Relationship of occlusal vertical dimension to the health of the masticatory system

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Changes in occlusal vertical dimension have been claimed to cause masticatory system disorders. Early articles on this subject were mainly limited to clinical case reports, and the more recent clinical studies have been flawed by the lack of control groups, blind evaluation, and by poor definition of criteria for evaluating the health of the masticatory system. Research with humans and animals has shown that if increases in occlusal vertical dimension are not extreme and the appliance used covers most of the dentition, there is a good possibility of adaptation. Current scientific knowledge does not support the hypothesis that moderate changes in occlusal vertical dimension are detrimental to the masticatory system.
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The evaluation and establishment of the occlusal vertical dimension (OVD) is regarded as particularly important in the treatment with complete dentures where no teeth are present to aid the dentist in the establishment of a therapeutic OVD. It may also be of special concern for dentulous on partially edentulous patients who may have a reduced OVD due to wear or loss of the posterior teeth.

Many controversial claims have been made about the role of OVD on the health of the masticatory system. This review assesses the relative scientific merit of those claims and provides a scientifically based conclusion about the relationship of OVD to the health of the masticatory system.

EARLY PATIENT REPORTS

An "overclosed or collapsed" OVD due to wear or loss of teeth has been claimed to cause atrophy and perforation of the temporomandibular joint (TMJ) "meniscus" and perforation of the tympanic plate, deafness, tinnitus, problems in respiration and deglutition, earache and paresthesia of the tongue and pharynx, facial and glossopharyngeal neuralgia, vertigo, xerostomia, loss of tone of muscles of mastication, sleeplessness, lack of concentration, and diseases of the ear, nose, and throat.[1-13] Although he was not the first, Costen[14] is perhaps the best known among this group of authors. He concluded from clinical observations made on 11 patients that mandibular overclosure caused posterior displacement of the condyles.[14] The condyles, in turn, were believed to exert pressure on the chorda tympani and auriculotemporal nerves, and on the eustachian tubes. According to Costen,[14] pressure on these anatomic structures caused headache, sinus pain, earache, tinnitus, a stuffy sensation in the ears, burning tongue, and metallic taste and deafness, a group of symptoms that became known as "Costen's syndrome." These authors' simplistic and erroneous cause-and-effect deductions were based either on inaccurate interpretations of anatomic and radiologic findings[1, 4, 6, 9] or a misinterpretation of clinical "success" in relieving some of the symptoms involved by "opening the bite."[2, 3, 5, 7, 8, 11-14]

Although this hypothesis was widely accepted at the time, it was brilliantly challenged as early as 1925.[15] Additional evidence against it was offered in the late 1930s and early 1940s,[16-19] and it was proved wrong on anatomic grounds by Zimmerman as mentioned in Sarnat,[20] and Sicher[21] in the 1950s, and on clinical grounds by Schwartz[22] in 1959. Schwartz[22] reported his work of 10 years with 2500 TMJ patients and observed that "the symptoms that we found were not those emphasized by Costen . . . we were unable to relate them to 'bite closure'." Nonetheless, Costen's hypothesis led to the restoration of many dentitions at an "increased" OVD to treat or prevent the so-called Costen's syndrome. This approach prompted a new series of writings describing problems that were presumably caused by this practice, especially if the increase in OVD obliterated the interocclusal distance (IOD) from postural rest position or "freeway space."

The problems that were described included increased

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tooth mobility, alveolar bone resorption, trauma and intrusion or depression of teeth, fatigue, undue strain on muscles of mastication, clenching and bruxism, increase in the stress on the supporting tissues or excessive stresses on periodontium, recession and atrophy of bone and soft tissues, and myofascial pain.[23-30] According to these authors, an increase in OVD beyond postural rest position caused an increase in muscle activity in the jaw-closing muscles, "as the musculature attempts to recover the individual's original interocclusal clearance."[27] Unfortunately, no scientifically compelling evidence was provided to support these claims, since clinical observations and patient reports exclusively could not be used to establish cause-and-effect relationships. However, these clinical observations gave rise to two closely related testable hypotheses: (1) interocclusal distance from postural rest position is fixed and unchanging throughout life, and (2) if OVD is increased beyond postural rest position, it produces muscle hyperactivity in an effort to reestablish the previous IOD with possible symptomatic sequelae in the TMJs, muscles of mastication, periodontal tissues, and teeth.

