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# Hemimandibular Hyperplasia – Hemimandibular Elongation

*Hugo L. Obwegeser, Miro S. Makek*

Maxillo-Facial Surgery Clinic (Head: H. L. Obwegeser, M.D., D.M.D.) and Institute of Pathology (Head: Chr. Hedinger, M.D., and J. R. Rüttner, M.D.) University Hospital Zürich, Switzerland

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**Summary**

Clinical and radiographic experience as well as histological findings leave no doubt that the term "condylar hyperplasia" refers only to hyperplasia of the condyle alone and should therefore not be used to mean the two hemimandibular anomalies as is the case in the literature today. There are two basically different malformations of one side of the mandible which we call hemimandibular hyperplasia and hemimandibular elongation respectively. We are convinced that there exist pure and mixed forms of both growth anomalies because we have observed several such clinical cases. The stimulus for the abnormal growth either lies within the fibrocartilaginous layer or is produced by it. Different histological patterns within the condylar growth zone were seen in the two anomalies. The pathophysiological bases of the abnormal growth are discussed. They seem to contribute to the understanding of the normal and abnormal mandibular growth and consequently also of many of the mandibular anomalies. The explanations are demonstrated by the illustrations of some cases.

**Key-Words**

Mandibular anomalies – Mandibular growth – Condylar hyperplasia – Hemimandibular hyperplasia – Hemimandibular elongation – Acromegalism

## Introduction

According to the literature (*Rushton*, 1951; *Broadway*, 1958; *Hovell*, 1963), the term "condylar hyperplasia" describes two clinically completely different form anomalies of the mandible. Both anomalies obviously derive their names from the resulting distinctive enlargement of the condyle. In our opinion, it is unjustifiable to label these two separate anomalies using the same term, because their pure forms occur as very typical but completely different anomalies of one whole side of the mandible; they also entail different therapeutic concepts. It is not simply a matter of distinguishing between these two anomalies. They must also be differentiated from other pathological processes. We reported our opinion on these abnormal forms of the mandible in the 7th congress of the EAMFS 1984 in Paris (*Obwegeser* and *Makek* 1984).
A detailed report follows on these two entirely different anomalies of the mandible, as well as their mixed forms.

## Basic Types

On the one hand, both anomalies occur very typically and consistently with the same signs, again and again. Thus, we assert that basic classical types of these anomalies exist. On the other hand, our hypothesis for their pathogenesis makes the existence of mixed forms inevitable. We have indeed observed such forms. According to our hypothesis, it is easily comprehensible that mixed forms with varying degrees of combinations should occur. We believe that our patient collection indeed contains such forms.

*A. Hemimandibular hyperplasia (H. H.)*
Hemimandibular hyperplasia (abbreviated H. H.) always exhibits the same typical presentation which, however, can vary in the degree of its development. This depends on the one hand upon the age of the patient during the initial stage of the abnormal growth, and on the other hand upon the degree of the abnormal growth.
Furthermore, it depends upon the duration of the abnormal growth of the mandible. Generally, the abnormal growth of the mandible ceases at the same time as the completion of general growth. It can also keep on growing beyond this point to produce grotesque pictures.

1. Typical hemimandibular hyperplasia (H. H.)
H. H. is characterized by a three-dimensional enlargement of one side of the mandible, i.e.: the enlargement of the condyle, the condylar neck and the ascending and horizontal rami. The anomaly terminates exactly at the symphysis of the affected side. That is why we call this anomaly which affects one half of the mandible, "hemimandibular hyperplasia".
Clinically and radiographically, the following details are discernible: In pronounced cases the striking increase in height of the affected side gives the face a rotated appearance (Fig. 1 a). The unilateral asymmetric increase in height of the face, though not well-marked, gives rise to a sloping rima oris. The mouth can be opened without restriction. The sloping rima oris and the facial asymmetry still remain discernible, even if they are less marked. Viewing the face from below discloses the cause of the facial asymmetry: a very striking unilateral downward projection of the ascending ramus and the angle in particular (Fig. 1 b). Since the anomaly commences before puberty, it is understandable why the maxilla follows the downwards growth of the mandible. The teeth on the affected side thereby generally remain in occlusion on a lower level than the teeth on the normal side. This results in a tilted occlusal plane. Of course, the maxilla might not be able to follow the growth of the mandible when its downwards growth proceeds at a very rapid rate. In this case, an open bite becomes evident on the affected side. The elongation of the ascending ramus and the caudal displacement of its horizontal ramus, which take place solely unilaterally with a clear demarcation at the symphysis, give rise to a sharp kink of the mandible at the

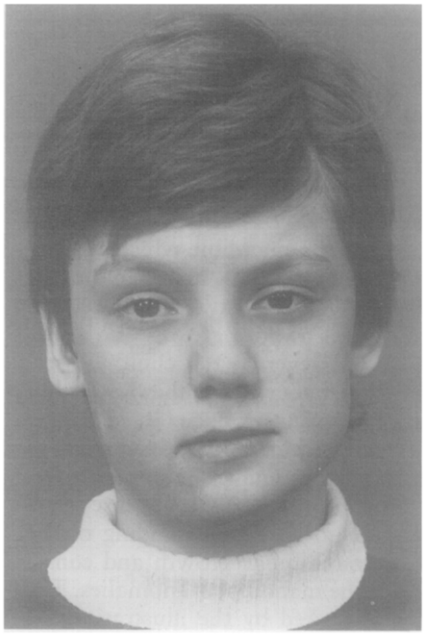

**Fig. 1 a**

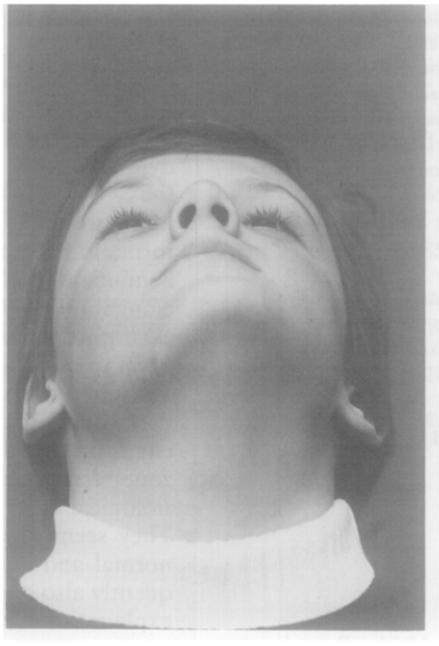

**Fig. 1 b**

**Figs. 1 a + b** demonstrate the increase in vertical height of the middle and lower facial thirds on the affected side.

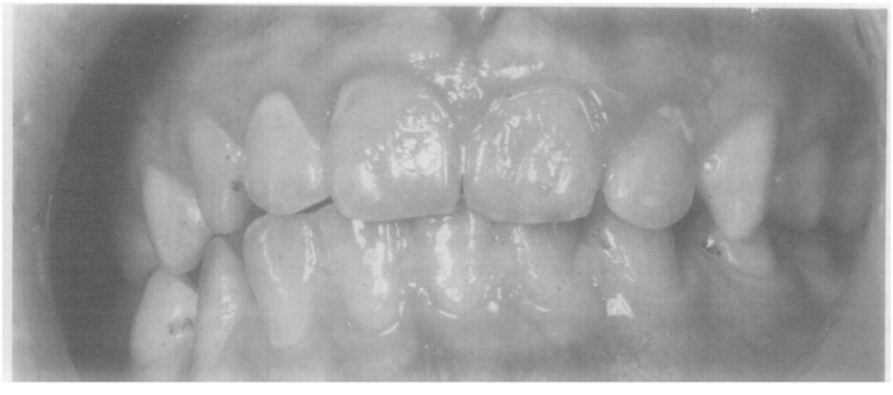

**Fig. 1 c** shows the rotated and tilted occlusion, including the anterior teeth and the displacement of the midline of the lower dental arch towards the affected side.

symphysis. This is also clearly disclosed by viewing the mandible from below (Fig. 1 b). As a result there is tilting of the anterior teeth to the affected side. The caudal displacement of the mandible and the growth thrust of the one side obviously bring about a contralateral relative reduction in height of the horizontal ramus and lingual tilting of the lower dental arch on that side. The upper arch can obviously maintain the occlusion by growing downwards more on the affected than the opposite side. The upper arch on the unaffected side can grow downwards bypassing the lingually inclined lower arch and ending up in buccal non-occlusion. This gives rise to a twisted occlusal plane, which is a reflection of the similarly distorted facial appearance (Fig. 1 c).

The orthopantomogram in Fig. 1 d reveals typical pathognomonic findings: the ascending ramus is clearly elongated. This vertical elongation is expressed by the enlargement of the condyle and the elongation and thickening of the condylar neck. The angle is characteristically rounded off and the mandibular lower border is bowed downwards and positioned at a lower level than that on the other side. The essential increase in height of the horizontal ramus is expressed by the increased distance between the tooth roots and the mandibular canal. The latter is displaced down towards the lower border of the mandible. In that region it seems that only the alveolar process is increased. This is always manifested in the area of the premolars and molars. The horizontal ramus of the contralateral side shows a relatively reduced vertical height, also in the area of the molars and premolars. The enlargement of the affected side of the mandible which terminates exactly at the symphysis, can be clearly seen, especially in the orthopantomogram. It can also not be missed on the postero-anterior view of the mandible. The elongated ascending ramus and the coarse and thickened trabecular structure of the affected side are particularly striking in that view (Fig. 1 e). However, that difference cannot be missed in all x-ray-projections when compared with the normal side (Figs. 1 d–g). The elongation of the ascending ramus and the increased height of the horizontal ramus together with the consequent compensatory downward growth of the maxilla with its sinus constitute the essential skeletal basis of the vertical height of the

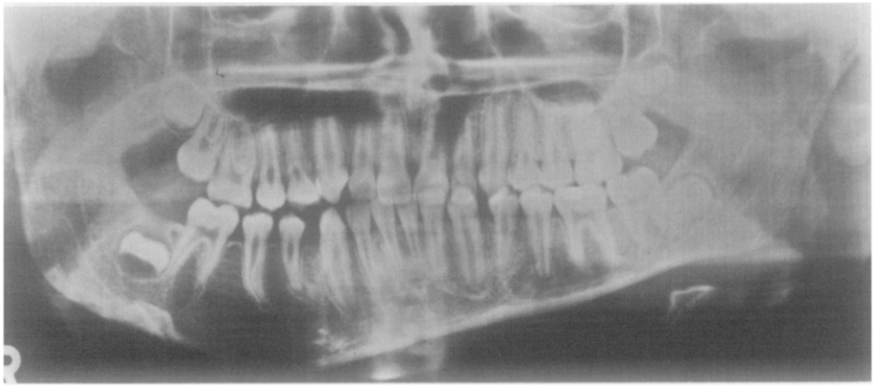

**Fig. 1 d**

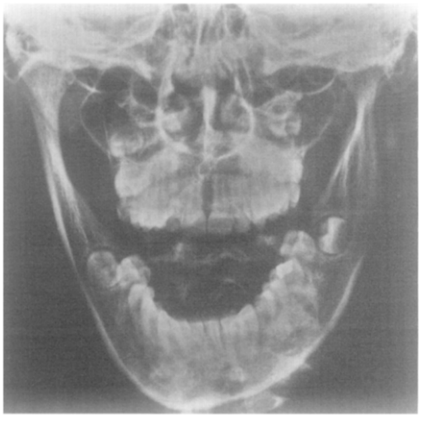

**Fig. 1 e**

**Figs. 1 d + e** Radiographs showing the skeletal basis of the unilateral enlargement of the mandible in all its dimensions together with its downwards growth which the maxilla including maxillary sinus follows. The mandibular canal is displaced to the lower border of the mandible; compared with the other side the coarse trabecular structure of the affected side can be clearly seen.

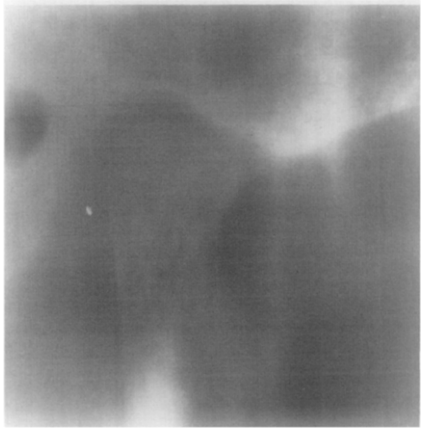

**Fig. 1 f**

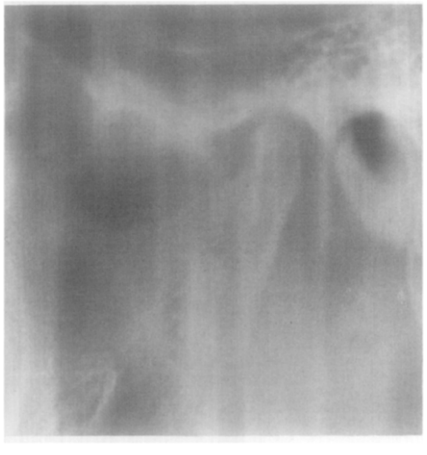

**Fig. 1 g**

**Figs. 1 f + g** show the differences in form and size of the two condyles. On the affected side the enlargement of the condyle, the thickening of the condylar neck and also the coarse trabecular structure is clearly visible.

**Fig. 1** A 12-year old patient with hemimandibular hyperplasia.

affected side of the face and consequently of the facial asymmetry. In pronounced cases, these skeletal changes which take place essentially unilaterally, bring about the well-known and typical rotation effect on the face previously mentioned.

The very enlarged condyle is usually irregularly deformed; the condylar neck is thickened and elongated and occasionally kinked (Fig. 1 f). These findings are particularly noticeable in comparison with the unaffected side (Fig. 1 g). The trabecular structure of the affected side is coarse, well-marked and wide-meshed compared with the opposite side (Figs. 1 d–g). Some trabeculae appear as if they were sclerotically enlarged, a picture which always is seen whenever a noxious stimulus, be it inflammation, a malignant osteogenic tumour, irradiation or any other cause, gives rise to osseous restructuring and production. The structure is very similar to the radiographic findings in uninfected areas of irradiation damage to the mandible. The ascending ramus and condyle of the unaffected side appear especially fine-structured. This might be due to the thrust exerted by the enlarged side.

This type of mandibular anomaly we have always observed to be unilateral. We find only rarely a strikingly clear, but often slight, mixture with the second type on the same side. We have never observed a case where both sides have been affected by this anomaly. In our opinion, such a case should exist, theoretically at least.

2. Differential diagnostic characteristics of hemimandibular hyperplasia

H.H. which affects one side of the mandible and terminates at the symphysis, must be clearly distinguished from the solitary and *exclusive hyperplasia of the condyle (Figs. 2 a–f)*. The latter involves only the condyle, which is homogeneously enlarged, showing radiographically a coarse structure (Figs. 2 d, e) when compared with the normal side (Fig. 2 f). Clinically, the facial appearance is likewise distorted (Figs. 2 a, b). There is a slight decrease in height of the affected side. The prominence of the chin is shifted to the unaffected side; but not as pronounced in H.H. An open bite might exist on the abnormal side (Fig. 2 c). This depends, on the one hand, on the rate of the

Fig. 2a

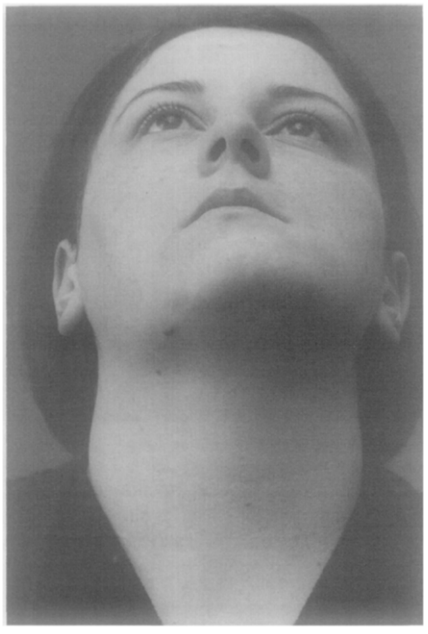

Fig. 2b

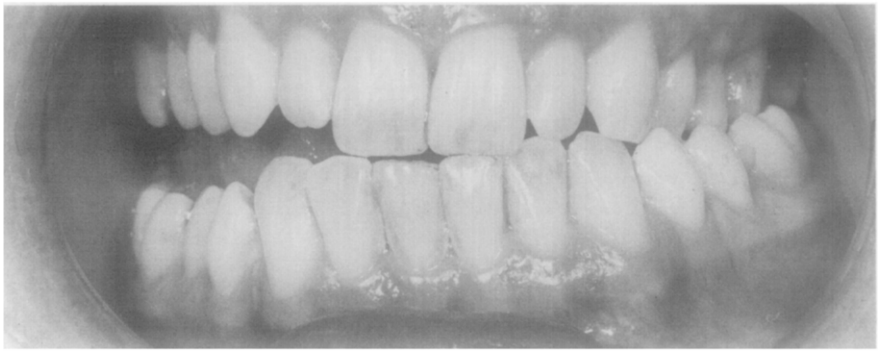

Fig. 2c

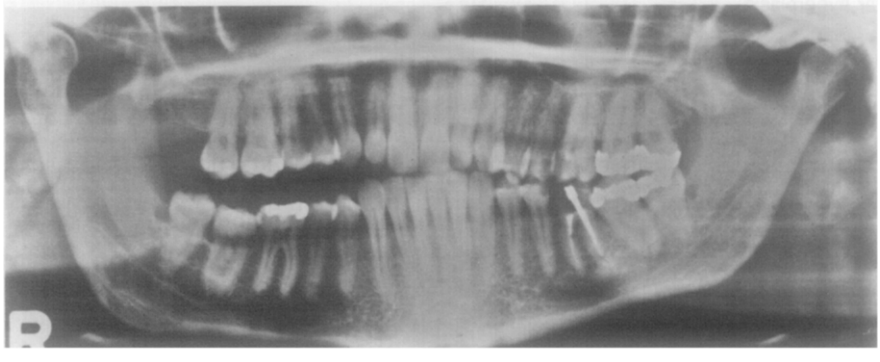

Fig. 2d

increasing enlargement of the condyle and, on the other hand, on the downward growth of the maxillary alveolus and teeth. Radiography shows, unlike hemimandibular hyperplasia, nothing but a massive enlargement of the condyle; the horizontal ramus is not increased in height; the mandibular canal is not displaced; no line of demarcation can be seen in the symphyseal region; the angle is not rounded, it rather shows an exaggerated rightangle; the horizontal ramus on the other side shows no relative reduction in height.

