SPECIAL ARTICLE

The postural basis of malocclusion: A philosophical overview

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Over the last 100 years, many theories have attempted to explain the cause of malocclusion. Most have stated that it is inherited, but, more recently, greater emphasis has been placed on the influence of the environment, especially the activity and the posture of the oral soft tissues. Unfortunately, it is not possible to measure long-term posture with any precision, and this has reduced its perceived importance. When some evidence is missing and much of the rest conflicting, there is merit in moving from the traditional "prove-it" attitude to philosophical reasoning to separate the probable from the improbable. We do not know to what extent posture and parafunction might be inherited, but there can be no doubt that facial and dental structures are, at times, strongly influenced by the soft tissues and that some malocclusions appear to have a postural basis. This article undertakes a philosophical examination of the conflicting strands of evidence that link oral posture with malocclusion, hoping to create a theory based solely on the restricted evidence that is broadly accepted by all sides in this age-old debate. (Am J Orthod Dentofacial Orthop 2004;126:729-38)

In the past, clinicians such as Edward Angle[1] believed that "Orthodontic treatments are very unlikely to succeed, if the functional disorders are still going on." However, later research did not provide material support for this view, and it is currently thought that the influence of the soft tissues is limited to teeth and alveolus, which are believed to lie in a position of balance between tongue, cheeks, and lips. Unfortunately, long-term oral posture is almost impossible to measure; little information is available to guide clinicians when diagnosing parafunction or forecasting its consequences.

Deductive reasoning has always been the backbone of science. The philosopher Karl Popper[2] taught his students to first put forward a hypothesis and then test it. Unfortunately, the variability implicit in the biological sciences and especially orthodontics renders objective research difficult. If such variables cannot be controlled, false negatives might arise, sometimes leading to invalid positive assumptions. The problem can be compounded in clinical situations when other major variables might be introduced, such as operator skills and patient cooperation. Under these circumstances, even random, controlled trials[3] might not offer much help.[4]

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When essential data is either unobtainable or confused by large variables, Popper considered the *best-fit* approach more appropriate. Accepting that no amount of data can ever prove a hypothesis for certain, he recommended that we reverse the procedure and test all the hypotheses to see which fit the evidence best. The best-fit system has won approval in other fields, especially in situations such as ours with large confusing variables. In an effort to seek consensus, this overview will first consider the evidence that is quoted in support of the various theories and then test each of them against the restricted evidence that is broadly accepted by all sides.

When considering the etiology of malocclusion, most textbooks list the possible factors without providing a working hypothesis with which to balance their clinical relevance. Even projects that have been set up with the specific objective of establishing a hierarchy among the many factors have failed to find consensus and in the end just provide another list.[5] Congenital defects and trauma are always listed but are generally accepted as responsible for less than 5% of malocclusions. The influence of muscle activity and posture is usually included but listed under "local factors," and little guidance is given about the extent of their impact or how they can be assessed. The multifactorial concept is often advanced as an explanation, but some doubt has been cast on its mathematical credentials,[6] and it distracts attention from the search for the principal precipitating factors.

Without a clear understanding of the etiology of any condition, there is a risk that treatment becomes empir-

ical or symptomatic. Any theory must be based on a wide range of observations, and the more respected term *hypothesis* should perhaps be reserved for theories that accommodate all, or at least most, of the available evidence. Surprisingly few current theories can pass this test; this article attempts to identify the single most likely hypothesis. Some years ago, I put forward the following definition.

> A sound hypothesis needs to fit all the available evidence rather than rest on part of it. It needs to be both logical and specific. It is additionally convincing if it is compatible with evidence other than that upon which it was based, especially if no additional corollaries are required. The final test is if new prognostications can be drawn from it, which when tested are found to agree with both existing and future findings. In retrospect, the truth is usually simple.

It might be helpful to consider how well past theories fit this definition. Most are centered around the belief that the form and the size of the dental skeleton is primarily inherited, but the position of the alveolar bone and the teeth is open to environmental influences. The reasoning is as follows.

INHERITED FACTORS

The evidence of twins. There are many studies of monozygote twins, most of whom show striking similarities between their malocclusions. This is true even if they have been separated soon after birth and brought up in different environments. Some studies show a high rate of concordance in twins for specific malocclusions such as Angle Class II Division 2,[7] but the issue is confused by nontwin studies that suggest that this type of malocclusion is not genetic.[8,9] Horowitz et al,[10] in a classic study of 35 pairs, concluded that "[h]ighly significant hereditary variations occur in anterior cranial base, mandibular body length, total face height and lower face height ($P = 0.001$)," and many subsequent studies have reported broadly similar findings. These particular measurements appear to reflect variations in the direction of growth of the facial bones (either horizontal or vertical) rather than their inherited forms. This possibility is supported by the work of Lobb,[11] who, in a study of 30 pairs of identical twins, found that "the greatest variation in each group was in the spatial arrangement of the component parts of the craniofacial complex rather than within these components" and noted that, even when identical twins had identical occlusions, there was often a "considerable variation in the bony components." One problem in this type of research is that inherited variations of overall size cannot easily be separated from variations in arch form during growth,[12] adverse or otherwise, but newer work with Procrustes superimpositions might improve this situation in the future.[13]

Overall, the twin evidence is not as conclusive as some suggest, with clear differences of opinion among accepted leaders in genetics. We are left with no explanation for the fact that some monozygote twins have substantially different skeletal relationships[10,11] because it is clearly impossible for these to have been carried in the genes.

