The Aetiology of Malocclusion

Can the Tropic Premise Assist our Understanding?

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The relative responsibilities of the inherited and environmental components of facial growth are examined. While the underlying control of facial growth is accepted as genetic it is clear that this is frequently tempered by environmental factors, sometimes to a point where skeletal malocclusion results. What is not clear, is how often this point may be reached. It is suggested that posture may influence inherited bone growth by a 'tropic' mechanism which guides the maturation of the dental tissues. Because muscle postures are also inherited, this premise provides a convenient explanation for the mixed genetic and environmental nature of malocclusion.

FEW things can seem more certain to the clinical orthodontist than that the morphology of the facial skeleton is inherited and yet, the greater our knowledge of genetics, the less we can be sure of the strength of the aphorism.

This review will consider the reason for this and suggest how many of the apparantly contradictory viewpoints can be reconciled by a simple hypothesis.

The Problem

For convenience, the causes of malocclusion can be considered under 3 headings: (1) congenital failures, (2) inherited deficiencies, (3) environmental influences. It is fairly simple to isolate the 'congenital failures' such as cleft palate or partial anodontia and compare them to similar conditions in similar proportions in the rest of the animal kingdom (Colyer, 1936). However, it is more difficult to separate the inherited and environmental factors from each other, for instance enlarged tonsils or abnormal tongue and lip posture. If, for convenience, these 2 are linked together, one finds little parallel in feral malocclusion where they hardly exist, in contrast to their overwhelming presence in modern man. Certainly the human environment has changed dramatically over the last 10,000 years, but whether our present level of malocclusion is related to this, or to some genetic deficiency is a matter of much controversy.

History

Until recently, the environment had been given little weight as a major cause of malocclusion. This had been so since the days of Angle (1907), who undoubtedly overplayed this aspect with phrases such as 'Faulty environment in which the ideal alignment of the teeth as intended by the great "Watchmaker" did not reach full expression.' Lack of convincing evidence sapped their cause and the futility of accommodating 32 teeth in every mouth was exposed by their treatment failures. Furthermore, the advent of lateral skull x-rays in the 1930s seemed to show that orthodontic treatment had little influence on the hard tissues other than the teeth and alveolus (Brodie, 1938). This swing of the pendulum led to the concept of genetic immutability which was later consolidated in the comprehensive review of Brash (1956). As a result, most orthodontic treatment was, until a few years ago, carried out on the assumption that the problem was one of skeletal inheritance and techniques were tempered accordingly.

Alternate Viewpoints

From an extreme point of view, these contrasting ideas could be expressed as either:

'The form, size and position of the jaws is entirely genetically predetermined leaving only the position of the teeth and alveolus open to the adaptive influence of environmental factors';

or: 'Environmental influences of both intra- and extra-oral origin can produce major changes in the facial skeleton.'

Most people's beliefs, and also, presumably, the truth, are dispersed somewhere between these two extremes. However, there are difficulties in accepting either viewpoint as the following discussion will show.

Discussion

The Recent Increase in Malocclusion

There has been an evolutionary reduction in the size of the jaws from the time of our pre-hominid cousins 10 million years ago until the arrival of modern *Homo sapiens* about 35,000 years ago (Wolpoff, 1975). However, the reduction over this period was not accompanied by any significant increase in malocclusion which remained around the 10 per cent level, comparable with monkeys (Colyer, 1936). Then, within a short period, in settled communities all round the world we find much higher proportions of malocclusion. Dickson (1970) in his excellent assessment of several surveys concluded, 'malocclusion is, and has been for many generations past, an almost universal state in *Homo sapiens*.'

It has been suggested that a continuing evolutionary change is taking place in the size of the jaws as a result of the reduced pressures of selection, as we no longer need good occlusion to survive; or, in other words, the reverse of Darwin's theory of improvement by natural selection. However, the time period is too short. Papazian (1967) inferred that a minimum of 300,000 years is necessary for a major evolutionary change and yet there has been a significant further change in jaw size within the last 400 years, a mere 16 generations (Moore *et al.*, 1968). This would suggest that such changes are environmental. In any case, the mere removal of a selective factor such as effective dentition could not be expected to have much influence unless it became a positive disadvantage, and there is no indication that this is the case.

