

CLASS III MALOCCLUSION - ORTHOPAEDIC TREATMENT
IN THE DECIDUOUS AND MIXED DENTITIONS

by

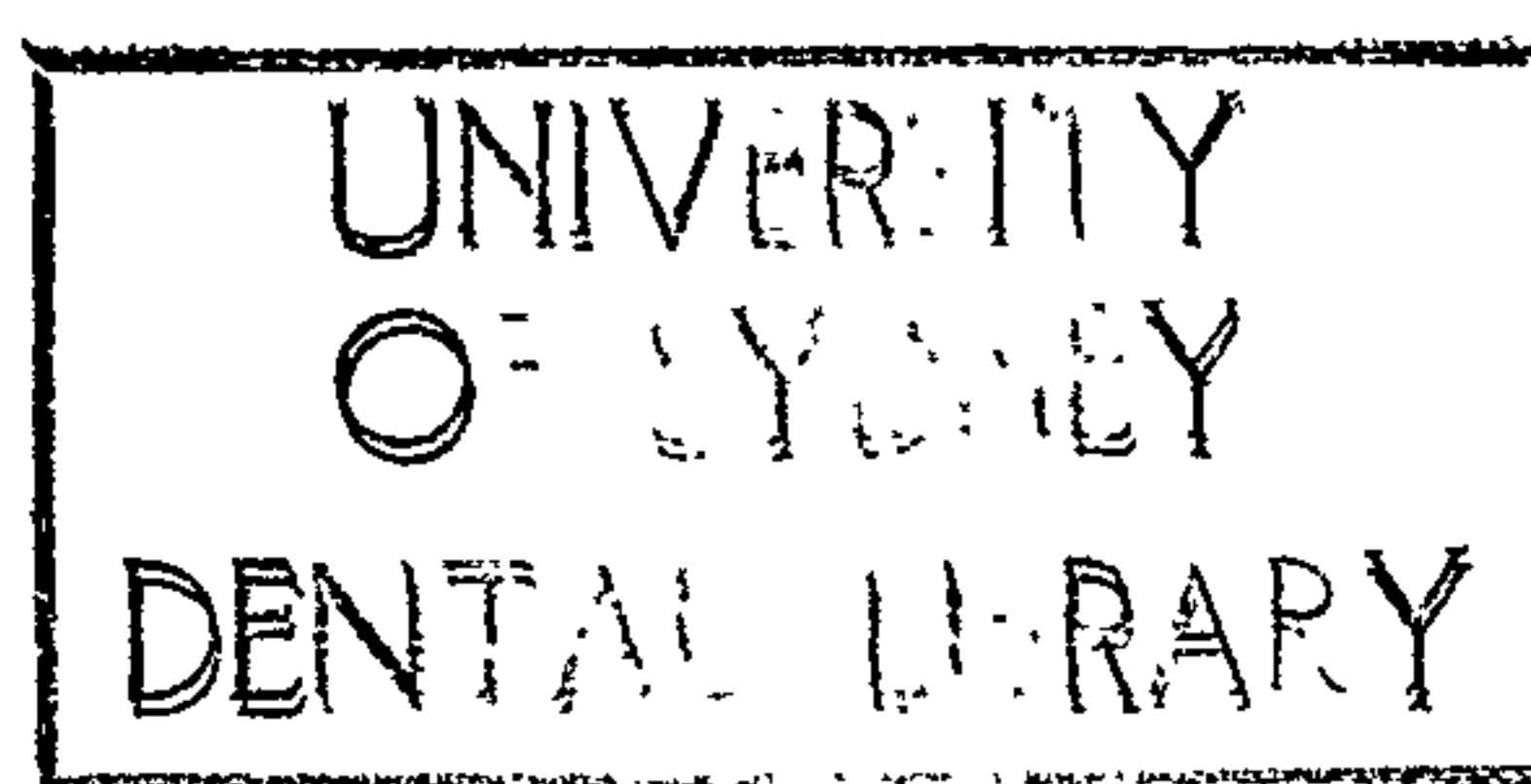
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A treatise submitted in partial fulfilment
of the requirements for the degree of
Master of Dental Surgery

DEPARTMENT OF PREVENTIVE
DENTISTRY

UNIVERSITY
OF SYDNEY

1986



Acknowledgments

I wish to thank Associate Professor Keith Godfrey who, as my supervisor, provided encouragement and direction during the writing of this treatise. The rapidity of his corrections (frequently at short notice) is greatly appreciated.

