role, it is elaborate carbohydrates, particularly sugar during the Industrial Revolution, that have been identified as the principal cause of increased caries.[16]

Paleontologists struggle to identify periodontal disease, especially in its earliest stages of gingivitis, and where alveolar bone loss may have been caused post mortem. Consequently, little is known of the history and development of this disease. Notwithstanding, as is the case with decay, food likely plays a significant role in periodontal disease,[17] and prevalence remains high in industrialized populations at about 75% to 96%.[15]

Dental crowding and malocclusion are also considered "diseases of civilization," affecting more than 60% of the population.[18] They have not, however, been identified in fossilized hominins.[19] For instance, third-molar impaction occurs ten times more frequently in Industrial Age samples than in hunter-gatherers, and reports exist of a decrease in size of the supportive bone structures and not of teeth.[20] Despite the lack of consensus explaining the high prevalence in Western society, food consistency has been identified as playing a major role.[21]

In support of this, further evidence exists in anthropologic observations dating to the early 20th century by many scientists exploring remote areas of the planet. Probably the most detailed was by Price,[22] who analyzed the oral health of multiple "primitive" populations (eg, Australian aborigines and indigenous circumpolar peoples) yet to be influenced by the modern soft diet. Oral condition in these populations was surprisingly healthy despite the nonexistent notion of oral hygiene, and people mostly presented worn dentition even at a young age. According to Price,[22] "diseases of civilization" appeared quickly when individuals turned to Western diets.

Tooth wear and dental occlusion

Tooth wear observed in any individual is the result of a combination of multiple etiologic factors, ie, physiologic and pathologic, which determine erosion, abrasion/attrition, and abfraction on dental surfaces.[23] In archaic populations, environmental factors (eg, the presence of raw and abrasive diets and the consistency of food) and individual oral habits (ie, teeth also serving as tools) affected the extensiveness of occlusal abrasion in flattening the occlusal surfaces of teeth.[24] In contrast to these observations, it has now been concluded that this phenomenon radically decreased and changed in modern society, which, to the contrary, mostly displays tribo-chemical wear aspects.[25]

On the basis of these observations, Begg[26] sought to explain the role of masticatory activity in the development of malocclusion. He assumed that interproximal dental wear led to a reduction of the dental arch perimeter, adjusting it to that of the jaw bones and solving the problem of tooth-arch size discrepancies. He assumed that "correct occlusion is not a fixed or particular anatomic state, but a changing functional process undergoing continual modification and adjustment during the whole life of both deciduous [primary] and permanent dentitions." His theory was later developed by Kaifu et al,[27] who defined it as "attritional occlusion."

In both ancient and modern populations, different compensatory mechanisms have been documented as determining functional modifications of human dentition in response to progressive wear, and ensuring the maintenance of occlusal functionality throughout the life of the individual: ie, mesial drift, continuous eruption, and incisor lingual tipping.[25] Consequently, there is a transition from scissor to edge-to-edge anterior occlusion during growth, following changes in two components: overbite (vertical overlap) and overjet (horizontal overlap). Vertical overlap decreases as a result of occlusal wear on anterior teeth, and horizontal overlap decreases primarily as a result of the difference in the amount of incisor lingual tipping between the jaws.[28] Moreover, modifications to occlusal planes are also detectable. When unworn dentition is viewed from the front, a helicoidal plane may be observed, resulting from the combination of two distinct components defined as compensation curves: The cusp tips of the posterior teeth usually conform to a smooth curve in the antero-posterior direction that is referred to as the "curve of Spee"; a transverse occlusal curve also exists normally for each pair of right- and left-side teeth that is concave above and convex below, referred to as the "curve of Wilson." Compensation curves tend to diminish with increasing wear, resulting initially in a leveling of occlusal planes. The curve of Wilson is generally more susceptible, and, in heavy-worn dentitions, it may assume a reversed orientation, in which the occlusal planes of the first molar region incline downward to the buccal region (Fig 1). Such modifications allow for maximal occlusal contact area in a scheme of "fully balanced" occlusion.[27]

In summary, anthropologic observations pinpoint tooth wear as a physiologic process influenced by environmental factors and capable of inducing a functionalization of occlusion during the maturation and aging of an individual. Only late stages of tooth wear may be classified as pathologic. Consequently, depending on the age of an individual, multiple occlusal configurations may be considered physiologic.

Dental occlusion and gnathology

The term "gnathology" was first proposed in 1924 by Stallard,[29] who described the science of analyzing and treating the SS as a whole, drawing on anatomy, histology, physiology, and pathology. Its fundamentals later included the concepts of centric relation (CR), vertical dimension of occlusion (VDO), anterior and lateral guidance, the scheme of posterior occlusal contacts (OCs), and the relationship of the determinants of mandibular movements.

Centric relation

CR was first defined by Hanau in 1929.[29] Around that time, dental activity was mostly oriented to edentulous patients, and a reliable method was needed to define a reproducible maxillo-mandibular relationship for producing prostheses meeting both functional and esthetic criteria. Very soon this clinical reference position for prosthodontic rehabilitation started, albeit gradually, to be considered as a physiologic ideal: "centric relation occlusion" (CRO; later mostly referred to as centric occlusion, or CO), corresponding to a coexistence of a CR and a maximum intercuspation position (MIP).[30] Consensus converged around an individuation of most posterior CR positions, assuming the ideal jaw position in CO was restricted to purely rotary movement about the transversal hinge axis.[31] Hence, where the MIP was irreproducible or where a discrepancy appeared between the CR and MIP, corrective dental treatment was required. Accordingly, occlusal treatment, ranging from prosthodontics[32] to orthodontic[33] and orthognathic surgery[34] was consistently promoted, even in asymptomatic patients.

