Vertical malocclusion article raises questions

Editor:

In the Winter 1991 edition of the Angle Orthodontist, you requested letters to the editor, so I thought I would take this opportunity to air one of my pet peeves. In that same issue, Dr. Ib Leth Nielsen presented an article (Vertical malocclusions: etiology, development, diagnosis and some aspects of treatment. Angle Orthod 1991;61:247-260). In discussing the deep bite, low mandibular plane angle cases with reduced anterior face height, Dr. Nielsen states several times that a patient (or subject) has a retrognathic mandible (page 247, second column, line 13; page 256, second column, lines 18 and 26). This statement or judgment is, as near as I can tell, strictly arbitrary. He does not mention any kind of analysis to back up his statements (not that I know of any). When I look at the headplates and photographs of the same subject, I don't see a short mandible, I see lower teeth that are too far posterior on the mandible, making point b retrusive. This, in turn, allows the lower lip to curl inside the upper incisors (with the teeth in occlusion), creating a deep-appearing mentolabial sulcus.

Dr. Nielsen is not alone in this judgment; I have many colleagues who feel the same way. It is shameful that this opinion is so widespread, as the clinician making the judgment will decide this is an orthognathic surgery case; this type of case is never indicated for orthognathic surgery as it can be corrected very nicely at almost any age, nonextraction, with orthodontic treatment alone. I have numerous cases several years out of retention (adults and adolescents) to prove it. In this same article, Dr. Nielsen exhibits a similar case treated by another clinician using the Begg appliance, which was never successfully completed (his Figure 17). I don't know much about Begg techniques, but I can assure you that cases like this, approached correctly, are not difficult to treat with an edgewise appliance.

I have never seen orthognathic surgery cases of this type demonstrated out of retention, so who is to know if they are stable or not, if the surgery has any effect on the temporomandibular joint or if there are any other side effects. Even if we don't consider any postretention difficulties, the surgery requires an oral surgeon's fee, a hospital bill and the risks and pain of major surgery, on top of the orthodontic care and charges. In these days of escalating health insurance premiums, the public certainly does not need any more unnecessary charges. If the patient actually looked better after the surgery, it may be warranted, but that is strictly a matter of opinion.

One other thought on this article, in 29 years of treating adults, I have never been able to permanently increase the lower anterior face height in patients with very low mandibular plane angles. I have used bite plates in conjunction with elastics, attempting to open the mandibular plane angle, inevitably it closes to its original stance. In his Figure 21, Dr. Nielsen illustrates a female patient of this type, 14 years 9 months old, (hopefully she is past puberty) who has undergone orthodontic treatment and orthognathic surgery to advance her mandible and increase her lower anterior face height. Both of these goals were accomplished very nicely. I have never been able to permanently increase lower face height with orthodontic treatment alone, so when I read the article I wondered if it could actually be done with a combination of surgery and orthodontics. I conferred with my oral surgeon compatriot and he assured me that all of the fibers of the masseter and internal pterygoid muscles are left attached to the proximal segment (his term) or the angle and ascending ramus (my terms) of the mandible after the orthognathic surgery. As near as I can tell from his tracings, only the distal segment (his term) or the body minus the angle (my terms) was involved in opening the bite. As I can think of no other muscles with a vertical attachment from the skull to the distal segment that would tend to close the bite, I would assume that the vertical opening in this case might hold. In any event, it would be very interesting to see this case several years out of retention to find out.

Floyd W. Vallie, DDS, MS
Great Falls, Montana

Editor:

I am glad you asked for comments on published articles. Your request makes me feel free to comment on "Vertical malocclusions: etiology, development, diagnosis and some aspects of treatment" by Dr. Ib Leth Nielsen (Angle Orthod 1991;61:247-260). I highly compliment Dr. Nielsen on the treatment results shown. The orthognathic surgery case was particularly impressive. The surgeon did an excellent job.

I do not like the term "vertical malocclusion". We do not speak of "horizontal malocclusion". The former term is not an accurate description of a malocclusion which is primarily caused by excessive vertical growth or by a deficiency in vertical growth and it makes no distinction between

the two conditions.

Instead, the terms hyperdivergent and hypodivergent characterize vertical dysplasias which cause the malocclusion and distinguish between the two extremes. The term vertical malocclusion confuses malocclusion with facial type; they are not the same.