Associate Professor Peter Barnard provided assistance with the technical aspects of the treatise - particularly the format.

Dr Susan Howell, Head of Unit (Orthodontics) and Professor Keith Lester, Director of Dental Services, Dental Clinical School, Westmead Hospital, generously allowed time off for frequent library visits.

To my wife, Tristy, and our children, Nicholas, Aimee and Christopher, I owe thanks for undying support, encouragement and understanding during the period of writing.

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INTRODUCTION

Class III malocclusions provide one of the greatest challenges in orthodontics.

Their aetiology, diagnosis and treatment have been extensively researched and widely divergent views on treatment philosophies developed.

Early orthopaedic treatment of established and developing Class III malocclusions has undergone a resurgence in recent years in spite of the lack of consensus of opinion regarding its efficacy.

This lack of consensus includes the criteria by which the success of the treatment is assessed.

Ideally, a successful early treatment should fulfil two basic requirements.

1. elimination of acute problems associated with existing or imminent damage to the teeth, soft tissues, efficacy of the masticatory apparatus and psychological and social development of the child.

2. provide some long term benefit that will reduce (or eliminate) the complexity of future orthodontic or surgical treatment.

The aim of this treatise is to examine the hypothesis that Class III malocclusions can be successfully treated by orthopaedic means in the deciduous and mixed dentition, on the basis of the above requirements.

If the hypothesis is to be supported, ample evidence should exist in the literature to support the following statements.

1. Accurate diagnosis of specific forms of Class III malocclusion can be made and this diagnosis be related to specific treatment modalities.
2. Currently proposed growth mechanisms are consistent with observed clinical responses to treatment techniques in current usage.
3. Clinical alterations in the growth rate and direction of facial bones, and to skeletal morphology are consistently achievable, reproducible and predictable.

CHAPTER ONE

DESCRIPTION AND CLASSIFICATION

1.1 The Variability of Class III Malocclusions

Angle (1907) described the Class III malocclusion as one

where there is a mesial occlusion of the mandibular teeth by more than one half the width of a single cusp.

Although a widely used method of classifying malocclusion, it does not provide the information regarding the underlying skeletal morphology or functional activity of the mandible which is required to accurately identify various forms of Class III malocclusion. A characteristic of Class III's in general is their great variability, and a range of morphological types satisfy Angle's description.

Descriptions based on skeletal morphology have been proposed by a number of authors.

Kloepel (1970) stated that, originally, all forms of class III malocclusion were defined by the term "inferior prognathism".

A psychiatrist, Meier, used the term "progenia" in 1868 and believed that an excessively large mandible was a symptom of debility and epilepsy (Rakosi 1981).

During the Nineteenth Century, a number of terms were used (Table 1.1) to describe Class III malocclusions - some of which were decidedly uncom-

plimentary - though few defined the degree to which skeletal dysplasia was responsible for the malocclusion.

Author	Year	Terms
Majolin	1832	Inversion
Kneisel	1836	Oblique position
Carabelli	1842	Protruding set of teeth
Linderer	1834	Senile chin
Welker	1862	Opisthognathy
Magitot	1877	Anteversion
Wedl	1867	Dog's mouth
Iszlay	1891	Epharmosis
Meier	1868	Progenia

Table 1.1 Terms used to describe mandibular prognathism from Rakosi and Schilli (1981)

Belinfante (1982) described Class III malocclusion as a "syndrome composed of Class III malocclusion, vertical height dysplasia of the anterior mandible and midfacial (suborbital/zygomatic area) deficiency".