It is sometimes forgotten that, for new variants to spread outside their own demesne, a considerable proportion of the remaining population must perish. In other words, the percentage of the new variant in the population will remain the same unless something occurs to reduce the

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number of its rivals. This latter situation has occurred with myxomotosis in rabbits so that almost all the existing population is descended from a small number of resistant animals. However, there is no evidence of a human variant with malocclusion becoming established as a result of the death of most of the rest of the population; indeed, the gene pool of the human species has been for many years so vast that it is hard to see how any evolutionary change could fail to be 'washed out' in subsequent generations.

Calvin Case suggested in the last century that this sudden increase in malocclusion was due to mixed inheritance as a result of population movements with 'father's large teeth in mother's small jaws,' but as the majority of 'western' races are of similar ethnic background, this cannot be the whole explanation (Dickson, 1970). There is little, if any, evidence of disproportionate inheritance effecting other parts of the body, human or animal, although inherited pathologies may well do so (for example, the cartilaginous displasias suffered by some dogs). A moment's reflection would assure us that a very heavy selective pressure would have developed during the evolution of life to prevent disproportionate inheritance from different sized parents, and it is extremely unlikely that chromosome pairing would be so haphazard. Case's views have found little support in the recent literature on facial growth for reasons that will become clear later in this article.

The Constant Association of Malocclusion with Specific Levels of Civilisation

We know that this progressive increase in malocclusion has been associated not only with time but also with location. Groups living in primitive situations display, in common with those living in primitive times, a lower ratio of malocclusion. Research workers in different parts of the world have found the same pattern in each area and Lundstrom and Lysell (1953) in Scandinavia, Begg (1954) in Australia, Moore *et al.* (1968) in Britain have all described a similar sequence. Lundstrom's findings are especially significant in this respect as there had been little population movement in the intervening period. This again suggests the changes are environmental.

There is little opportunity in this day and age to study the comparative effects of refined or primitive diets on the facial skeleton. However, one should respect the earlier views of intelligent observers such as Weston-Price (1945), even if his comments on evolving malocclusion were rather subjective. One cannot help being impressed by the many cases he quotes from all over the world.

Although such clear-cut situations no longer exist, most recent scientific studies also show similar patterns at significant levels (Preston, 1979).

The Difficulty in Establishing a Pattern of Inheritance in the Spread of Malocclusion

If malocclusion is inherited, was at one time rare, and is now common, then at some time each specific genetic variation must have materialised as a dominant gene in an individual prior to spreading throughout the civilised world.

For example, a similar situation has occurred with sickle-cell anaemia, which provides near immunity to malaria. It has thus become firmly established in the genetic coding of certain populations where it is an asset for survival. The genes for this condition obey normal Mendelian laws and their progressive spread can be studied not just within family groups, but also from area to area.

If malocclusion obeyed the same genetic pattern, each variation from the norm would have occurred at a specific time and place, in which case its subsequent spread would have been evidenced by historical or anthropological findings. However, although certain types of malocclusion are more common in specific areas (Class III in the Far East), every type is found in every area with no sign of progressive spread. This distribution is not easy to reconcile with theories of inherited malocclusion.

Familial Malocclusion

The similarities of some parent and child malocclusions are so marked that it might be surmised that they are, in fact, genetic, although it must be borne in mind that soft tissue behaviour patterns are inherited as well as skeletal form (Lorenze, 1958). However, it was not proved easy to trace any pattern of Mendelian inheritance in familial malocclusion.

To explain this apparent anomaly, it was suggested that the sequence could be multi-factorial and Harris and Kowalski (1976) felt that this is why no clear Mendelian pattern is seen. Lavelle (1977) used a multivariate analysis in an attempt to separate the many factors he felt might be responsible, but with barely significant results and states: 'a simple Mendelian model is not compatible with most cranio-facial dimensions.' Smith and Bailil (1977), following a comprehensive survey, throw the issue open again by saying 'because of the very real possibility of environmental effects, there is no justification in concluding from the available literature that occlusal variables are polygenic.'

The Reduction in Size of the Jaws

The sequence of jaw reduction before the advent of *Homo sapiens* might well have been evolutionary, but can we assume that the reduction over the last 10,000 years is also? At no previous stage in our evolution did the reduction in size of the jaws fall far out of step with that of the teeth (Wolpoff, 1975) and yet, according to Lavelle and Foster (1969) only 2 per cent of our existing population are free from either crowding or spacing, much of it very severe.

One of the most frequent ingredients of modern malocclusion is the narrow maxilla, and a simple comparison with pre-historic skulls shows that not only is the modern jaw often narrower but also that the teeth are more likely to be lingually inclined. This is not easy to rationalise, for, if this were an inherited reduction in jaw size, one might expect to see the cheek teeth splayed outward rather than the reverse.