EARLY CLINICAL STUDIES

Several early studies provided apparent support for the first hypothesis, that is, the claim for constancy of IOD. Niswonger[31] reported having found an interocclusal distance of 3.175 mm (1/8 inch) in 85% of 400 subjects by use of a soft tissue instrument he called a "jaw relator," measuring between a point on the chin and the junction of the philtrum and septum of the nose. This procedure provided a "one-shot" cross-sectional study, and the author implied from his results that the IOD was the same for most subjects. These results have not been replicated in subsequent studies, which showed soft tissue facial measurements to be relatively inconsistent and inaccurate,[32,33] accounting for as little as only half of the skeletal movement involved, in measuring known alterations in OVD of up to 6 mm.[33]

Brodie,[34] in studying cranial development in children from 3 months to 8 years of age, reported a "constancy of position of the mandible, which position is maintained by muscle tensions." Thompson and Brodie[35] and Thompson[36] conducted long-term follow-up studies (up to 8 years) in which the postural rest position was measured by use of cephalometric radiographs. The authors reported postural rest position to be stable and "not affected by presence or absence of teeth,"[35] with an "average" value of 2 to 3 mm for the IOD.[36] Based on these findings, the clinical technique of measuring vertical dimension at postural rest position and reducing that measurement by 2 to 4 mm to allow for IOD has been used and is still advocated for establishment of OVD.[37-42]

Thompson and Brodie's findings[34-36] were not replicated in subsequent research by Olsen,[43] who conducted a one-session study, and Atwood[44-46] and Tallgren[47,48] who conducted longitudinal studies of complete denture–wearing patients using cephalometric radiographs. They found a large intrasubject and intersubject variability and that postural rest position can be affected by head posture, absence of teeth, absence or presence of a prosthesis, and the emotional state. These findings have since then been extensively confirmed.[49-59]

LABORATORY STUDIES WITH ANIMALS

The second hypothesis, that is, that an increase in OVD beyond postural rest position will produce an increase in masticatory muscle activity leading to pain and affecting the TMJs, muscles of mastication, periodontal tissue, and teeth has been tested in monkeys, rabbits, and rats.[60-68] The methodologies have included histologic or histochemical studies of the TMJ; morphologic, histologic, and histochemical studies of masticatory muscles; histologic studies of the periodontium; clinical dental examination; and radiographic-morphologic studies.[60-68] OVD has been increased 5 mm interincisally in monkeys by use of gold crowns on lower left first molars,[60] 3 mm by use of cast splints covering all posterior teeth in both dental arches but occluding only on the distal aspects of terminal molars[61]; 2, 3, 5, 10, and 15 mm with cast maxillary splints covering the canines and all posterior teeth[62]; 2 mm with cast mandibular splints[63]; 7 mm[64] and 15 to 18 mm[65] with cast maxillary splints covering all posterior teeth; and 0.25 mm with a unilateral left maxillary posterior splint.[66] The increase in OVD was 5 to 7 mm interincisally in the experiment with rabbits[67] and 1 to 2 mm between the molars in the experiment with rats.[68] The OVD-increasing devices were in place from 7 days[66] up to 3 years[64]; sometimes for varying amounts of time for different subjects within each experiment.

None of these studies directly tested the first part of the second hypothesis, that an increase in OVD will cause hyperactivity of the masticatory muscles. The studies did attempt to test the second part of the hypothesis, that an increase in OVD will produce pain and affect the TMJs, muscles of mastication, periodontium, and teeth.

It is hard to measure objectively whether these monkeys had any discomfort due to an increase in OVD, although the authors of one study reported the monkeys were initially irritated and had "gnashing" of their teeth until they became used to the situation.[60] Other authors also reported slight, if any, initial difficulty of the animals with eating, no significant problems, resumption of normal dietary behavior shortly after the experimental intervention, no weight loss or any other apparent health problem[62,63,65,66] and even a total gain of weight for the experiment and no apparent distress.[64] Although objective measurement of pain is difficult even in humans, the overall clinical picture seems to imply their situation was not extremely uncomfortable.

These experiments confirmed that increasing OVD may affect the different components of the masticatory system at a histologic and morphologic level. Nonetheless, the response is not one of collapse and breakdown, as the hypotheses would predict, but one of general compensation and adaptation.