As will be seen in following chapters, this rather broad statement does not apply to all Class III's. A characteristic of Class III malocclusion is its great variability and forms may be encountered which exhibit one or all of the features listed by Belinfante (1982).

Graber (1972) observed that most Class III's also exhibited anterior crossbite, and often lingually inclined lower incisors, constricted maxillary arch, maxillary arch length deficiency and maxillary dental arch crowding. These features are not necessarily always seen together in Class III's.

Bryant (1981) used mandibular morphology as a basis for classification. He described two groups

1. low gonial angle (deep overbite)
2. high gonial angle (open bite).

Similarly, Jacobson, Evans, Preston and Sadowsky (1974) divided mandibular morphology into two groups:

1. Divergent type which was characterised by
 - a. anteriorly divergent palatal, occlusal and mandibular planes
 - b. an obtuse gonial angle
 - c. anterior open bite.
2. Convergent type consisting of
 - a. less divergent (tending to parallelism), palatal, occlusal and mandibular planes
 - b. less obtuse gonial angle
 - c. deep overbite.

In addition, these authors claimed that Class III malocclusions differed from other types of malocclusion primarily in mandibular morphology and anterior cranial base length (reduced in Class III).

Schudy was quoted by these authors as describing the two groups as hyperdivergent and hypodivergent. These are probably more accurate terms as the planes mentioned by Jacobson et al. (1974) rarely converge.

Turpin (1981) (from Campbell, 1983) listed morphological features of the jaws that may be associated with Class III malocclusion.

1. large or protrusive mandible

2. deficient or retrusive maxilla
3. protrusive mandible and retrusive maxilla
4. protrusive mandibular dentition
5. retrusive maxillary dentition.

Pseudo Class III or postural mandibular protrusion (Grossman, 1970) occurs where the skeletal abnormality is minor or non-existent (Cozzani, 1981). These forms are essentially the result of anterior mandibular displacement due to the absence of a stable occlusal position in a Class I (Angle's) molar relationship.

Kawata, Kishingami, Yamashita, Uyama, Toda and Uemura (1982) assessed the position of the condyles in centric occlusion and related this to the dental relationship. True skeletal Class III was characterised by a Class III molar relationship with the condyles seated in their fossae. Postural Class III's exhibited forward positioning in the glenoid fossae and a Class III molar relationship, indicating anterior shift of the mandible.

The variety in terms and methods used to describe and classify (respectively) Class III malocclusions is indicative of the wide variation in presenting features found with this abnormality. This is graphically demonstrated in Fig. 4.1 (page 72). Here, Schwarze (1970) shows the morphogenetic expression of Class III features in a family with a strong tendency towards this form of malocclusion.

It could be concluded that for any description and classification to provide meaningful diagnostic and prognostic information, more features must be assessed. These should be related in some way to the aetiology and

treatment alternatives. From this some assessment of prognosis may be made for individual cases.

A more detailed review of the literature will be made in Chapters 2 and 4 with regard to these statements.

1.2 Early Treatment

In the context of this treatise, early treatment refers to treatment instituted in the deciduous and mixed dentition stages. It will be seen that "orthopaedic" techniques may involve a long treatment period, and may often be only the first phase of the overall treatment plan.

Class III malocclusions are generally a problem for everyone associated with them:

For the general practitioner, when to refer; for the orthodontist, when to treat and for the oral surgeon, the variety of forms and surgical procedures. (Moss, 1976)

Campbell (1983) claimed that failure to treat these patients early "predetermined that many young people must experience facial and dental disfigurements which could have far reaching psychological and physical ramifications during the most important formative years of their lives".

This view is subjective and not supported by evidence relating to the influence of Class III features alone on the psychological and social development of children. Nor is the assertion quantified in any way with respect to the degree of severity of the abnormality which is likely to cause these untoward effects.