The evidence of x-rays. The desire to measure explains our heavy reliance on x-rays, which provide a convenient 2-dimensional image. Cephalometric articles have dominated the orthodontic literature over the last 60 years and have, in general, supported Brodie's conclusion in 1938: "The most startling find was the apparent inability to alter anything beyond the alveolar process."[14] This set the foundation for the subsequent belief that environmental forces (including treatment) do not alter the inherited pattern of skeletal growth. This view is currently maintained by random controlled trials[15] that show minimal skeletal change after orthopedic treatment.

We know that sutural growth causes the bones of the skull to move as whole units, and that these changes are followed by extensive remodelling. This combination maintains the original contours while permitting an increase in size; this in turn makes superimposition on landmarks an unreliable means of evaluating craniofacial form.[13] Björk and Skieller[16] overcame this problem by using implants and noticed that the bodily movement of bones tended to be disguised by the subsequent remodelling. Isaacson et al[17] reassessed Björk's original implant work and concluded that "[t]his rotation was not obvious in the past since it is masked by an external surface remodelling that tends to restore the relationship of the jaws to their original morphology." This can also be seen after sagittal split osteotomies, when the mandible sometimes appears to relapse to its original position despite rigid fixation.[18] In these cases, the metal fixings act as bone markers and show that the true relapse is less than it appears because there is a concomitant process of peripheral adaptation. As a result, the lateral skull x-rays can give a misleading impression of the true jaw changes.

Situations such as these cloud the whole issue of using x-rays to identify tooth or bone movement. If whole bones remodel toward their original positions after movement, how can we assess changes in the base of the skull where bone markers cannot be used? For instance, we know that horizontal and vertical growth is associated with contrasting saddle angles.[10,19] Occasionally, patients show a change in the direction of

facial growth accompanied by an altered saddle angle.[20,21] In this situation, an analysis of x-rays superimposed along sella-nasion would give a misleading impression of the change in growth direction. Battagel[22] found that "vertical changes are not easily detected by conventional cephalometric investigations."

It is unlikely that any part of a growing skull remains static,[23] and Ricketts et al[24] attempted to overcome this by constructing midpoints along the general direction of growth to increase the predictability of forecasts. This has proved very accurate on average, but individual forecasts have been less so.[25]

Because Brodie et al[14] could detect only minimal movement of the skeleton during treatment, he and many since assumed that the movement had no clinical significance. However, if remodelling masks such changes, x-rays alone must be a less-than-certain means of determining their true extent. No doubt the situation will become clearer with the development of modern scanning techniques.[13]

Parent/child similarities. Rightly or wrongly, most clinicians consider malocclusions to be inherited because of similarities between parents and their children. Although many children have the same malocclusions as their parents, many do not, and severe Class II and Class III malocclusions can appear and disappear in a single family over a few generations. Lavelle[26] was one of the first to use a multivariate analysis to separate the many factors he thought might be responsible. However, his findings, like those of many since, were barely significant, and he concluded that "a simple Mendelian model is not compatible with most craniofacial dimensions."

Much publicity has been given to a series of studies on the inheritance of mandibular prognathism in the Habsburg kings.[27] These opinions, originally written in German, were based on interpretations of royal portraits, most of which were three-quarter views. Mayoral[28] provided an English translation and observed that throughout the Habsburg family "superior micrognathism is a more constant feature than inferior prognathism." It is known that the whole family was heavily interbred. Thompson[29] believed that they suffered from craniosynostosis, which would be an equally valid, and possibly more rational, explanation for the inherited maxillary hypoplasia. Similar inherited pathologies are found in bulldogs. It would seem that geneticists currently stand on the middle ground that "[a]t least half of the phenotypic variation in this sample is due to environmental differences,"[30] but there appears to be no strong evidence either way.

Mixed inheritance. One hundred years ago, Case[31] suggested that malocclusion was due to disproportionate inheritance of the skulls, jaws, and teeth from parents of different racial backgrounds. Although supported by some orthodontists, this theory is at odds with the geneticist's teaching that inbreeding is more likely to cause genetic disproportion than outbreeding.[32] I know of no evidence to support the idea that large parts from 1 parent are mixed with small parts from the other. Consider, for example, a single species with a contrasting gene pool such as the dog. A Chihuahua can be mated with a Saint Bernard that is 100 times as large and very different in appearance,[33] but, even if both had been purebred for many generations, malocclusion in any offspring is extremely unlikely. Not only will the tooth size be matched to the jaw size for each animal, but also both jaws themselves will also be matched. There will be some intermediate-sized offspring whose growth will also have been balanced even though their parents were purebred extremes. Where did their genes come from? Most evolutionary biologists would reject the concept of disproportionate inheritance on the dual grounds that it is virtually unknown in animals and hard to relate to Darwinian theory.

Attrition. Begg and Kesling[34] thought that lack of attrition in modern diets results in crowding. Although this is an environmental concept to explain dental crowding, Begg still believed that skeletal form was inherited. They reported that the wear between the teeth of primitive aborigines was equivalent to a premolar's width in each quadrant and suggested that much of this was due to grit in their food. However, Corruccini[35] questioned this concept after finding that the mesiodistal dimensions of the teeth "did not relate to crowding." He found that few of Begg's samples suffered from wear to this extent, even in old age. Primitive food certainly contained more grit, but modern bruxers eating a diet of refined foods might achieve equivalent wear; this makes it more likely that much of the wear is due to chewing rather than grit. Although Begg's theory explains some aspects of crowding, his own figures show the reverse of what might be expected, because both crowding and attrition increased with age.