These experiments resulted in relatively minor changes and eventual adaptation in the masticatory system of the animals as long as the increases in OVD were not extreme and the devices used covered all or most of the teeth. The more severe responses, for example, tooth mobility, intrusion or extrusion,[60, 64] TMJ remodeling,[61] and other occlusal, skeletal, and muscular changes[62, 65, 67] occurred only when the increases in OVD were extreme[62, 65] or when the device used did not cover the dentition completely.[60, 61, 64, 67] The nature and severity of these experimental changes are quite different from those used in routine dental practice. The result of relatively large increases in OVD, with only partial coverage of the dentition, cannot be generalized to any increase in OVD or occlusal change. Interestingly enough, in spite of the severity of these changes, most of the experimental animals seemed to adapt to them successfully. We do not think that these results should be interpreted as implying that changes in occlusion or OVD are irrelevant to the health of the masticatory system. However, they do not prove that small or moderate changes in OVD as a result of clinical restorative dental treatment are a major cause of pain or other symptoms of temporomandibular disorder (TMD).

CLINICAL STUDIES WITH HUMAN SUBJECTS

Human studies in which OVD has been experimentally increased have been few since ethical concerns limit the type of intervention and amount of information that can be obtained. Nonetheless, findings of these studies are more clinically relevant, because pain and other subjective symptoms can be more easily evaluated in humans.

Christensen[69] and Carlsson et al.[70] increased OVD in dentulous subjects by means of bilateral splints covering all molars[69] and all teeth posterior to the canines[70] over a 3- to 7-day experimental period. There was no control group in either of these experiments. As mentioned, results obtained by increasing OVD with partial-coverage restorations may not necessarily be the same as those obtained with complete coverage of the dentition. The extent of opening was beyond the interocclusal distance[69] and approximately 4 mm interincisally.[70] Both experiments reported initial symptoms such as headaches, clenching and grinding, muscle and joint fatigue, soreness of teeth covered by the splint, cheek biting, and problems with chewing and speech articulation. Carlsson et al.[70] reported that all symptoms diminished in intensity after 1 to 2 days for all six subjects except one. They tested the hypothesis that increased OVD would cause masticatory muscle hyperactivity by electromyographic recordings. They found no evidence to support this hypothesis, and concluded that a moderate increase in vertical dimension of occlusion does not seem to be a hazardous procedure, provided that occlusal stability is maintained.[70] Besides the subjective symptoms, Christensen[69] reported an increase of tenderness to palpation in all masticatory muscles and concluded that increasing OVD "apparently deranged the function of muscles and joints."

A group of 22 complete denture–wearing patients also participated in Christensen's experiment, having their OVD increased to the clinically determined postural rest position. Rosas et al.[71] conducted a similar experiment with 15 complete denture–wearing patients, in which five served as controls and two groups of five subjects each had the OVD in their dentures either increased or decreased by 7 mm. Their results and conclusion were similar to Christensen's; that is, subjects in both experimental groups developed significant symptoms that the authors reported as supporting the "critical value of properly established vertical dimension in complete dentures."[71] The length of these experiments was 6 to 8 days.[70, 71]

There are several problems with Christensen's study. First the dentulous subjects were all dental students. Besides not being an appropriate control group to compare with edentulous patients, the dental students may have had preconceived notions about the experimental procedure in which they were participating. This problem raises the possibility of serious confounding factors. Second, the finding of increased tenderness to palpation, defined as "blink reflex elicited by moderate pressure"[69] is of questionable value in light of the vagueness of the definition. The lack of a double-blind evaluation design raises the possibility of unconscious researcher bias as an additional confounding factor.

Unfortunately, not enough details about the experimental design are given in the abstract by Rosas et al.[71] Subject expectation may affect the outcome in an experiment of this type. The way the experimental procedure is presented to the subjects may influence their reaction. In addition, for the use of a control group to be significant, the examining clinician or researcher must be unaware of the group to which the examined subject belongs; for example, control or experimental. Otherwise, unconscious researcher bias may play a factor in the evaluation of the results. In addition, control and experimental groups should be matched for age, sex, and other confounding factors. Unfortunately, without appropriate clarification of these issues, the conclusions presented by the authors of this study are unsubstantiated and questionable.

These three studies[69-71] do not provide sufficient evidence to support the hypothesis that increases in OVD may cause symptoms of TMD. Indeed, the study by Carlsson et al.[70] suggested the possibility of human adaptability, similar to that found in animals studies, to increases in OVD.