Contradictory results soon emerged, however. In the 1970s, doubts arose as to the validity of this treatment, and alternative definitions were formulated to identify the most appropriate explanation.[35] The consensus on the placement of the condyle in relation to the glenoid fossa, defining the CR, has shifted (from the most retracted position to the most superior, and then, to the most antero-superior); meanwhile controversy and confusing ideas proliferated.[36] In the most recent version of the glossary of prosthodontic terms,[37] CR is defined as "a maxillo-mandibular relationship, independent of tooth contact, in which the condyles articulate in the anterior-superior position against the posterior slopes of the articular eminences." However, recent surveys have been conducted by some researchers from the field of prosthodontics seeking agreement on the definition of CR.[38] Moreover, the emphasis must be placed on an apparent lack of agreement among and within different groups of specialists in oral care regarding this topic.[39]

One change of paradigm in defining CR was proposed by Jankelson et al.[40] Instead of referring to bones, condyles, and glenoid fossae, they referred to the relationship of muscle balance. This novel approach, termed "muscular centric relation" (MCR), is a position induced on the isotonic closure path of the

Figs 1a to 1j Progression of tooth wear observed in five jaw samples collected from a homogenous population of central Italy (Section of Anthropology and Ethnology, Museum of Natural History, Florence, Italy) dating from the 18th and 19th centuries. The samples belong to people of different ages and consequently show different levels of tooth wear. In the same jaw, different teeth also present different degrees of abrasion, because adult teeth do not erupt at the same time: teeth erupting earlier show more abrasion. All samples are shown from two angles: frontal and occlusal. The white dotted lines highlight the orientation of the compensation curve of Wilson. Missing teeth were all lost post mortem. *(a and b)* A sample with nearly perfect mandibular dentition. Third molars are not completely erupted, reflecting the young age of the individual (17 y). Occlusal compensatory curves are well defined and the Wilson curve shows an accentuated upward orientation. *(c and d)* A sample of an adult (25 y) with lightly worn dentition. Wear is concentrated on teeth involved in anterior and lateral guidance (canines and incisors) and on first molar cusps. Occlusal compensatory curves are detectable and the Wilson curve shows a slight upward orientation. *(e and f)* A fully developed adult (33 y) with signs of dental wear and exposed dentin on almost all teeth but with different ranges. Occlusal compensatory curves are not detectable and occlusal planes are flat. *(g and h)* A fully developed adult (49 y) with an advanced state of dental wear, with large surfaces of exposed dentin on all teeth. Even in this case, wear on the first molars is more advanced than on other molars, and the anterior teeth no longer present a chisel shape, where continuous wear has transformed their occlusal surface into a flat one. The Wilson curve presents an inversed downward orientation. *(i and j)* An older individual (60 y) with massive dental wear. On the first and second molars, abrasion has exposed the pulp chamber and signs of apical lesion due to endodontic complications. The Wilson curve shows an accentuated downward orientation, resulting in occlusion being largely destroyed.

mandible from its rest position after muscle relaxation obtained through transcutaneous electric neural stimulation (TENS).[41] Nonetheless, these methods have received very little attention in the international literature; there is no scientific evidence, and very few—albeit encouraging—clinical applications have been reported.[42]

Fig 2 Illustration of the localization of the three most cited CR positions in the history of dentistry: the most retracted position (red point), the most superior (orange point), and the most antero-superior (yellow point). The green zone represents the possible new conception of CR, no longer interpreted as a point but as an area within which the individual is able to find natural adaptation.

Various studies have also highlighted the many technical flaws in the concept of CR, bringing its clinical utility into question.[43] Firstly, magnetic resonance imaging (MRI) revealed a lack of clinical trustworthiness, as condyles are not predictably locatable in the positions assumed by certain clinicians.[44] Secondly, there is a significant amount of error associated with the use of instruments to exploit CR registrations with an accuracy estimated at 0.3 mm,[45] and with transfer of records (eg, articulators,[46] facebows,[47] and condylar position indicators[48]). Meanwhile, it has been demonstrated that neither rational basis nor scientific evidence support the use of the hinge axis model to describe physiologic jaw movement.[49] Indeed, the axes of rotation of the mandible during motion are not determined by SS bone structures, but by muscle contraction and the maintenance of an optimum condyle-disk-fossae relationship. This condition has been obtained using muscular deprogramming procedures of varying types and effectiveness, but consensus is still lacking as to the most appropriate technique.[50]

In conclusion, attempts to couch CR as a mechanical concept in physiologic terms has never found scientific validation despite considerable efforts. In the history of dentistry, there have been more than 26 definitions of CR,[30] and many have been applied clinically. This has overshadowed the relevance of the concept but, at the same time, highlights the high capacity of human beings to adapt to occlusal changes. However, the identification of a maxillomandibular relation remains an unavoidable step in all occlusal rehabilitation, and its technical feasibility will always depend on the selection of a specific position. From this perspective, there is potential for a radical conceptual change to CR, shifting from a specific "point" to an "area," within which the SS is able adapt naturally: the higher the individual's capacity to adapt, the larger the area (Fig 2).

Vertical dimension of occlusion (VDO)

In dentate individuals, the VDO is the "distance between two selected anatomic or marked points (usually one on the tip of the nose and the other on the chin, located on the midline) when in the maximal intercuspal position"[37] and is largely determined by the occluding dentition. In 1934 the American otologist Costen assumed that, in the absence of molar support, the strong elevating mandibular muscles could push the condyle upward and backward, compressing the delicate anatomical area situated between the condyle and the acoustic meatus, rich in vessels and nerves, and provoking temporomandibular disorder (TMD).[51] His hypothesis was widely used to promote the treatment of dentitions at an "increased" VDO to correct and even prevent this pathology. However, descriptions of the undesirable consequences of this practice ensued. Consistent VDO augmentation was suspected to obliterate the interocclusal distance (IOD), or "freeway space," which was presumed to increase jaw-elevator activity.[52] These observations gave rise to two closely related convictions: *(1)* At rest, the individual determines the IOD, which is fixed for life, and *(2)* if the VDO is increased beyond its physiologic limit, it may provoke muscular hyperactivity in attempting to reestablish the original IOD and provoke TMD.[53]

Animal experimentation on monkeys was conducted in search of answers. After modifying the VDO with fixed prosthodontic interventions, animals were initially irritated until they accepted to the new condition.[54] These studies showed that incremental VDO interventions influence different histologic and morphologic constituents of the SS, provoking not a collapse of the system but a general compensation and adaptation. Undesirable reactions occurred only in extreme VDO increases, a condition far from daily clinical reality.[55]

Studies in human subjects are scant due to ethical constraints, and most suffer from a lack of randomization and controls, limited sample sizes, and difficulties in achieving objectivity of signs and symptoms.[56] That notwithstanding, the general outcomes reflect the results reported in animals.[57] In fact, contrary to traditional suggestions of increased VDO leading to muscle strain, electromyographic (EMG) studies have demon-