Functional matrix. The functional matrix hypothesis was put forward by Moss,[36] who suggested that the soft tissue units guide the hard to an extent that renders skeletal genes superfluous. Essentially a genetic theory, this explains how the soft tissues mold the hard. However, it does not explain all aspects of bony development and has proved difficult to test. It has little favor with embryologists who point out that the long bones from chick embryos will develop normally without soft tissues. Moss's theory also suggests that the nasal cartilage influences the growth of the mid-face, but this is unproven. Figure 1 shows the changes in the

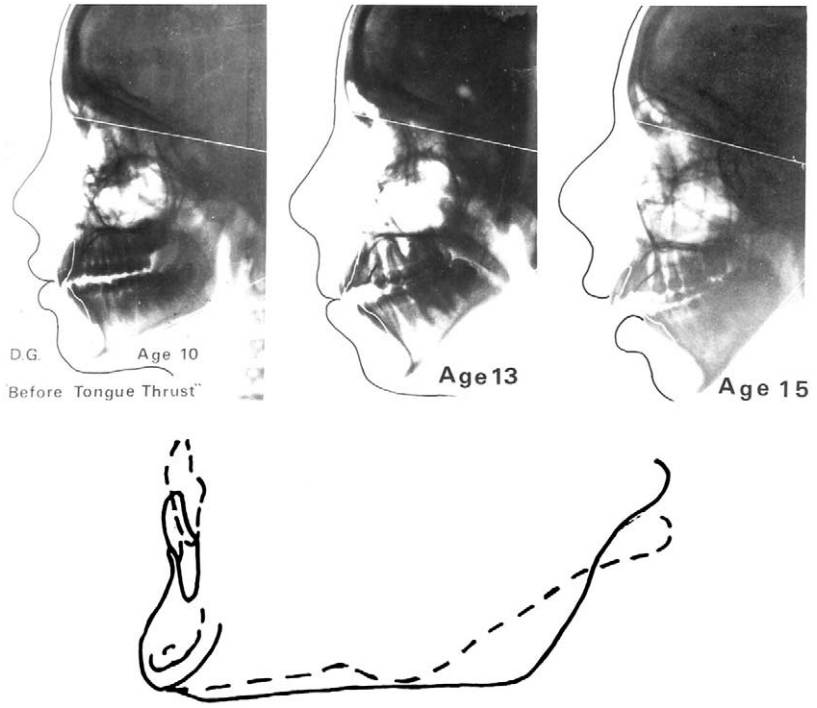

Fig 1. Ten-year-old girl who developed tongue-between-teeth swallow. Note changes in shape of mandible, superimposed on inner symphyseal outline. (Used with permission of British Journal of Orthodontics 1981;8:203-11)

mandible of a 10-year-old girl who developed a parafunctional swallow. As is often seen with vertically growing faces (Fig 2), the vertical ramus has remodelled forward, in this case shortening the horizontal ramus by a third, during maximum growth. There are few muscle attachments on the anterior or posterior borders of the ascending ramus, and the trigger for this massive remodelling seems to have been nothing more than gonion sliding back passively between the investing tissues of the pharynx. No other bone in the human body changes its relative position (or form) to this extent and, in view of the lack of muscle activity, the embryologists' concept that tissues grow and adapt in response to positional information[37] from neighboring cells provides a more understandable explanation than the functional matrix. Although Moss's theory fits some situations well, it does not seem able to explain all aspects of malocclusion.

Evolutionary change. It has been suggested that a genetic shift is causing the jaws to become progressively smaller.[38] However, it is hard to understand how this could happen because genetic shifts can occur only if the gene pool itself changes; there is no evidence for this. A genetic change would have required selective pressure that eliminated those with larger jaws. Nothing suggests that this has occurred; in any case, change in the genes themselves would require 100,000 years or more.[39]

Soft tissue drag. In 1977, Solow and Kreiborg[40] suggested that some differences in craniofacial morphology could be explained by the drag of the soft tissues on the facial skeleton caused when the mouth is dropped open. Solow suggested that there is a "dentoalveolar compensating mechanism"[41] that tends to restore the incisal relationship despite the skeletal disproportion. This theory is a good fit in many situations and has a more environmental slant, suggesting a sequence of nasal obstruction, craniocervical extension, and increased tissue drag. However, it does not explain how the alveolar compensation is effected or the origin of some malocclusions such as deep bites or Class III, which the authors appear to accept as genetic.[42]

Most of these 8 theories accept that malocclusion has a genetic basis, but this appears to be at odds with other evidence.

If malocclusion were inherited, one would expect a sign of its progressive spread in the historical, geographical, or epidemiological records, but this has not occurred. Instead, we find the classic malocclusions appearing in diverse populations wherever civilization

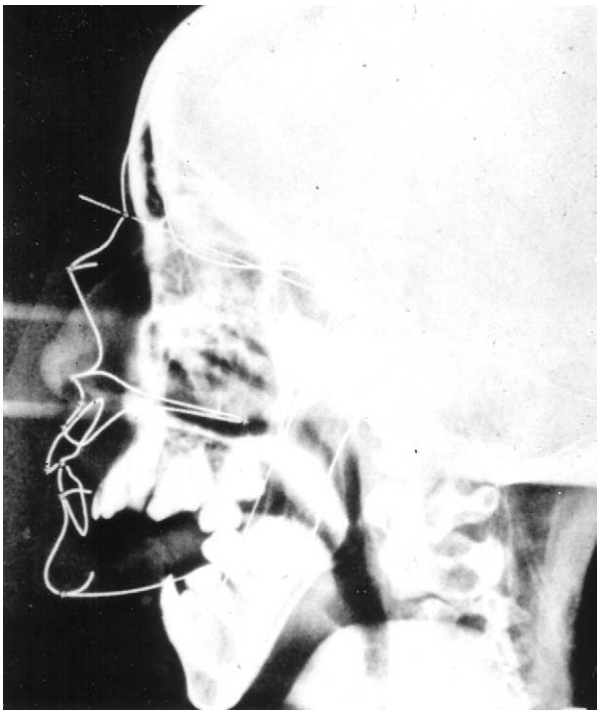

Fig 2. Radiograph of 12-year-old girl with muscular dystrophy, with average outline for her age superimposed. (Used with permission of author and publisher. Kreiborg et al. American Journal of Orthodontics 1978; 74:121-41)

progresses above a certain level. Although skeletal form certainly varies from 1 location to another, significant malocclusions in each area are restricted to the last 20,000 years.