In addition to these purely experimental procedures, OVD has also been therapeutically changed in many patients by orthognathic surgical procedures. Wessberg et al.[72] evaluated neuromuscular adaptation to orthognathic superior repositioning of the maxillae in 15 subjects, with clinical and radiographic features of "maxillary vertical excess." They had a control group of 10 adult women with normal dentofacial morphology. The subjects in both groups were evaluated before and 3 months after surgery by computerized morphometry, mandibular kinesiography (MKG), and EMG measurements.

The authors found no statistically significant difference between presurgical and postsurgical trials, with respect to mean interocclusal distance from postural rest position or masticatory EMG activity ($p > 0.05$). This finding provides further evidence for the possibility of adaptability to changes in OVD in the human masticatory system.

Additional evidence along those lines has been provided by clinical treatment studies in TMD. Manns et al.[73] studied the influence of OVD in the reduction of myofascial pain-disorder (MPD) symptoms and masseter muscle EMG activity[74] in subjects with TMD. For the first of these studies[73] they had 75 subjects with range in age of 13 to 53 years, with MPD symptoms as described by Laskin,[75] and no previous use of occlusal splints or removable partial dentures. They used full-coverage "centric relation," that is, nonanterior repositioning acrylic resin splints with canine disclusion and anterior guidance, for 3 hours daily and all night for 3 weeks.

The subjects were randomly assigned to three groups. In the first group, OVD was increased by 1 mm with the splint. In the second group, OVD with the splint was increased halfway between OVD in centric occlusion and vertical dimension of minimal masseter EMG activity. The mean increase in OVD with the splint was 4.42 mm. For the third group, OVD was increased to that of minimal EMG activity in the masseter muscles. The mean increase in OVD with the splint was 8.15 mm. The experiment lasted 3 weeks. All of the subjects had a gradual decrease in symptom intensity, but the decrease was faster for groups 2 and 3, up to twice as fast for some symptoms.

For the second study,[74] they used a similar methodology, with 60 subjects randomly divided into three groups. In this study, they evaluated masseter EMG activity at postural rest position and with "slight tooth contact" on the splints. Both of these readings gradually decreased for groups 2 and 3, but remained about the same for group I.

Replication of these results by other researchers is not available; however, these results do not support the traditional belief that an increase in OVD beyond postural rest position will produce masticatory muscle hyperactivity and symptoms of TMD. These results are, indeed, the complete opposite of what the hypothesis would predict.

The evidence from these studies is not sufficient to prove that thicker splints work better than thinner splints in the treatment of TMD or that "improper" OVD plays no etiologic role on TMD. It appears, therefore, that the issue is still an open one and further research is justified.

CRITICAL REVIEW OF SOME TECHNIQUES TO EVALUATE OVD

OVD has been long regarded as important in dentistry as evidenced by the long list of methods and techniques proposed to establish it.[76] None of the proposed techniques has been proved scientifically accurate or superior, and scientific research has only succeeded in destroying old myths. Regretably, alternative concrete, practical, and accurate guidance for clinicians has not been provided although clinical realities demand methods that are predictable, scientifically tenable, and universally applicable. A brief examination of a few alternate approaches should help to substantiate our conclusions.

OVD AND BITE FORCE

The influence of OVD on bite force has been studied[77, 78] while testing Boos' hypotheses[79] that there is an optimum vertical dimension for development of masticatory force, one which occurs close to the postural rest position of the mandible which should be used as a therapeutic OVD. Mackenna and Turker's results with 23 cats,[78] and the Manns et al.[77] results in eight humans agreed with the first part of Boos' hypothesis, but their findings for optimal length were 15 to 20 mm of opening[77] and when the mouth was "almost completely open,"[78] disagreeing with the second part.

Although interesting, these findings provide no evidence for the third and most critical part of the hypothesis, that the jaw opening where maximal closing force is developed is the most appropriate for a therapeutic OVD. There is no clinical indication for the use of this technique at this time.

THE COMFORT ZONE APPROACH OF ESTABLISHING OVD

Tryde et al.[80, 81] used a different approach for the determination of OVD in edentulous patients. They had an adjustable screw jack inserted with wax occlusal rims. The screw jack was closed down until the reduced height was perceived by the subjects as being too low; then it was opened, until the increased height was perceived by the subjects as being too high. This procedure was repeated to obtain 22 pairs of high and low readings during one session,[81] or two pairs of high and low readings during each of 15 separate recording sessions done over a 3-week period.[80] The term "comfort zone" for the range betwen the high and low readings was used. This zone measured approximately 1 mm on the one-session experiment[81] and 1.3 mm on the multiple session experiment.[80]

These experiments were well designed, especially the multiple-session experiment[80] in which two different investigators made separate measurements, and the order of the investigator making the measurement and the height of the screw jack were randomized. These results appear to contradict the possibility of adaptability suggested by previous experiments, because the average comfort zone turned out to be a relatively narrow one, 1.3 mm. The clinical implication would be that complete dentures constructed at an OVD outside of this comfort zone may cause "discomfort" of the masticatory system.