*Osseous tumour and exostosis of the condyle* show similar clinical, radiographic and occlusal pictures as does exclusively condylar hyperplasia. Therefore, these clinical pictures cannot be confused with hemimandibular hyperplasia and not at all with hemimandibular elongation. In these cases, a clear-cut distinction is perceptible.

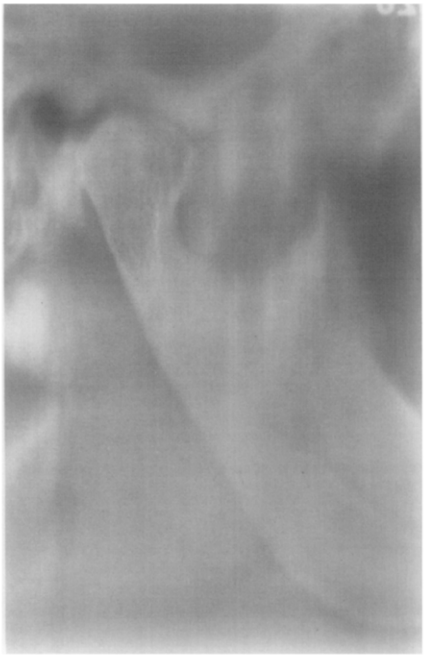

Fig. 2e

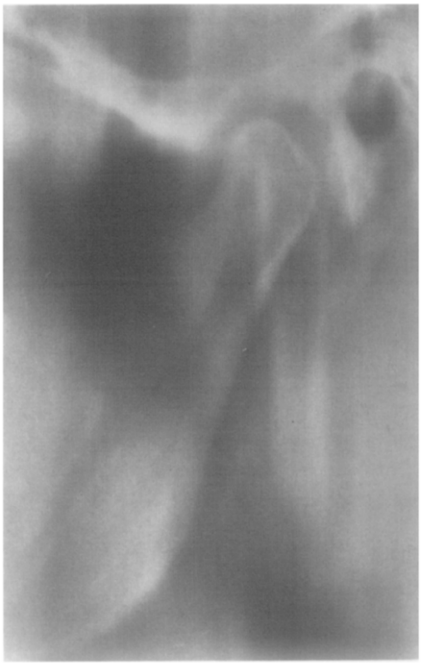

Fig. 2f

**Fig. 2** A 31-year old female patient showing a right sided hyperplasia of the condyle. The appearance (a+b) reminds one of hemimandibular hyperplasia. The occlusal picture (c) shows an open bite on the affected side and a displacement of the midline to the unaffected side where there is a cross-bite. The twisted occlusion characteristic of hemimandibular hyperplasia is missing. The orthopantomogram (d) and the tomographs of the joints (e+f) show unmistakable enlargement of the condyle alone and its coarse structure without involvement of the ramus and corpus. One is therefore dealing with hyperplasia of the condyle alone which has no effect on the growth of the other parts of the affected side of the mandible.

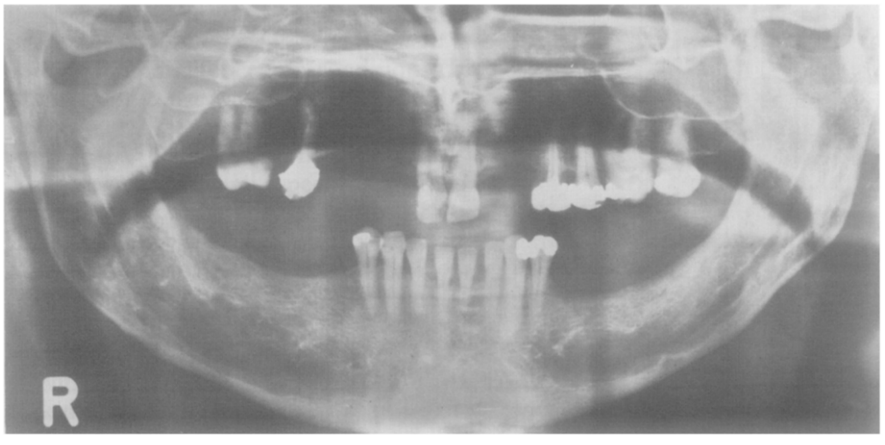


Fig. 3a

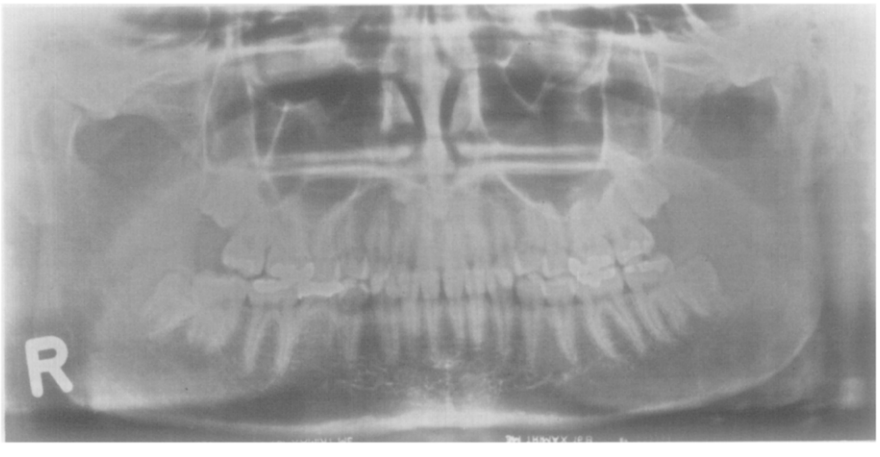


Fig. 3b

**Fig. 3** Bilateral and homogeneous enlargement of the mandible: a) In acromegalism a bilateral hemimandibular hyperplasia seems to exist, however, without the striking asymmetry of the unilateral cases; b) Bilateral macrogonion also exists in the case of deep overbite. The abnormality lies mainly in the existence of very large and rounded angles of the jaw and lack of height in the chin area. It is easily distinguishable from hemimandibular hyperplasia.

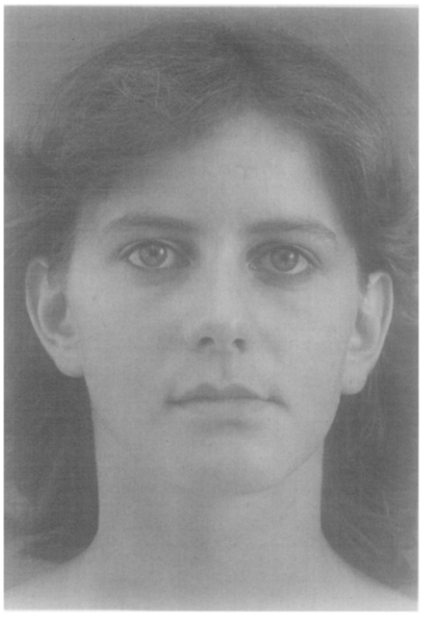

Fig. 4a

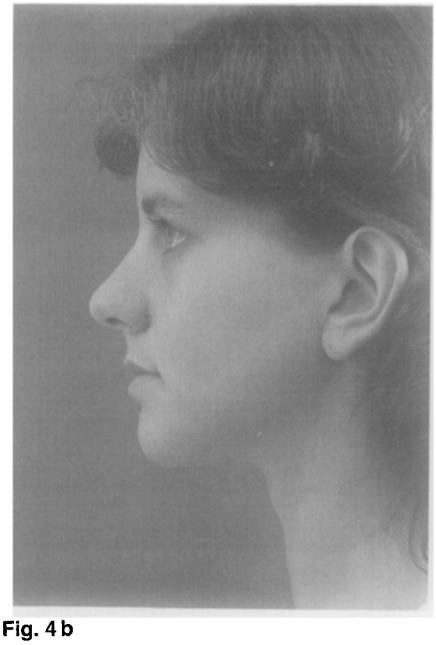

Fig. 4b

**Figs. 4a + b** The photographs reveal displacement of the chin prominence towards the unaffected side. However, no progenic profile has been produced.

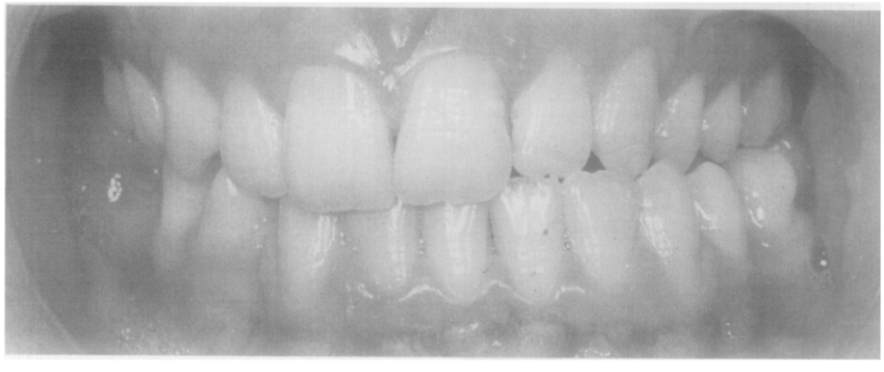

**Fig. 4c** shows typical occlusal findings in a case of hemimandibular elongation, i.e. displacement of the mandible and the dental midline to the unaffected side with the resulting cross-bite on the unaffected side.

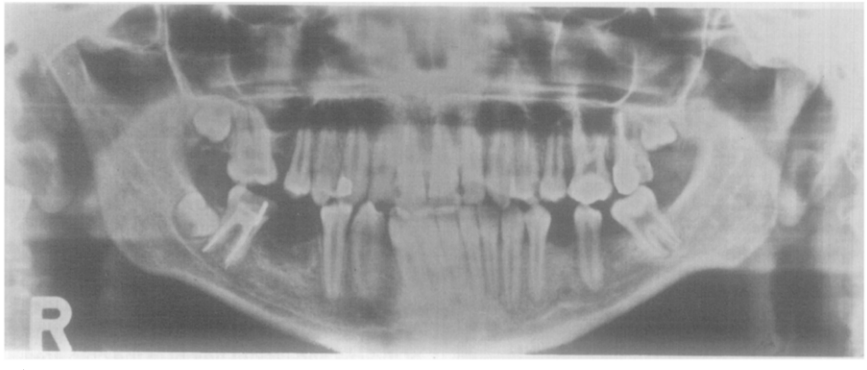


Fig. 4d

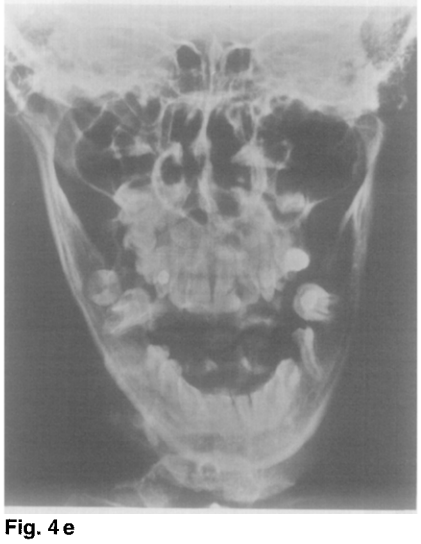

Fig. 4e

**Figs. 4d + e** Radiographs showing the skeletal basis of the anomaly. Typical elongation of the affected side involving the condylar neck and the widening of the mandibular angle.

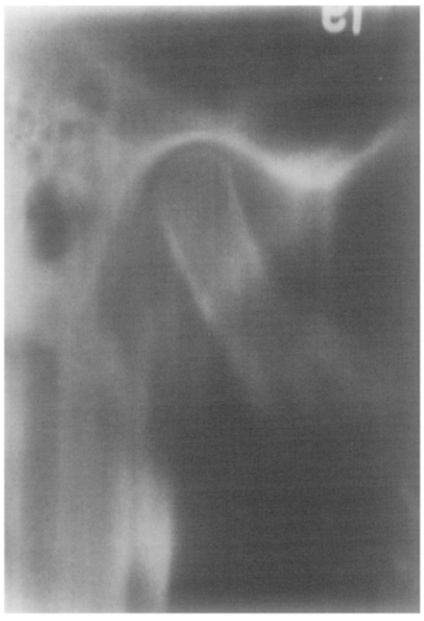

**Fig. 4 f**

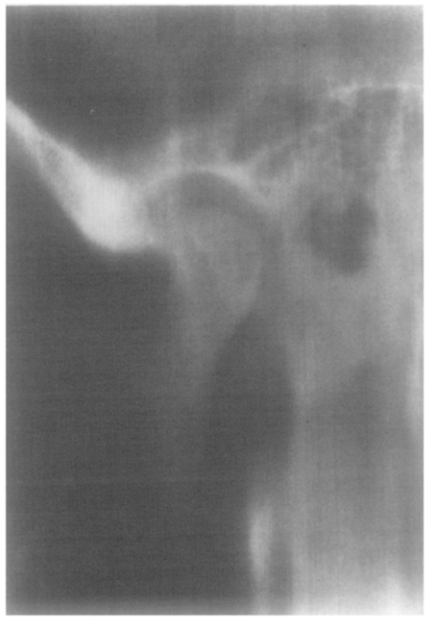

**Fig. 4 g**

**Figs. 4 f + g** Radiographs showing the big difference which exists between the condyles and condylar necks of the affected and unaffected sides of the mandible.

**Fig. 4** An 18-year old female patient showing right sided hemimandibular elongation.

*Bilateral and homogeneous enlargement of the mandible* is observed mainly under two conditions (Figs. 3 a, b):
*Acromegaly* causes an enlargement and elongation of the whole mandible which assumes an interesting shape in relation to bilateral H.H. and/or H.E. The mandible becomes enlarged in all aspects and it grows longer. It has a shape as if it could be a combination of a bilateral H.H. and H.E. Since both sides are influenced there is no such asymmetry as seen in H.H. or H.E. cases. However, all parts of the mandible seem to be enlarged and lengthened in all dimensions including the condylar neck and head. The angle is rounded off, the horizontal ramus is deeper and the ascending ramus is thicker than normal, and there is a strikingly coarse and thickened trabecular structure. The vertical enlargement of the horizontal ramus is mainly due to increase of height above the mandibular canal. All these are signs which are also seen in the cases of typical H.H. We miss, of course, the downwards-bowed lower border of the mandible and the typical tilting of the teeth. The absence of these signs of H.H. is due to the almost symmetrical bilateral growth. There are also cases of acromegaly which seem to consist mainly of a bilateral H.E., with, however, a fair amount of influence of either H.H. or additional bone apposition as in long bones.
Since acromegaly may start long after general growth has ceased certain differences of shape between left and right side may be due to conditions which have happened before acromegaly started or are due to an unilateral, mainly hyperplastic and contralateral, mainly elongation-type of acromegalic enlargement of the two halves of the mandible (Fig. 3 a).
Bilateral and homogeneous enlargement of the mandible is also observed in *cases of deep overbite and in cases of masseter hypertrophy*. These types of mandibular enlargement pose no differential diagnostic problems (Fig. 3 b). Here we are dealing primarily with a bilateral and homogeneous enlargement in the angle region which involves the horizontal and ascending rami. The mandibular angle is almost a right-angle; the typically rounded angle of H.H. is absent. Similarly, the low positioning of the bowed mandibular lower border which extends to the midline is also absent. Of course, the enlargement of the condyle, the elongation and thickening of the condylar neck and the increase in height of the ascending ramus are also absent. The anomaly always seems to occur bilaterally and symmetrically. Thus, it is not difficult to distinguish it from hemimandibular hyperplasia.