Extragroup differences between humans are surprisingly small even between white and black people, suggesting that our subspecies are genetically very similar. However, intragroup variations of the facial skeleton are often large, making it probable that epigenetic factors are interceding.

Malocclusion is also relatively less frequent in modern man living in primitive conditions, and Corruccini et al[43] found that deep bites were 9 times as common in a sample of privileged children in India as in their close relatives who were poor. In the modern industrialized world, malocclusion has worsened[44] so that it is now endemic.[45]

Recent work on the human genome[46] appears to confirm that our genes have been handed down over the last 30,000 years or more, with little alteration.

ENVIRONMENTAL THEORIES

Other theories are primarily environmental.

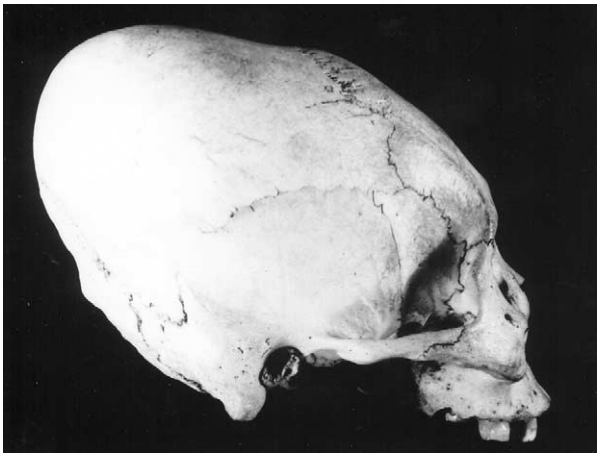

Fig 3. Skull of North American Indian whose head was bound as child. Kindly photographed and loaned by Smithsonian Institution, Washington, DC.

Muscle tone and activity. Modern diets are relatively soft, and it is suggested that this has led to a reduction in muscle strength.[47] Figure 2 shows a 12-year-old girl who suffered from muscular dystrophy, compared with the average profile for her age. Her maxilla appears to have collapsed downwards, possibly under the force of gravity and drag from the "soft tissue mask,"[41] to create a horrendous skeletal malocclusion. This has caused the mandible to hinge back and has been associated with a massive restructuring of that bone, although clearly no force was involved. There is now wide agreement that muscle weakness is linked to increased vertical growth.[48] Figure 3 shows the skull of a North American Indian whose head was bound as an infant. It is clear that these light forces caused a massive and permanent change that involved most of the bones in the cranium. This applies to the sphenoid and temporal bones that received no direct pressure but nevertheless underwent substantial remodelling. The teeth and their supporting structures are known to be highly resistant to short-term force, but the 2 subjects illustrated (Figs 2 and 3) show that light forces over an extended period can have a dramatic effect on the skeletal bones, especially in younger children. This would suggest that muscle posture is more influential than function.

There are many ways of assessing muscle force, and earlier research tended to rely on short-term clench tests that might or might not reflect long-term posture. Muscle thickness is probably a better guide than force measurements.[48] Benington et al[49] found that "a steeply inclined mandibular plane, a small posterior face height and an increased gonial angle were strongly related to

short, thin masseter muscles of low volume." The effect of the muscles depends on the product of both time and force, together with the distribution of types 1 and 2 fibers, which can vary within a muscle over time. All these variables can influence muscle thickness, and failure to allow for each factor separately can render research results questionable. It is sometimes suggested that poor occlusal contacts inhibit firm muscle contraction, but Ingervall[50] found that "strong muscles with resultant hard biting and increased function cause better occlusal stability," suggesting the reverse. Electromyography is helpful in determining long-term muscle contraction but tends to be qualitative rather than quantitative.

Bone is a rigid and inflexible structure, and changes such as these can occur only by organized apposition and resorption. We do not know how bones can adapt in this way, but embryologists believe that positional information is of crucial importance during organogenesis,[37] again suggesting that posture is more important than function.

Open-mouth postures. The term *mouth breather* can be misleading because Vig et al[51] showed that many people who keep their mouths open still breath nasally. Fratto et al[52] suggested that "mouth breathing may be due to acquired habits following previous rhino-pharyngeal obstruction." It would seem that these changes might be habitual rather than obligate. For this reason, I have always preferred "open-mouth posture" rather than "mouth breather" or "incompetent lips." Research that ignores these distinctions is liable to provide flawed results.

The natural lip seal of most healthy infants in industrialized populations has been lost by the age of 4,[53] and, for them, open-mouth postures must be considered normal. As they reach puberty, some, for social reasons, will develop a competent lip seal, but this will tend to be achieved with voluntary rather than involuntary muscle fibers, and many will still leave their lips apart for long periods, especially at night.

