Factors Influencing Mandibular Growth

J. R. C. Mew

A clinically oriented presentation on growth mechanisms and the influence of posture and cellular communication on mandibular growth, with clinical illustrations.

KEY WORDS: • GROWTH • HEREDITY • MALOCCLUSION • • MANDIBLE • TROPIC PREMISE •

While it is generally accepted that the mandible grows to a genetically predetermined size and shape, it also appears that a range of physiologic, pathologic, and mechanical factors can influence this growth. Opinion is divided, however, as to the significance of this environmental overlay.

Some authorities consider that any influence is restricted to the alveolus (MILLS 1978), while others believe that it can at times be more extensive (PETROVIC 1975, MCNAMARA 1980, AND PANCHERZ 1982).

This paper examines some of the findings in a field where the research material is growing almost faster than one man can read. An hypothesis is presented concerning the clinical variations in mandibular growth, and an extension of this hypothesis suggests that such growth could be artificially increased or decreased. Clinical examples are used to illustrate its application in various situations.

Embryogenesis

Human children, like most animals and plants, develop from a single fertilized cell. They reach their full complement of many millions of separate and specialized cells through a process of repeated division. This phenomenon is undoubtedly one of nature's greatest achievements, but the details are still among her best kept secrets.

Author Address:

J. R. C. Mew
The Purley School of Orthodontics
21 Foxley Lane
Purley, Surrey CR2 3EH
ENGLAND

Recent thinking assumes that developing systems are controlled by electrical, chemical or hormonal stimulae, but stops short of explaining how those signals can be interpreted differentially by apparently similar receptor cells. Such agents must presumably vary quantitatively rather than qualitatively, and it is hard to understand how they can elicit different responses from neighboring cells.

For instance, during early embryogenesis before the central control system has differentiated, cells appear able to pass information among each other before dividing to undertake their various specialized activities (GRANT 1978). Even during the later stages of growth, cells still possess considerable autonomy, relying on positional and other information from their neighbors (WOLPERT 1978).

It seems that the expression of the genetic pattern remains very much at the cellular level (GURDON 1966). One has only to follow the growth of a complex bone such as the mandible to recognize the infinite variability it displays in response to changing situations. This enables constancy of form and spatial relationships to be maintained during growth, even with the rotation of the inner mass of this bone that we know occurs (ISAACSON AT AL. 1976).

Remote Control

It seems unlikely that the complex resorption and deposition that must be involved in mandibular growth could be controlled from beyond the mandible, either centrally or peripherally. Every cell in the periosteum would require subtly different information, and even if an outside center of control existed, how could it distribute such instructions or receive feedback on progress achieved? More inexplicably, from where would it receive its own guidance?

Local Control

If remote control systems appear to be illogical, how else can the phenomena of facial growth be understood? For instance, tooth eruption could be understood in terms of:

- Physical parameters, such as blood pressure or elongation of the root
- Chemical agents, such as blood-borne hormones or morphogens
- Electrochemical agents, with the message conducted via the nerve supply

While all of those factors are probably involved, it is obvious that they lack the necessary finesse to supervise a total phenomenon such as tooth eruption, where every cell within a wide area appears to participate. Each cell is apparently provided with information about the activity of others and about the ultimate objective. The death of cells in the path of eruption and deposition of bone elsewhere is part of an overall, but nevertheless variably responsive, pattern rather than a series of local reactions.

It seems obvious that the cells themselves are reacting one to another.

If an erupting tooth goes off course, the cells of its follicle appear to enforce the message to bone cells in an area which would not in normal circumstances have been anywhere near a tooth, yet they obediently die or form periodontal membrane as the case may be. If the errant tooth comes into contact with some adjacent structure which has different positional information, then cell activity slows down, possibly because of conflicting instructions. There may then be destructive changes such as intermittent deposition and resorption of calcified tissues, as though a confrontation had taken place between the two groups of cells.

Successfully transplanted teeth can develop a lamina dura that appears iden-

tical to that found around normal teeth (Fig. 1) (GARDINER 1979). What agency could promote such a growth in normal bone cells?

If a mandibular condyle is completely removed, it may regenerate to near its original form, with peripherally aligned fibrocartilage cells like those seen in a normal condyle (ADLER 1981). Situations like these also suggest that control rests with cells in the immediate vicinity.

Genetic Variations

If, as the evidence suggests, the control of growth rests largely with individual cells which contain identical genetic information, then how can we explain the variations which occur between twins, and even between the right and left sides of the body? We know from research on identical twins (KRAUS ET AL. 1959) that their bones can vary both in shape and position and, in certain pathologies, these variations can be considerable.

Obviously, other factors must influence the interpretation of genetic information. If logic refutes the existence of remote control centers, it must be assumed that any such variations are contrived by the local cells themselves. It seems possible that they have the facility of accommodating to situations such as local damage or pressure so that a functional unit is produced whenever possible.

— Control of Facial Growth —

In their search for the "rules" that govern facial growth, researchers have tended to search for cause-and-effect relationships on a macroscopic scale. Almost every element of the face and jaws has been found to be involved in some way or another.