*B. Hemimandibular elongation (H.E.)*
Hemimandibular elongation is characterized by horizontal displacement of the mandible plus chin towards the unaffected side. The horizontal rami of both sides lie on the same level. The facial distortion becomes very noticeable when viewed from below. A comparison of the side views of the face reveals the more or less obtuseness of the angle and the unilateral elongation of the mandible terminating at the symphysis. The corner of the mouth on the unaffected side shows a furrow effect without causing its depression. The typical and striking lateral displacement of the mandible is also clearly manifested by the occlusal picture (Figs. 4 c, 5 b). In dentate cases, the axial alignment of the teeth in the strikingly slender horizontal ramus generally shows no essential tilting. Occasionally, a slight tilting towards the abnormal side is noted. The displacement of the mandible towards the unaffected side is identifiable by the contralateral displacement of the midline of the lower dental arch and the resulting crossbite on this side. Since there is no vertical elongation of the ascending ramus, no depressed positioning of the arch on the affected side occurs.
This prevents the development of a tilted occlusal plane. In a classical case, the lower arch is simply displaced towards the opposite side. Unlike the occlusal picture in H.H., the

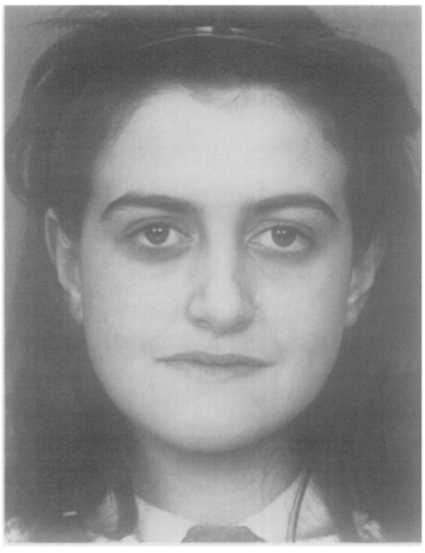

Fig. 5a

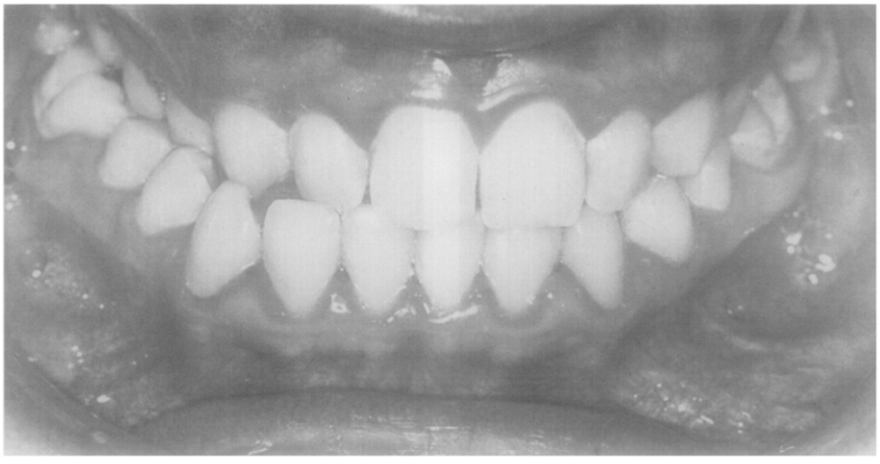

Fig. 5b

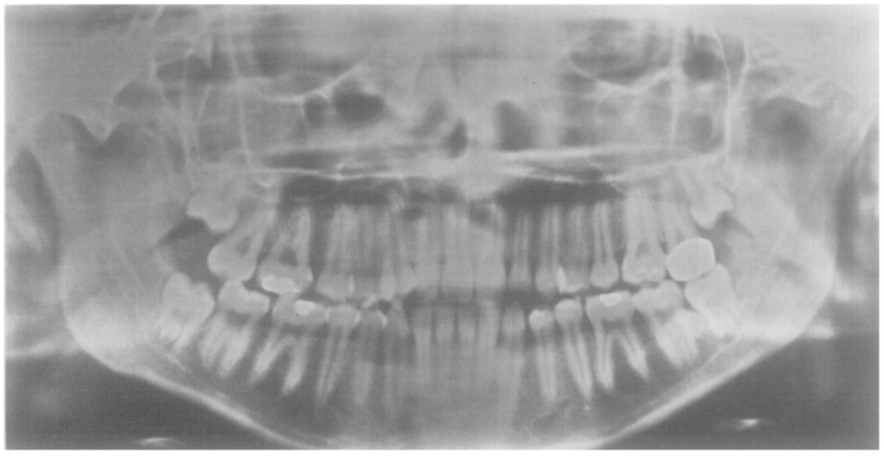

Fig. 5c

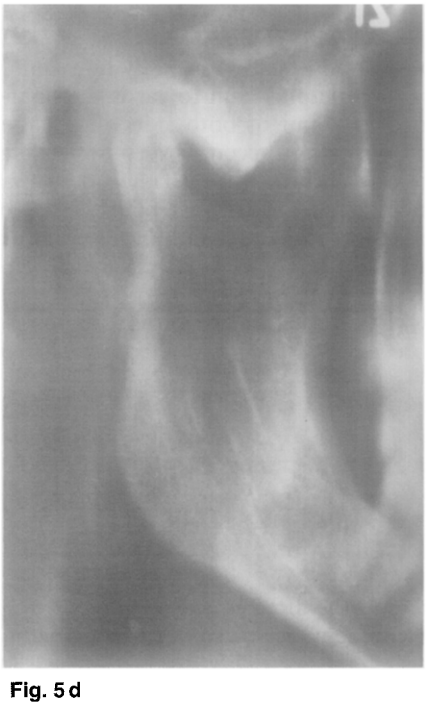

Fig. 5d

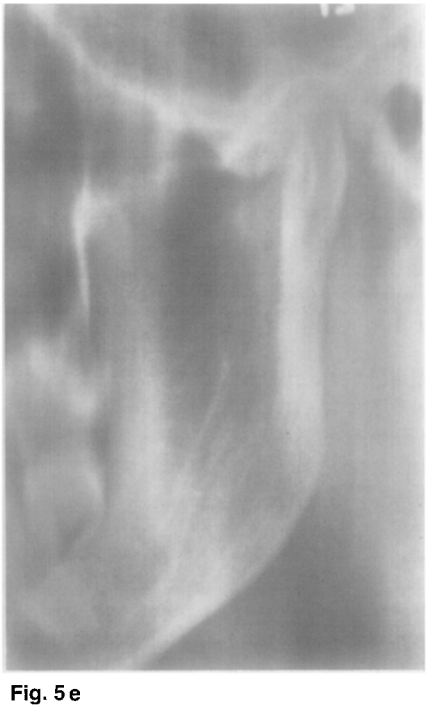

Fig. 5e

**Fig. 5** A 19-year old female patient showing left sided hemimandibular elongation with a degree of hemimandibular hyperplasia.
The front view (a) and the occlusal picture (b) reveal the unmistakable picture of hemimandibular elongation. The radiograph (c) shows that there is a hint of rounding of the mandibular angle despite its widening. Although the condyle shows a nearly normal form, the condylar neck is thickened (d, e). These are all signs indicating the possible presence of a degree of hemimandibular hyperplasia.

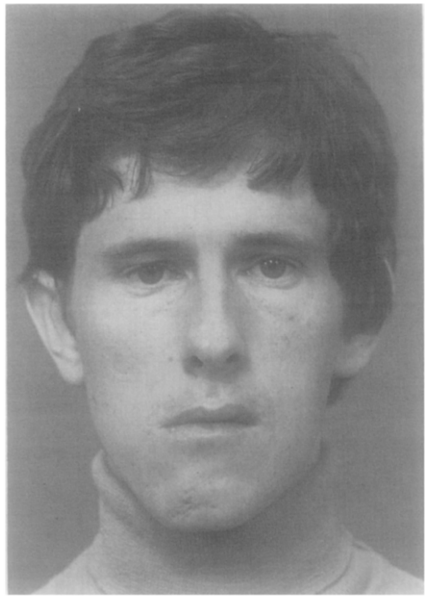

Fig. 6 a

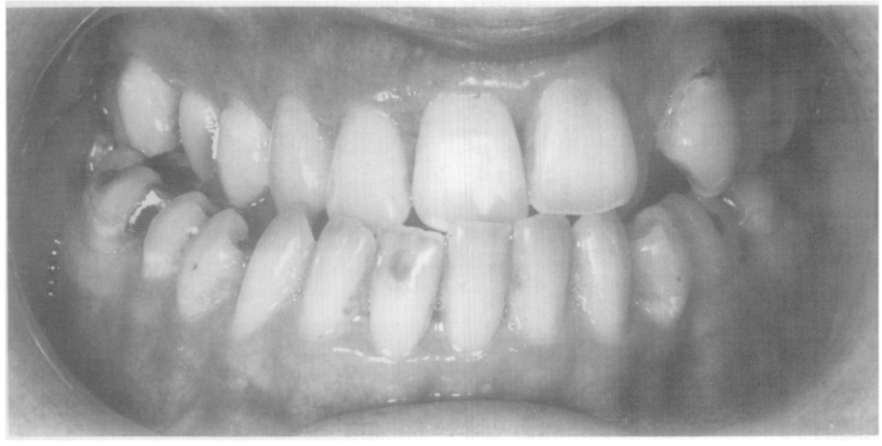

Fig. 6 b

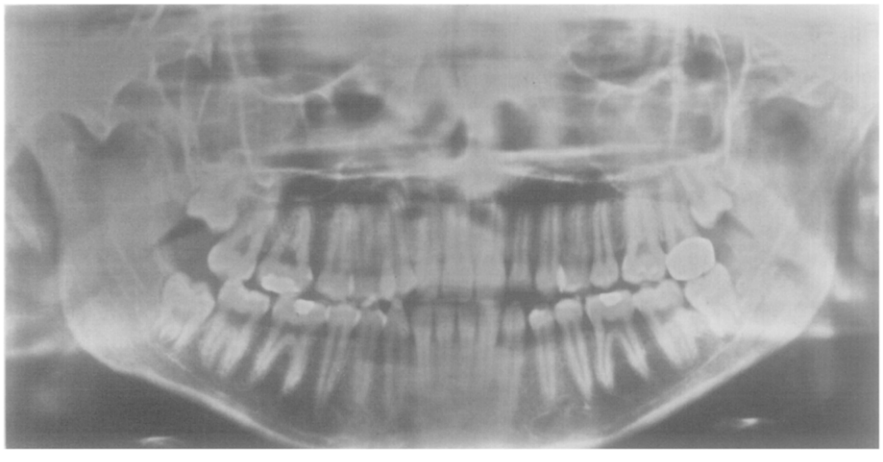

Fig. 6 c

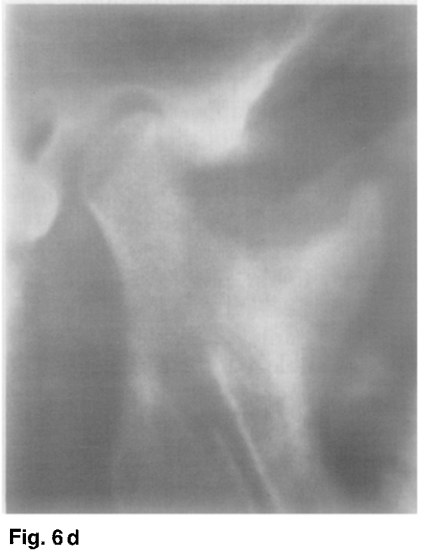

Fig. 6 d

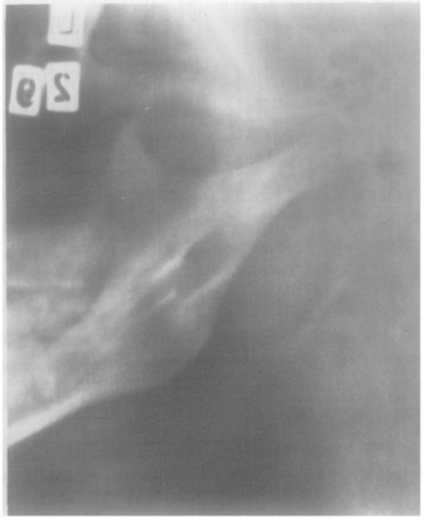

Fig. 6 e

**Fig. 6** A 25-year old patient showing asymmetrical bilateral hemimandibular elongation. A front view of the face (a) reveals the appearance of so-called asymmetric prognathism. The occlusal picture (b) shows an anterior cross-bite typical of asymmetric bilateral hemimandibular elongation in the presence of a normal maxilla (the maxilla is normal in size and position). There is displacement of the mandibular midline towards the less active side. On the radiograph (c) the more active side shows a slender condylar neck and a wider mandibular angle than on the less active side. The tremendous difference in size and shape of the two condyles is striking (d + e).

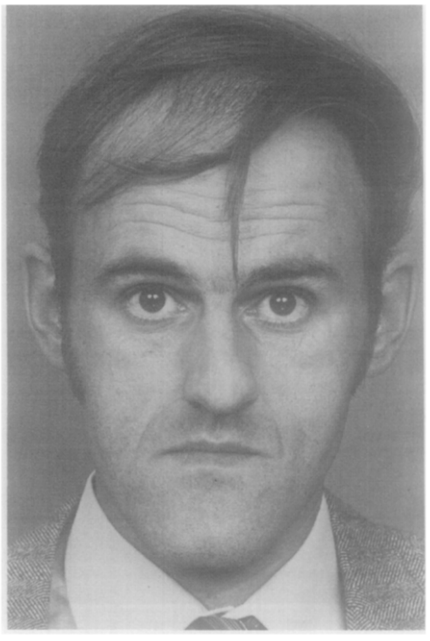

Fig. 7 a

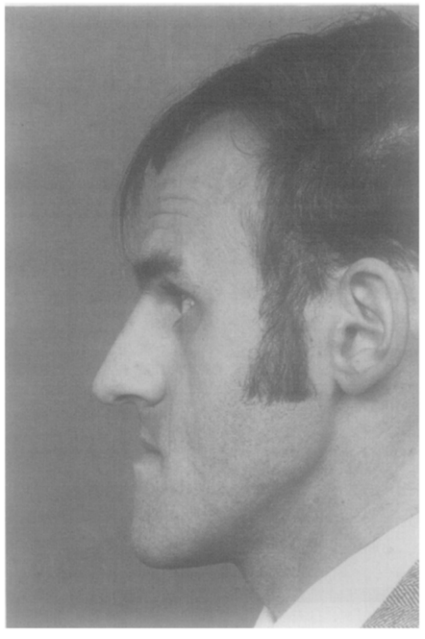

Fig. 7 b

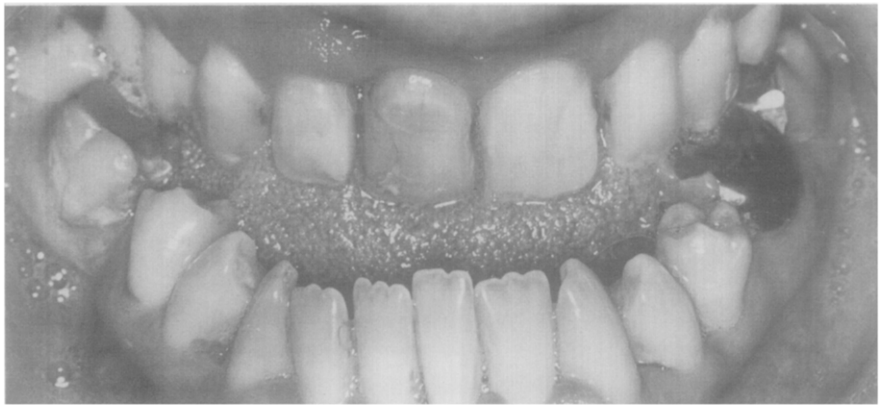

Fig. 7 c

upper and lower arches do not seem to have a tendency to remain in occlusion. Radiographically, the unilateral elongation of the mandible is noticeable on the orthopantomogram and p.a.-projection in a maximally opened position of the mouth (Figs. 4 d, e). Thus, extraorally and occlusally, one sees the picture of laterognathia which is also occasionally labelled asymmetric or unilateral prognathism. Despite the unequivocal elongation of one side of the mandible, the profile of true progenia is not produced (Fig. 4 b).