Saggital Anomalies

Apart from the reduction in the size of the jaws, an additional variable has arisen within most civilised societies: the pre-normal and post-normal relationship of one jaw with the other. However, it is hard also to see this as an evolutionary trend, regressive or otherwise.

Evidence of Twins

Identical twins provide clear insight to the genetic control of growth and the many surveys have all produced similar

results. To quote but one (Horowitz *et al.*, 1960) 'highly significant hereditary variations occur in anterior cranial base, mandibular body length, total face height and lower face height ($P = 0{\cdot}001$).' Lundstrom (1955) summarised 'when common non-genetic factors can modify this relationship, we ought to be able to improve disturbing deviations with out comparatively powerful therapeutic measures.' In support of this rationalisation, Mew (1977*a, b*) described a case where 19-year-old twins had marked skeletal differences but became near identical following orthopaedic treatment to one of them.

It would seem that the genetic control of growth is, in general, imprecise and nowhere does this appear more obvious than in the face. Also, as will be discussed this is the area that seems especially susceptible to environmental influence.

The Adaptability of Bone

That bones are responsive to environmental stimuli has never been in doubt, and the literature is full of examples from primitive native customs to modern surgical appliances, many of which produce permanent distortion. Experiments on unfortunate monkeys have distorted practically every tissue and Harvold *et al.* (1972) have summarised 'any common type of dental irregularity can be produced experimentally on monkeys with normal dentitions.' Harvold also showed (1968) that mere changes of tongue posture could result in extensive alterations to the occlusion and Schulmacher (1977) demonstrated that a change in the head posture of rats produced remodelling of the base of the skull. If rats are fed on a semi-liquid diet, they develop a narrowing of the maxilla and lengthening of lower face height (Salik and Schneider, 1977), a situation not unfamiliar to the clinical orthodontist.

Human research seems to confirm that posture, especially that of the tongue and mandible, may influence facial growth. Foster *et al.* (1977) have shown that the development of the maxilla appears to be related to factors of this type and the reported cases of conditions such as muscular dystrophy would suggest that this also applied to the mandible (Kreibourg *et al.*, 1978) (fig. 1). Head posture also seems influential but whether this is a primary or secondary factor to that of the mandible and facial mask has not been established (Woodside and Linder-Aronson, 1979).

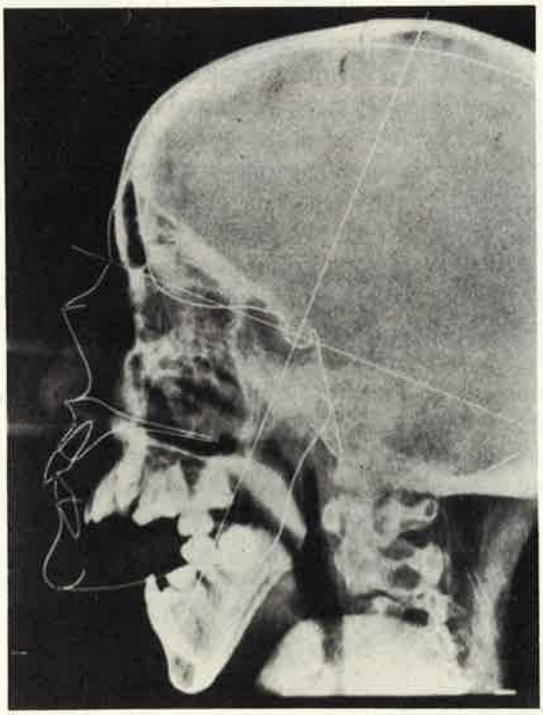

Fig. 1.—Taken from Kreibourg *et al.* (1978). A 12-year-old Danish girl with muscular dystrophy. The white line indicates the average for Danish girls of an equivalent age. *(By permission of the authors and publishers.)*

Biologists in the field of facial growth (Enlow *et al.*, 1977) are now broadly agreed that changes in the facial skeleton produced by indirect agencies such as rheumatoid arthritis or myasthenia gravis are largely environmental. Enlow anthologises 'the total deformity is 10 per cent pathology and 90 per cent biology.' This is very significant as the pathology may be quite unrelated to the skeleton and yet may produce changes so pronounced that individual bones are barely recognisable. These distortions are presumably the result of no more than the very mild forces produced by changes in the posture and form of the adjacent diseased tissues (fig. 1). If bone is so freely adaptable, this must increase the likelihood of malocclusion being environmental.