Other authors have offered subjective views supporting early treatment.

Nanda (1980) described prognathism as a handicapping disfigurement.

Rakosi and Schilli (1981) claimed that first impressions of people are often gained from facial appearance and proportion and quoted Lavater (1802) as stating that "a face is dull when the lower part is bigger than one of the two upper parts."

The potentially damaging effects of developing Class III malocclusion is not restricted to the psychological effects of poor aesthetics.

Graber (1975) asserted that there is a tendency for Class III faces to become more prognathic and that this may have adverse effects on muscle function and tooth position. Further, he stated that the upper lip is active in Class III cases and is drawn against the maxillary anterior teeth and that this may have the effect of restricting normal maxillary growth. Fränkel (1972) claimed that the effect on the upper anterior teeth may be due more to a hypofunctional lower lip than to a hyperactive upper lip.

The inference drawn from these two views is that correction of the aberrant labial muscle activity may retard the development of Class III features.

The influence of soft tissue function on growth is an active field of research. The significance of soft tissue activity on the development of Class III malocclusions is discussed more fully in Chapters two and three.

The elimination of occlusal interferences associated with anterior and lateral functional mandibular shift has been advocated to remove restrictions on normal alveolar growth in the maxilla (Bell and Le Compte, 1981; Nakashima, Ichinose, Nakata and Takahama, 1982, Bell, 1982).

Correction of the aberrant pattern of mandibular movement at an early age would, these authors claim, allow maximum use of the maxillary growth period and thus reduce the extent of the skeletal discrepancy.

This controversial view warrants further discussion, and this is presented in Chapters 2 and 3.

This review gives an indication of the range of reasons for instituting early treatment in Class III cases. They all rest on the basic assumption that developing Class III growth patterns may be intercepted, corrected or reduced at an early stage.

This view has opponents as well as supporters.

Mills (1983) asserted that the amount of condylar growth is unaffected by extrinsic factors and thus early orthopaedic treatment of mandibular prognathism is unwarranted.

Björk and Skieller (1983) examined in detail mandibular growth patterns in longitudinal studies. Their conclusions conformed with the view that excessive mandibular growth is an expression of the genetically predetermined growth potential.

It can be seen from the foregoing that the decision to treat early in Class III cases may, in many cases, be largely subjective, and based on the attitude that "we must do something" rather than "we can treat this patient now because there is scientific evidence to prove the treatment is potentially successful".

The following chapters will aim to provide a basis to help rationalise early treatment of Class III problems, with particular emphasis on orthopaedic techniques.

The emphasis on evaluation of orthopaedic appliances is justified on the basis that my literature review has revealed that the evidence relating to their effectiveness is controversial and frequently inconclusive.

CHAPTER TWO

AETIOLOGY OF CLASS III MALOCCLUSIONS

Of Class III malocclusions, Angle (1907) asserted:

Deformities under this class begin at about the age of the eruption of the first permanent molars, or even much earlier, and are always associated at this age with enlarged tonsils and the habit of protruding the mandible, the latter probably affording relief in breathing ...

Further, he stated:

The time honoured custom of attributing these conditions to heredity and degeneracy has, in the author's opinion, no substantial support.

Since Angle's time a considerable amount of speculation and effort has been directed towards attempting to elucidate the relationship between genetic and environmental factors and the development of Class III malocclusions.

2.1 Genetic Factors

There are numerous references to the hereditary basis for Class III malocclusions, although frequently these are subjective observations that provide little evidence to support the statements and little or no understanding of the mechanisms of inheritance.

Pac (1970) quoted Hauple as stating that Class III malocclusion is an hereditarily conditioned overdevelopment of the lower jaw.

Jacobson, Evans, Preston and Sadowski (1974) asserted that there

appeared to be a strong genetic influence determining the occurrence of Class III characteristics.

Rakosi and Schilli (1981) stated simply that in Class III malocclusions, growth of the mandibular base is predetermined by heredity.