1. Slender form of hemimandibular elongation
This unilateral elongation of the mandible can clearly affect all three parts of it: the condylar neck, the ascending and horizontal rami. The increased obliquity of the angle is thereby very marked and seems to be responsible for the elongation of the one side. The trabecular structure of the affected side shows no irregularity. The condyle is occasionally somewhat enlarged (Figs. 4 f, g) but it is clearly of minor degree and less deformed than in the case of H.H. The condylar neck is clearly elongated and slender.

2. Non-slender form of hemimandibular elongation
Very often, the condylar neck is not noticeably elongated although on external viewing and radiologically the unmistakeable clinical picture of H.E. is present (Figs. 5 a–c). Also the form and size of the condyle appear nearly normal but somewhat rather bigger than that of the slender form, and the flattening out of the angle is not so pronounced. Equally, a clear reduction in height of the horizontal ramus is absent too. These are all clear signs that are so obvious in the slender form. The clear displacement of the midline of the chin and the dental arch to the unaffected side, the typical cross-bite of H.E. on the unaffected side and the elongation of the horizontal ramus cannot be missed. Accordingly, we consider it possible that there exists a small proportion of hyperplastic component in the non-slender form of H.E.

3. Bilateral hemimandibular elongation
Unlike H.H., bilateral occurrence of hemimandibular elongation is not rare. It is often more or less asymmetrical,

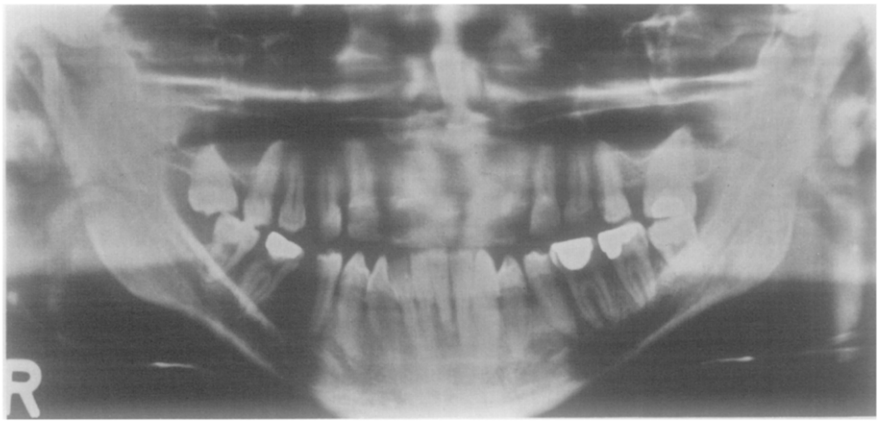


**Fig. 7 d**

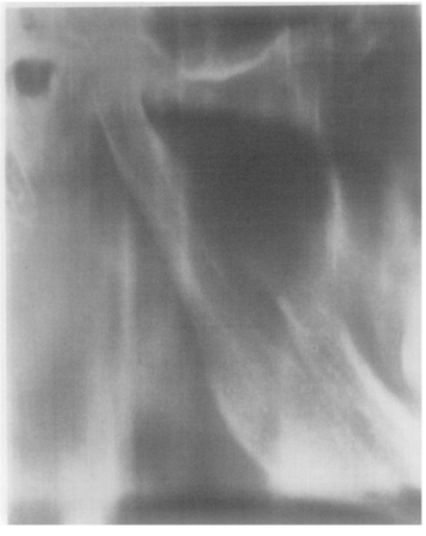

**Fig. 7 e**

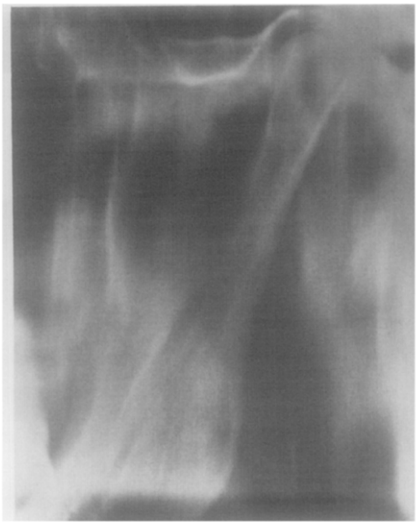

**Fig. 7 f**

**Fig. 7** A 33-year old patient, showing a bilateral asymmetric hemimandibular elongation in the presence of micromaxillism. The pictures (a + b) reveal the flattening of the midface and retro-positioning of the maxilla on the one hand, and on the other the asymmetric elongation of the mandible together with marked displacement of the midline of the chin is unmistakable. The micro-retro-maxillism and the elongation of both sides of the mandible give rise to a well-marked progenic occlusion (c). The radiographs (d, e, f) clearly show the skeletal elongation of the condylar necks and the widened mandibular angle together with the slender horizontal rami. The short and retro-positioned base of the maxilla is also visible.

however. Both the slender and non-slender forms can therefore be present unilaterally or bilaterally. Clearly, a less involvement of one side gives rise to the characteristic anterior crossbite and likewise a noticeable asymmetric chin prominence (Figs. 6 a–e). In the case presented the orthopantomogram shows unmistakably a slender form on the left side and a non-slender form on the right. In the case with this bilateral form, though asymmetrical, the progenic appearance of the mandible is a necessary consequence.
When both sides are markedly affected, a progenic appearance, symmetrical or asymmetrical, characterized by an extremely oblique angle, a long horizontal ramus and a very protruding chin prominence is produced. In the case with the bilateral slender form, the whole mandible looks noticeably thin. In the presence of an associated micro- or retromaxillism, there is a marked progenic appearance (Figs. 7 a–f).

4. Differential diagnosis
Differential diagnostically, there is only the possibility that a *hemimandibular hypoplasia of the opposite side* simulates, with the normal length of the horizontal ramus of the opposite side, an apparent lengthening. The former then gives the impression of H.E. of the other side. Cases of otomandibular dysostosis are typical such examples. The chin likewise is displaced to the side of the hypoplasia; but the lengthening of the three parts of the mandible, the obliquity of the angle of the opposite side and the contralateral cross-bite are, however, missing. The class II malocclusion on the hypoplastic side discloses clearly the micromandibulism of this same side.
A diagnostically difficult situation arises when the mandible is affected by a *true H.E. on the one side* and a *true hemimandibular shortening on the other side*. Then, due to the shortening of the contralateral side, the H.E. appears excessively marked. For therapeutic reasons this clear distinction is very important. Such a unilateral hypoplasia in association with a contralateral elongation is illustrated in Figs. 8 a–f. On the orthopantomogram and TMJ-tomographs the hypoplasia of the condyle of the right side and with it the corresponding side of the mandible are equally identifiable as H.E. of the left side. The case illustrated of right-sided hemimandibular hypoplasia is part of a right-sided facial hypoplasia, which is discernible in the front view. It is, therefore, a case of unilateral facial hypoplasia in association with contralateral H.E.

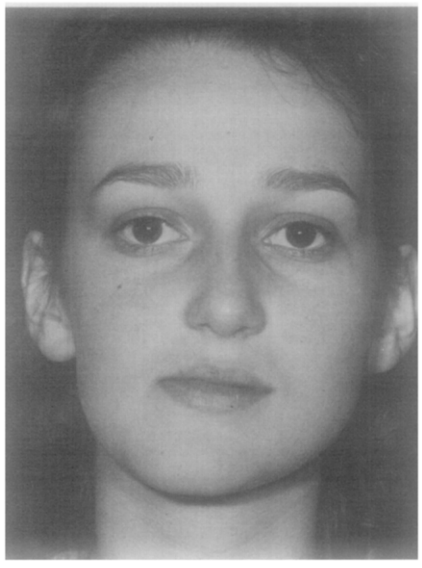

**Fig. 8a**

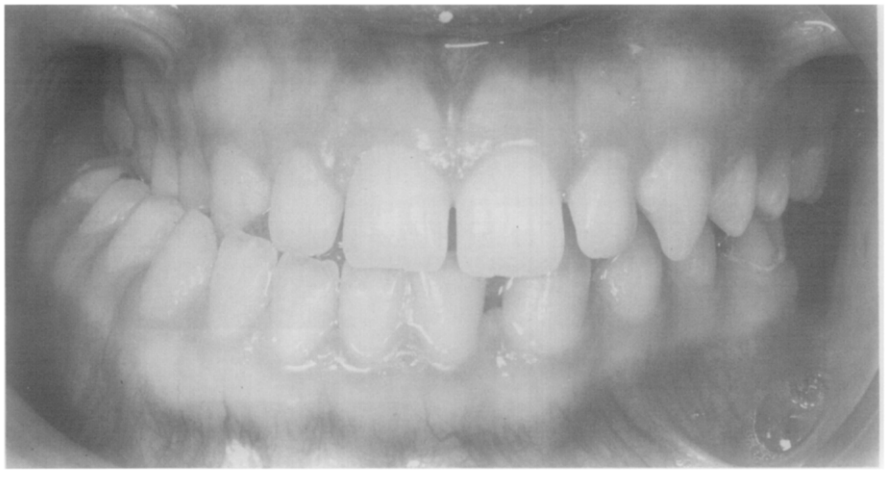

**Fig. 8b**

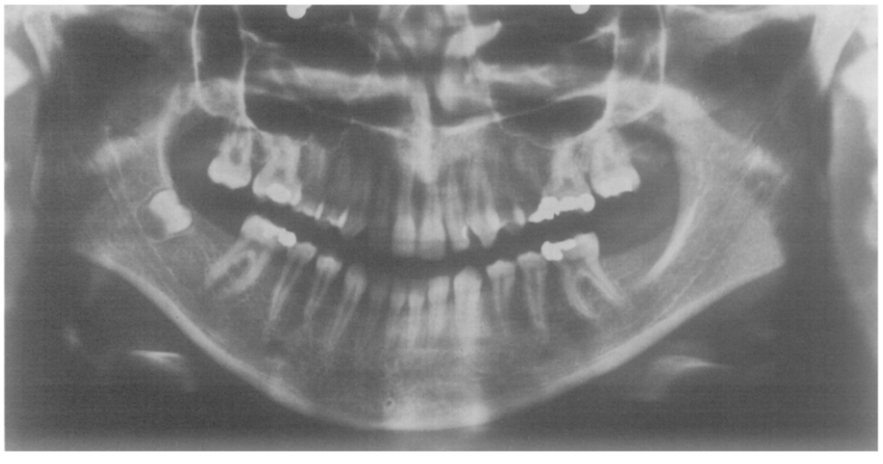

**Fig. 8c**

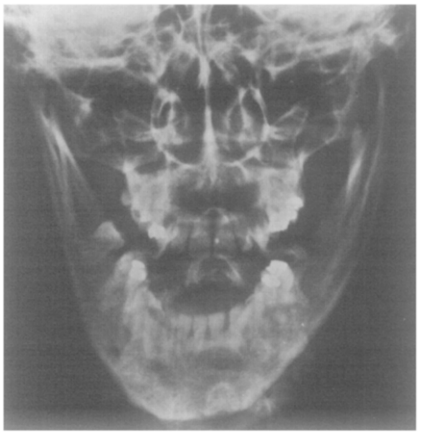

**Fig. 8d**

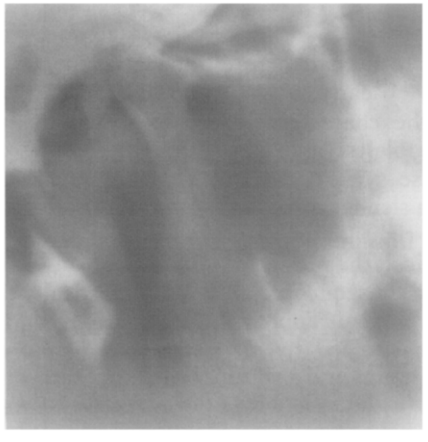

**Fig. 8e**

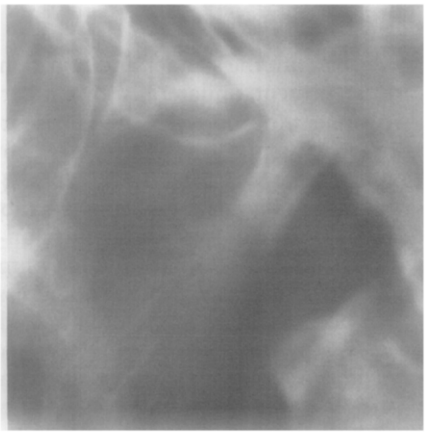

**Fig. 8f**

**Fig. 8** A 16-year old female patient, showing leftsided hemimandibular elongation and a right-sided facial hypoplasia.
This combination leads to extreme deviation of the chin prominence towards the unaffected side (a). Also the occlusal picture clearly reveals the displacement of the mandible by 1 + 1/2 tooth widths (b). The orthopantomogram (c) shows hemimandibular elongation on the left which extends exactly to the midline and on the right the hypoplasia of the condyle as well as the short ascending ramus. The shape of the angles and ascending ramus of this case are similar to that of a bilateral condylar hypoplasia case, however, with a very pronounced hemimandibular elongation of the left side. The hypoplasia of the right side of the mandible is particularly well-demonstrated in the p.a.-projection (d). The tomograms of left and right TMJ including ascending ramus (e + f) demonstrate the tremendous difference between the two.

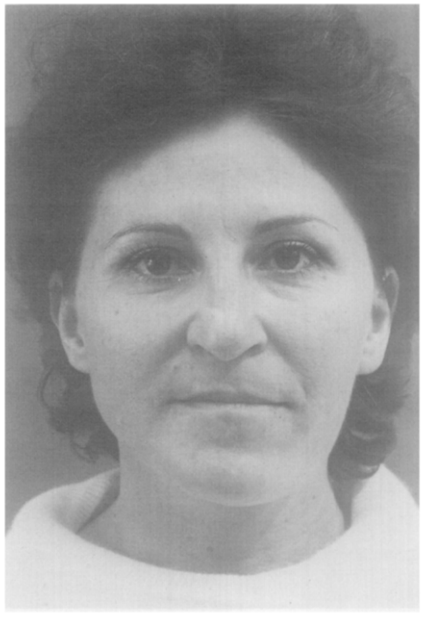

Fig. 9 a

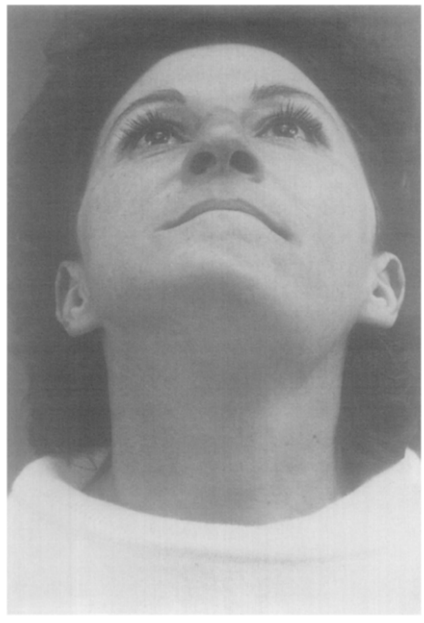

Fig. 9 b

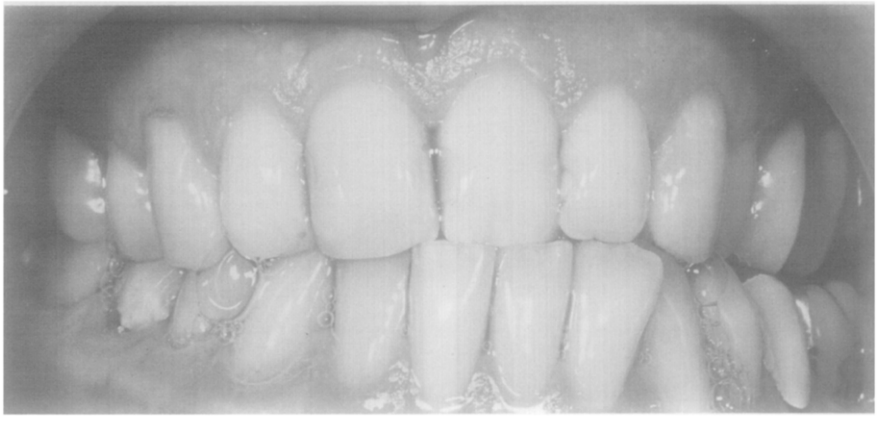

Fig. 9 c

## Combined and Hybrid Forms

As stated earlier, according to the hypothesis which will be elaborated later, two different growth stimuli or factors are discernible: there must exist a unilateral hybrid form and bilateral combined forms. The variability of these forms is explained by the fact that they can occur in association with the various forms of normal mandibles of the opposite side, or even a micromandible on one side might exist – this happens mostly in the case of hypoplasia of the condyle – and one of the two described hemimandibular anomalies on the other side, in either pure or hybrid form, as shown in Figs. 8 a–d.