Radiographic Evidence

Despite the bone changes that occur naturally in humans, or have been induced in animals by orthopaedic appliances, there is little evidence to show that similar changes can be induced in man (Mills, 1978). This may be because clinical forces are of a more gentle nature. We know that during the growth of the mandible, the body often rotates forward, but that adaptive growth recontours the margins so that the overall relationships appear undisturbed. Isaacson *et al.* (1976) state of the mandible in their secondary study of Bjork's implant cases 'It is also apparent that this rotation was not obvious in the past, since it is masked by external surface remodelling that tends to restore the relationship of the jaws to their original external morphology.'

It would obviously be possible for remodelling of this type to disguise overall treatment changes in the position of the bones if any occurred. If this were so, the bone would appear to remain stationary while the teeth were moving although, in fact, the teeth and bone could have moved together prior to the remodelling.

If adaptive changes do indeed blurr the accuracy of lateral skull radiographs (Moyers and Bookstein, 1979), the keystone to the extreme philosophy of skeletal immutability is removed and the evidence of animal experiments and twin studies becomes more significant.

Variations in the Direction of Facial Growth

Those who believe that the dental skeleton is under direct genetic control are faced with the difficulty of explaining the changing directions of facial growth. These changes, which may have a forward, downward or even backward vector, have defied forecasts by even the most sophisticated computers. While the mandible does display many inherited characteristics it is hard to provide any genetic explanations for such haphazard variations in its direction of growth.

Summary Assessment

We can condense the foregoing discussion as follows:

(1) The history and presentation of malocclusion is in

many ways incompatible with concepts of skeletal inheritance.

(2) Studies of identical twins appear to show that the facial bones are freely responsive to environmental influences.

(3) Applied forces and created postures can produce major and permanent changes in the facial skeleton.

Current Clinical Attitudes

Does this mean that purely environmental factors could precipitate whole aspects of clinical malocclusion such as a narrow maxilla or a retruded mandible? While, at first thought, this seems improbable, there is really little evidence to refute it, other than that of x-rays which, as we have seen is suspect. What perhaps has not been given due consideration, is the influence of posture during the long period from infancy to puberty in contrast to the research evidence of function or appliances over much shorter periods around the age of puberty itself. Most of the dramatic changes achieved with monkeys have been with animals less than 30 months old, the equivalent to a human child of under 8 years. A considerable number of clinicians feel that the susceptibility of the human dental skeleton decreases rapidly after the age of 10 (Weislander, 1976; Graber, 1977; Wertz and Dreskin, 1977) and, as yet, little orthopaedic research has been carried out in younger age groups. Either way, it is impossible to align the facts with the concept of genetic immutability and, for some years now, there has been a softening of attitudes in this respect.

It is now broadly accepted that environmental influences such as thumb-sucking and artificial agencies such as orthopaedic therapy can, and do, have some permanent effect on the bones as well as the teeth and alveolus. Nevertheless, it is argued by some (Mills, 1978) that the proportion of change attributable to any orthopaedic movement is insignificant when related to overall treatment and growth changes. Moss and Salentijn (1969) have suggested that bone is not under direct genetic control but is guided by the growth of the tissues around it. He considers that these act as individual units or 'functioning matrixes.' They cite many instances where physiological and pathological forces cause reshaping of bone. Although there is much to support this hypothesis, there are also aspects of bone growth which display inherited features. The real problem is as Barnabei and Johnson (1978) say: 'functioning spaces are easy to talk about but hard to study.'

The Precision of Growth

We know very little about the control of cellular growth, but the dissimilarities between twins, and the frequent differences between the two sides of the body indicate the laxity of genetic control. For example, the anterior teeth of a crocodile need to meet exactly and yet there is some 5 feet of supporting tissue between the upper and lower contacts. It would not be surprising to find that some additional controlling factor had evolved to maintain the correct incisal relationship throughout the period of growth. There is support for this idea in figure 2, taken from Bjork (1947), which shows the distribution of overjets among conscripts. Chance alone produces a very characteristic pattern of deviation around a mean value (as shown by the dotted line). Bjork himself states 'the distribution is characterised by a greater number of variants around the mean value than occurs in a normal distribution and by an abnormal number of extreme deviations.' Presumably, there was an additional influence which caused most of the overjets to be correct but permitted some of them to be very wrong. Just such an influence could be exerted by a control system that occasionally went wrong.