The authors' conclusions are prudent ones as they observe that: "Further studies are needed before a clinical method applying a screw jack can be recommended."[80] In addition to acknowledged limitations in their experimental approach, the authors recognize the questionable merits of transferring the observed results to a clinical situation, since the possibility of patient adaptation to complete dentures constructed with an OVD outside of the comfort zone was never tested.

The existence of a comfort zone measured under artificial laboratory conditions was well established, but whether that comfort zone had any relationship to a physiologic or therapeutic OVD used for clinical restorative dental treatment was not explored. This should be considered a separate hypothesis and could be tested by making dentures with OVD outside of the comfort zone and developing means for quantifying how well patients do with them. Research of this kind should be done if the concept of "comfort zone" is to be regarded as scientifically tenable.

DISCUSSION

This review suggests that the state of the science in this field has been found wanting. However, because OVD is an integral part of routine prosthodontics treatment, a state-of-the-art analysis is necessary.

The hypotheses that postural rest position is fixed and unchanging throughout life and that restorations or prostheses that encroach on the postural rest position may cause pathologic sequelae appear to be untenable.

It seems reasonable to accept an alternate hypothesis that the correct or physiologic OVD can better be described as a range instead of a fixed point or position for most subjects.

Nonetheless, the width of that comfort zone may vary among individuals and in a single individual at different times, since individual adaptive capacity is unknown. It is also unknown whether what constitutes the optimum range for OVD in a fully dentulous state with diverse craniofacial morphofunctional features is carried over to, or modified by, different stages of partial edentulism or complete edentulism.

Most techniques for clinical establishment of OVD in complete denture–wearing patients are based on the premise that the OVD the patient had with natural teeth is likely to be the best one for the new edentulous state and therefore should be reproduced. This premise has not been studied scientifically, and remains an unproved assumption. If optimum OVD for the edentulous state were different—and this is certainly within the realm of possibility—the clinical establishment of OVD may have to be reevaluated. It should be conceded, however, that traditional clinical wisdom in establishing OVD on a routine basis and the clinical outcome achieved, appears to satisfy most dentists.

Nonetheless, the absence of compelling scientific evidence to prove or disprove the routinely employed clinical techniques should not be regarded as a justification for careless or haphazard approaches to the issue. Considering the amount of work involved, expense, and relative irreversibility of extensive prosthetic treatment, the adequate establishment and evaluation of OVD continues to be an important issue.

This issue may be especially critical when fixed prostheses are prescribed, because in none of the studies reviewed was the OVD increased by use of fixed, individual, unsplinted restorations. The effect of increases of OVD by use of metal or acrylic resin splints may not necessarily be the same as the effect of an increase by individual fixed restorations. This issue has not been studied in a controlled manner.

SUMMARY

A review of a large number of human and animal experiments failed to provide substantial evidence that moderate changes in OVD cause hyperactivity of the masticatory muscles and symptoms of TMD. Nor have published studies demonstrated that the postural rest position of the mandible is fixed and unaffected by a variety of extrinsic and intrinsic factors.

No technique to establish OVD has been shown to be scientifically accurate or superior to any other. Most of the assumptions on which popular techniques have been based, for example, "constancy of postural rest position," have not been supported by controlled research. The belief that appropriate OVD in the dentulous state is the same as OVD for the edentulous state also has not been shown. This is one of the many aspects in dental practice in which clinicians rely mainly on empirical knowledge.

It can be agreed that the application of this empirical knowledge has enabled and continues to enable the dentist to provide successful treatment. After all, OVD, like other quantifiable aspects of a body or system function, such as blood pressure per weight, for example, is unlikely to be a rigid, specific, and unchangeable entity; hence, the conventional clinical wisdom of regarding it as a variable range. Nonetheless, research that reconciles clinical techniques with basic science information should underscore the chosen methods of routine clinical application. OVD, like its allied and perhaps inseparable horizontal maxillomandibular considerations, deserves a more scientific approach to its understanding.