By its nature, posture is long term, and thus accurate measurements have been difficult to record or monitor, resulting in many negative findings. However, within the last few years, several articles have supported the concept that closed-mouth postures are beneficial.[54] Linder-Aronson et al[55] studied postadenoidectomy patients and found that those who developed a natural lip seal showed substantial forward movement of the incisors and increases in arch length. Gross et al[54] found that patients with "high levels of open-mouth posture manifested significantly smaller growth of the maxillary arch." These findings have been supported by many others,[12,57,58] with few to the contrary.

Many clinicians and especially occlusionists believe that a resting freeway space of several millimeters is normal. If "rest" were synonymous with "minimal muscular activity," then the study of Rugh and Drago[59] would suggest that the mouth should be open 8.6 mm; this seems excessive. In this context, normal might be misleading, because there is little evidence to suggest that large freeway spaces exist outside the industrialized world.

Few would doubt that letting the mouth hang open will encourage overeruption of the teeth (Fig 2), and, if Proffit and Sellers's work[60] on animals is linked to their work on humans,[61] it is obvious that the teeth must be in contact for a sufficient period each day to prevent them from overerupting. Once overeruption has taken place, the increase in facial height is likely to maintain a broken lip seal (Figs 1 and 2) and so perpetuate a vertical pattern of growth. On the other hand, if the teeth are in contact for long enough with enough force, they will not overerupt, and the occlusion will be excellent.[62,63] There seems strong evidence to show that open-mouth postures are associated with both increased vertical growth and malocclusion.

Tongue between teeth swallowing and parafunction. It was accepted for many years that the tongue should suck against the palate when swallowing, but Rix[64] demonstrated that many people swallow with their tongue between their teeth. These people will usually recruit the lips and buccinator muscles to seal the margins of the tongue, and this is associated with visible parafunction and hypertrophy of the muscles concerned. Levine[65] found that nonnutritive sucking (eg, thumb sucking) might be as high as 95% in many western countries but is almost unknown in primitive cultures, where breastfeeding often continues for 3 or 4 years.[66]

There is universal agreement that the teeth and alveolus lie in a position of balance between the cheeks, lips, and tongue; however, research in general has found that the tongue is the more powerful. To some extent, this is at odds with the observed fact that the teeth in many malocclusions are inclined lingually. Lundeen and Gibbs[62] and Melsen et al[63] have all noted that those who rest and swallow with their teeth in contact have less malocclusion, suggesting that tongue-between-teeth postures could also cause malocclusion.

The effect of suction during swallowing was debated at length in the 1930s but subsequently ignored because of inconclusive findings. Harvold et al[67] experimented with lumps of plastic placed against the palates of monkeys. This appeared to have little imme-

diate effect other than to displace the tongue from its normal position. However, in the longer term, there were widespread occlusal and skeletal consequences, and "in every juvenile animal the maxillary arch was considerably reduced in width."[67] This suggests that changes in muscle posture are more influential than muscle function. At times, as we have seen, this will result in massive restructuring of the dental skeleton, compared with which the genetic contribution to skeletal variation must be accepted as small.

DISCUSSION

A sound hypothesis must be able to accommodate all the evidence. We have just considered 8 hypotheses largely based on inheritance and 3 environmental theories, none of which can stand alone. This leaves us in the same position as many previous assessments, with some partial explanations but no agreed hierarchy of probabilities. The objective of this article was to find the single most likely explanation.

The lack of positive evidence confirming genetic influence is surprising. There must be some inherited features somewhere, and, if so, why can they not be clearly demonstrated? In other research, Harvold et al[68] took groups of young monkeys with perfect occlusions and changed their oral posture by blocking their noses. Every monkey developed a severe malocclusion, but the pattern of irregularity varied between groups. Here we have an identical environmental assault precipitating different malocclusions. This is highly relevant to our discussion, and we should consider how this information fits the human model.

We have just discussed how young children from industrialized countries leave their mouths open most of the time.[53] This presumably creates a similar situation to the open-mouth postures in Harvold's monkeys that produced a malocclusion every time. But why should a single environmental factor, such as open-mouth posture, produce a range of different malocclusions, the characteristics of which appear to run in families?

We know that muscle patterns are inherited,[69] especially of the tongue,[70] and that the teeth, the alveolus, and even the skeletal bones are very sensitive to muscle posture. Is it possible that bizarre tongue patterns are expressed only if the environment is prejudiced in some way—eg, if the child keeps his mouth open or develops a dislike for hard food? Sharma and Corruccini,[71] in their study of identical twins, came to a similar conclusion, that "the muscular balance achieved by the lips, cheeks and tongue may be disturbed by abnormal or habitual behaviour patterns, perhaps shared in families." A similar situation exists with a familial tendency to heart disease, where the actual cause is more probably obesity, smoking, or lack of exercise. These hidden genetic differences might surface only when the person is put under environmental stress. Philosophically, this is a more rational explanation, which appears in keeping with the evidence, and would explain why skeletal malocclusion does not obey Mendelian rules.[26] This could be stated: "environmental factors disrupt resting oral posture, increasing vertical skeletal growth and creating a dental malocclusion, the occlusal characteristics of which are determined by inherited muscle patterns, primarily of the tongue." This theory could offer a simple explanation for the mixed genetic and environmental features of many, if not most, malocclusions.

Figures 1 and 2 demonstrate that muscle function and posture can at times produce a large but unpredictable disruption of facial growth. Current scientific protocol precludes single-case evidence; however, in many studies, variables of this size might increase the standard deviation so that there is little chance that existing statistical techniques can identify the much smaller genetic differences. This might be why this debate has remained unresolved for so long.