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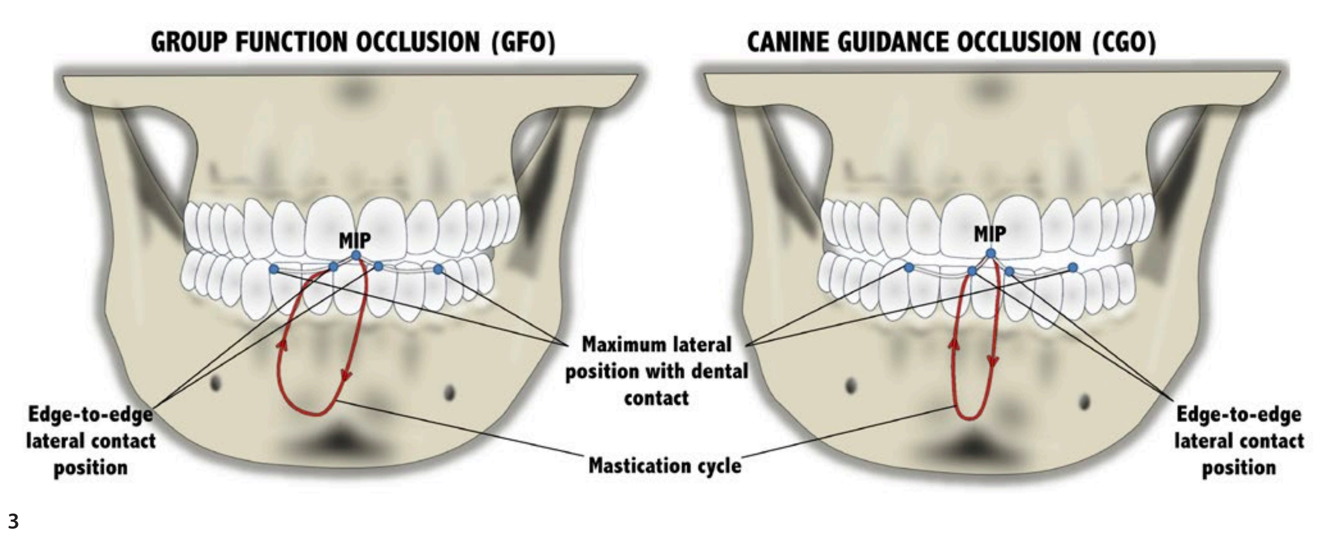

Fig 3 Pattern of movements achieved with different occlusal lateral guidance. CGO is associated to vertical masticatory cycles (red line) and lateral excursions (white lines) to lower EMG activity, whereas GFO shows more horizontal patterns and higher EMG activity. MIP, maximum intercuspation position.

strated the opposite. Likewise, IOD presents a minimum gamut of between 10 and 16 mm of the mouth opening, showing that clinical rest position does not correspond to muscle rest position.[58] This is caused by the constant activity of the masticatory muscles to balance the jaw against the forces of gravity.[59] Consequently, the concept of a rest "position" may evolve into a rest "range" with characteristics intrinsically related to posture.[60]

To date, no compelling evidence contraindicates techniques involving VDO alteration, and several authors have declared multiple clinical advantages.[61] However, no study indicates either precisely "how" or "by how much" this parameter can be increased, although the limit of 5 mm at the interincisal level is nowadays considered trustworthy,[56] despite an individual's degree of variability being preferable.

Anterior and lateral guidance

Christensen[62] first described the phenomenon of disclusion, referring to the distancing of antagonistic posterior teeth during eccentric movements of the jaw. Protrusive movement is controlled by the sliding of opposing incisal surfaces, where the steepness of the guidance is a function of horizontal and vertical overlap, ie, overjet and overbite (reported to be ideally between 2 and 3 mm). Different schemes have been described: complete anterior, incisal, and mesio-incisal guidance.[63] For lateral movement, possible guidance schemes are canine-guided occlusion (CGO), group function occlusion (GFO), and bilateral balanced occlusion (BBO). To date, there is no scientific evidence supporting the superiority of any one scheme.[64]

BBO associated with a posterior balanced occlusion during protrusive movements has been defined as "fully balanced" occlusion.[65] Although this concept was initially more diffuse because of the higher stability it lent to complete dentures, the gnathologic map soon encompassed a "mutually protected" theory, where anterior teeth acted as protectors of posterior teeth from tangential forces and posterior teeth were recognized as shock absorbers of vertical forces.[66] As was the case with CR, a rehabilitative concept was soon adopted as a physiologically ideal condition, stating that unguided occlusions could lead to not only disruptive effects for dentition but also TMD.[67] Muscular activity was demonstrated to be open to direct influence by varying disclosure guidance, which EMG analysis seemed to support. For example, there were claims of complete anterior guidance successfully reducing TMD symptoms,[68] because with CGO a significantly lower muscular activity was detected compared to other schemes.[69] These observations led to indicate CGO as the schema for preventing not only TMD but also parafunctional activity, eg, bruxism.[70] However, no scientific confirmation has been found. On the contrary, it is today widely accepted that parafunctional activity like bruxism is not connected to any characteristics of dental occlusion.[71] Indeed, reports have described significant variability in disclosure guidance for healthy and non-restored dentition, contrasting with past statements and assuming that physiologic function and patient acceptance appear to be minimally influenced by lateral and anterior occlusion.[72] Moreover, long-term patient acceptance appears minimally influenced by different kinds of lateral guidance, suggesting the role of this parameter may have been overestimated in early literature.[73]

Finally, anterior guidance does exert an influence on the shapes of masticatory cycles. Steeper anterior guidance determines the narrowest movement pattern,[74] as well as CGO compared to GFO (Fig 3).[75] This can be attributed to increased vertical overlap between the anterior teeth. Clinically speaking, it involves disclosure movements with oblique stress of lower magnitude for posterior teeth.[76]

In conclusion, a more horizontal disclosure scheme enables increased jaw stability during lateral and protrusive excursion, and brings efficacy to a system that, producing stronger forces, would otherwise be more destructive to dental integrity. On the contrary, vertical force vectors reduce the masticatory efficacy of the SS, but protect dental tissue integrity by reducing the muscular forces exerted during functional activity. However, this consideration does not apply to parafunctional activity, which is unrelated to occlusion schemes.