*A. Bilateral combination forms*

Actually, this form requires no more explanation, because it is clear that both anomalies, H.H. and H.E., can develop in the mandible of the same patient, one on the left side and the other on the right.

Figs. 9 a–g illustrate a typical case of bilateral combination form. On the right side there is H. E., and on the left side there is a moderate but well-developed H.H. The frontal view of the female patient discloses a noticeable asymmetry of the mandible described as a right-sided H.E. (Fig. 9 a). The chin prominence is clearly displaced to the left of the facial midline. Moreover, the level of the two facial sides is distorted; this is perceptible especially when viewed from below (Fig. 9 b). The occlusal picture shows displacement of the midline to the left and an open bite in front. On the left side a slight open bite in the region of the molars and premolars is present (Fig. 9 c). This can be seen clearly in the orthopantomogram taken with the teeth in complete centric occlusion (Fig. 9 d).

Thus the combination form is also demonstrated by the picture of centric occlusion and the position of the teeth: on the one side, the displacement of the midline of the lower dental arch by almost the width of a tooth to the opposite side reflects the main occlusal sign of H.E., and on the other side, the tilting of the teeth of the opposite side with the slightly open bite in the region of the molars present the occlusal picture of H.H. on this side. Both forms are clearly demonstrated by the radiographs, especially the orthopantomogram (Fig. 9 d). On the right side, the almost normal form of the condyle with its slender and prolonged condylar neck, the oblique angle, the elongation of the whole side

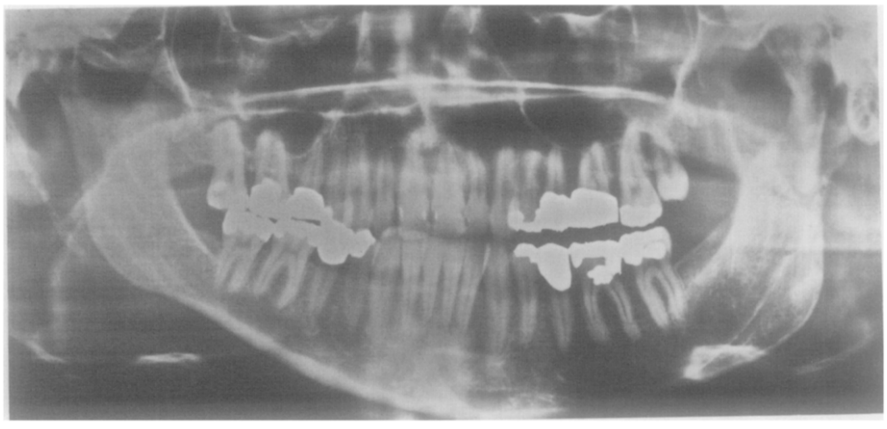

**Fig. 9 d**

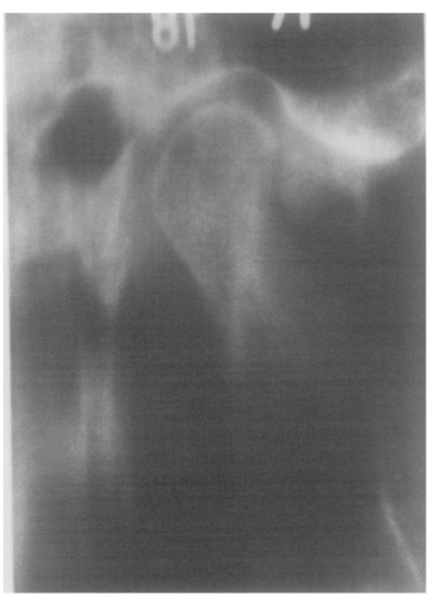

**Fig. 9 e**

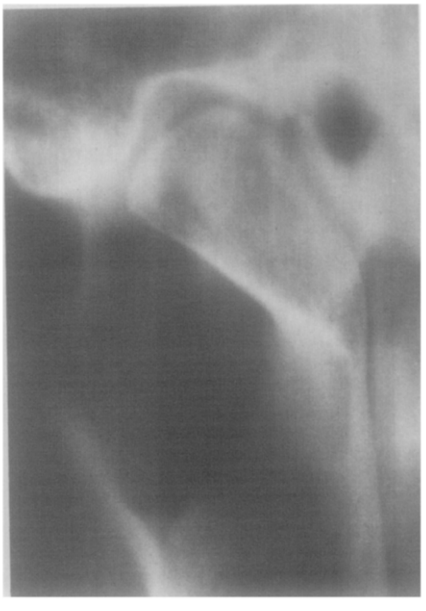

**Fig. 9 f**

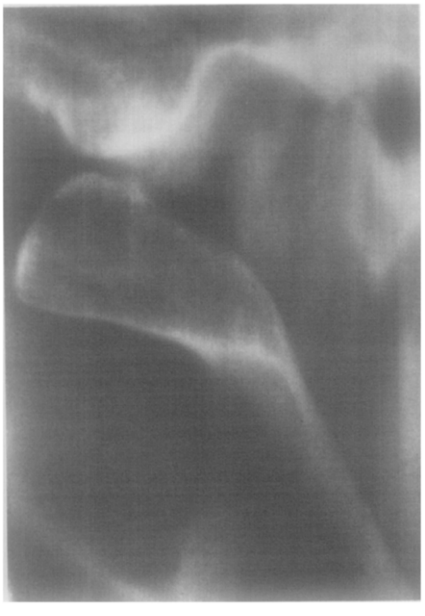

**Fig. 9 g**

**Fig. 9** A 39-year old female patient showing leftsided hemimandibular hyperplasia and right-sided hemimandibular elongation.
The front view (a) and the view from below (b) give a clear picture of the elongation of the right horizontal ramus together with shifting of the chin prominence towards the unaffected side and hyperplasia and low level of the left side of the mandible. The occlusal picture also demonstrates the combination of the two anomalies: The midline is shifted towards the unaffected side where a cross-bite exists. The hyperplasia leads to an open bite in the affected side. The elongation of the mandible and the widening of the mandibular angle on the right side and the characteristic findings of unilateral hyperplasia on the left side are identifiable on the orthopantomogram (d). Displacement of the mandibular canal towards the mandibular lower border, the increase in height of the horizontal and ascending rami, enlargement of the condyle and elongation and thickening of the condylar neck are all present. The TMJ-projections show on the one hand a normal right condyle with an elongated condylar neck (e) and on the other hand an enlarged right condyle with an elongated and thickened condylar neck (f). Despite the condylar enlargement, the TMJ function is normal (g).

and the reduced body of the mandible, the displacement of the chin prominence and midline of the dental arch to the other side are unmistakable skeletal signs of H.E. The trabecular structure of that side of the mandible is inconspicuous. On the left side, the shapeless enlargement of the condyle, the thickening and prolongation of the condylar neck and with it the prolongation of the ascending ramus with the rounded-off angle, the increased height of the horizontal ramus which extends to the midline, and the displacement of the mandibular canal to the lower border of the mandible are unmistakably cardinal skeletal signs of H.H. There is also unmistakably a different trabecular structure than on the elongated side. It is coarse and thickened, not only in the condyle and ascending ramus but also obvious in its horizontal part including the alveolar bone. The tomograms of the TMJ demonstrate not only the two typical forms of the condyle and its neck but also the clear structural difference between the two. The elongated side (right) shows an inconspicuous fine trabecular structure of the condyle (Fig. 9 e). The condyle on the hyperplas-

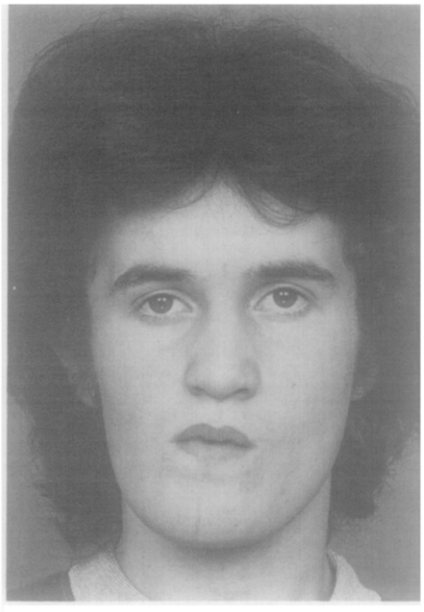

**Fig. 10 a**

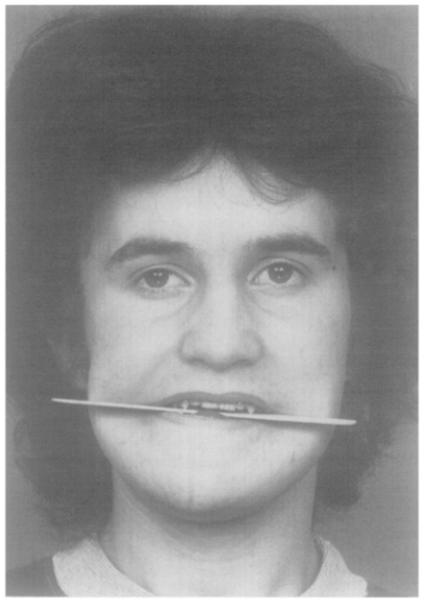

**Fig. 10 b**

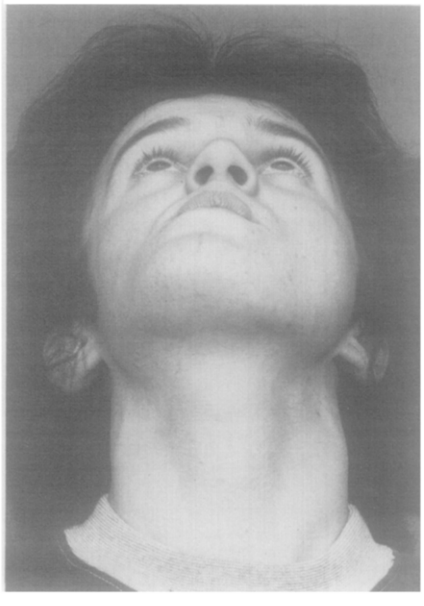

**Fig. 10 c**

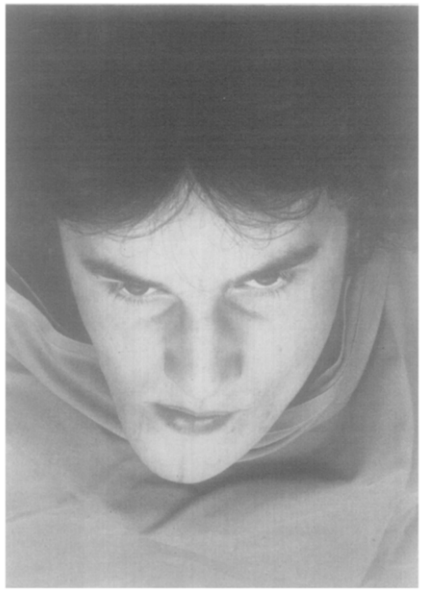

**Fig. 10 d**

tic side (left) (Figs. 9 f, g) shows the coarse and thickened trabecular structure. In spite of its huge condyle which does not fit into the glenoid fossa there is reasonable function of the joint (Figs. 9 f, g). The displacement of the chin prominence to the unaffected side is a major sign of H.E. when viewed from in front. The increase in height of one side of the face is the major sign of H.H. Of course, the latter sign also applies to unilateral hyperplasia of the condyle and to an osseous tumour of the TMJ; but the orthopantomogram also illustrates, apart from the enlargement of the condyle, a thickening and prolongation of the condylar neck, the clearly rounded-off angle and the essential increase in height of the horizontal ramus in the region of the molars and premolars with the downward displacement of the mandibular canal. In the case of this female patient one is thus dealing with an unequivocal bilateral combination form expressed by a clearly marked H.E on the right side and a moderately developed H.H. on the left.

*B. Unilateral hybrid forms*

In the case of unilateral hybrid forms (Figs. 10 a–g) the appearance must be more grotesque than that in the case of a unilateral pure form of either of the two possibilities and the bilateral combined forms because both abnormalities are summated on one side. The affected side of the face has a surplus in height and is elongated in the region of the horizontal ramus. This is more clearly perceived in the views from below and from above (Figs. 10 c, d) than in the view from in front (Fig. 10 a). Accordingly, the rima oris is slanting and the occlusal plane is tilted as in the case of H.H. (Fig. 10 b). The occlusal picture of the patient undergoing preliminary orthodontic treatment clearly shows on

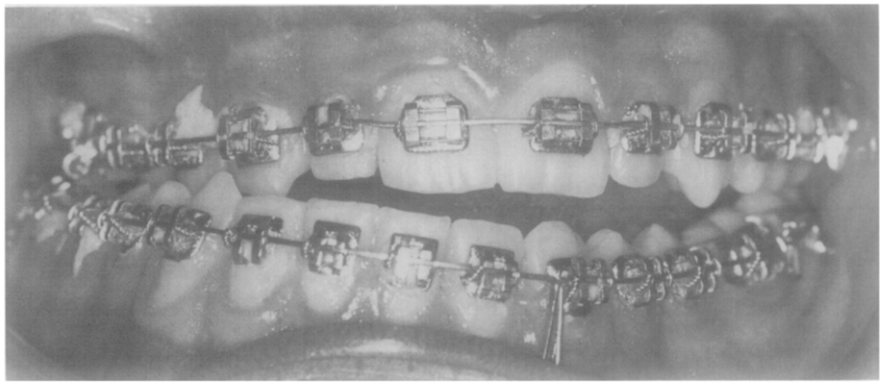

Fig. 10e

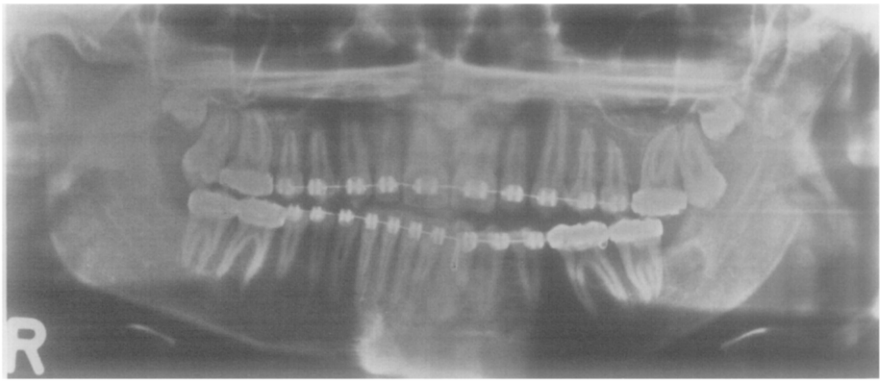


Fig. 10f

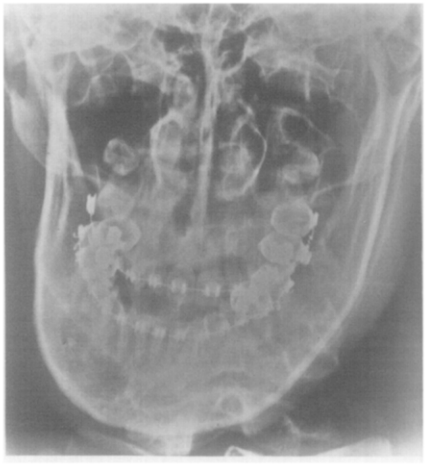

Fig. 10g

Fig. 10h

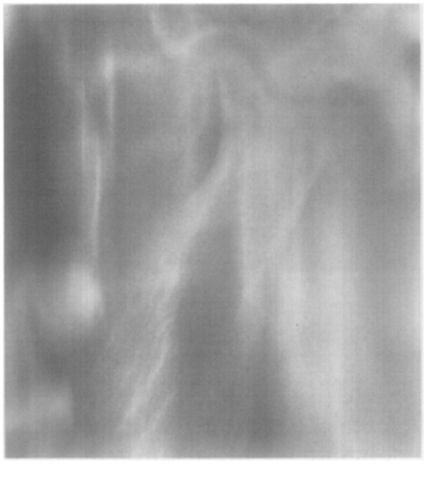

Fig. 10i

**Fig. 10** A 16-year old female patient showing a left-sided hybrid form composed of hemimandibular elongation and hemimandibular hyperplasia. The H.E. is manifested by the clear displacement of the chin prominence towards the unaffected side (a–d), the unilateral hyperplasia is expressed by the prominence of the lower two facial thirds on the affected side, by the low level of the angle of mouth, the occlusal plane and the mandibular angle together with the horizontal ramus (a–c). The occlusal picture (e) shows, despite the current orthodontic treatment, the marked displacement of the midline of the lower teeth amounting to almost two incisor tooth widths. There is a cross-bite on the unaffected side and at the same time an open-bite predominantly on the affected side, as a manifestation of the hyperplastic component. This unilateral hybrid form, composed of the two anomalies, is also unmistakably identifiable on the orthopantomogram (f): the anomaly ends exactly at the midline of the chin and lower dental arch. The mandibular angle is clearly widened and rounded off however. The increased height of the corpus ends at the midline. The condyle and the condylar neck are enlarged and elongated. The right side of the mandible appears to be completely normal. The p.a.-projection shows the anatomical basis of this grotesque appearance particularly clearly (q). The striking difference of the two condylar processes is seen in the tomograms of the right and left joint including ascending ramus (h + i).