Factors Influencing Plant Growth

Plants respond to certain physical and chemical stimulae such as light or water by the release of 'auxins' which redirect cellular growth. Botanists consider this growth separately from any genetic potential and call it a 'tropism.' For example (fig. 3), 4 similar bean seeds were planted, one the right way up, one upside down, and one on each side. On germination, they each responded to the tropic force of gravity and, as can be seen, the seedlings are of completely different form and size despite the fact that they could have been genetically identical. Subsequent growth is likely to restore the plants to a broadly similar form as normal genetic control takes over, but any continuation of abnormal tropic influences is certain to result in permanent deformation. Could these epigenetic or tropic responses be paralleled in the animal kingdom,

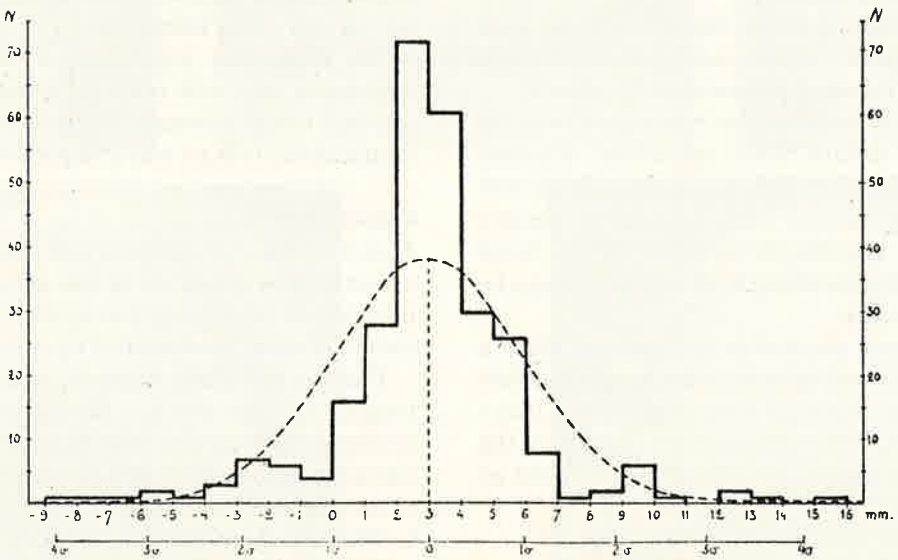

Fig. 2.—Taken from Bjork (1947). This shows the distribution and standard deviation of overjets among conscripts in the Swedish army. *(By permission of the author and publishers.)*

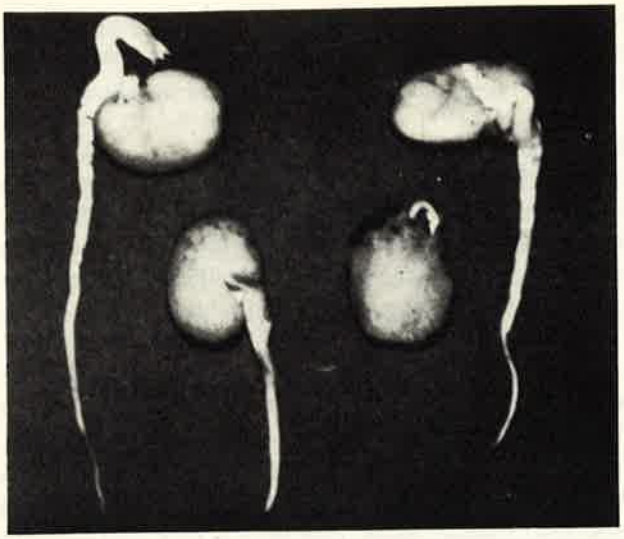

Fig. 3.—Four similar bean seeds planted, one the right way up, one upside down and one on each side.

having evolved to compensate for the inaccuracies that we know exist in the genetic control of growth?

Factors Influencing Growth in Animals

There has been an enormous amount of work done on animals in an effort to discover 'rules of facial growth,' and almost every element of the face and jaws has been found influential in one way or another. While it is sometimes difficult to distinguish between the response to experimental damage and normal physiological changes, the following features stand out clearly and constantly:

(a) The maxilla will move, tilt or widen in response to pressure from different directions (Harvold, 1968; McNamara, 1973; Petrovic *et al.*, 1975; Schulmacher, 1977).

(b) Lack of tongue contact against the maxilla restricts the development of the upper dental arch (Harvold, 1968; Salik and Schneider, 1977).

(c) The mandible responds to altered posture by extensive remodelling (Isaacson *et al.*, 1976), and can be induced to grow as much as one sixth longer than comparable controls (Petrovic, 1979).