Posterior occlusal contact schemes

Defining "normal occlusion" was first proposed by Angle[77] in 1899, interpreted as the ideal to be attained in the treatment of malocclusion. Prosthetic necessities, however, called for the identification of different occlusal models and theories, to obtain a maximal stability for complete dentures, eg, "Bonwill triangle," "Hanau's Quint," "Thielemann's formula" and "lingualized occlusion." The ideal design of posterior OCs presented surfaces that were reliable in terms of eccentric movements of the mandible, with an inclination of cusps and antagonistic fossae derived through the recording of condylar pathways. These shapes were considered ideals, contributing to jaw and denture stability in dynamic movements and creating a "fully balanced" occlusal scheme.[78] At first, complete dentures were inescapably constructed following arbitrary decisions, and all occlusal schemes, albeit non-essential as with the physiologic model of SS, were at some time abused, seen as representing nature rather than from the position of prosthetic dentistry needing standardization and predictable outcomes.

The development of fixed prosthodontic techniques was a crucial step forward for dentistry, mostly because achieving prosthetic stability stopped being the treatment goal, resulting in the introduction of anterior disclusion theories. Occlusal designs for ridge and groove direction made little sense, because the posterior teeth were supposed to disclose during eccentric movements. Emphasis was thus placed on the importance of determining the location, distribution, and number of occlusal contact points in the model of "tripodism."[79] Under this rationale, the design of occlusal surfaces of posterior teeth could control the distribution of axial forces imposed by function and maximize the stabilization of the jaw and occlusion, providing the highest masticatory efficiency.[80] However, the pursuit of this ideal proved a challenging clinical exercise, potentially ending in involuntary creation of posterior interferences.[81]

With the classical gnathologic concept outdated, a simplified pattern of OCs spread in order to streamline clinical procedures and satisfy the following criteria: homogenous contact distribution, adequate esthetics, and occlusal stability.[82] The adaptability of all SS components suggests individuals may be able to accept more than one form or arrangement of OC, on the condition they meet the range of patient needs and deliver predictable results.

Historical summary and discussion

Anthropologic research and observations of isolated human populations from the beginning of the 20th century provide us with a somewhat homogenous response: Human dentition, both ancient and modern, is primarily "designed" for heavy-wear environments. The reduction in tooth wear is preventing contemporary humans from accomplishing the "attritional occlusion" described by Begg.[26] In fact, if we admit attritional occlusion as the product of evolutionary adaptation, its absence is none other than the consequence of changes in the environmental context. In displaying this, the SS exhibits a high potential of adaptability.[83] These notions are important in understanding how the SS matures, but nothing insists the attritional occlusion model be applied. First, flat occlusal surfaces and the edge-to-edge features proposed are incompatible with contemporary esthetic standards. Second, the modern diet is soft and will most probably remain so. Absent changes to this crucial factor, Begg's occlusion model not only cannot be naturally realized but it is conceivable that it cannot naturally maintain itself stable in time. The high efficiency promoted by this model is not the main priority with the foodstuffs available these days, especially considering that restorative materials available to repair teeth are still not as resilient as natural structures.[84]

To avoid further misinterpretation of gnathologic concepts, a comprehensive understanding of their historical background

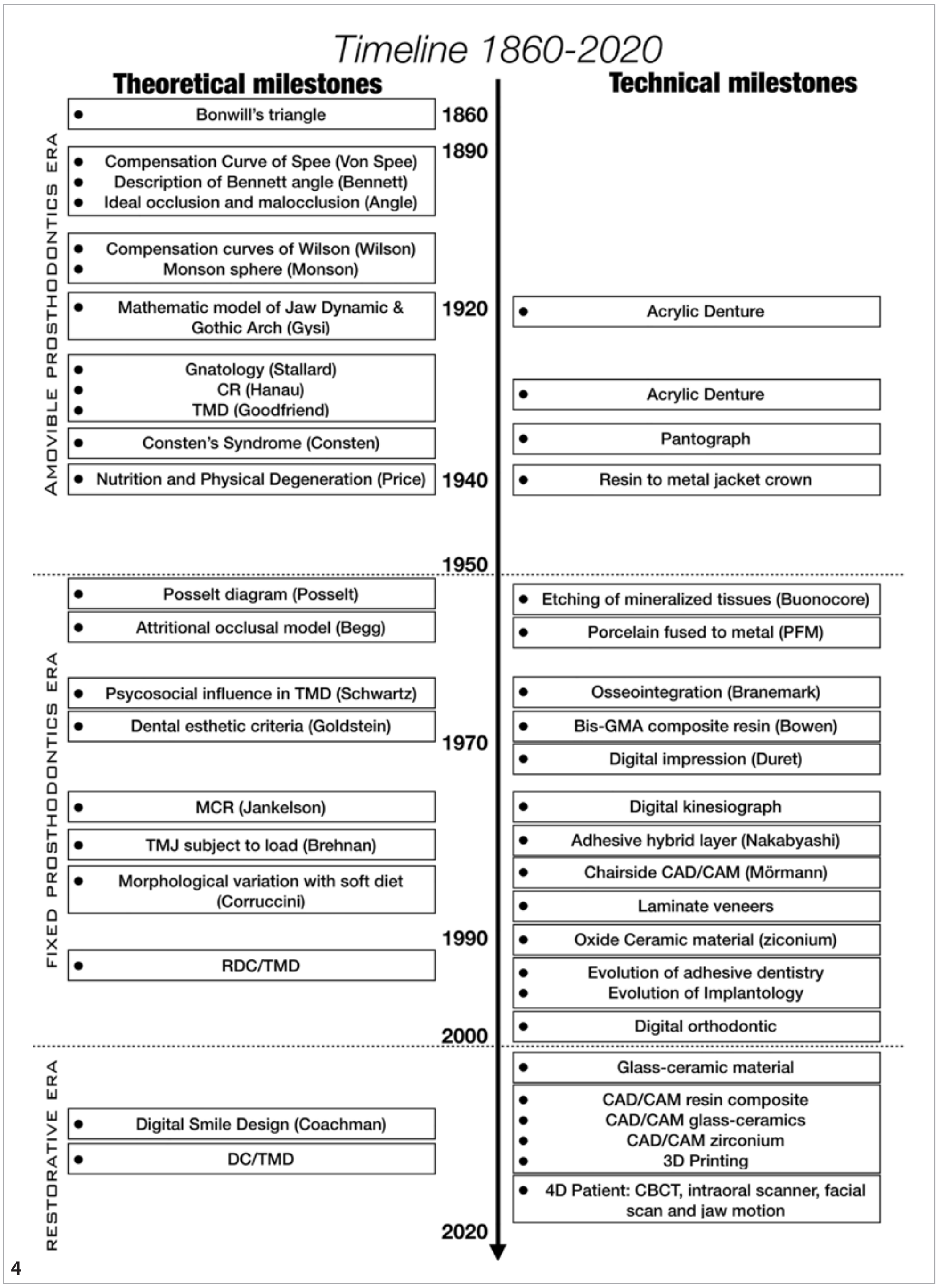

Fig 4 Timeline of milestones in dentistry from 1860 to 2020 with, on the left, the most relevant theoretical concepts, and, on the right, significant technical developments.