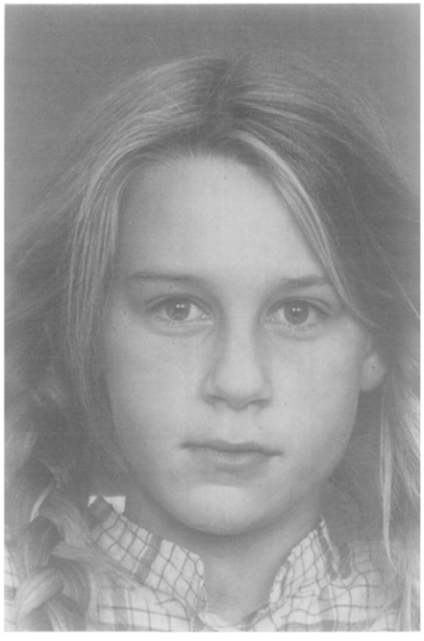

**Fig. 11 a**

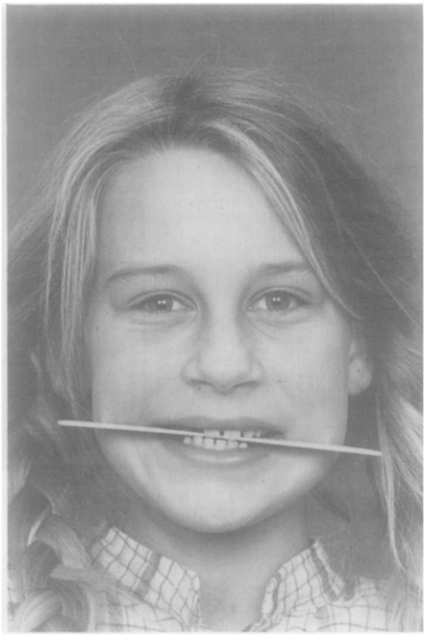

**Fig. 11 b**

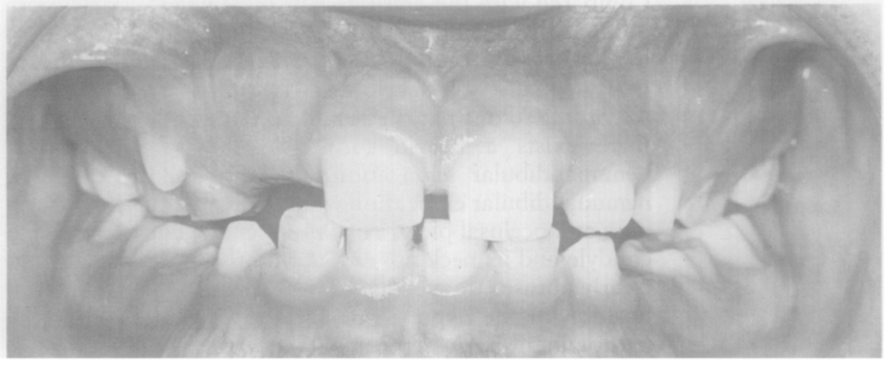

**Fig. 11 c**

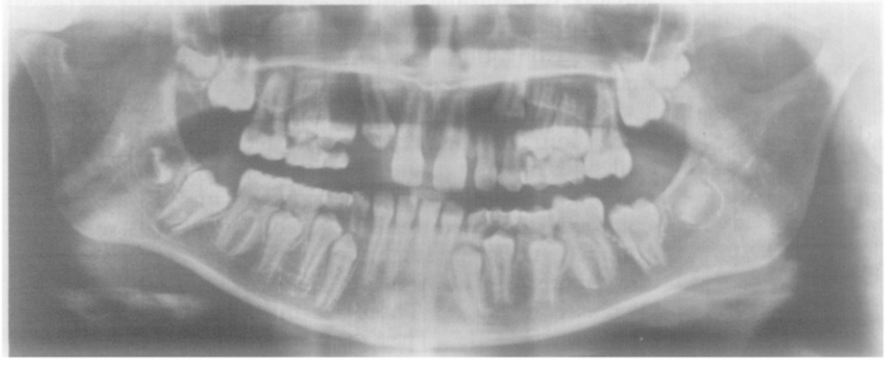

**Fig. 11 d**

the one hand the lateral displacement of the mandible to the right side by two lower incisor tooth widths and a cross-bite on this same side, and on the other hand an anterior open-bite more obvious on the left side (Fig. 10 e).

Radiographically, the orthopantomogram clearly shows the skeletal morphology of both forms of anomalies on the left side of the mandible: the affected side is caudally convex and is elongated towards the opposite side. This results in extreme displacement of the chin prominence and the midline of the lower dental arch. The increased height and caudal convexity of the horizontal ramus are the cardinal signs of hemimandibular hyperplasia. The elongation of the ascending ramus together with the pronounced displacement of the chin prominence to the opposite side constitute the cardinal signs of hemimandibular elongation.

In the region of the angle the anomaly poses a peculiar

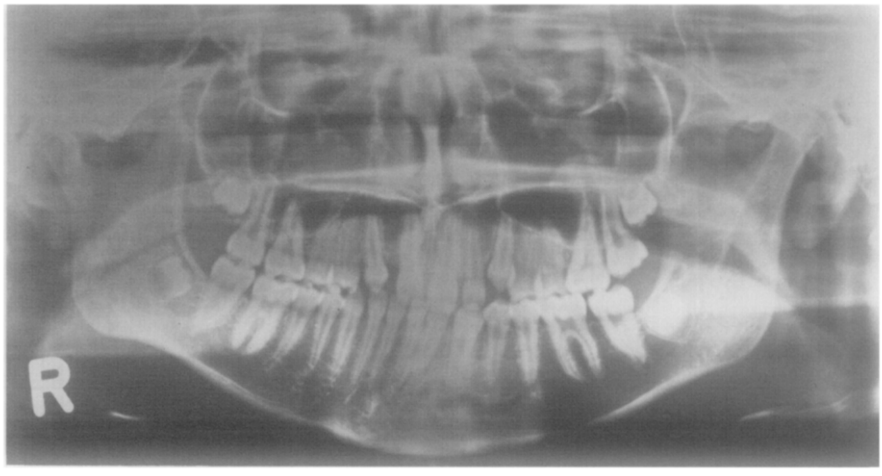


Fig. 11 e

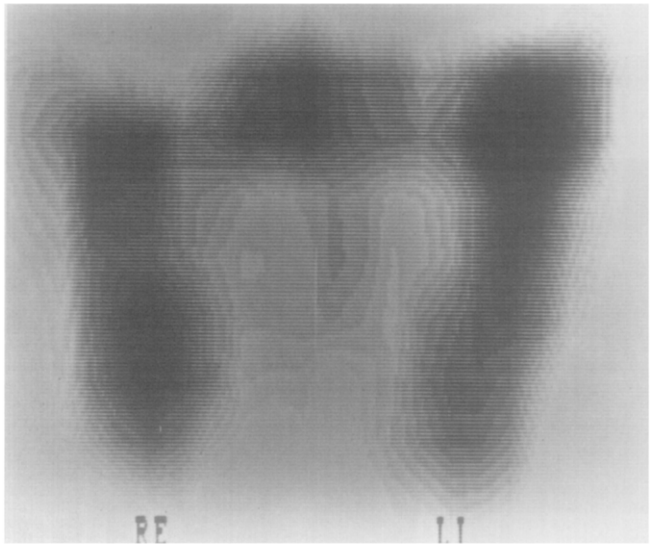


Fig. 11 f

**Fig. 11** An 11-year-old female patient showing a predominant hemimandibular elongation with a degree of hyperplasia present. There is typical displacement of the mandible and chin prominence towards the unaffected side (a). The low-set maxillary teeth and a tilted occlusal plane are evidence of hyperplasia (b). The occlusal picture of the primary dentition (c) exhibits no typical findings, only the displacement of the midline. But the orthopantomogram (d) clearly shows the left-sided H.E. with a thickened condyle and a rounded off angle however. The slightly increased height of the corpus in the premolar region and the low level of the maxillary alveolus are indicative of hyperplasia on the affected side of the mandible. They are even more obvious in an orthopantomogram taken 18 months later (e). Scintigraphy with Technetium$^{99}$ (f) reveals very striking hyperactivity in the region of the condyle.

incongruity which is expressed by a well-rounded but obtuse angle. This is very well demonstrated radiographically. In the radiographic p.a.-view of the mandible the hybrid form looks especially grotesque (Fig. 10 g). The coarse trabecular structure of the affected side is also identifiable. The tomogram of the right TMJ plus ramus shows a completely normal condyle and its neck (Fig. 10 h). The condyle of the abnormal side shows some sort of double condyle formation: one part anteriorly and the other part posteriorly (Fig. 10 i). The posterior part lies in the proper fossa, the anterior part seems to have developed its own fossa. The condylar neck is almost as thick as the enlarged width of the condyle and is noticeably long. Compared with the opposite side the angle is clearly more obtuse. All the evidence points, without doubt, to a unilateral hybrid form comprising H.H. and H.E. Both forms are nearly equally well-developed.

According to our patient population, we do not doubt that there exists a hybrid form with highly predominant hemimandibular elongation. Possibly, those cases of hemimandibular elongation which present a typical appearance and occlusal picture of this anomaly, but in which the condyle and its neck are rather plump and the angle is not clearly obtuse, are such hybrid forms with a minor superimposition of the hyperplastic form. The expression of the predominant form of the anomaly on the affected side also dominates the clinical and radiographic picture.

Under some circumstances such hybrid forms are probably already recognizable during childhood (Figs. 11 a–f). An eight-year-old girl exhibited a predominantly hemimandibular elongation, due to which the midlines of the chin and the lower dental arch were shifted to the opposite side. Clear signs of the hyperplastic component were also identifiable: initial tilting of the anterior teeth towards the abnormal side; inferior positioning of the left dental arch and a slanting occlusal plane; the condyle and its neck were enlarged, thickened and elongated; the angle is somewhat rounded and at the same time obtuse, and, compared with the opposite side the horizontal ramus is somewhat increased in height. These are the typical findings which characterize H.H. They are obviously much less pronounced because of the highly predominant H.E., to the classical picture of which they do not belong. The orthopantomogram taken 18 months later permits more clearly the recognition of both anomalies on the left side: the mandible is shifted further over to the right side; the left angle area has become more rounded and the increase in height of the horizontal ramus in the premolar and molar area has become very obvious (Fig. 11 e). Both anomalies terminate at the symphyseal region which is defined by the midline between the roots of the lower central incisors. The hyperactivity in the condylar region can be clearly identified by means of Technetium$^{99}$ (Fig. 11 f).

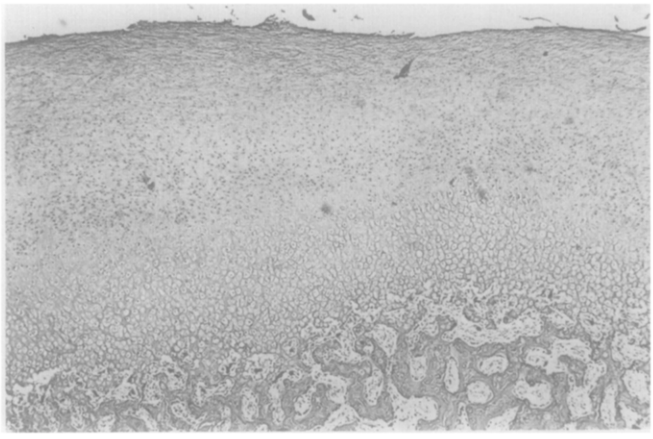

**Fig. 12a** A low-power histological picture from a case of H.H. indicating a homogenous, but diffuse widening of the entire fibrocartilaginous layer. An extremely active ossification process taking place can also be seen (HE, 40 ×).

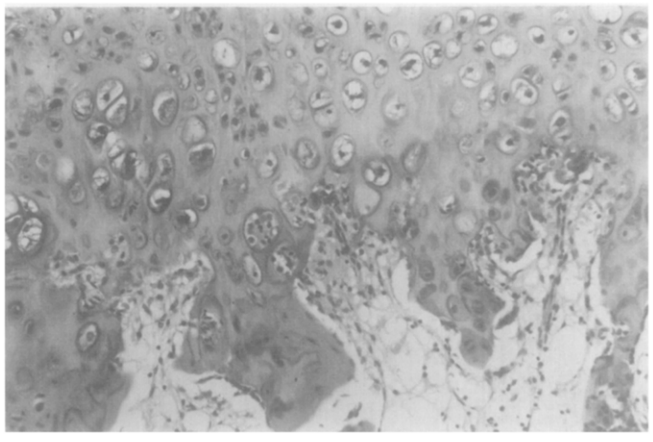

**Fig. 12b** The wide cartilaginous layer within the transition zone showing initial mineralisation of the newly formed osteoid (HE, 130 ×).

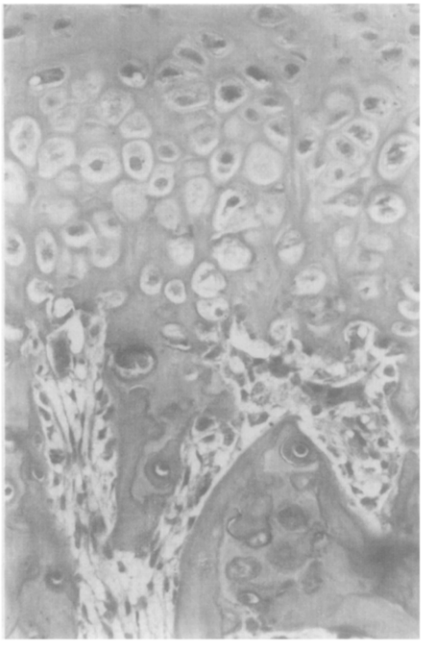

**Fig. 12c** A well-vascularized remodelling area containing multinucleated osteoclasts (HE, 250 ×).

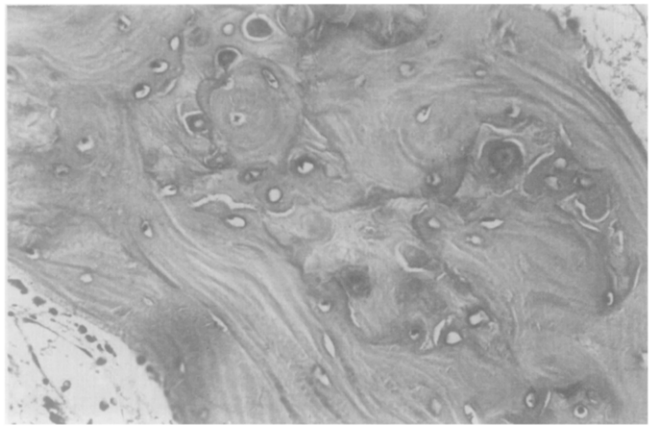

**Fig. 12d** The already mineralized cancellous bone with persisting cartilage islands. Ossification taking place intermittently, leading to the formation of mosaicing (HE, 275 ×).

**Fig. 12** Histology of H.H.

### Histopathological Findings

There are basic clinical and radiographic differences between H.H. and H.E. Since, clinically, both forms originate from the topmost layers of the condyle, a micromorphological examination should enable us to detect the distinctions. It is understandable that these distinctions are possibly only detectable during the prevailing hyperactivity. After the completion of growth histological findings corresponding to the picture of the norm are found in the abnormal side. The macroscopic changes, particularly the enlargement of the condyle, remain discernible. Since most of these anomalies present for correction after the completion of growth, the extirpation of the condyle is no longer necessary because it is histologically less expressive. Despite the few condyles we have examined during the hyperactivity state, we believe that we are able to identify clear distinctions between the two forms.