Muscle attachments have been moved, nerves cut, cartilages destroyed, tongues sectioned, teeth removed, tissues distorted, and every conceivable force applied in almost every direction. While this has been of help in identifying some cause-and-effect relationships, it does little to elucidate the true control of organogenesis.

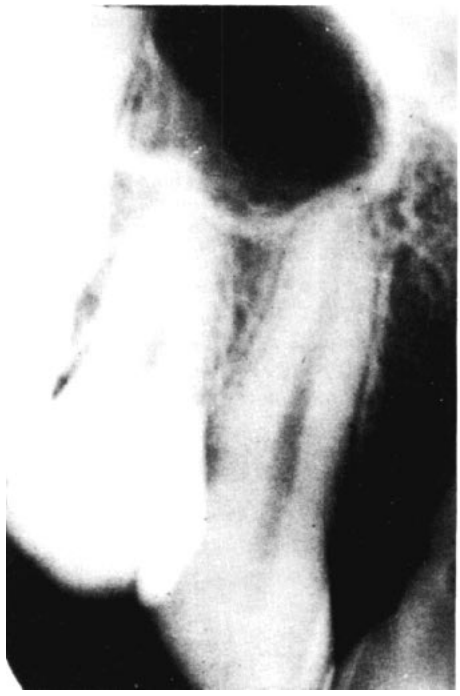


Fig. 1
A transplanted cuspid which has generated a "lamina dura."
G. T. Gardiner

If the genetic formulae are stored within the cells themselves, perhaps we should not be looking only at nerve pathways or hormones, but at the contacts between the individual cells as they spread across a particular field.

Tropic Premise

We know that the face shows more genetic variation than any other part of the body (SUSANNE 1975, HARRIS AND SMITH 1980), and the Author has previously put

forward the *tropic premise* (trōpic — a turning or change of direction) to explain this (MEW 1981).

The tropic premise suggests that the genetic latitude of facial growth has evolved to permit the skeleton in this area to respond to functional demands, including occlusion. "A delicate tropic mechanism overlays the genetic control of facial growth to guide the teeth and jaws into a satisfactory occlusion. The mandible grows to suite the position in which it is normally postured, and both jaws tilt to balance the anterior and posterior contact forces."

Other theories have been put forward by Wolfe (WEINMANN AND SICHER 1955, and Moss 1962). The latter suggests that bone growth is influenced and perhaps controlled by neighboring tissues and their "Functional Matrices." However, this still begs the existence of communicating systems to relay functional information, and provides no answer for the regeneration of a condyle.

If, as seems likely from the earlier discussion, the genetic interpretation of growth lies within the cells themselves, then a different and perhaps more simple system of control (such as the tropic premise) would suffice, relying on no more than information passed from cell to cell.

— Clinical Application of the Hypothesis —

If the *tropic premise* is a valid hypothesis, aberrations in mandibular growth could be the result not of inherited bony form but of changes in posture. It is rare in medicine for an hypothesis to be "proven" — lack of disproof is much more likely. In contradistinction, "belief" is usually based on clinical reality. The following cases illustrate various relevant points.

Case L. M. (Figs. 2-4)

From a very young age, this child postured her jaw forward and to the right. Figure 2 shows her at ages 2, 4, 6, and 8. When she was referred for an orthodontic opinion at the age of 9, there was a full crossbite on the right side, with a negative overbite anteriorly (Fig. 3). The lower midline was deviated 2mm to the right, with a further shift of 2mm on closure. She was treated with some "semi-rapid expansion" (MEW 1977), and the deviation was then corrected with a cast silver splint and flange.

Within two months, the mandible was occluding centrally and she was given a course of myotherapy to correct her posturing habit. She was also provided with an upper appliance with a lower labial bow referred to as a *Purley wire*, which contacts the mucosa below the lower incisors whenever the mandible is moved forward or to one side.

At 15 months out of retention, she has ceased posturing her mandible, and shows no sign of recurrence of the deviant mandibular growth (Figs. 3 and 4).

Case Discussion

PERSSON (1973) reports that mandibular deviation tends to be inherited. However, prognathism itself is only sporadically inherited, and transmission over several generations is exceptional (SCHULZ 1979). The *tropic premise* suggests that in this case the genetic factor, if there was one, was most likely the posturing.

Fig. 2 L. M.

Childhood photographs at the ages of 2, 4, 6 and 7, showing the consistent tendency to posture her jaw forward