The most revealing data has been gathered from investigations of monozygotic (MZ) and dizygotic (DZ) twins. In addition, studies of siblings, parents and children of Class III patients has provided significant information with regard to the genetic basis of malocclusion (Nelson 1969).

MZ twins develop from a single ovum, separating symmetrically early in embryonic life and thus are genetically identical. A rare exception to this is when non-dysjunction of the chromosomes occurs during anaphase of meiosis with the result that unequal amounts of chromosomal material is distributed to the embryos (Nelson 1969).

It should be possible to assume that morphological differences between MZ twins are due to environmental influences on the developing dentition or skeleton or, as an epigenetic factor, on the genomic expression.

DZ twins develop from two separate ova and thus are no more alike than siblings (Nelson, 1969).

Markovic (1970) stated that a high degree of concordance for a certain trait in MZ twin pairs compared with a low degree of concordance for the same trait in DZ twin pairs supports the view that genetic factors are causative in the development of that trait. However, if a similar

concordance rate is observed in both types of twins then non-genetic (environmental factors) should be considered.

2.1.1 Family investigations

Probably the most famous family investigation of Class III malocclusion was that carried out into the Hapsburg family who demonstrated class III characteristics over eleven generations (Haeker, 1911; Brash, 1956).

This family was characterised by having a long narrow mandible and a small maxilla. In conjunction with the large beaked nose, the appearance of those members of the family affected was quite distinctive.

These features were evident in family portraits from Emperor Maximillian I in the Fifteenth Century down to Alfonso XII of Spain who died in 1941.

With the exception of twin studies there have been very few other family investigations relating specifically to Class III hereditary patterns.

Pac (1970) quoted Rubrecht and Korkhaus (no reference provided) as observing the appearance of Class III malocclusion through several generations.

Schulze and Wise (1965, in German from Schwarze 1970) made similar observations during "their genetic research on several families".

Suzuki (1961) examined a large sample of 1362 persons in 243 Japanese families and found:

1. in families with a history of prognathism, 34.3% of the family demonstrated the trait;
2. in families with no history, 7.5% of the family was affected;
3. in five families where both parents were affected, 40% of the children were also affected;
4. with one prognathic parent, 20.2% of children were prognathic.

He concluded that a complicated hereditary mechanism was involved.

Iwagaki (1938) examined 2461 students and found an incidence of 6%.
He found:

1. when the mother was prognathic, 18% of children were also prognathic;
2. with the father only prognathic, 31% of children were prognathic;
3. when neither parent was prognathic, 4% of children demonstrated prognathism.

Iwagaki concluded that prognathism is familial and perhaps Mendelian recessive, although the latter conclusion was not statistically tested.

Eismann (1976) assessed "fifteen essential criteria" and assigned numerical values to these. The summation provided a score for each subject of the investigation which was an indicator of the presence of Class III characteristics.

He found that the scores were highest in subjects, followed by siblings, mothers and lowest in fathers.

He stated that

If multifactor polygenic inheritance controls these variations the number of children affected and the extent of morphological abnormality must show a dependence in family investigations.

On the basis of the apparent relationships listed above he concluded that the results were consistent with a polygenic multifactorial inheritance system.

Whilst the connection between his conclusions and the results of his family investigation as he presented them is somewhat tenuous, there did at least, appear to be a family association of Class III characteristics.

Litton, Ackerman, Isaacson and Shapiro (1970) studied 58 probands and 51 families of these patients to attempt to assess the mode of inheritance. Of the probands, 28 were male and 30 female over 15 years of age. A total of 374 relatives were assessed for Class III features. The assessments were carried out on models of the subject's dentition, and they acknowledged the potential inaccuracies inherent when basing assessment of skeletal dysplasias on dental analyses.

The authors discussed their results by considering four patterns of inheritance:

1. Autosomal dominant

Approximately 50% of the probands had one parent affected. Autosomal dominant transmission would require at least one parent affected in all cases.