Available hypotheses

I am aware of 4 theories that offer viable explanations for the etiology of malocclusion, those of Begg and Kesling,[34] Moss,[36] and Solow and Kreiborg,[40] already quoted, and my own "tropic premise."[72] This states that "Because the genetic control of skeletal growth is not precise, the articulation of the teeth and jaws depends upon additional guidance from oral posture." It is further suggested that "If the tongue at rest is against the palate with the lips lightly sealed and the teeth in or near contact, there will be ideal facial and dental development." This posture is rare in industrialized societies, but the same could be said of the number of adults with ideal occlusions, possibly for the same reason. It seems to be restricted to those who have good muscle tone and swallow with their tongues sucking against the palate. There is a simple logic to the tropic premise, because if the tongue rests against the palate with the lips sealed, the erupting teeth have little option but to slide down the space between them, until the cusps contact and guide them into occlusion. If, on the other hand, the tongue rests between the teeth, they will have no guidance and are likely to erupt haphazardly as is seen in aglossia. According to this hypothesis, every type of malocclusion, including Class III[73] and temporomandibular disorders,[74,75] is closely linked to specific oral postures.

Begg and Kesling's attrition theory,[34] Moss's func-

tional matrix,[36] Solow and Kreiborg's soft tissue drag,[40] and the tropic premise all offer an explanation for the etiology of malocclusion. At this point, we can test them using Popper's best-fit method[2] on the basis of the broadly accepted evidence we have just discussed

1. Our direct ancestors of 30,000 years ago had little malocclusion.
2. There is no evidence that genes have changed significantly since then.[46]
3. Fewer than 5% of humans suffer from inherited dentofacial deformities.
4. Malocclusion is endemic in industrialized countries.
5. There has been no genetic pattern in its geographical or historical spread.
6. Skeletal and dental structures are highly resistant to short-term heavy forces.
7. The skeletal and dental structures are very pliant to long-term light forces.
8. On occasion, changes in oral soft tissue function and especially posture are followed by major alterations in the growth of the facial skeleton.

Begg's theory explains crowding but not skeletal disproportion. Moss's theory suggests that soft tissues carry the genes causing the malocclusion; this is hard to relate to factors 2 and 5 above. Solow and Kreiborg's theory[40] fits all the above but does not explain the different types of malocclusion. The tropic premise appears to be the only theory that is compatible with all 8, and it also offers an explanation for the inherited variations in malocclusions. This is not confirmation that it is correct, but it does seem to fit the available evidence and appears to be a sound basis for further research. It replaces the many partial explanations that now exist with a single hypothesis covering 95% of malocclusions. Having used it for many years,[75] I think clinicians will find it a helpful guide in most circumstances.

The final test of the tropic premise will be if new prognostications can be drawn from it, which, when tested, are found to agree with both existing and subsequent research results— eg, whether specific tongue and lip postures are found to be associated with certain malocclusions (Fujiki et al[76]). If malocclusion is inherited, then our armamentarium is limited to mechanics and surgery, but, if the tropic premise is valid, then correcting oral posture at a young age might offer a permanent cure.

There is little new in these concepts, which were put forward by Roux,[79] Kingsley,[80] Angle,[1] and Herbst[81] at the turn of the previous century. Orthodontists tend to concentrate on clinical cures, but, as Helman[77] suggested in 1921, we should pay more attention to the "biologic laws underlying the development of malocclusions." In 1968, Harvold[78] said, "For years clinicians have realized that the tongue and facial muscles are the factors which determine the size of the dental arches and the crowding and spacing of teeth and that the skeleton has a subordinate role." However, Harvold's powerful research had little influence on clinical practice, and, as Machiavelli (1469-1527) said, "The reformer has enemies in all those who profit from the old order, and only lukewarm defenders in all those who might profit from the new."

CONCLUSIONS

Our current environment and diet are dramatically different from those of our ancestors of 30,000 years ago when malocclusion was rare; no evidence suggests that there has been a significant change in our genes since then. This questions all hypotheses that are based on the long-held belief that most malocclusions are inherited. There is strong scientific, logical, and clinical evidence to suggest that the weak muscles and open-mouth postures that are now endemic in our society can cause increased vertical growth, whereas parafunction of the tongue, lips, and cheeks is known to displace both the teeth and their supporting bone. Such a malocclusion would be a postural deformity.

Perverse oral postures have proved difficult to measure, diagnose, and treat, and, in these circumstances, we should perhaps be guided by basic research in preference to clinical evidence. The tropic premise seems the best fit of the hypotheses that have been considered, and no evidence appears to disprove it. It suggests that environmental factors disrupt resting oral posture, increasing vertical skeletal growth and creating a dental malocclusion, the occlusal characteristics of which are determined by inherited muscle patterns, primarily of the tongue. The last entry in Victor Hugo's diary states "There is one thing stronger than all the armies in the world; and that is an idea whose time has come."

REFERENCES

1. Angle E. Treatment of malocclusion of the teeth. 7th ed. Philadelphia: S. S. White; 1907.
2. Popper KA. Conjectures and refutations. London: Raubledge & Kea Paul; 1963.
3. Sackett D. Clinical epidemiology. 2nd ed. Boston: Little, Brown; 1991.
4. Mew JRC. Are random controlled trials appropriate for orthodontics? Evid Based Dent 1991;3:35-6.
5. Brash J. The aetiology of irregularity and malocclusion of the teeth. London: The Dental Board of the United Kingdom; 1956.