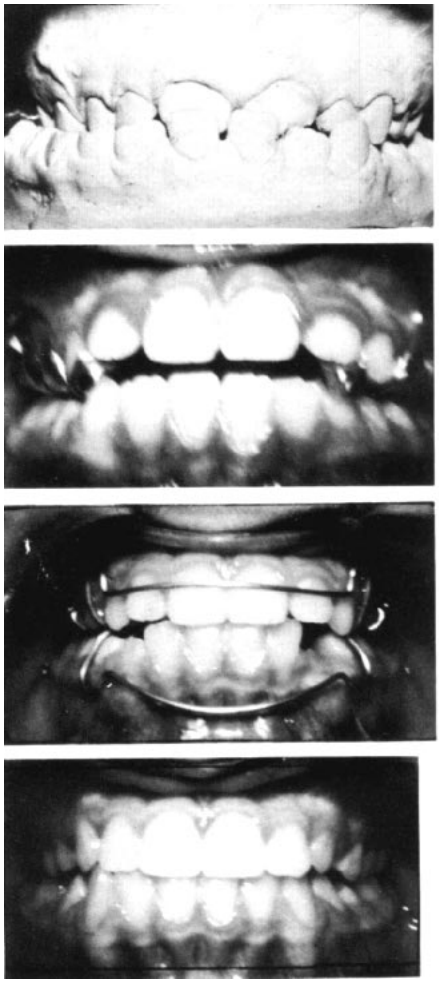


Fig. 3 L. M.

Dental casts showing right crossbite and negative overjet at the age of 9

Cast silver splint to correct deviation

Two months later, mandible occluding in retruded centric position with a Purley wire to discourage posturing

Age 13½, 15 months out of retention

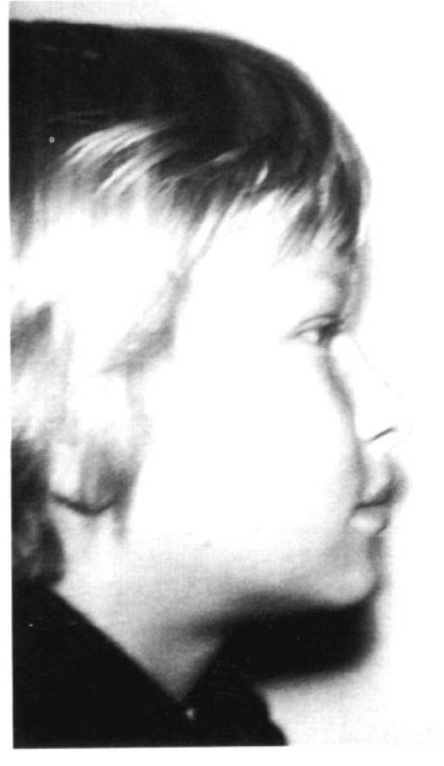


Fig. 4 L. M.

Age 13½, 15 months out of retention

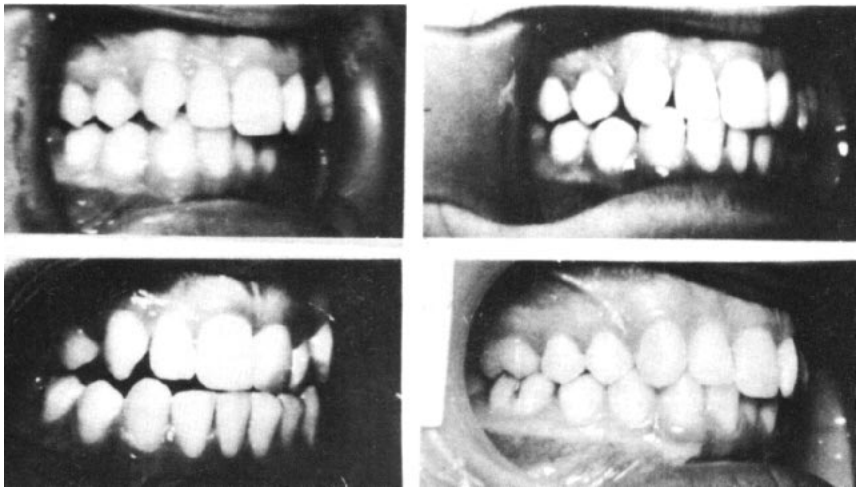


Fig. 5 A. H.

Age 13, before treatment	Edge-to-edge bite apparently resulting from continued use of activator
Overjet nearly corrected with an activator	Spontaneous resolution 15 months out of retention

Case A. H. (Fig. 5)

This 13yr-old boy was referred with a mild Class II[1] malocclusion and slight crowding. He was treated with semirapid expansion followed by a protrusive activator to encourage mandibular growth. This had corrected the postnormality after six months. Unfortunately, he then missed two appointments, and when seen four months later, had developed into an edge-to-edge relationship.

Case Discussion

It would be most unusual for a Class I case of this type to change spontaneously into the relationship seen in the third photograph in Fig. 5, and it seems likely that the forward posture induced by the activator precipitated the prognathism. The mandible could not be manipulated distally at that time, although the overcorrection had corrected spontaneously two years later.

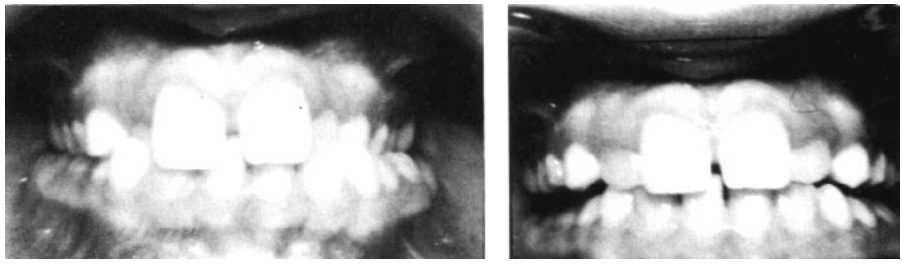


Fig. 6 P. O.

left — Age 10½, before treatment; 6mm midline deviation

right — Three months later, with the right buccal segments in full crossbite and the left side nearly so; midline deviation is 4mm.