is necessary (Fig 4). Anthropologic perspective barely influenced early gnathologists developing explicatory SS models, simply because occlusal notions were initially oriented to rehabilitate edentulous patients, as restorative treatment was inconsistent at the beginning of the 20th century. The need arising in prosthodontics was mainly the achievement of maximal occlusal stability to enhance the immobility of removable complete dentures. At that time, scientific evidence-based concepts were still not part of dentistry, and practitioners had to rely upon the lowest level of scientific proof to elect methods for oral rehabilitation. Many among the prosthetic dentistry elite introduced their own theories of occlusion and designed specific articulators to reflect those theories. These events fed mechanistic viewpoints, and early considerations on TMD also reinforced the mechanistic interpretation of the SS as being ideal and physiologic.

The subsequent enhancement of fixed prosthodontic techniques triggered a paradigm shift for treatment plans. First, prosthetic stability was no longer a major problem. That notwithstanding, the construction of an ideal occlusal scheme did become much more challenging, especially in extended rehabilitation, due to the influence of the original position of the prepared teeth. Second, the biomaterial initially employed for fixed prosthodontics responded to mechanical requirements but were not mimetic with natural tissues. An increased demand for higher esthetic outcomes drove the further development of manufacturing techniques, fusing tough and resistant metal to more esthetic ceramics and acrylics. Naturally, the poor mechanical properties of porcelain led to the inevitable mechanical failure of prostheses. In these circumstances, a full mouth in fixed rehabilitation became almost mission impossible. Prosthodontic rehabilitation needed to be technically easier, and managing the relationship of only one contact point during eccentric movement was obviously much easier than managing multiple points. Accordingly, in the 1950s, occlusal philosophies evolved from "fully balanced" to "mutually protected." Many treatment philosophies, not only prosthetic and orthodontic, but also those attempting to treat TMD, introduced the element of a posterior disclusion dictated by anterior and lateral guidance. Observations on the maturation of natural dentition seemed to lend scientific support to this conclusion.[85]

The ideal model was finally completed and the goal of gnathology became establishing occlusion free from interference, providing for posterior disclusion, cusp to fossae relationship, uniform centric contact and tripodism, narrow occlusal table, maximum cusp height and fossae depth with supplemental anatomy, forces along the long axes of the teeth, and centric (relation) occlusion. Natural dentitions not satisfying these criteria were considered pathologic.[86]

This mechanistic description reached the height of its recognition in the early 1970s, but, soon after, the fallacies emerged. Rather than providing simple-to-follow therapeutic protocols, gnathologic principles were too difficult to implement in daily practice, and, pursuing the otherwise charming idea, therapeutic procedures of an irreversible nature were chosen, often with unfavorable biologic and financial cost-to-benefit ratios. Some have since been modified or reformulated multiple times but have only added to the confusion and ambiguity. Meanwhile, the scientific community was unable to justify their use sufficiently, and clinical evidence debunked most of these principles relegating some of them "old-fashioned dogmas" of dentistry, clearly downsized in relevance and only employable as an idealistic concept in teaching.[87]

Following a period of stagnancy, events of recent decades have revitalized the debate on these principles in the dental community. First, dental practitioners have seen an upward trend in the spread of non-carious lesions (NCLs), produced by abrasion, attrition, erosion, and abfraction, which, especially in combination, provoke a significant loss of sound tooth substance.[88] This trend came hand-in-hand with a growing demand for esthetic treatment,[89] but also a tremendous development of minimal or noninvasive principles of treatment and remarkable progress in the development of dental materials and adhesive systems.[90] Indeed, minimally invasive approaches have found a valuable clinical advantage in modifying VDO, establishing new maxillomandibular relationships and designing new occlusal schemes. As in the past, practitioners promote a variety of different clinical procedures to modify VDO, most still remaining empirically guided given their basis in clinical experience rather than evidence-based science. Last but not least, noteworthy enhancements have been accomplished by the dental community in framing the question of TMD. Psychosocial context, stress, and anxiety were quickly identified as major causes of increased tension in masticatory muscles and causative of TMD, unseating dental occlusion.[91] What was a purely mechanistic and morphologic problem gradually evolved into an orthopedic musculoskeletal condition, influenced by a psychologic component that may impact etiology and therapeutic management. However, the modern understanding of TMD etiology remains incomplete, and available data may prove to be controversial. The available evidence indicates a low influence of occlusion on the development of TMD.[92] Nevertheless, recent research analyzing the neuro-pathophysiology of TMD pain with functional magnetic resonance image (fMRI) showed structural and functional alterations in multiple neural structures, eg, peripheral trigeminal nerve roots, brainstem, and thalamus, providing support for a peripheral origin of TMD.[93] Moreover, it indicates splint therapy as able to alleviate TMD-related symptoms by inducing functional brain changes. After all, occlusal appliances are still considered a valuable solution in any attempt to tackle TMD.[94]

One possible motivation for the continuing controversy surrounding occlusion and TMD has been proposed to be definition-based.[95] Currently falling within the term TMD is a heterogenous group of musculoskeletal disorders with varying etiologies and physiopathologies,[96] whereas considering them as a unique group of pathologies leads to interpretation errors. Work to find international consensus on the definition of a group of problems, and consequently to define the treatment approach, is unquestionably a step in the right direction.[97] However, models describing the progression and diagnosis of degenerative soft and hard tissue changes in the temporomandibular joint have yet to be clearly elucidated.[98]