*A. Hemimandibular hyperplasia*

Histologically, the affected condyle of these cases is covered by a very broad layer of fibrocartilage (Fig. 12 a). The outer fibrous layer is relatively thin and the cells are loosely distributed, spindle-shaped and run parallel with the surface. The next underlying intermediate proliferation zone is

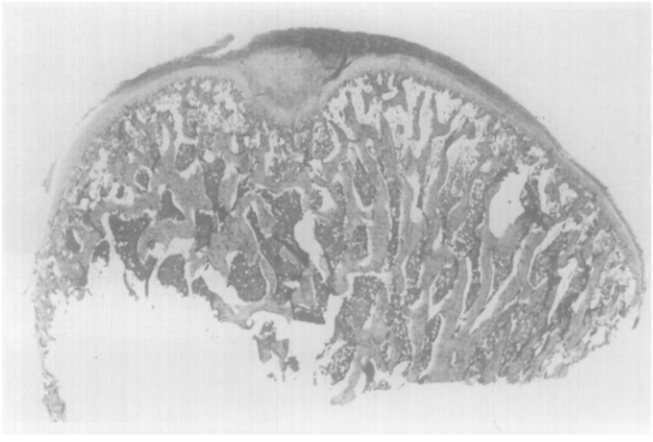

**Fig. 13 a** The picture shows the resected condyle typical of H.E., with a focus of central, cuneiform hyperactive growth extending deep into the spongiosa (HE, 9,5 ×).

**Fig. 13 b**

**Fig. 13 c**

**Figs. 13 b + c** A highly magnified view of the cuneiform hyperactive growth site, showing the lateral (b) and apical (c) parts of it (b + c HE, 40 ×).

**Fig. 13** Histology of H.E.

very broad, cell-rich and exhibits occasional myxoid areas. The transition to the underlying very broadened fibrocartilaginous layer is not clearly defined. The cells are large, the cytoplasma is vesicular and there is abundant newly produced cartilage matrix between the cells (Fig. 12 b). The regions adjacent to the bone show active resorption by many multinucleated giant-cells which lie in small and large lacunae (Fig. 12 c). These areas are well vascularized and sometimes show fresh haemorrhages. In parallel with the osteoclasis there is osteogenesis induced by osteoblasts which lie close together. This network of young and newly formed trabeculae contain soft and ramified cartilage islands at their centres which react positively to Alcian blue (Fig. 12 d). Such cartilaginous structures are also present in the distant spongiosa lamellae which still exhibit active remodelling processes. In the case of H.H. the above described fibrocartilaginous layer is distributed in a diffuse but regular manner all over of the condylar head.

*B. Hemimandibular elongation*

In these cases the fibrocartilaginous layer shows, to a great extent, a normal depth. Depending on the age of the patient it shows either normal active proliferation zones, as in the case of juveniles, or assumes a static stage as in the case of older patients. Unlike H.H. the pathological hyperactive growth focus is characteristically located in the centre of the condyle (Fig. 13 a) and has a cuneiform structure with the tip lying in the newly formed spongiosa (Figs. 13 b, c). The regions of active growth show the same growth and transformation processes as H.H.

*C. Hybrid forms*

There is a hypothetical possibility that the same patient might exhibit hyperactive growth which begins in the form of activity areas (as in the case of H.E.) and then comprises the whole surface of the condyle. This is in fact the case: there are joints in which semblances of cuneiform sclerosed and static bony structures have been observed. Cartilaginous vestiges clearly demarcate these structures from the rest of the bony tissues (Figs. 14 a, b).

## Aetiology

Aetiologically, we could not find any cause for the abnormal development in the case history of these patients. A possible genetic factor seems to be present only in a few

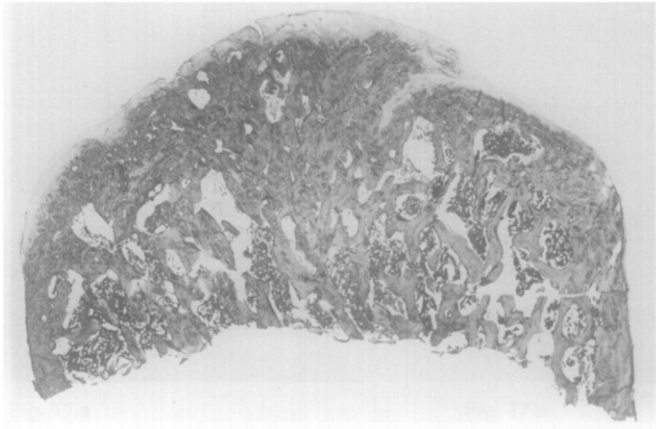

**Fig. 14 a** A histological picture of a resected condyle of the hybrid form (unilateral elongation and hyperplasia) showing growth almost completed. Centrally, a cuneiform sclerosis of the bone (corresponding to the ossification of the pre-existing growth matrix) with a slightly bulging cartilage layer can also be seen. This area is demarcated from the adjacent bone by a thin cartilage layer (HE, 7,5 ×).

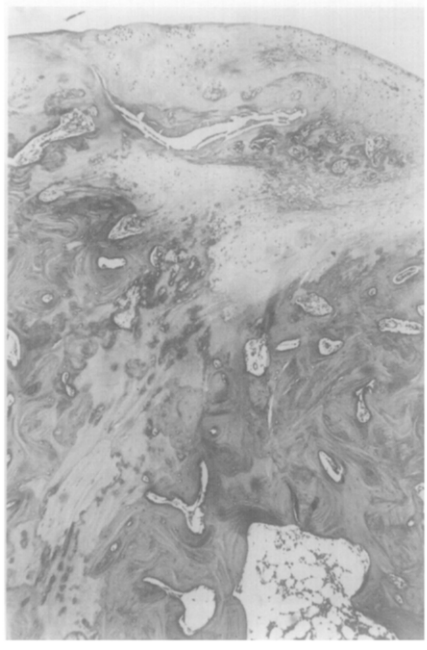

**Fig. 14 b** A part of the picture in Fig. 14 a indicating secondary degenerative alteration taking place in the superficial cartilaginous layer of the condyle (HE, 40 ×).

**Fig. 14** Histology of a hybrid form.

cases of bilateral H.E. These are cases which, associated with micro- or retromaxillism, give the distinct impression of progenia. We do not, however, have positive proof of a genetic aetiology for this bilateral H.E.

## Growth Behaviour

According to information obtained from the patients or their parents, the presence of both anomalies at birth or shortly thereafter is not ascertainable. Generally, they appear at the age of 5 to 8 years and occasionally later. During puberty they are always readily detectable. On average, their growth ceases at the same time as the completion of normal growth or, rarely, only a few years later.
The abnormal formation is arrested after operative removal of the condyle (high condylectomy) of the affected side. It is thus easy to provide clear clinical proof that these anomalies come in to being as a result of an abnormal growth stimulation located within the condyle.

## Therapeutic Principles

It is not the intention of this paper to go into therapeutic details. These are, after completion of growth, the results of exact diagnosis of the prevailing anomaly. The anatomical abnormalities and their therapeutic consequences are always, in the cases with the pure form of one or other of the two anomalies, typical for each case. Therefore, we apply standardized procedures for their correction. In the case of the hybrid forms these standard procedures must be modified in each individual case. Two essential viewpoints must be mentioned however: These concern, on the one hand, therapeutic measures in a case diagnosed during childhood and, on the other hand, in the post-puberty period.
During childhood, the cause of hemimandibular disturbances can be removed early by high condylectomy. Then the mandible must be provided with adequate splinting appliances to obtain normal mouth opening and a normal intermaxillary relationship. This ensures normal movement of the mandible in all directions and normal further growth. The early elimination of the cause is necessary in order to prevent secondary growth disturbances of the contralateral side and of the maxilla; above all to prevent severe disturbance of dental alignment which is difficult to correct. This occurs mainly in the case of H.H.
During puberty and the post-puberty years when facial deformity leads to psychological and social problems for the patient, condylectomy with subsequent treatment by means of the above-mentioned orthodontic splinting should be carried out only when clear scintigraphic evidence of hyperactivity in the articular region is present. Otherwise, we dispense with the operation on the joint and proceed as in the case of adults and inform the patient of possible partial relapse which can necessitate a second operation. This procedure is, above all, indicated in the relatively frequent case of H.E. The correction of the relapse then consists of a unilateral sagittal osteotomy only. The above-

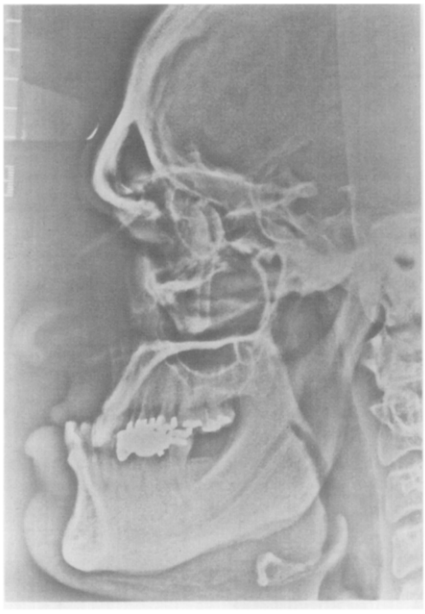

**Fig. 15a**

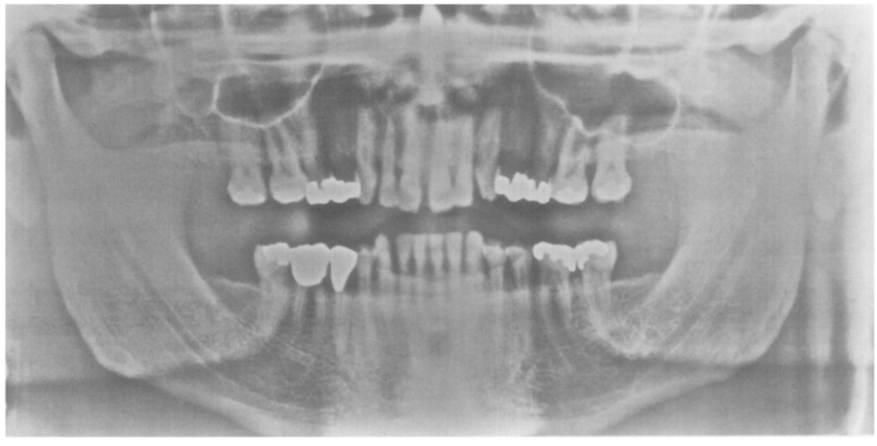

**Fig. 15b**

**Figs. 15a + b** Case of acromegaly with bilateral mainly H.H. shape of mandible with rounded angles and very high mandibular body.

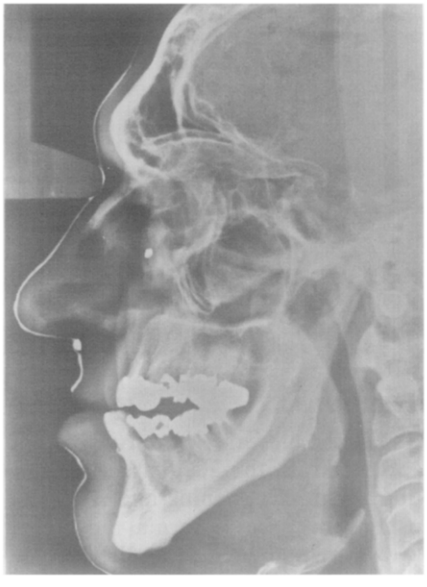

**Fig. 16a**

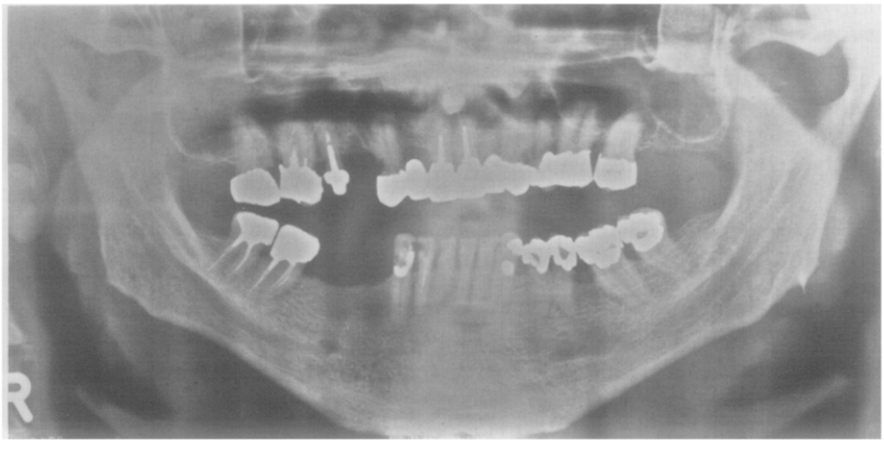

**Fig. 16b**

**Figs. 16a + b** Case of acromegaly with bilateral mainly H.E. shape of mandible with streched angles and almost normal height of mandibular body.

mentioned operation and necessary subsequent treatment has helped the patient to avoid having an operation on the joint.

Possibly, radiotherapy could also arrest the hyperactivity. Radiotherapy would make sense only if it could eliminate the hyperactivity selectively without damaging normal condylar growth and the adnexa.

## Discussion

So-called "condylar hyperplasia" – i.e. H.H. and H.E. – is a condition which is not at all rare, and without a definite aetiology. *Rushton* (1946) presumed a cartilaginous exostosis to be a cause. *Reichenbach* and *Seidler* (1948) on the other hand presumed articular arthrosis to be a cause.