REFERENCES

1. Prentiss HJ. Preliminary report upon the temporomandibular articulation in the human type. Dent Cosmos 1918;60:505-12.
2. Reisner SE. Temporomandibular articulation: its consideration in orthodontic diagnosis. Int J Orthodont 1936;22:1-30.
3. Schopper AF. Lost vertical dimension: causes and effects; diagnosis and various treatments recommended. J Missouri Dent Assoc 1938;18:217-22.
4. Bleiker RF. Ear disturbances of temporomandibular origin. J Am Dent Assoc 1938;25:1390-9.
5. Decker JC. Traumatic deafness as a result of retrusion of the condyle of the mandible (preliminary report). Ann Otol Rhinol Laryngol 1925; 34:519-27.
6. Harris HL. Anatomy of the temporomandibular articulation and adjacent structures. J Am Dent Assoc 1932;19:584-90.
7. Fowler EF. Deafness associated with dental occlusal disorders in contrast with deafness definitely not so associated. NY J Dent 1939;9: 272-6.
8. Jamieson CH. Mandibular position and the partial denture. J Am Dent Assoc 1940;27:3-10.
9. Wright WH. Deafness as influenced by malposition of the jaws. J Natl Dent Assoc 1920;7:979-92.
10. Smith T. A review of the literature on the eustachian tube with data indicating a relation to the dental occlusion. J Am Dent Assoc 1925;12:671-81.
11. Monson GS. Impaired function as a result of closed bite. J Natl Dent Assoc 1921;8:833-9.
12. Greene E. Temporomandibular joint: dental aspect. Ann Otol Rhinol Laryngol 1937;46:150-7.
13. Batson OV. The closed bite and related clinical problems. Dent Cosmos 1938;80:1191-6.
14. Costen JB. Syndrome of ear and sinus symptoms dependent upon disturbed function of the temporomandibular joint. Ann Otol Rhinol and Laryngol 1934;43:1.
15. Connors MC. Tissue reactions in the temporomandibular articulation following loss of teeth. Dent Cosmos 1925;67:124-35.
16. Gillis RR. Roentgen-ray study of temporomandibular articulation. J Am Dent Assoc 1935;22:1321-8.
17. Higley LB. Practical application of a new and scientific method of producing temporomandibular roentgenograms. J Am Dent Assoc 1937;24:222-30.
18. Chor H. Neurologic aspects of temporomandibular disorders. J Am Dent Assoc 1938;25:1033-46.
19. Shapiro HH, Truex RC. The temporomandibular joint and the auditory function. J Am Dent Assoc 1943;30:1147-68.
20. Sarnat GB, ed. The temporomandibular joint. Springfield: Charles C Thomas, 1951;107.
21. Sicher H. Positions and movements of the mandible. J Am Dent Assoc 1954;48:620-5.
22. Schwartz L. Disorders of the temporomandibular joint. Philadelphia: WB Saunders Company, 1959;22.
23. Tench RW. Dangers in dental reconstruction involving increase in the vertical dimension of the lower third of the human face. J Am Dent Assoc 1938;25:566-70.
24. Mershon JV. Bite-opening dangers. J Am Dent Assoc 1939;26:1972-9.
25. Schuyler CA. Problems associated with opening the bite which would contraindicate it as a common practice. J Am Dent Assoc 1939;26:734-40.
26. Miller SC. The practical solution to the prevention and cure of temporomandibular joint disturbances. J Dent Med 1953;8:43-8.
27. Beyron HL. Characteristics of functionally optimal occlusion and principles of occlusal rehabilitation. J Am Dent Assoc 1954;48:648-56.
28. Schweitzer JM. Restorative dentistry—a half century of reflection. J Prosthet Dent 1974;31:22-51.
29. Dawson PE. Evaluation, diagnosis and treatment of occlusal problems. St Louis: CV Mosby Co., 1974;275-8.
30. Monteith B. The role of freeway space in the generation of muscle pain in complete denture wearers. J Oral Rehabil 1984;11:483-98.
31. Niswonger ME. The rest position of the mandible and centric relation. J Am Dent Assoc 1934;21:1572-82.
32. Swerdlow H. Roentgencephalometric study of vertical dimension changes in immediate denture patients. J Prosthet Dent 1964; 14:635-50.
33. Tryde G, McMillan DR, Christensen J, Brill N. The fallacy of facial measurement of occlusal height in edentulous subjects. J Oral Rehabil 1976;3:353-8.
34. Brodie AG. On the growth patterns of human head from third month to eighth year of life. Am J Anat 1941;68:209-62.
35. Thompson JR, Brodie AG. Factors in the position of the mandible. J Am Dent Assoc 1942;29:925-41.
36. Thompson JR. The rest position of the mandible and its significance to dental science. J Am Dent Assoc 1946;33:151-79.
37. Gillis RR. Establishing vertical dimension in full denture construction. J Am Dent Assoc 1941;28:430-6.
38. Schlosser RO. Methods of securing centric relation and other positional relation records in complete denture prosthesis. J Am Dent Assoc 1941;28:17-21.
39. Pleasure MA. Correct vertical dimension and freeway space. J Am Dent Assoc 1951;43:160-3.
40. Boucher CO, et al. Prosthodontic treatment for edentulous patients. 7th ed. St Louis: CV Mosby Company, 1975;273-5.
41. Heartwell CM, Rahn AO. Syllabus of complete dentures. 2nd ed. Philadelphia: Lea & Febiger, 1975;243-5.
42. Samant A, Martin JO, Cinotti WR, Moy F. Vertical dimension of the face and muscle tone. Compendium Contin Educ Dent 1986;7:755-9.
43. Olsen ES. A radiographic study of variations in the physiological rest position of the mandible in seventy edentulous individuals [Abstract]. J Dent Res 1951;30:517.
44. Atwood DA. A cephalometric study of rest position of the mandible. Part I. J Prosthet Dent 1956;6:504-19.
45. Atwood DA. A cephalometric study of rest position of the mandible. Part II. J Prosthet Dent 1957;7:544-52.
46. Atwood DA. A cephalometric study of rest position of the mandible. Part III. J Prosthet Dent 1958;8:698-708.
47. Tallgren A. Changes in adult face height due to aging, wear and loss of teeth and prosthetic treatment. Acta Odontol Scand 1957;15(suppl 24):1-122.
48. Tallgren A. The reduction in face height of edentulous and partially edentulous individuals during long-term denture wear. A longitudinal roentgenographic cephalometric study. Acta Odontol Scand 1966; 24:195-239.
49. Hickey JC, Williams BH, Woelfel JB. Stability of mandibular rest position. J Prosthet Dent 1961;11:566-72.
50. Reference deleted in galleys.
51. Preiskel HW. Some observations on the postural position of the mandible. J Prosthet Dent 1965;15:625-33.
52. Dombradγ L. Investigation into the transient instability of the rest position. J Prosthet Dent 1966;16:479-90.
53. Carlsson GE, Ericson S. Postural rest height in full denture wearers. Acta Odontol Scand 1967;25:145-62.
54. Kleinman AM, Asheppard IM. Mandibular rest levels with and without dentures in place in edentulous and complete denture-wearing subjects. J Prosthet Dent 1972;28:478-84.
55. Bando E, Fukushima S, Kawabata H, Kohno S. Continuous observations of mandibular position by telemetry. J Prosthet Dent 1972; 28:485-90.
56. Sheppard IM, Sheppard SM. Vertical dimension measurements. J Prosthet Dent 1975;34:269-77.
57. Gattozzi JG, Nicol BR, Somes GW, Ellinger CW. Variations in mandibular rest position with and without dentures in place. J Prosthet Dent 1976;36:159-63.
58. Feldman S, Leupold RJ, Staling LM. Rest vertical dimension determined by electromyography with biofeedback as compared to conventional methods. J Prosthet Dent 1978;84:216-9.
59. Troendle R, Troendle K, Rugh JD. Electromyographic and phonetic rest position changes with head posture [Abstract]. J Dent Res 1980; 59:494.
60. Waerhaug J, Hansen ER. Periodontal changes incident to prolonged occlusal overload in monkeys. Acta Odontol Scand 1966;24:91-104.
61. Gianelly AA, Ruben MP, Risinger R. Effects of experimentally altered occlusal vertical dimension on temporomandibular articulation. J Prosthet Dent 1970;24:629-35.
62. McNamara JA Jr. An experimental study of increased vertical dimension of the growing face. Am J Orthod 1977;71:382-95.
63. Maxwell LC, Carlson DS, McNamara JA Jr, Faulkner JA. Adaptation of the masseter and temporalis muscles following alteration in length with and without surgical detachment. Anat Rec 1981;200: 27-37.