Another possible drawback is the analytical methods applied. First, functional studies on the mastication process and oral dysfunction are necessarily observational in nature. It would be unethical and/or impractical to use experimental designs with irreversible outcomes in this field. Some basic information may be obtained from experiments on animals, but studying the mode of functioning of the human SS requires a different approach. In fact, in view of the complexity, a pluralistic approach to causality is needed, identifying latent relationships between variables and collecting different types of evidence that lend to conclusions of a causal nature, particularly concerning complex exposures.[99] Generally speaking, most data are, therefore, best described as real-world data, ie, derived from a number of sources associated with outcomes in a heterogenous patient population in real-world settings. Their main goal is to generate real-world evidence, where observational studies compare causes and effects among variables in order to confirm or refute the proposed causal relationships. Looking at TMD, for example, multiple categories are commonly associated to limitation or interference in jaw movement. Altering jaw movement is also a parameter that can be influenced with different occlusal schemes. Consequently, the modification and modifiability of jaw mobility is a common variable that may indirectly link anatomy to functional or dysfunctional clinical situations. Therefore, it is possible to abandon approaches that study those causes of a complex problem like TMD with only a partial or even direct correlational link (without causal direction) between variables, with a view to greater reliance on statistical methods of approaching the so-called causal inference. In conclusion, dental occlusion can no longer be considered directly responsible for TMD, but as often happens in human history, there is the tendency to shift from one extreme to another: It is self-contradictory to support a total absence of connection between the morphologic aspect of anatomy (eg, dental occlusion) and the physiologic mode of functioning (and consequently pathologic dysfunction), as it denies a principle underpinning human physiology. It would be wiser to explore the errors that have obfuscated explicative cause-effect models linking structure and function. Although dental practitioners benefit from room for maneuver, it must not excuse careless or haphazard approaches. The SS does have a remarkable capacity to adapt to various stimuli, but currently the biologic cost of changes to occlusion is neither known nor measurable. ▦

Conclusions

Most gnathologic concepts were initially conceived to solve technical difficulties in a period when oral care and therapeutic means were completely different to today. A sense of confusion resulted from the assumption—necessary to define and correlate morphology and function—that these principles were physiologic stereotypes. It has taken almost a century of debate to overcome this point, and today, despite the misgivings still circulating in the dental community, the available evidence supports a flexible and adaptable model of occlusion rather than a preconceived and rigid theory. For instance, dental occlusion can no longer be considered directly responsible for TMD, and most conventional gnathologic arguments have been relegated to become of exclusively technical relevance in oral rehabilitation, for example:

- Use of the CR, MCR, or MIP may all be envisaged, but come with multiple technical considerations that must be analyzed and anticipated in oral rehabilitation.
- Modifying VDO appears to be a safe clinical procedure, within certain limits.
- Different schemes of anterior and lateral disclosure are equally acceptable when restoring anterior dentition, but muscular activity and jaw movement patterns must be considered as they may be directly affected.
- Different forms and arrangements of OCs may be equally accepted on the condition that the selected schemes meet sufficient homogenous contact distribution, and adequate esthetic and occlusal stability.

These considerations should be interpreted with caution, and clinical decisions must be adequately justified in the light of the clinical necessity of each case.

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References

1. World Health Organization (WHO). World Oral Health Report 2003. https://apps.who.int/iris/handle/10665/68506. Accessed 4 April 2021.

2. Lund JP. Mastication and its control by the brain stem. Crit Rev Oral Biol Med 1991;2:33–64.

3. Van der Bilt A. Assessment of mastication with implications for oral rehabilitation: a review. J Oral Rehabil 2011;38:754–780.

4. Farooq M, Sazonov E. Automatic measurement of chew count and chewing rate during food intake. Electronics (Basel) 2016;5:62.

5. Vozzi F, Favero L, Peretta R, Guarda-Nardini L, Cocilovo F, Manfredini D. Indexes of jaw muscle function in asymptomatic individuals with different occlusal features. Clin Exp Dent Res 2018;4:263–267.

6. Gibbons A. Evolutionary biology. An evolutionary theory of dentistry. Science 2012;336(6084):973–975.

7. Raia P, Boggioni M, Carotenuto F, et al. Unexpectedly rapid evolution of mandibular shape in hominins. Sci Rep 2018;8:7340.

8. Shimelmitz R, Kuhn SL, Jelinek AJ, Ronen A, Clark AE, Weinstein-Evron M. 'Fire at will': the emergence of habitual fire use 350,000 years ago. J Hum Evol 2014;77:196–203.

9. Zink KD, Lieberman DE. Impact of meat and Lower Palaeolithic food processing techniques on chewing in humans. Nature 2016;531(7595):500–503.

10. Bock W, Von Wahlert G. Adaptation and the form-function complex. Evolution 1965;9:269–299.

11. Itan Y, Powell A, Beaumont MA, Burger J, Thomas MG. The origins of lactase persistence in Europe. PLoS Comput Biol 2009;5(8):e1000491.

12. Ungar PS, Sorrentino J, Rose JC. Evolution of human teeth and jaws: implications for dentistry and orthodontics. Evol Anthropol 2012;21:94–95.

13. Grine FE, Gwinnett AJ, Oaks JH. Early hominid dental pathology: interproximal caries in 1.5 million-year-old *Paranthropus robustus* from Swartkrans. Arch Oral Biol 1990;35:381–386.

14. Larsen C. Biological changes in human populations with agriculture. Ann Rev Anthropol 1995;24:185–213.

15. Frencken JE, Sharma P, Stenhouse L, Green D, Laverty D, Dietrich T. Global epidemiology of dental caries and severe periodontitis – a comprehensive review. J Clin Periodontol. 2017;44(Suppl 18):S94–S105.

16. Meyer BD, Lee JY. The confluence of sugar, dental caries, and health policy. J Dent Res 2015;94:1338–1340.

17. Colombo APV, Tanner ACR. The role of bacterial biofilms in dental caries and periodontal and peri-implant diseases: a historical perspective. J Dent Res 2019;98:373–385.

18. Kragt L, Dhamo B, Wolvius EB, Ongkosuwito EM. The impact of malocclusions on oral health-related quality of life in children: a systematic review and meta-analysis. Clin Oral Investig 2016;20:1881–1894.

19. Varrela J. Masticatory function and malocclusion: a clinical perspective. Semin Orthod 2006;12:102–109.

20. Brown T, Townsend GC, Richards LC, Burgess VB. Concepts of occlusion: Australian evidence. Am J Phys Anthropol 1990;82: 247–256.

21. Corruccini RS, Beecher RM. Occlusal variation related to soft diet in a nonhuman primate. Science 1982;218(4567):74–76.

22. Price W. Nutrition and Physical Degeneration: A comparison of primitive and modern diets and their effects. 6th ed. Lemon Grove: Price-Pottenger Nutrition Foundation, 1939.