(d) Young animals respond more quickly and more permanently to all these factors.

These research findings indicate that not only the teeth and alveolus, but also the jaws of young animals are able to respond to a wide range of pressures and postures.

Many of these experiments are destructive and even the control animals need to have 'sham' operations. We must therefore separate those factors we know can distort growth ('disturbance' factors) from the 'genetic' factors which guide normal growth. If, as seems likely, facial growth also has an adaptive element, this could perhaps be found as a 'tropic' factor.

While it is obviously impossible to draw too close a parallel between plants and animals, it does seem that they each present similar situations of force couplets and tissue responses. Presumably, some pressures act directly on the cells concerned while others are relayed by chemical or electrical communicators.

A Hypothesis

The following hypothesis might therefore be considered:

'The Tropic Premise'

'A delicate tropic mechanism overlays the genetic control of facial growth to allow adopted postures to guide the jaws and teeth into a satisfactory occlusion.'

It is suggested that the mandible will adapt itself to suit the posture most frequently adopted. In addition, the maxilla is able to reposition itself by slippage against its neighbouring bones in response to pressure from either the tongue or occlusal contacts. It is important to realise that if the tropic factor exists, it is epi-genetic, additional to, and quite separate from, genetic growth. Although all bones are known to respond to pressure, it assumes that the facial bones are specifically sensitive to, and perhaps even dependent upon, those gentle forces and postures that are generated during normal occlusion. As such, they are open to deformation by any incorrect postures that may be adopted.

We have already considered how unnatural postures associated with animal experiments and human disease can cause major skeletal deformities, and many clinicians might accept that good posture, with the tongue constantly against the palate with the teeth and lips in mutual contact from early childhood, could be related to good occlusion. Failing tropic stimulation from such postures, the facial skeleton may be unable to attain its full genetic potential. There is an underlying logic to a premise which would enable us to blame nature, not for her imperfect growth, but for leaving the system so delicate that minor environmental changes can upset the balance. Those malocclusions that so frequently accompany nasal obstruction, provide a convenient example. The mouth-breathing that results deprives the maxilla of the pressures that may well be one of its main tropic stimulants. Also, if a deep bite developed, this would restrict the forward posturing of the mandible which, according to the tropic premise, would result in associated under-development in the lower jaw to create a classic Angles Class II division 1 malocclusion.

Lorenze (1958) has shown patterns of muscular behaviour are inherited in just as precise a way as some more physical characteristics. So, presumably, tongue and jaw postures are also predetermined and, as Lorenze suggests 'will stubbornly resist learning.' According to the tropic premise, malocclusion would thus be inherited by way of soft tissue behaviour.

The increasing popularity of so-called 'functional' appliances may well result in greater importance being ascribed to the influence of the environment, for genetic immutability is in no way compatible with their action.

Clinical Relevance

Figure 4 illustrates the facial and mandibular changes that occurred in a girl who, at the age of 10, following the insertion of an orthodontic appliance, developed a 'sore throat swallow' as described by Atkinson (1966).

This, as the name suggests, is associated with a low tongue and jaw posture during swallowing with little synchronous muscular contraction and an atonic posture. The x-rays between the ages of 10 and 15 bear mute witness to the massive structural changes which followed. She was a doctor's daughter but, despite extensive tests, no pathology of any kind was disclosed. The fact that the horizontal ramus of the mandible was reduced in overall

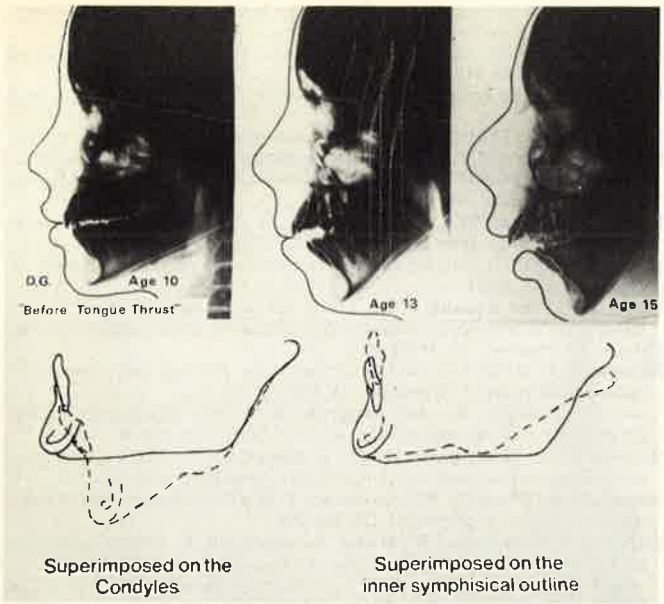

Fig. 4.—Three lateral radiographs taken at the ages of 10, 13 and 15, of a girl who developed a flacid swallow. Inset is a tracing of the mandible taken from the first and last radiographs (age 10·5 continuous line, age 14·11 intermittent line).