64. Ramfjord SP, Blankenship JR. Increased occlusal vertical dimension in adult monkeys. J PROSTHET DENT 1981;45:74-83.
65. Carlson DS, Schneiderman ED. Cephalometric analysis of adaptations of the masseter muscle after lengthening in adult rhesus monkey, Macaca mulatta. Arch Oral Biol 1983;28:627-37.
66. Kubo Y. The uptake of horseradish peroxidase in monkey TMJ synovium after occlusal alteration. J Dent Res 1987;66:1049-54.
67. Sergl HG, Farmand M. Experiments with unilateral bite planes in rabbits. J Periodontol 1975;45:108-14.
68. Akagawa Y, Nikai H, Tsuru H. Histologic changes in rat masticatory muscles subsequent to experimental increase of the occlusal vertical dimension. J PROSTHET DENT 1983;50:725-32.
69. Christensen J. Effects of occlusion-raising procedures on the chewing system. Dent Pract Dent Rec 1970;20:233-8.
70. Carlsson GE, Ingervall B, Kocak G. Effect of increasing vertical dimension on the masticatory system in subjects with natural teeth. J PROSTHET DENT 1979;41:284-9.
71. Rosas P, Saavedra M, Barghi N, Rey R. Effects of increased and decreased vertical dimension of complete dentures [Abstract]. J Dent Res 1982;61:127.
72. Wessberg GA, O'Ryan FS, Washburn MC, Epker BN. Neuromuscular adaptation to surgical repositioning of the maxilla. J Maxillofac Surg 1981;9:117-22.
73. Manns A, Miralles R, Santander H, Valdivia J. Influence of vertical dimension in the treatment of MPD-dysfunction syndrome. J PROSTHET DENT 1983;50:700-9.
74. Manns A, Miralles R, Comsille F. Influence of vertical dimension on masseter muscle EMG activity in patients with mandibular dysfunction. J PROSTHET DENT 1985;53:243-7.
75. Laskin DM. Etiology of pain-dysfunction syndrome. J Am Dent Assoc 1969;79:147-53.
76. Turrel AJW. Clinical assessment of vertical dimension. J PROSTHET DENT 1972;28:238-43.
77. Manns A, Miralles R, Palazzi C. EMG, bite force, and elongation of the masseter muscle under isometric voluntary contractions and variations of vertical dimension. J PROSTHET DENT 1979;42:674-82.
78. Mackenna BR, Turker KS. Twitch tension in the jaw muscles of the cat at various degrees of mouth opening. Arch Oral Biol 1978;23:917-20.
79. Boos RH. Intermaxillary relation established by biting power. J Am Dent Assoc 1940;27:1193-9.
80. Tryde G, Stoltze K, Morimoto T, Salk D. Long-term changes in the perception of comfortable mandibular occlusal positions. J Oral Rehabil 1977;4:9-15.
81. Tryde G, Stoltze K, Fujii H, Brill N. Short-term changes in the perception of comfortable mandibular occlusal positions. J Oral Rehabil 1977;4:17-21.