23. Lussi A. Dental Erosion: From diagnosis to therapy. 1st. ed. Augusta: GM Whitford, 2006.

24. Young WG. Anthropology, tooth wear, and occlusion ab origine. J Dent Res 1998;77:1860–1863.

25. D'Incau E, Couture C, Maureille B. Human tooth wear in the past and the present: tribological mechanisms, scoring systems, dental and skeletal compensations. Arch Oral Biol 2012;57:214–229.

26. Begg P. Stone age man's dentition: With reference to anatomically correct occlusion, the etiology of malocclusion, and a technique for its treatment. Am J Orthod 1954;40:298–312.

27. Kaifu Y, Kasai K, Townsend GC, Richards LC. Tooth wear and the "design" of the human dentition: a perspective from evolutionary medicine. Am J Phys Anthropol 2003;Suppl 37:47–61.

28. Reinhardt GA. Attrition and the edge-to-edge bite. An anthropological study. Angle Orthod 1983;53:157–164.

29. Pokorny PH, Wiens JP, Litvak H. Occlusion for fixed prosthodontics: a historical perspective of the gnathological influence. J Prosthet Dent 2008;99:299–313.

30. Rinchuse DJ, Kandasamy S. Centric relation: A historical and contemporary orthodontic perspective. J Am Dent Assoc 2006;137:494–501.

31. Moulton GH. The importance of centric occlusion in diagnosis and treatment planning. J Prosth Dent 1960;10:921–926.

32. Moulton GH. Centric occlusion and the free-way space. J Prosth Dent 1957;7:209–215.

33. Roth R. Functional occlusal for the orthodontist. J Clin Orthod 1981;15:32–51.

34. Hohl TH. Occlusal concepts for the orthognathic surgeon. Int J Oral Surg 1978;7:197–207.

35. Turp JC, Greene CS, Strub JR. Dental occlusion: a critical reflection on past, present and future concepts. J Oral Rehabil 2008;35:446–453.

36. Goldstein G, Andrawis M, Choi M, Wiens J, Janal MN. A survey to determine agreement regarding the definition of centric relation. J Prosthet Dent 2017;117:426–429.

37. The Glossary of Prosthodontic Terms: 9th edn. J Prosthet Dent 2017;117(5S):e1–e105.

38. Wiens JP, Goldstein GR, Andrawis M, Choi M, Priebe JW. Defining centric relation. J Prosthet Dent 2018;120:114–122.

39. Truitt J, Strauss RA, Best A. Centric relation: a survey study to determine whether a consensus exists between oral and maxillofacial surgeons and orthodontists. J Oral Maxillofac Surg 2009;67:1058–1061.

40. Jankelson B, Sparks S, Crane PF, Radke JC. Neural conduction of the myo-monitor stimulus: a quantitative analysis. J Prosthet Dent 1975;34:245–253.

doi: ##.####/j.qi.a#####

41. Bracco P, Deregibus A, Piscetta R. Effects of different jaw relations on postural stability in human subjects. Neurosci Lett 2004;356:228–230.

42. Cooper BC, Kleinberg I. Establishment of a temporomandibular physiological state with neuromuscular orthosis treatment affects reduction of TMD symptoms in 313 patients. Cranio 2008;26:104–117.

43. Kandasamy S, Greene CS, Obrez A. An evidence-based evaluation of the concept of centric relation in the 21st century. Quintessence Int 2018;49:755–760.

44. Kandasamy S, Boeddinghaus R, Kruger E. Condylar position assessed by magnetic resonance imaging after various bite position registrations. Am J Orthod Dentofacial Orthop 2013;144:512–517.

45. Hellmann D, Etz E, Giannakopoulos NN, Rammelsberg P, Schmitter M, Schindler HJ. Accuracy of transfer of bite recording to simulated prosthetic reconstructions. Clin Oral Investig 2013;17:259–267.

46. Rinchuse DJ, Kandasamy S. Orthodontic dental casts: the case against routine articulator mounting. Am J Orthod Dentofacial Orthop 2012;141:9–16.

47. Maveli TC, Suprono MS, Kattadiyil MT, Goodacre CJ, Bahjri K. In vitro comparison of the maxillary occlusal plane orientation obtained with five facebow systems. J Prosthet Dent 2015;114:566–573.

48. Lindauer SJ, Sabol G, Isaacson RJ, Davidovitch M. Condylar movement and mandibular rotation during jaw opening. Am J Orthod Dentofacial Orthop 1995;107:573–577.

49. Chen J, Katona TR. The limitations of the instantaneous centre of rotation in joint research. J Oral Rehabil 1999;26:274–279.

50. Weinberg LA. The role of muscle deconditioning for occlusal corrective procedures. J Prosthet Dent 1991;66:250–255.

51. Consten J. A syndrome of ear and sinus symptoms dependent upon disturbed function of the temporomandibular joint. Ann Otol 1934;43:1–15.

52. Beyron H. Characteristics of functionally optimal occlusions and principles of occlusal rehabilitation. J Am Dent Assoc 1954;28:648–659.

53. Woda A, Pionchon P, Palla S. Regulation of mandibular postures: mechanisms and clinical implications. Crit Rev Oral Biol Med 2001;12:166–178.

54. Carlson DS, Schneiderman ED. Cephalometric analysis of adaptations after lengthening of the masseter muscle in adult rhesus monkeys, *Macaca mulatta*. Arch Oral Biol 1983;28:627–637.

55. Maxwell LC, Carlson DS, McNamara JA Jr, Faulkner JA. Adaptation of the masseter and temporalis muscles following alteration in length, with or without surgical detachment. Anat Rec 1981;200:127–137.

56. Abduo J. Safety of increasing vertical dimension of occlusion: a systematic review. Quintessence Int 2012;43:369–380.

57. Rivera-Morales WC, Mohl ND. Relationship of occlusal vertical dimension to the health of the masticatory system. J Prosthet Dent 1991;65:547–553.

58. Manns A, Miralles R, Guerrero F. The changes in electrical activity of the postural muscles of the mandible upon varying the vertical dimension. J Prosthet Dent 1981;45:438–445.

59. Michelotti A, Farella M, Vollaro S, Martina R. Mandibular rest position and electrical activity of the masticatory muscles. J Prosthet Dent 1997;78:48–53.

60. Moreno-Hay I, Okeson JP. Does altering the occlusal vertical dimension produce temporomandibular disorders? A literature review. J Oral Rehabil 2015;42:875–882.