2. Autosomal recessive

This mode of transmission would in theory result in 25% of the offspring affected in a group. The study revealed only 15% of affected offspring of non-affected parents. Statistically considered this may not be significantly different from 25%.

3. Sex-linked inheritance

This would result in unequal sex distribution of features. Approximately equal numbers of males and females were affected.

4. Polygenic or Multifactorial inheritance

Litton and associates assumed a 1% incidence of Class III in Caucasians. They claimed that a 1% incidence would result in a 10% expression of Class III characteristics in first degree relatives of probands under this system of inheritance (Edwards, 1960). The 13% incidence observed in the study in first degree relatives was consistent with a polygenic multifactorial mode of inheritance.

As can be seen from the variety of conclusions presented, the mode of transmission is, as yet, unproven.

It does, however, appear that there is a strong familial tendency and the evidence seems to implicate a multifactorial system.

Schulze (1973 from Eismann, 1976) listed characteristics that distinguish polygenic multifactorial inheritance:

1. As a rule, features with a polygenic multifactor basis are more frequent;
2. The concordance in MZ twins is several times greater than in DZ twins;

3. The degree to which patients are affected is shown by a normal distribution curve.
4. Within families and kinsfolk a manifold distribution is found but:
 - a. if both parents are affected the number of children affected is especially high;
 - b. if only the father or mother is affected the number of children affected decreases;
 - c. if none of the parents is affected, in general the number of children affected is a minimum.

Nakasima, Ichinose, Nakata and Takahama (1982) examined 104 Class III patients and their parents.

They found high correlations between the two for concave profiles and skeletal cephalometric measurements, and concluded that there was good agreement with a polygenic system of inheritance.

Although the studies discussed here are in general agreement with the principles of polygenic multifactorial inheritance, direct proof for this system is lacking.

2.1.2 Twin studies

Markovic (1976), whilst acknowledging that twin studies provide information relating to the genetic or environmental basis of traits, stated that they do not assist in identifying mechanisms by which these traits are transmitted. He discussed also the relative paucity of published studies of twins relating specifically to Class III malocclusions - a situation that has persisted until the present.

He quoted a study by Schulze and Wiese (1965 in German) as demonstrating a concordance of 80% in MZ twin pairs and a discordance of 100% in DZ twin pairs for Class III malocclusion.

Markovic (1976) studied 16 pairs of twins with Class III malocclusion and assessed incisor and molar relationship (from dental casts), skeletal relationships (from lateral cephalograms) and made inter- and intra-pair comparisons of the concordance-discordance rate.

He found a 93.3% concordance in MZ twin pairs compared with a 14.3% concordance in DZ twin pairs.

Variation in Class III characteristics was, he stated, less between the MZ twin pairs than between the DZ twin pairs.

Finally, he concluded that "the results ... strongly suggest that genetic factors play a predominant role in the aetiology of Class III malocclusion".

As can be seen from the literature a strong hereditary factor exists in Class III malocclusion although the mode of transmission is not yet firmly established.

In addition the current level of understanding is clouded by the absence of studies of specific forms of Class III malocclusion, the significance of variation in forms of Class III within families and by our lack of knowledge of the extent to which environmental factors may modify expression of genetic information.

2.2 Skeletal Components

Skeletal factors are contributory in nearly all Class III malocclusions (Foster, 1976). This is most obvious in the anteroposterior direction, although intermaxillary relations may also be disturbed in the vertical and transverse planes (Foster, 1976, Bryant, 1981).

Class III malocclusions have frequently been attributed to a differential growth rate of the jaws (Grossman, 1970; Litton et al., 1970; Turpin, 1981; Cozzani, 1981) and the possible mechanisms associated with this are discussed in detail in Chapter 3 of this treatise.

As discussed in Chapter one, Class III malocclusions have been divided into two basic types: viz. pseudo and true Class III's. This division is often somewhat arbitrary as they are often difficult to distinguish and true skeletal Class III's may be misdiagnosed as pseudo or functional Class III's (Kawata, Kishigami, Yamashita, Uyama, Toda and Uemura, 1982).