6. Mew JRC. The aetiology of temporomandibular disorders: a philosophical overview. Eur J Orthod 1997;19:249-58.
7. Markovic M. At the crossroads of oral-facial genetics. Eur J Orthod 1992;14:469-81.
8. Kerr WJS. The variability of some cranio-facial dimensions. Angle Orthod 1991;61:205-10.
9. Pancherz H, Zieber K, Hoyer B. Cephalometric characteristics of Class II Division 1 and Class II Division 2 malocclusions: a comparative study in children. Angle Orthod 1997;67:111-20.
10. Horowitz EP, Oxbourne RH, de George FC. Cephalometric study of craniofacial variations in adult twins. Angle Orthod 1960;30: 1-5.
11. Lobb WK. Craniofacial morphology and occlusal variation in monozygous and dizygous twins. Angle Orthod 1987;57:219-33.
12. Betzenburger D, Ruf S, Pancherz H. The compensatory mechanism in high angle malocclusions: a comparison of subjects in the mixed and permanent dentition. Angle Orthod 1999;69:27-32.
13. Mclntyre GI, Mossey PA. Size and shape measurement in contemporary cephalometrics. Eur J Orthod 2003;25:231-42.
14. Brodie AG, Downs WB, Goldstein A, Myer E. Cephalometric appraisal of orthodontic results—a preliminary report. Angle Orthod 1938;8:266-89.
15. Tulloch JFC, Phillips C, Profitt WR. Benefit of early Class II treatment: progress report of a two-phase randomized clinical trial. Am J Orthod Dentofacial Orthop 1998;113:62-72.
16. Bjork A, Skieller V. Growth of the maxilla in three dimensions. Br J Orthod 1977;4:53-64.
17. Isaacson RJ, Worms FW, Spiedel TM. Measurement of tooth movement. Am J Orthod 1976;70:290-303.
18. Carels C, Willems G, Verdonck A, Bossuyu M, Verbeke G, Kiekens RM. Post surgical changes in mandibular morphology after bilateral sagittal split osteotomies. Eur J Orthod 1994;16: 442.
19. Jarvinnen SHK. Posterior cranial base and sagittal jaw relationship. Eur J Orthod 1993;15:448.
20. Karlsen AT. Association between facial height development and mandibular growth rotation in low and high MP-SN angle faces: a longitudinal study. Angle Orthod 1997;67:103-10.
21. Singh GD, McNamara JA, Lozanoff S. Thin-plate spline analysis of the cranial base in subjects with Class III malocclusion. Eur J Orthod 1997;19:341-53.
22. Battagel JM. The use of tensor analysis to investigate facial changes in treated Class II Division 1 malocclusions. Eur J Orthod 1996;18:41-54.
23. Forsberg C. Facial height and tooth eruption in adults—a 20 year follow-up investigation. Eur J Orthod 1991;13:251-4.
24. Ricketts RM, Schulhof RJ, Bagha L. Orientation-sella-nasion or Frankfort horizontal. Am J Orthod 1976;69:648-54.
25. Mitchell DL, Jordan JF, Ricketts RM. Arcial growth with metallic implants in mandibular growth prediction. Am J Orthod 1975;68:655-9.
26. Lavelle CLB. An analyses of the craniofacial complex in different occlusal categories. Am J Orthod 1977;71:574-82.
27. Schulz C. Zur Atiologie de Progenie. Fortacher Kieferorthopadie 1979;40:87-104.
28. Mayoral J. Inferior prognathism in the Spanish kings of the house of Austria. Dent Rec 1931:610.
29. Thomson EM. Another family with the Habsburg jaw. J Med Genet 1988;25:838-42.
30. Cassidy MC, Harris EF, Tolly EA, Keim RG. Genetic influence on dental arch form in orthodontic patients. Angle Orthod 1998;68:445-54.
31. Case C. The techniques and principles of dental orthopedia. Chicago: Case Company; 1908.
32. Corruccini RS. How anthropology informs the orthodontic diagnosis of malocclusion's causes. Lewiston (NY): Edward Mellen Press; 1999.
33. Kennel Club illustrated breeds standards. London: Random House; 2003.
34. Begg PR, Kesling PC. Malocclusion in aboriginal and civilized man. Begg othodontic theory and technique. Philadelphia: W. B. Saunders; 1977.
35. Corruccini RS. Australian aboriginal tooth succession, interproximal attrition, and Begg's theory. Am J Orthod Dentofacial Orthop 1990;97:349-57.
36. Moss ML. The functional matrix. Vistas in orthodontics. Philadelphia: Lea & Febiger; 1962.
37. Grant P. Biology of developing systems. New York: Holt Rinehart and Winston; 1978.
38. WalpoffWH. Determinants of mandibular form and growth. Ann Arbor: Center for Human Growth and Development; University of Michigan; 1975. p. 34-7.
39. Pepazian HP. Modern genetics. London: Weidenfeld & Nicholson; 1967.
40. Solow B, Kreiborg S. Soft tissue stretching: a possible factor in craniofacial morphogenesis. Scand J Dent Res 1977;85:505-7.
41. Solow B. The dento-alveolar compensating mechanism. Br J Orthod 1981;7:145-61.
42. Solow B, Sandham A. Cranio-cervical posture: a factor in the development and function of the dentofacial structures. Eur J Orthod 2002;24:447-56.
43. Corruccini R, Keul SS, Chopra SRK, Karosas J, Larsen MD, Morrow C. Epidemiological survey of occlusion in North India. Br J Orthod 1983;10:44-7.
44. Moore WJ, Lavelle CLB, Spence TF. Changes in the size and shape of the human mandible in Britain. Br Dent J 1968;125: 163-9.
45. Dickson CG. The natural history of malocclusion. Dent Pract 1970;20:216-32.
46. Sykes B. The seven daughters of Eve. New York: W. W. Norton; 2001.
47. Kiliaridis S, Kalebo P. Masseter muscle thickness measured by ultrasonography and its relation to muscle morphology. J Dent Res 1991;70:1262-5.
48. Kiliaridis S, Mejersjo C, Thilander B. Muscle function and cranio-facial morphology: a clinical study in patients with myotonic dystrophy. Eur J Orthod 1989;11:131-8.
49. Benington PCM, Gardener JE, Hunt NP. Masseter muscle volume measured using ultrasonography and its relationship with facial morphology. Eur J Orthod 1999;21:659-70.
50. Ingervall B. Correlation between maximum bite force and facial morphology in children. Angle Orthod 1997;67:415-22.
51. Vig PS, Sarver DM, Hall DJ, Warren BN. Quantitative evaluation of airflow in relation to facial morphology. Am J Orthod 1981;79:272-3.
52. Fratto G, Barbato E, Proietti D, Poggesi MP, Cannoni D. Breathing tests in children with and without mouth breathing signs. Eur J Orthod 1999;21:587.
53. Glatz-Noll E, Berg R. Oral disfunction in children with Down's syndrome: an evaluation of treatment effects by means of video-registration. Eur J Orthod 1991;13:446-51.
54. Bresolin D, Shapiro GC, Shapiro PA, Dassel SW, Furuawa CT, Pierson WE, et al. Facial characteristics of children who breath through the mouth. Paediatrics 1984;73:622-5.
55. Linder-Aronson S, Woodside DG, Hellsing G, Emerson W.