61. Calamita M, Coachman C, Sesma N, Kois J. Occlusal vertical dimension: treatment planning decisions and management considerations. Int J Esthet Dent 2019;14:166–181.

62. Christensen C. The problem of the bite. Dent Cosmos 1905;47:1184–1195.

63. Schuyler CH. The function and importance of incisal guidance in oral rehabilitation. 1963. J Prosthet Dent 2001;86:219–232.

64. Abduo J, Tennant M. Impact of lateral occlusion schemes: A systematic review. J Prosthet Dent 2015;114:193–204.

65. Schuyler C. Fundamental principles in the correction of occlusal disharmony, natural and artificial. J Am Dent Assoc 1935;22:1193–1202.

66. Thornton LJ. Anterior guidance: group function/canine guidance. A literature review. J Prosthetic Dent 1990;64:479–482.

67. Ramfjord S. Dysfunctional temporomandibular joint and muscle pain. J Prosthet Dent 1961;11:353–374.

68. Kerstein RB, Wright NR. Electromyographic and computer analyses of patients suffering from chronic myofascial pain-dysfunction syndrome: before and after treatment with immediate complete anterior guidance development. J Prosthet Dent 1991;66:677–686.

69. Belser UC, Hannam AG. The influence of altered working-side occlusal guidance on masticatory muscles and related jaw movement. J Prosthet Dent 1985;53:406–413.

70. Akören AC, Karaağaçlioğlu L. Comparison of the electromyographic activity of individuals with canine guidance and group function occlusion. J Oral Rehabil 1995;22:73–77.

71. Lobbezoo F, Ahlberg J, Raphael KG, et al. International consensus on the assessment of bruxism: Report of a work in progress. J Oral Rehabil 2018;45:837–844.

72. Abduo J, Tennant M, McGeachie J. Lateral occlusion schemes in natural and minimally restored permanent dentition: a systematic review. J Oral Rehabil 2013;40:788–802.

73. Carlsson GE. Critical review of some dogmas in prosthodontics. J Prosthodont Res 2009;53:3–10.

74. Ogawa T, Koyano K, Suetsugu T. The influence of anterior guidance and condylar guidance on mandibular protrusive movement. J Oral Rehabil 1997;24:303–309.

75. Salsench J, Martínez-Gomis J, Torrent J, Bizar J, Samsó J, Peraire M. Relationship between duration of unilateral masticatory cycles and the type of lateral dental guidance: a preliminary study. Int J Prosthodont 2005;18:339–346.

76. Wang M, Mehta N. A possible biomechanical role of occlusal cusp-fossa contact relationships. J Oral Rehabil 2013;40:69–79.

77. Angle E. Classification of malocclusion. Dental Cosmos 1899;4:248–264.

78. Price RB, Kolling JN, Clayton JA. Effects of changes in articulator settings on generated occlusal tracings. Part I: Condylar inclination and progressive side shift settings. J Prosthet Dent 1991;65:237–243.

79. Walther W. Determinants of a healthy aging dentition: maximum number of bilateral centric stops and optimum vertical dimension of occlusion. Int J Prosthodont 2005;18:287–289.

80. dos Santos J Jr, Blackman RB, Nelson SJ. Vectorial analysis of the static equilibrium of forces generated in the mandible in centric occlusion, group function, and balanced occlusion relationships. J Prosthet Dent 1991;65:557–567.

81. Wiskott HW, Belser UC. A rationale for a simplified occlusal design in restorative dentistry: historical review and clinical guidelines. J Prosthet Dent 1995;73:169–183.

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

83. Sessle BJ. Biological adaptation and normative values. Int J Prosthodont 2003;16(Suppl):72–73.

84. Saratti CM, Rocca GT, Krejci I. The potential of three-dimensional printing technologies to unlock the development of new ‘bio-inspired’ dental materials: an overview and research roadmap. J Prosthodont Res 2019;63:131–139.

85. Sillman J. Dimensional changes of the dental arches: Longitudinal study from birth to 25 years. Am J Orthod 1964;50:824–841.

86. Mohl ND, Ohrbach R. The dilemma of scientific knowledge versus clinical management of temporomandibular disorders. J Prosthet Dent 1992;67:113–120.

87. Carlsson GE. Some dogmas related to prosthodontics, temporomandibular disorders and occlusion. Acta Odontol Scand 2010;68:313–322.

88. Lee A, He LH, Lyons K, Swain MV. Tooth wear and wear investigations in dentistry. J Oral Rehabil 2012;39:217–225.

89. Blatz MB, Chiche G, Bahat O, Roblee R, Coachman C, Heymann HO. Evolution of aesthetic dentistry. J Dent Res 2019;98: 1294–1304.

90. Frankenberger R, Van Meerbeek B. Editorial: Adhesive dentistry – no future? We don't think so! J Adhes Dent 2017;19:3.

91. Ohrbach R, Dworkin SF. The evolution of TMD diagnosis: past, present, future. J Dent Res 2016;95:1093–1101.

92. Olliver SJ, Broadbent JM, Thomson WM, Farella M. Occlusal features and TMJ clicking: A 30-year evaluation from a cohort study. J Dent Res 2020;99:1245–1251.

93. Yin Y, He S, Xu J, et al. The neuro-pathophysiology of temporomandibular disorders-related pain: a systematic review of structural and functional MRI studies. J Headache Pain 2020;21:78.

94. Greene CS, Menchel HF. The use of oral appliances in the management of temporomandibular disorders. Oral Maxillofac Surg Clin North Am 2018;30:265–277.

95. de Kanter R, Battistuzzi P, Truin GJ. Temporomandibular disorders: "occlusion" matters! Pain Res Manag 2018;2018:8746858.

96. Slade GD, Ohrbach R, Greenspan JD, et al. Painful temporomandibular disorder: decade of discovery from OPPERA studies. J Dent Res 2016;95:1084–1092.

97. Schiffman E, Ohrbach R, Truelove E, et al. Diagnostic Criteria for Temporomandibular Disorders (DC/TMD) for Clinical and Research Applications: recommendations of the International RDC/TMD Consortium Network and Orofacial Pain Special Interest Group. J Oral Facial Pain Headache 2014;28:6–27.

98. Tanaka E, Detamore MS, Mercuri LG. Degenerative disorders of the temporomandibular joint: etiology, diagnosis, and treatment. J Dent Res 2008;87:296–307.

99. Pearl J. Causality: Models, reasoning, and inference. Cambridge: Cambridge University Press, 2000.

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