Dr. Nielsen states 'One particularly important factor in the development of deep bite and open bite is the pattern of growth of the mandible." I must state categorically that the vertical growth of the maxilla, or lack of it, is the primary cause of vertical dysplasias. I have discussed this several times in my writings and have presented a great deal of objective evidence to substantiate it. He attributes the downward growth of the palatal plane to sutural growth. There are no sutures between the orbits and the palatal plane and therefore the growth must be by bone absorption and addition. Otherwise, the orbits would increase in size as much as the palatal plane moves down.

Fred F. Schudy, DDS
Houston, TX

Author's response

Dr. Schudy states that he is opposed to the term 'vertical malocclusion' because "we do not speak of horizontal malocclusions." In today's teaching, we do in fact divide malocclusions into those that are horizontal or sagittal (Class II, III etc.), those that are vertical and those that are transverse. We also divide malocclusions into dentoalveolar and skeletal. The etiological factors associated with the development of the malocclusion are many and act in different combinations. We intentionally do not blend the etiology of the occlusal problem with the symptoms in the description of the malocclusion. In other words, we clearly separate cause from effect in the diagnosis. That is not to say we are not interested in the etiology of the problem and its development.

Along similar lines, Dr. Schudy also states that open bites and deep bites are caused by hyper- or hypodivergency of the face. That is not necessarily the case. More extreme vertical deviations in facial form may predispose a patient to a vertical malocclusion, however the malocclusion may never develop if the dentoalveolar compensation is adequate. It also should not be forgotten that not all open bites and deep bites are skeletal; some are dentoalveolar in nature and the result of functional factors such as a previous or present fingersucking habit or tongue dysfunction.

The role of the maxilla in the development of skeletal open bite and deep bite is claimed by Dr. Schudy to be the primary cause of the malocclusion. There is ample evidence in the implant studies of Björk and Skieller[1,2] of untreated subjects that the mandibular growth pattern is a primary etiological factor, with the maxilla's role being secondary. Further support for this notion can be found in a study by Isaacson et al.[3] comparing skeletal and dental relations in high angle, average and low angle cases. Their study demonstrates that while upper anterior face height on average is almost identical in all three groups, lower anterior face height differs significantly. This suggests a similar combined effect of sutural lowering and resorptive lowering of the nasal floor in all three groups and therefore does not lend support to the idea that the maxilla is the cause of the development of open bites and deep bites. This study further shows that the main differences between the three groups were found in the lower anterior face height and posteriorly in the dentoalveolar development in the maxilla. The difference in posterior maxillary dental height observed in the study by Isaacson et al.[3] is of clinical significance as it is this factor that we as orthodontists attempt to control with vertical mechanics, i.e. high-pull headgear, transpalatal arches, etc., and in the extreme case, correct through maxillary surgical impaction. The same clinically observed problem is undoubtedly what Dr. Schudy is referring to when he states that the maxilla is the cause of vertical dysplasias.

Finally, Dr. Schudy is correct when pointing out that there are no sutures between the orbits and the palatal plane. We disagree, however, as to the contribution of sutural growth and resorptive lowering of the nasal floor to vertical maxillary displacement. The nasal floor is lowered during growth in relation to the anterior cranial base by two mechanisms: sutural lowering and resorptive lowering (Björk and Skieller[2]). Concomitant with the lowering of the nasal floor, there is appositional growth in the opposite direction at the orbital floor which on average accounts for about 50% of the sutural lowering of the nasal floor.

Dr. Vallie writes that I described the patients illustrated in my Figures 1A and 16 (in the original article) as having mandibular retrognathia. The subject in Figure 16 is clearly in the retrognathic end of the spectrum with several cephalometric analyses, Björk, Steiner, etc. The subject in figure 1A has a mandible that is retrognathic in relation to the maxilla, but not to anterior cranial base. It is important to remember