Normalization of incisor position after adenoidectomy. Am J Orthod Dentofacial Orthop 1993;103:412-27.
56. Gross AM, Kellum GD, Franz D, Michas K, Wlaker M, Foster M, et al. A longitudinal evaluation of open mouth posture and maxillary arch width in children aged 5 to 9. Angle Orthod 1994;64:419-24.
57. Trotman CA, McNamara JA Jr, Dibbets JM, van der Weele LT. Association of lip posture and the dimensions of the tonsils and sagittal airway with facial morphology. Angle Orthod 1997;67: 425-32.
58. Lofstrand-Tidestrom B, Thilander B, Ahlqvist-Rasted J, Jakobss O, Hultcrantz E. Breathing obstruction in relation to craniofacial and dental arch morphology in 4-year-old children. Eur J Orthod 1999;67:425-32.
59. Rugh JD, Drago CJ. Vertical dimension: a study of clinical rest position and jaw muscle activity. J Prosthet Dent 1981;45:670.
60. Proffit WR, Sellers KT. The effect of intermittent forces on the rabbit incisor. J Dent Res 1986;65:118-22.
61. Lee CF, Proffit WR. The daily rhythm of tooth eruption. Am J Orthod Dentofacial Orthop 1995;107:38-47.
62. Lundeen HC, Gibbs CH. Advances in occlusion. Boston: John Wright; 1982.
63. Melsen B, Attina L, Suntueri M, Attina A. Relationships between swallowing pattern, mode of respiration and developing malocclusion. Angle Orthod 1987;57:113-9.
64. Rix RE. Deglutition and the teeth. Dent Rec 1946;66:103.
65. Levine RS. Briefing paper: oral aspects of dummy and digit sucking. Br Dent J 1998;186:108-43.
66. Larsson E. Sucking, chewing, and feeding habits and the development of crossbite: a longitudinal study of girls from birth to 3 years of age. Angle Orthod 2001;71:116-9.
67. Harvold EP, Chierier G, Vargervik K. Experiments on the development of dental malocclusion. Am J Orthod 1972;61:38-44.
68. Harvold EP, Tomer BS, Vargervik K, Chierici G. Primate experiments on oral respiration. Am J Orthod 1981;79:359-72.
69. Lorrenz KZ. The evolution of behavior. Sci Am 1958;199:67-8.
70. Darlington CD. The genetic component of language. Heredity 1947;1:269-86.
71. Sharma K, Corruccini R. Genetic basis of dental occlusal variations in northwest Indian twins. Eur J Orthod 1986;8:91-7.
72. Mew JRC. The aetiology of malocclusion: can the tropic premise assist our understanding? Br Dent J 1981;151:296-302.
73. Mew JRC. Factors influencing mandibular growth. Angle Orthod 1986;56:31-48.
74. Mew JRC. The aetiology of temporomandibular disorders: a philosophical overview. Eur J Orthod 1997;19:249-58.
75. Mew JRC. Biobloc therapy. Braylsham Castle, Sussex, United Kingdom: author; 1986.
76. Fujiki T, Inoue M, Miyawaki S, Nagasaki T, Tanimoto K, Takano-Yamamoto T. Relationship between maxillofacial morphology and deglutitive tongue movement in patients with anterior open bite. Am J Orthod Dentofacial Orthop 2004;125: 160-7.
77. Helman M. Studies on the etiology of Angle Class II malocclusal manifestations. Transactions of the American Society of Orthodontists; Saint Louis; 1921. p. 76-97.
78. Harvold EP. The role of function on the etiology and treatment of malocclusion. Am J Orthod 1968;61:38-44.
79. Roux W. Entwick der Organismen, Bd I and II. Leipzig: Englemann; 1895.
80. Kingsley NW. A treatise on oral deformities as a branch of mechanical surgury. London: H. K. Lewis; 1880.
81. Herbst E. Dreissigjahrige Erfahrugnen mit dem Retention-Schanier. Zahnaerztl, Rundschau 1934;43:1515-24.