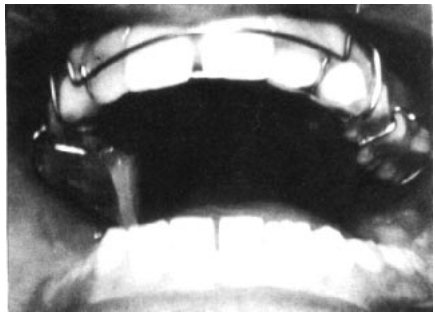


Fig. 7 P.O.
Flange fitted to overcorrect the midline

Case P. O. (Figs. 6-8)

This 10yr-old boy had a Class III malocclusion with the lower midline 6mm to the left, and a buccal crossbite on that side. Treatment was with semirapid expansion to open up the upper arch, which resulted in a full buccal crossbite on the right hand side. An acrylic flange was then provided to posture the mandible to the right (Fig. 7). Within 4 weeks, the center line had adjusted 2.5mm in retruded centric (Fig. 8).

Case Discussion

When using one of the flanges illustrated in Fig. 7, it is important that it be high enough to prevent the lower teeth from occluding under it at rest. A "lopsided" activator will rarely succeed on its own.

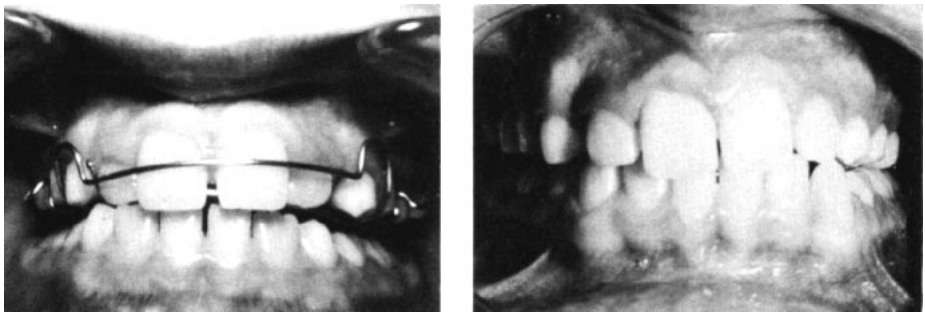


Fig. 8 P.O.

left — Teeth closed, showing how the flange overcorrects the midline

right — The retruded condylar position four weeks after the flange had been placed

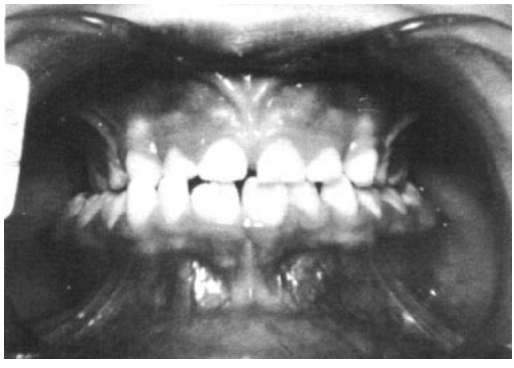


Fig. 9 S. B.

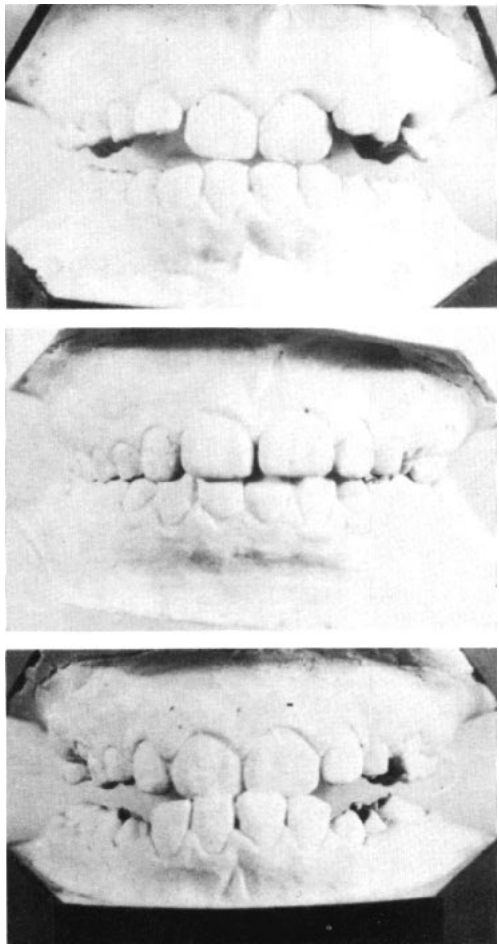
Age 6½, before treatment

Case S. B. (Figs. 9 and 10)

This girl was first seen at the age of 6½, with a Class III tendency and a right crossbite (Fig. 9). As is often the case, a request for an early photograph brought evidence that she had been posturing from early childhood (Fig. 10).