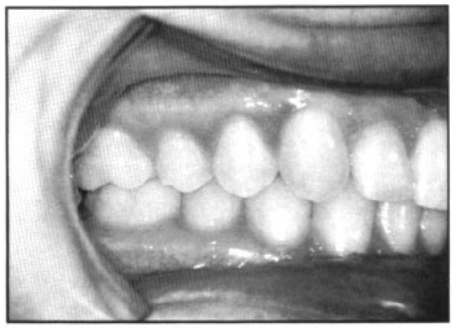

Figure 1A

Figure 1B

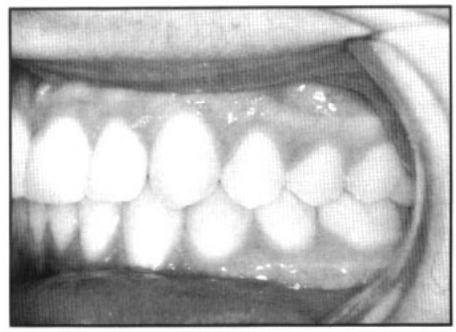

Figure 1C

that most cephalometric analyses relate jaw position to anterior cranial base. Measurements such as S-N-Pg do not describe the length of the mandible but merely its relative position to anterior cranial base.

Another comment made by Dr. Vallie relates to whether or not a case is "surgical". The decision to treat an orthodontic patient with a surgical procedure is based on a number of different factors, among which is the relative position of the mandible to cranial base. Just as important, however, are the relationship of the mandible to the maxilla, the soft tissue profile and the severity of the malocclusion as well as the patient's needs and desire for an esthetic change.

The choice of mechanics (Begg) for the treatment of the patient BK, illustrated in Figures 15-21 in the original article and in Figures 1 and 2 here, was based solely on that particular clinician's experience with the appliance. The lack of improvement in jaw position and occlusion during the initial phase of treatment does not reflect inadequacy of the mechanics but is a function of the severity of the skeletal discrepancy, the patient's musculature and the extreme occlusal problems. The stage of maturation and expected amount of residual growth after the initial treatment phase, where the patient was post-pubertal, additionally indicated that surgery was the treatment of choice in the second phase. Dr. Vallie questions the stability of the surgical mandibular advancement in patient BK.These intraoral photos (Figure1A-C) were taken two years posttreatment and demonstrate that the occlusion held up very well indeed. The operation performed on this patient was a modified sagittal split osteotomy which does not result in lengthening of the masseter and pterygoid muscles. Minimal tendency to posttreatment vertical relapse, therefore, can be expected which may explain why this case did not relapse vertically. In contrast, when skeletal deep bite cases are treated by orthodontic means alone to achieve an increase in anterior face height, the extrusion of posterior teeth is often seen to relapse posttreatment as the muscle fibers generally are incapable of lengthening sufficiently to accommodate the skeletal change.

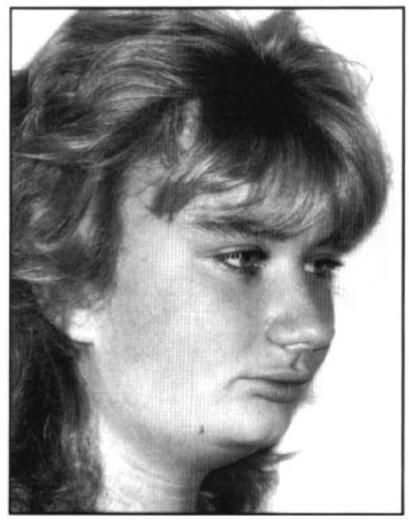

Figure 2A

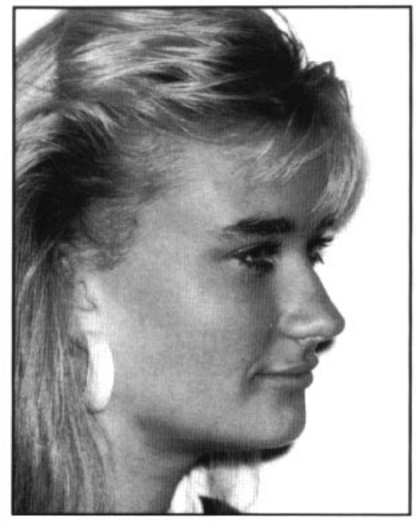

Figure 2B

Figure 1A-C
Case BK, 2 years posttreatment.

Figure 2
Case BK, pretreatment (left) and 2 years posttreatment (right).