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Power spectral analysis of electromyographic signal of masticatory muscles at rest position and habitual clench

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Thirteen normal subjects were selected for this investigation. At the vertical dimension of rest position and habitual clench, their myoelectric activities of the masseter, anterior and posterior temporal, and digastric muscles on the left and right sides were simultaneously sampled and processed by computer, and the mean power frequency (MPF) and the mean amplitude (MA) of the myoelectric signal were calculated. At rest position, the temporal muscles were major muscles and the MPF differences of various masticatory muscles were not significant. At habitual clench, with the increase in MA, the MPF of various muscles increased. The results show that MPF may have application in describing a central tendency of myoelectrical signal frequency distribution and in evaluating recruitment of motor units. MPF and MA are the quantitative indices reflecting masticatory muscle function. (J PROSTHET DENT 1991;65:553-6.)

Electromyogram (EMG) has been analyzed quantitatively from duration to frequency domain. Frequency analysis is done by a digital computer using a fast Fourier transform algorithm (FFT) to generate the power spectral density function to describe the power spectra of the signal. Spectral analysis involves the breakdown of the continuous EMG signal into a range of different frequency components; the power spectrum is a type of histogram showing the amount of power (activity) at each frequency.[1]

In reviewing power spectral analysis of the EMG of masticatory muscles, many research results are of interest. First, muscle fatique during sustained isometric contraction of the jaw muscles causes a shift to low frequencies in the EMG frequency power spectrum. The shift is due to increase in the low frequency range and decrease in the high frequency range, and it induces the mean power frequency (MPF) to decrease.[2,4] Second, an increase in bite force

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