Fig. 10 S. B.

At 1 year of age, showing mandibular posturing



Before first treatment

Four months later, with correction

Five months after treatment, showing redevelopment of the Class III relationship

Fig. 11 S. M. age 11

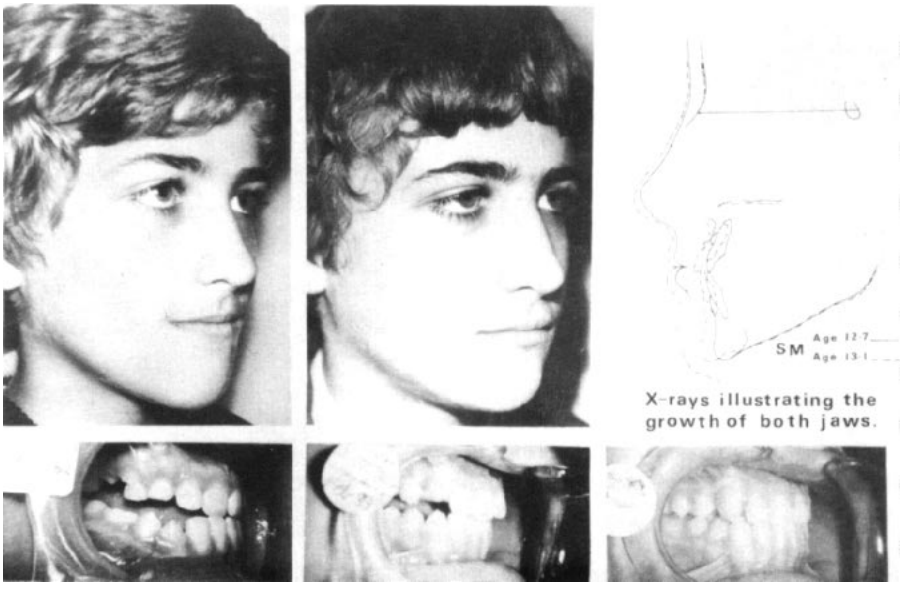
Case S. M. (Figs 11-13)

This 11yr-old boy had a prior history of an anterior crossbite (Fig. 11). A simple appliance had previously been provided to move the upper incisors forward to the position shown in the center photograph within 4 months. On referral 5 months later, he had again developed a negative overjet with the bilateral open bite shown in the lower photo of Fig. 11.

Experience suggests that this situation would be likely to worsen, especially in a boy approaching puberty. As in the previous cases, early photographs showed a tendency to posture the mandible forward (Fig. 12). He was retreated with expansion of the maxilla, chin traction, and a Purley wire to discourage posturing. Changes after five months of additional treatment are seen in Fig. 13.

Fig. 12 S. M.

Early photo, showing protrusive posturing of the mandible



Left and center, at age 12, before and after a second course of treatment lasting five months

Age 14, two years after the second course of treatment

Fig. 13 S. M.

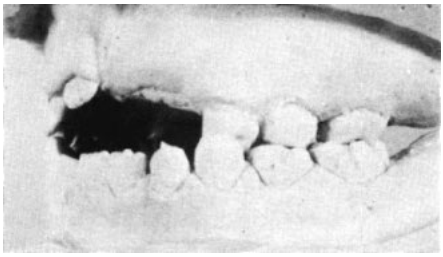


Fig. 14 N. B.

Age 7, with a buccal crossbite and Class III relationship

Fig. 15 N. B.

Age 1 and age 5, showing an early tendency to mandibular protrusive posturing

Case N. B. (Figs. 14 and 15)

This 7yr-old girl was referred with a buccal crossbite and Class III incisor relationship (Fig. 14). A request for early photographs showed that she had also been posturing her mandible from infancy (Fig. 15).

Discussion of Cases S.B., S.M., and N.B. (Figs. 9-15)

Although the *ropic premise* suggests that Class III malocclusions can be caused by postural habits, this Author had not heard this suggestion made before. Patients and their parents are frequently unaware of the posturing, yet the Purley wire will sometimes create an ulcer at a position which indicates long-term downward and forward posturing of several millimeters (Fig. 16).

Patients respond well to counselling against this, and most are able to stop the habit. Once it is stopped, the rate of mandibular growth appears to slow down, and it does not seem to accelerate unless posturing is resumed. The cure sounds simplistic in view of the predictions given for these cases, but it is effective and in accordance with the *tropic premise.*

The hypothesis suggests that the Class III relationship us likely to recur is forward posturing is resumed, so even apparently successful cases should be kept under close supervision for an extended period.