Ib Leth Nielsen, DDS
San Francisco

References

1. Björk A, Skieller V. Facial Development and tooth eruption. Am J Orthod 1972;62:339-383.
2. Björk A, Skieller V. Postnatal growth of the maxillary complex. In: Factors affecting growth of the midface. J.A. McNamara Jr, Editor. Center for Human Growth and Development, Ann Arbor, Michigan, 1976.
3. Isaacson JR, Isaacson RJ, Speidel TM, Worms FW. Extreme variations in vertical facial growth and associated variation in skeletal and dental relations. Angle Orthod 1972;41:219-235.

Correction

The description of the patient in Figure 1A on page 248 of Dr. Nielsen's original article (Vertical malocclusions: etiology, development, diagnosis and some aspects of treatment. Angle Orthod 1991;61:247-260) should have read: "The direction of mandibular growth, as expressed at the chin, is mostly horizontal."

Other reasons for second opinions

Editor:

I enjoyed your recent editorial on second opinions and I have several thoughts relevant to your essay.

First, the recession is real and Americans are being more careful with health dollars. Since the cost of orthodontic treatment can vary from $2,500 to over $4,000, it would only seem reasonable that people would shop around.

Second, one topic excluded from your list of controversial ones is the issue of cosmetics versus pathology. Indeed, Consumer Reports dealt with that topic in its health section last year and debunked the pathology rationale.

Finally, a recent article in the American Journal of Orthodontics and Dentofacial Orthopedics on the consistency of orthodontic treatment decisions evaluated the need for extensive information gathering by orthodontists before making treatment decisions (1991;100:212-219). It appears that "shoppers" have a legitimate right to question that process, based on the article's conclusions.

Keep up the interesting and provocative editorials.

Peter Lax, DMD
Portland, Oregon

Braces: Health or beauty?
(Reprinted from Consumer Reports)

Q: Most children in my son's class have braces on their teeth, and our orthodontist is suggesting we have our son fitted, too. Are there good medical and dental reasons for giving children perfectly straight teeth, or is the main motivation cosmetic?

A.M.G
Bala Cynwid, PA

A: It's mostly cosmetic. Crooked teeth can certainly cause emotional distress, particularly in appearance-conscious teens. But the dental reasons usually given for straightening a child's teeth -- to prevent cavities and gum disease -- have been questioned by studies that failed to show a protective effect. Nor have researchers convincingly linked crooked teeth to temperomandibular (sic) joint (TMJ) syndrome. Only a severely disordered bite is likely to cause such physical problems as difficulty in chewing or gum disease.

Abstract
(Reprinted from the American Journal of Orthodontics and Dentofacial Orthopedics, 1991;100:212-219.)

Consistency of orthodontic treatment decisions relative to diagnostic records
Unae Kim Han, DMD, MPH, MS; Katherine W.L. Vig, BDS, D.Orth., FDSRCS(Eng.), MS; Jane A. Weintraub, DDS, MPH; Peter S. Vig, BDS, PhD, D.Orth. FDSRCS(Eng.); and Charles J. Kowalski, PhD

The purpose of this study was to evaluate how incremental information obtained from different types of diagnostic records contributes to the determination of orthodontic treatment decisions. Pretreatment records of 57 orthodontic patients were assessed by five orthodontists who were part-time faculty members and also in private practice. This sample consisted of dental school orthodontic patients who had Class II malocclusions and included patients at three different dental developmental stages. The following diagnostic records were used: study models (S), facial photographs (F), a panoramic radiograph (P), a lateral cephalogram (C), and its tracing (T). Five combinations of diagnostic records were presented to the orthodontists in the following sequence: (1) S; (2) S + F; (3) S + F + P; (4) S + F + P + C; and (5) S + F + P + C + T. The simultaneous interpretation of all diagnostic records (S + F + P + C + T) was used as the "diagnostic standard." There was a diagnostic standard for each of the patients and for each of the orthodontists. The diagnositic standard was achieved: (1) S = 54.9%, (2) S + F = 54.2%, (3) S + F + P = 60.9%, and (4) S + F + P + C = 59.9%. Thus, in a majority of cases (55%), study models alone provided adequate information for treatment planning, and incremental addition of information from other types of diagnostic records made small differences.