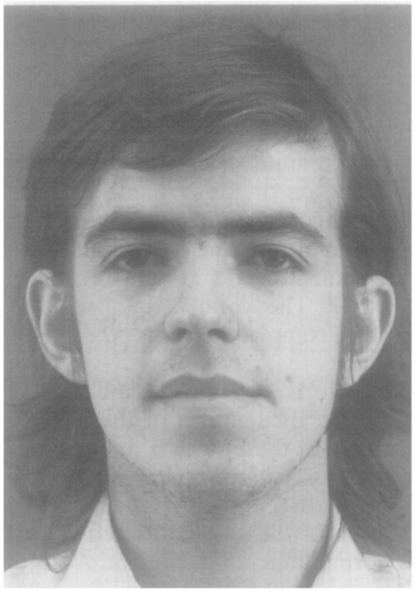

Fig. 17a

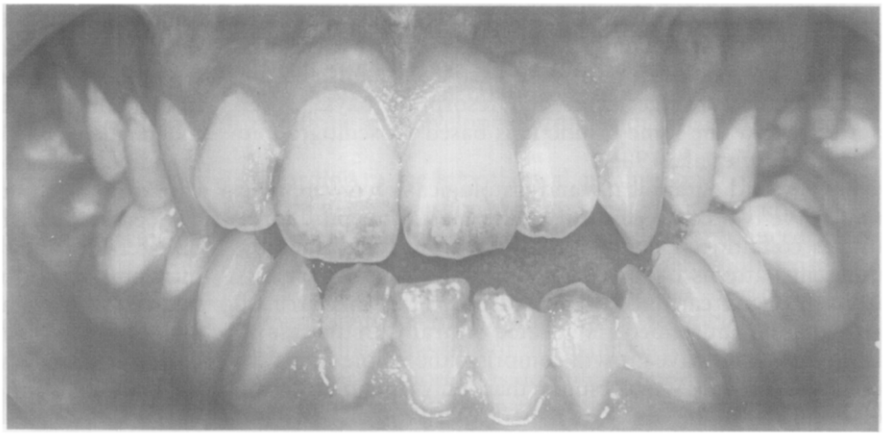

Fig. 17b

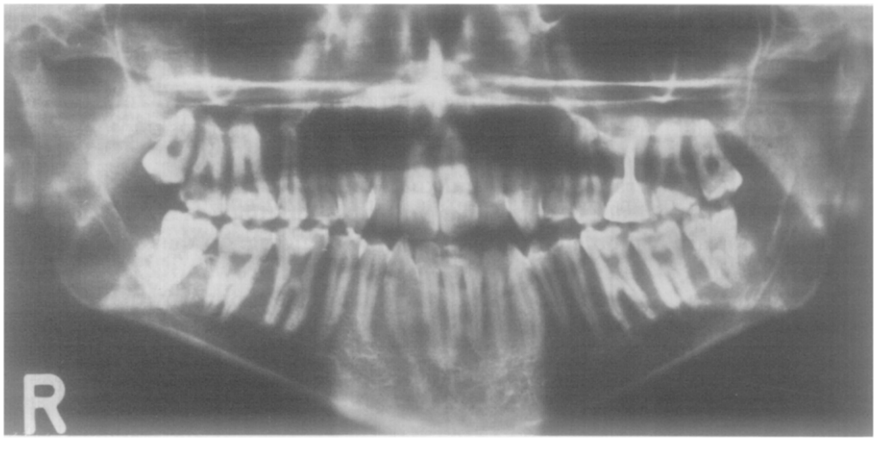


Fig. 17c

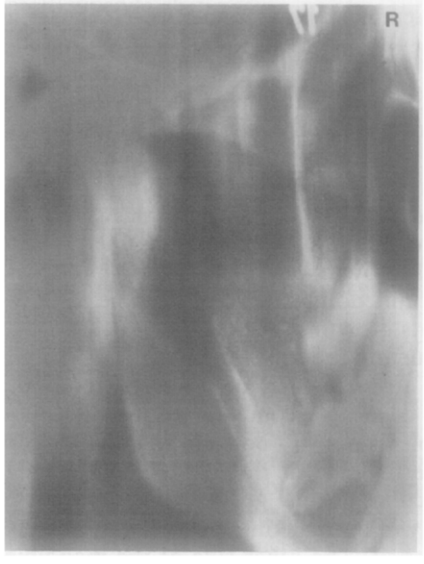


Fig. 17d

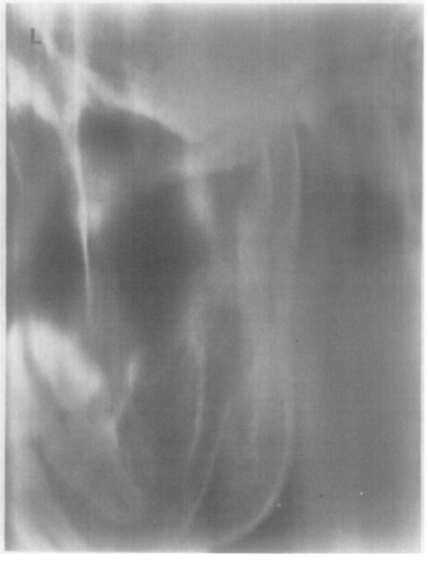


Fig. 17e

**Figs. 17a–e** A 21-year old patient showing excessive right-sided hemimandibular elongation.
As a result of excessive unilateral growth there is a characteristic front appearance showing marked mandibular asymmetry (a). In the case of unilateral excessive growth there is not only midline displacement of the lower dental arch but also an open bite in the incisor region of the unaffected side (b). The orthopantomogram (c) and tomograms of the condyles (d + e) show clearly that, although there is a striking elongation of the right mandible and a displacement towards width left by a tooths width, the condylar neck is relatively plump and there is only a suspicion of widening of the mandibular angle. We interpret this as a sign of minor participation of the hyperplasia factor.

*Egyedi* (1969) believed that vascular disturbances in the condylar region such as arteriovenous anastomosis and fractures were responsible for this condition. In all histologically well documented and observed cases, only actively growing cartilage could be demonstrated (*Rushton*, 1946; *Van Zile*, 1954; *Broadway*, 1958; *Sear*, 1972). *De Burgh Norman* and *Painter* (1980) make histological distinctions between active and inactive forms of condylar hyperplasia based on the existing intraosseous cartilage islands and the depth of the cartilage layers and resorption bays. Other authors make the same distinctions based on scintigraphic data (*Hampf* et al., 1985).

Clinically, radiographically and histologically we were able to observe two completely different types of the pure form of this condition. Histologically they are distinguished by different patterns of distribution of the areas of hyperactively growing cartilage. In the case of H.E. this area of growth is circumscribed and located centrally. In the case of H.H. the hyperactive growth comprises the whole fibrocartilaginous layer. We presume that only the pattern of distribution of the areas of hyperactive growth (focal or diffuse) within the same histological picture of active areas is responsible for the different clinical appearances. The combinations and variability of the clinical appearances could also be explained by our hypothesis.

## Hypothesis of the Pathogenesis

It is a fact that when H.E. and H.H. develop there is a scintigraphic evidence showing clear hyperactivity in the condyle on the affected side of the mandible. It is a fact, furthermore, that the resection of this condyle leads to the arrest of the abnormal growth. It is, therefore, in the clinical experiment as well as histologically provable that the stimulation mechanism of these two hemimandibular anomalies lies unequivocally within the top layer of the condyle. Thus, to us it seems justifiable to assume that these two anomalies must be the result of hyperactivity or hyperproduction of two different growth regulators which, lying within the fibrocartilaginous layer of the condyle, affect either the longitudinal or the expansile growth of the same side of the mandible. This fact seems to uphold the hypothesis that "the condyle is a major field of growth, . . .. serving as a master centre" (*Enlow*, 1982). From our clinical and histological observations we draw the conclusion that the genetically determined form of one side of the mandible develops only if there exists adequate equilibrium not only between the two growth regulators within the condyle itself but also between the other growth regulators of the body as a whole.

Clinical experience in traumatology and in the treatment of TMJ-ankylosis during childhood, arthritis of the TMJ during infancy, and experience in the reconstructive surgery of mandibular defects seem to confirm the hypothesis that on the one hand growth regulators are situated in the condyle and on the other hand that growth stimulation of the mandible depends on the functional matrix. Thus, the mandible continues its normal growth after resection of the condyle on account of ankylosis during early childhood, and restoration of normal mandibular function. The remaining growth deficiency amounted only to that existing prior to the ankylosis operation. Untreated ankylosis leads to increasing hypoplasia of the mandibular side in question, despite the presence of a traumatically dislocated condyle. The unaffected side of the mandible continues to grow normally despite the functional arrest. This leads to an asymmetrical bird-face. Thus, on the one hand, the condyle constitutes a growth regulator independent of the functional matrix as long as it maintains a normal anatomical relationship to the mandible; on the other hand, the condyle loses its regulatory capacity when it dissociates itself from the articular fossa and lies in the soft tissues. But mandibular function also constitutes a growth regulator, independent of the condyle. The condyle by itself and mandibular function seem to complement each other in the case of loss of one or other.

The growth of the mandible is retarded in the case of unilateral or bilateral juvenile rheumatism of the TMJ or inflammation of other cause. In these cases a unilateral or bilateral micromandible arises whereby all parts of the mandible lag behind in growth. This refers to the loss of condylar growth stimulation of the whole affected side of the mandible, although normal function is present. According to radiographic and histological findings the inflammatory damage takes place only in the superficial layer of the condyle.

If, during childhood, in the case of tumour of the condyle, one side of a mandible including the periostium is resected and reconstructed by means of a bone graft from the iliac crest, then normal growth of the transplant occurs if perfect function is attained. The bone graft grows in just the same manner as does the contralateral half of the mandible. This has been shown by the results in several cases in our clinic (*Sailer*, 1976).

Traumatology and the example of ankylosis show that a dislocated condyle with loss of function is not able to stimulate the growth of the mandible on that side. But the other side with its intact condyle grows to a great extent normally despite the loss of function. But growth regulation functions normally after the restoration of function despite the removal of the dislocated or ankylosed condyle. Even the example of growth of an iliac crest bone graft proves that the functional growth regulator of the mandible of a child must not lie within the mandible itself. Function itself induces resorption and apposition on the bone graft and brings about its growth.

In cases of reconstruction of large mandibular defects in childhood we have never seen the development of an anomaly like H.H. and H.E. arising within the grafted area. Such a case has also never been reported. The excessive longitudinal growth of a costo-chondral graft following its use for reconstructing the ascending ramus and condyle during childhood cannot be presented as proof of a functionally induced excessive longitudinal growth of a given bone graft. The costo-chondral graft contains the growth centre of a rib. This growth centre of the rib produces growth even without function. It could at most be equated to the condyle with its growth potential.

The facts are consequently the following:

1. H.H. and H.E. are arrested when the condyle is resected.
2. The ankylosed or dislocated condyle, lacking the function of the mandible, is not in a position to influence the growth of the affected side of the mandible. After the restoration of function, normal growth of the mandible proceeds though without making up for the already existing growth deficit. The other side of the mandible grows normally despite the lack of function and results in

an asymmetrical bird-face, if the condyle on this side is intact.

3. A bone graft from the iliac crest used to reconstruct one side of a mandible, even without possessing a condyle, follows the normal growth pattern of the other intact side if good function is guaranteed.
4. Cases of H.H. or H.E. within a reconstructed mandible or within a mandible whose condyle has been resected have never been reported.

On the basis of these facts we are of the opinion that the condyle and mandibular function are growth regulators which can complement each other during the course of pattern-specific growth. Furthermore, we are convinced that the condyle is the sole centre of stimulation for growth patterns of the mandible different from that determined genetically. Thus, we dare assume that the fibro-cartilaginous layer of the condyle does possess a growth programming ability. Many authors deny this fact.

In consequence of our observations, it is suggested that the condyle can be the pacemaker of mandibular growth but not the sole one. The condyle is certainly the regulator of abnormal growth, be it excessive or deficient. The condyle also possesses the potential to produce forms different from those determined genetically. We must, therefore, acknowledge that the fibrocartilaginous layer of the condyle possesses, without doubt, the function of a growth zone. We thus uphold the statement of *Enlow* (1982) "The current thinking is that the condylar cartilage does have a measure of intrinsic genetic programming".

More important is the following question: how does the fibrocartilaginous layer influence the growth form? It is certain that there is no bone production starting in the condyle and extending to the ramus and corpus. But what enables this growth centre to influence the form of the mandible decisively by producing typical forms?

It is generally accepted (*Enlow*, 1982) that the growth of the mandible proceeds not from a growth centre as is the case with rib or long bone, but by means of apposition and resorption from the front towards the joint. Accordingly, this apposition and resorption must be controlled by the growth regulators in such a way that H.H., H.E. and the hybrid form or a genetically determined or hypoplastic mandible arises. The latter occurs regularly in the case of early juvenile rheumatism or after inflammatory processes or after irradiation of a child's TMJ.

Since we have detected noticeable differences among the histological pictures of the fibrocartilaginous layer with the production of bone covering the whole of the condyle or just centrally located, we assume, without doubt, that the growth regulators are contained within the cells of these histological variations. Since direct longitudinal growth coming from the joint cannot cause the above described variations, the cause must be another agent. We know such an agent in the case of acromegaly in which a hormonal stimulation of the growth of the mandible induces its overgrowth. Is it conceivable that some factors or stimuli, which can influence the growth of the affected side of the mandible, might be produced in the growth zone of the condyle?

According to the hypothetical assumption that two factors or stimuli, one responsible for the longitudinal growth and the other for the increase in bulk, are produced in the condyle, it would be conceivable that there could exist more or less predominant production of one or other. If the hyperactivity or overproduction of the two stimuli takes place in the same side of the mandible, then a hybrid form arises. Furthermore, it is doubtless conceivable that each of the two anomalies can affect any side of the mandible independent of the other to give rise to the combined form. Further, one and the same form of anomaly can have the same or different degrees of expression in both sides of the mandible. The hypothesis of the production of growth stimuli would also explain how production of one of the growth stimuli can take place at a varying intensity thus permitting the development of various degrees of the same anomaly.

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In both anomalies pronounced growth can lead to an open-bite. In the hyperplastic form the open bite is on the affected side particularly in the molar region. Here the growth of the alveolar processes of the maxilla and mandible cannot bring about occlusal compensation for the excessive longitudinal growth of the ascending ramus. In the case of unilateral hemimandibular elongation it can likewise lead to an open bite if there is a rapid longitudinal growth thrust. Depending on the intensity of the longitudinal growth the open bite can be variably situated. It arises rather in the region of the canine of the unaffected side, or more in the anterior than in the molar region of the abnormal side (Figs. 17 a–e).

Then the protrusive growth of the affected side and its displacement towards the opposite side, together with the increasing opening of the mandibular angle, seem to enter into conflict with the unaffected side. Due to the exclusive lateral displacement of the mandible it seems that the power of the growth thrust of the abnormal side cannot be compensated for any more. The same also applies to the unilateral hybrid form (Fig. 10).

### Anlage-Induced Anomaly or Disease

For insurance purposes it is important to put one more question. Are we dealing with a malformation of congenital or anlage-induced nature or is it, rather, a special clinical entity? We tend unequivocally to the assumption that we are dealing with products of abnormal local growth stimulation because, as stated earlier, we have not found any positive genetic cause for it. Thus, they are equivalent abnormalities of the mandible to those skeletal abnormalities which are due to a central disturbance of growth stimulation as in acromegaly which is induced by a pituitary adenoma. Accordingly, we are of the opinion that it is a

real disease like acromegaly and not a form of anlage-induced malformation of the mandible.

## Conclusions

According to our cases and clinical observations there is no doubt that hemimandibular elongation and hemimandibular hyperplasia are two different clinical entities of one side of the mandible. They originate from the condylar fibrocartilaginous layer. This is due to the fact that H.H. and H.E. arise only in the presence of an intact condylar cartilage and are arrested after the removal of the condylar cartilage. Histologically and scintigraphically a clear hyperactivity is demonstrable in the region of this cartilaginous layer during the active stage of the pathogenesis of the mandibular malformation. Since hyperactivity by itself cannot directly induce one of the two anomalies by means of local bone production, as is the case in long bones or rib, it must either constitute a stimulus for the abnormal growth or produce one which has an effect on the whole side of the mandible. The growth stimulus is unequivocally contained within the cells showing hyperactivity. Therefore, these cells must be able to produce two different stimuli, one responsible for the longitudinal growth and the other responsible for the increase in bulk. Since the production of the stimuli, even if the extent varies, takes place in the same spot, the emergence of hybrid forms, as we have found clinically is possible. It is further conceivable that the hyperactivity can show varying intensity which then leads to different degrees of severity of the abnormal shape.

We believe that our clinical, radiographic and histological observations of the two forms of abnormal growth on the one hand and their comparison with other pathological forms of the condyle on the other hand can contribute to the understanding of mandibular growth. Our hypotheses on the development of the two anomalies would permit the bringing of some light into the darkness of the understanding of a large number of mandibular abnormalities.

For better understanding of mandibular growth and of the two above described clinical pictures and, may be, for the sake of early treatment, it would be desirable if such cases could be reproduced in an experimental animal study.

In the case of florid growing forms the therapeutic approach consists of early removal of the condylar growth zone and post-operative functional treatment. Radiotherapy exclusively for the elimination of the hyperactivity seems to us to be of doubtful benefit.

### References

*Broadway, R. T.:* Two cases of Unilateral Hyperplasia of the Mandibular Condyle. Proc. R. Soc. Med. 51 (1958) 23

*De Burgh Norman, J. E., D. M. Painter:* Hyperplasia of the Mandibular Condyle. A Historical Review of Important Early Cases with a Presentation and Analysis of 12 Patients. J. max.-fac. Surg. 8 (1980) 161

*Egyedi, P.:* Aetiology of Condylar Hyperplasia. Austr. Dent. J. 14 (1969) 12

*Enlow, D. H.:* Handbook of Facial Growth. 2nd Edition. Saunders, Philadelphia, 1982

*Hampf, G., A. Tasanen, S. Nordling:* Surgery in Mandibular Condylar Hyperplasia. J. max.-fac. Surg. 13 (1985) 74

*Hovell, J. H.:* Condylar Hyperplasia. Br. J. Oral Surg. 1 (1963) 105

*Obwegeser, H. L., M. S. Makek:* Hemimandibular Hyperplasia/Hemimandibular Elongation. Abstr. European Association for Maxillo-Facial Surgery, 7th Congress, September 1984, Paris

*Reichenbach, E., H. Seidler:* Über die sogenannte Hypertrophie des Kieferköpfchens. Dtsch. zahnärztl. Z. 3 (1948) 806

*Rushton, M. A.:* Unilateral Hyperplasia of the Mandibular Condyle. Proc. Royal Soc. Med. 39 (1946) 431

*Rushton, M. A.:* Unilateral Hyperplasia of the Jaws in the Young. Int. Dent. J. 2 (1951) 41

*Sailer, H. F.:* Zum Problem des Wachstums von Knochentransplantaten zum Unterkiefer. Fortschr. Kiefer- u. Gesichtschir., Vol. 21 Thieme, Stuttgart 1976

*Sear, A. J.:* Intra-oral Condylectomy applied to Unilateral Condylar Hyperplasia. Br. J. Oral Surg. 10 (1972) 143

*van Zile, W. N.:* Unilateral Hyperplasia of the Mandibular Condyle. J. Oral Surg. 12 (1954) 275

*Prof. H. L. Obwegeser, MD, DMD*
*Clinic of Maxillofacial Surgery*
*University Hospital*
*Frauenklinikstraße 10*
*CH-8091 Zürich/Switzerland*

*M. S. Makek, MD*
*Institute of Pathology*
*University Hospital*
*Schmelzbergstraße 12*
*CH-8091 Zürich/Switzerland*