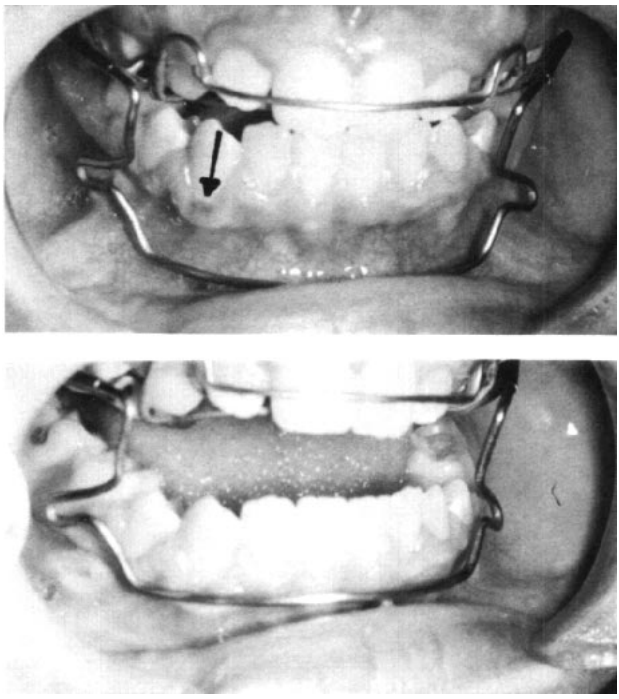


Fig. 16

A patient wearing a Purley wire with the mandible closed (above) and in the protrusive posture (below). Note the position of the ulcer (arrow) in the upper photograph.

— General Discussion —

Several authors have commented on the variations of mandibular form that may be associated with unusual posture.

KREIBORG ET AL. (1978) described a girl with congenital muscular dystrophy (Fig. 17). Although this condition has no direct influence on bone tissue, the mandible of this girl was grossly distorted.

SCHULHOF (1978) described a boy with nasal stenosis and open-mouth posture, with mandibular distortion and antegonial notching. Similar notching was described by ATTIA AND RUTHSTROM (1978), and they report that this extreme notching disappeared after the anterior open bite had been reduced forcibly with a chin cap.

This Author described a remarkable case of a girl with normal form and posture who subsequently developed a tooth-apart swallow with an open mouth posture (Figs. 18 and 19) (MEW 1981) . Between the ages of 10 and 15, the mandibular angle increased from 36° to 63°, and the horizontal ramus, judged from the radiographs, actually shrunk from 63mm to 51mm.

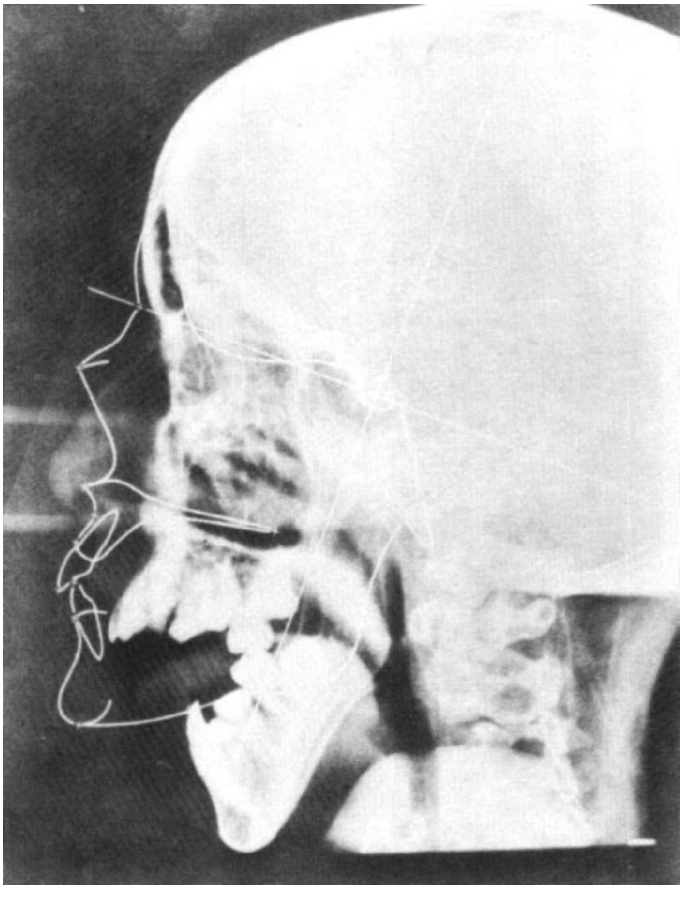


Fig. 17

A 12yr-old girl with muscular dystrophy. The white outline indicates the average outline for her age. *By permission of Kreiborg et al. and The American Journal of Orthodontics.*

Fig. 20 shows a boy who developed complete nasal stenosis after the age of ten. This was corrected surgically, but he remained a habitual mouth breather. The changes in mandibular and facial form, and the sagittal fall-back of the face following the blockage, are similar to the conditions seen in Figs. 18 and 19.