length by about one-third makes it highly improbable that this was part of a normal growth change and it would seem that it must be environmental. Support for this view may be gained by comparing the x-rays of this case with those in figure 1. Not only are the changes similar but, in each case, they are associated with reduced muscular tone. Also Harvold (1968) induced comparable bony changes in monkeys following an alteration in tongue posture alone.

Figure 5 shows how quickly the bony form can be recontoured during orthopaedic treatment. In this instance, expansion was used to open the suture and reposition the alveolus following which a modified activator was fitted to correct the saggital relationship. The whole treatment took less than 6 months.

Figure 6 shows the facial and dental changes that took place in a 12-year-old girl during a course of orthopaedic

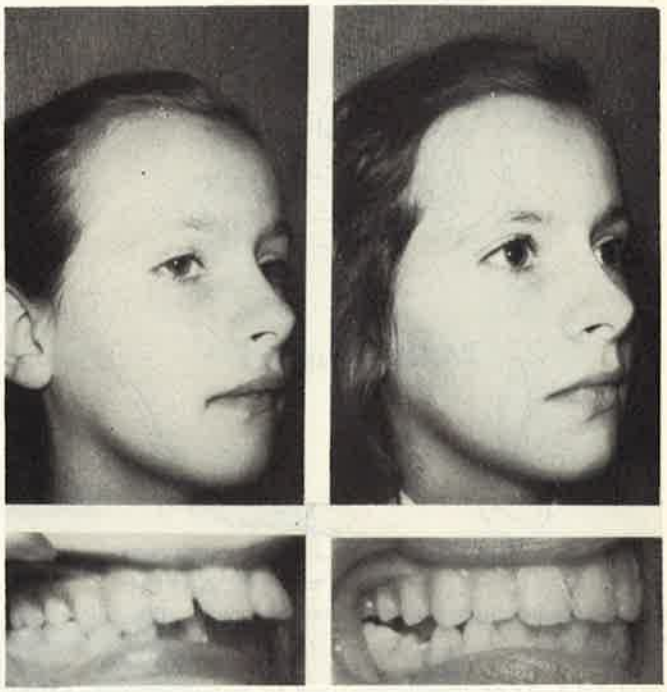

Fig. 6.—Photographs of a 12-year-old girl who underwent a course of orthopaedic treatment lasting 5 months 23 days.