TOMER AND HARVOLD (1982) observed that when monkeys were provided with bite-raising appliances " . . . the angle between sella-nasion and the posterior border of the ramus remained stable throughout the experiment in all the animals." Because the mandible remained open, the remodeling resorption at the posterior border produced an change in the gonial angle, which must have commenced as soon as the mandible was held open. What agency other than local cel-

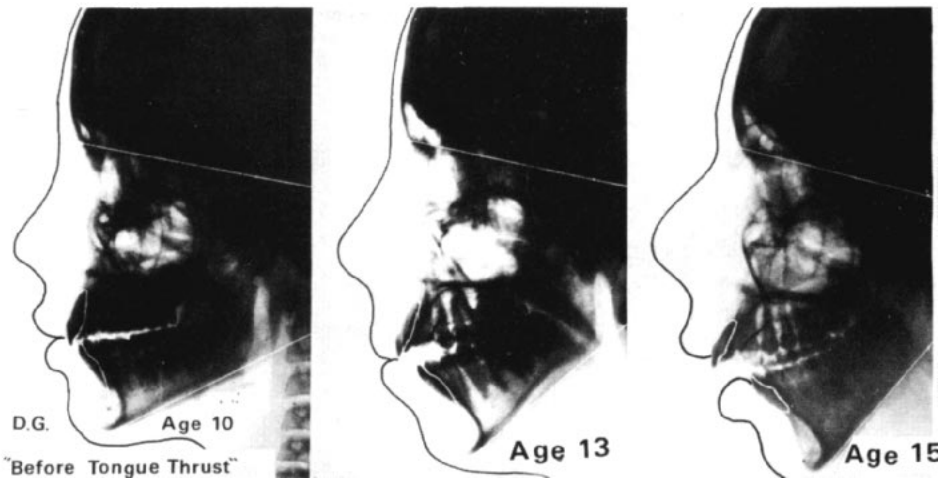
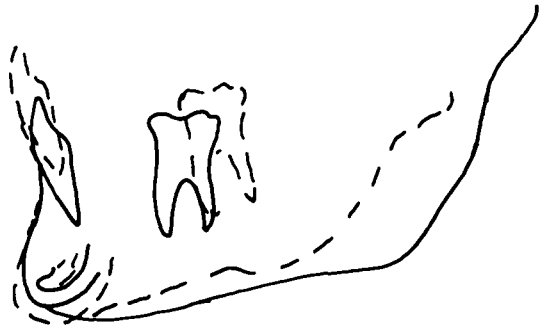


Fig. 18 A 10yr-old girl (left) who subsequently developed a forward tongue posture. Note changes at age 13 (center) and at age 15 (right).

Fig. 19

The outline of the mandible of the patient shown in Fig. 19 at age 10yr 5mo (solid line) and 14yr 11mo (broken line)

Superimposition is on inner cortical structures



lular response could have achieved this change?

Tomer and Harvold postulate that " . . . the masticatory muscles and the ramus area of the mandible constitute a functional system relatively independent of the facial and suprahyoid muscles acting on the anterior part of the mandible." Björk and Skieller (1983) use the terms "Matrix" and "Intra matrix" to differentiate the two types of mandibular growth.

The idea that the muscles are the principal molding influence is supported by many research workers and clinicians, and Petrovic (1975) put forward an ingenious cybernetic model to explain this. There are others, however, who consider posture more significant (Proffit and Mason 1975).

The *tropic premise* suggests that both muscle posture and muscle force are involved, but this still does not explain

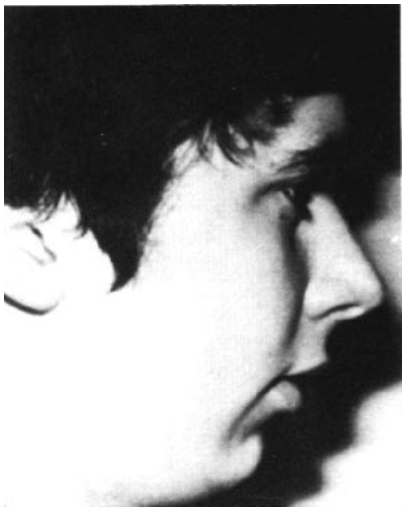


Fig. 20 A 10yr-old boy who developed nasal stenosis, with in the facial changes shown below at age 17.

Left, age 10

Below, age 17

the cybernetics. It was suggested earlier that the control of growth is likely to be embodied within the cells themselves, through their ability to respond to positional information from cells around them.

If the position of the mandible is changed, the peripheral cells receive new positional information. It seems possible that in this situation they might recontour the bone toward its original position in relation to nearby structures. ISAACSON ET AL. (1976) came to this conclusion after reorientating some of the implant cases that had originally been documented by BJÖRK AND SKIELLER in 1972. They concluded that " . . . this rotation was not obvious in the past since it is masked by

external surface remodeling that tends to restore the relationship of the jaws to their original morphology."

This may explain the changes in mandibular form such as occurred in the girl whose jaw shortened from 63mm to 51mm. When the jaw is dropped, the chin maintains its relationship with the tissues immediately around it, but the vertical ramus slips back between the enveloping soft tissues. Thus, positional information received by the cells of the horizontal ramus remains fairly constant, while those of the vertical ramus respond to their changed relationships with the surrounding tissues so that, as TOMER AND HARVOLD (1982) observed, this part of the bone is recontoured.