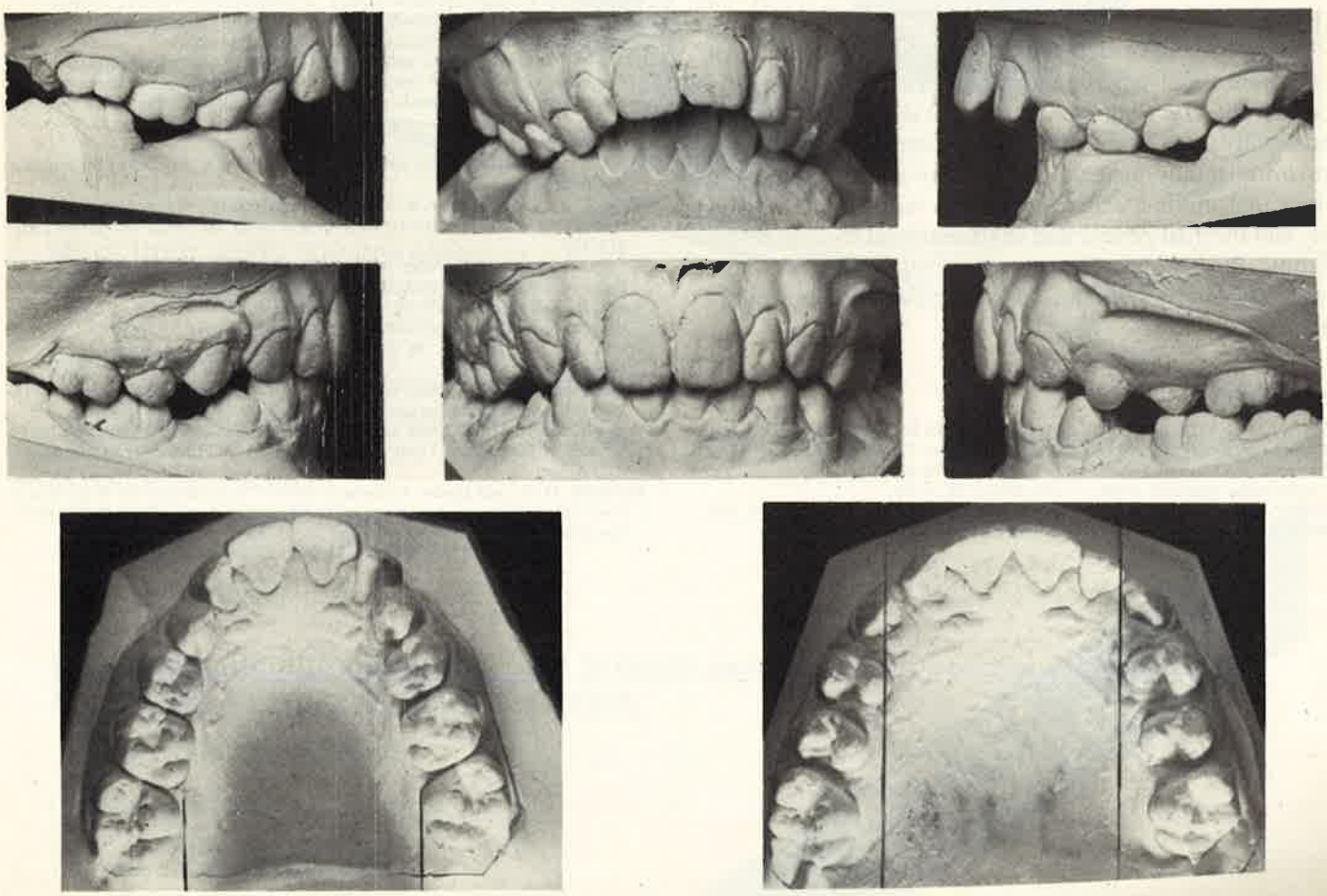

Fig. 5.—Models of a girl aged 10 who received less than 6 months' orthopaedic treatment.

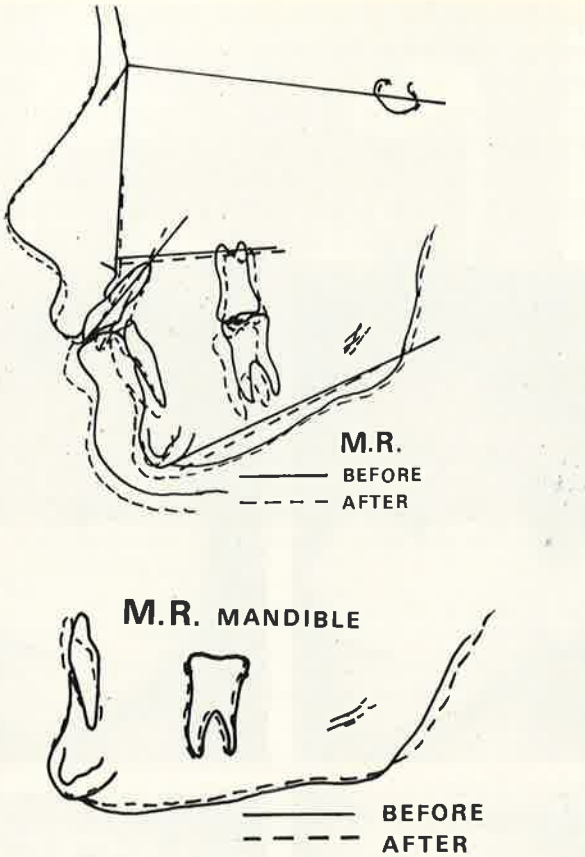

Fig. 7.—Tracings from the profile radiographs of M.R. before and after treatment.

treatment. Appliances were used to encourage growth in both upper and lower jaws during the period of 5 months 23 days. Figure 7 shows the bony and dental movements.

Conclusions

Philosophers have debated the relative importance of inheritance and environment since the issues were first conceived. Our interest is, of course, centred on the possibility that some of the classic malocclusions might be environmentally created. While there is no real evidence to show that this is so, there is much to suggest that it could be, and the dual genetic and environmental concept of the Tropic Premise provides a convenient bridge for those who have been standing on either side of this age-old divide.

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