This then leaves the horizontal ramus sloping downward with the characteristic antegonial notch where the two portions of the mandible meet. Any change may then be explained not by direct muscle force, but by individual cells in a frustrated effort to restore mandibular form to the genetic plan.

— Conclusions —

It appears that adopted postures can influence the growth of the mandible in all three planes of space independent of genetic control. Changes in posture elicited by an appliance such as an activator or a Purley wire, or by training, may perhaps accelerate or retard growth in any of these planes.

It is suggested that such changes are effected by the individual cells of the mandible acting in response to positional information that they receive from the tissues around them.

This hypothesis takes us very close to the concepts of EDWARD ANGLE (1907) who, in this Author's view, had a better understanding of the causes of malocclusion than anyone before or since. It would seem that he was lacking not in understanding but in armamentarium.

— Summary —

The *tropic premise* is considered in relation to the concepts of Wolf and Moss, and attention is drawn to some of the factors associated with abnormal mandibular growth. It is suggested that positional information from individual cells may have a greater influence than has been allowed for.

Clinical cases where mandibular posture appears to have influenced growth and effected changes in mandibular form are described and illustrated. Mandibular posture is regarded as a possibly significant factor in creating some Class III and other malocclusions.

REFERENCES

Alder, E. A. 1981. Regenerated human mandibular condyle. *Aust. Dent. J.* 26:216-217.

Angle, E. 1907. *Treatment of Malocclusion of the Teeth.* S. S. White, Philadelphia.

Attia, Y. and Ruthstrom, L. 1978. *Anterior malocclusion and tongue thrusting* Euro.Orthod. Soc. The Hague.

Björk, A. and Skieller, V. 1983. Normal and abnormal growth of the mandible. *Eur. J. Orthod.* 5:1-46.

Engel, G., Dumerell, J. M., McAlpine, J. and Walters, R. 1980. Treatment of deep bite cases. *Am. J. Orthod.* 77:1-13.

Gardiner, G. T. 1979. The autogenous transplantation of maxillary canine teeth; a review of 100 consecutive cases. *Brit. Dent. J.* 146:382-385.

Grant, P. 1978. *Biology of Developing Systems* Holt, Rinehart and Winston, New York.

Gurdon, J. B. 1966. Fertile intestinal nuclei *Nature.* 210:1240-1241.

Harris, E. G. and Smith, R. J. 1980. A study of occlusion and arch widths in families. *Am. J. Orthod.* 78:155-163.

Isaacson, R. J., Worms, F. W. and Spiedel, T. M. 1976. Measurement of tooth movement. *Am. J. Orthod.* 70:290-303.

Kraus, B. S., Wise, W. J. and Frei, R. M. 1959. Heredity and the cranio-facial complex. *Am. J. Orthod.* 45:172-217.

Kreiborg, S., Leth-Jensen, B., Moller, E. and Björk, A. 1978. Craniofacial growth in a case of congenital muscular dystrophy. *Am. J. Orthod.* 74:207-215.

McNamara, J. A. 1980. Functional determinants of cranio-facial size and shape. *Euro. J. Orthod.* 2:132-159.

Mew, J. R. C.

1977. Semi-rapid maxillary expansion. *Brit. Dent. J.* 143:301-306.

1981. The aetiology of malocclusion. *Brit. Dent. J.* 151:296-302.

Mills, J. R. E. 1978. The effect of orthodontic treatment on the skeletal pattern. *Brit. J. Orthod.* 5:133-143.

Moss, M. L. 1962. The functional matrix. *Vistas in Orthodontics.* Lee and Febiger, Philadelphia.

Pancherz, H. 1982. The mechanisation of Class II correction in Herbst appliance treatment. *Am. J. Orthod.* 82:104-113.

Persson, M. 1973. Mandibular asymmetry of hereditary origin. *Am. J. Orthod.* 63:1-11.

Petrovic, A. G., Stutzmann, J. J. and Oudet, C. L. 1975. Control processes in the postnatal growth of the condylar cartilage of the mandible. *Determinants of Mandibular Growth* Ed. J. A. McNamara, Center for Human Growth and Development, Ann Arbor, Michigan. p. 147.

Proffit, W. R. and Mason, R. M. 1975. Myofunctional therapy for tongue thrusting: background and recommendations. *J.A.D.A.* 90:403-411.

Schulhof, R. J. 1978. Consideration of airway in orthodontics. *J. Clin. Orthod. 12:440-444.*

Schulz, C. 1979. Zur atiologie de progenie. Fortschr. Kieferorthopadie 40:87-104.

Susanne, C. 1975. Genetic and environmental influences on morphological characteristics. *Ann. Hum. Biol.* 2:279-287.

Tomer, B. S. and Harvold, E. P. 1982. Primate experiments on mandibular growth direction. *Am. J. Orthod.* 82:114-119.

Wienmann, J., Pound, J. and Sicher, H. 1955. *Bone and Bones.* Henry Kimpton, London. p. 134.

Wolpert, L. 1978. The development of the pattern of growth. *Postgraduate Medical Journal 54* Suppl. 1, p. 15-21.