Turpin (1981) listed features that constituted true skeletal Class III:

1. large or protrusive mandible or
2. deficient or retrusive maxilla or
3. protrusive mandible and retrusive maxilla.

Fränkel (1970) stated that

The development of mesio-occlusion must be seen to be a consequence of an imbalance in the associated cranio-facial structures. There is a discrepancy in the antero-posterior dimension and/or position of the different parts of the skull such as the anterior cranial base, the naso-maxillary complex, the maxilla relative to mandibular dento-alveolar process and the base of the mandible.

Litton, Ackermann, Isaacson and Shapiro (1970) asserted that pseudo prognathism does not manifest itself through differential jaw growth. True prognathism, in contrast, manifested itself in centric relation (which may be co-incident with centric occlusion) and that any tooth guided functional shift on closure into centric occlusion is secondary to the underlying skeletal dysplasia.

Jacobson, Evans, Preston and Sadowsky (1974) studied mandibular morphology in 149 patients with Class III malocclusions, of which 32 and 34 were male and female adults respectively, 30 and 53 were respectively male and female children 6-16 years of age. On the basis of cephalometric tracings and subsequent measurement, they found that:

1. 50% of adults had mandibles beyond normal range in size;
2. 25% of adults had maxillae below normal range (retrusive);
3. 60% of children had mandibles within normal range.

They stated that these results did not indicate that the majority of Class III mandibles were larger than Class I mandibles.

Further they observed that Class III (as measured from condyle to Gnathion) mandibles were longer than Class I mandibles and that this was associated with an increased gonial angle thus indicating a morphologic rather than overall size difference between the two.

Jacobson et al. (1974) quoted Joffe (1964) as reporting similar findings in an assessment of surface areas in Class I and Class III mandibles, that is, there existed little difference between the two.

These results, whilst limited by the accuracy of cephalometric

measurements, admit the possibility of different growth patterns operating in the two groups.

Variations in the regulation of the complex growth-related rotations (Björk and Skieller, 1983) associated with mandibular development may be implicated in the formation of Class III characteristics.

In contrast to Jacobson et al. (1974), Solow (1980) relegated skeletal morphology to a position secondary to what he described as the dento-alveolar compensatory mechanism.

This is a process "by which the development of the dental and alveolar arches is controlled so as to secure occlusion of the teeth and adaptation to the basal parts of the jaws".

Solow (1980) claimed that skeletal discrepancies are not, on their own, responsible for the development of the malocclusion, but that a lack of dento-alveolar compensation is the primary cause. Further, he asserted that impairment of this compensatory mechanism occurs more readily as the severity of the skeletal discrepancy increases. The inference here being that the greater the interjaw discrepancy the greater the frequency of malocclusion. This seems to be a logical correlation, although it is an assumption that could be made even in the absence of a specific compensatory mechanism.

With regard to functional or pseudo Class III malocclusion, Solow offers no specific explanation for their occurrence other than discussing the variability in activity of the mechanism and the possible influence of oral and digital sucking habits on its effectiveness.

The basis for control and regulation of the mechanism was, unfortunately, not discussed.

Bimler (1970) observed what he termed "certain systemic growth disturbances" that seemed to be frequently associated with malocclusion.

He described three such growth disturbances with regard to their possible role as aetiological factors in the development of Class III malocclusion although it is often difficult to identify the three types.

1. Micro-rhnic dysplasia involves underdevelopment in the fronto-naso-maxillary complex associated with upward rotation of the palatal plane and forwards and upwards rotation of the mandible. Most frequently this results in a Class II malocclusion. However, occasionally Class III malocclusions may result.

With the mandible in rest position, a large freeway space is observed due to the vertical height deficiency in the fronto-naso-maxillary complex. Hyperflexion of the mandible is required to achieve centric occlusion and this hyperflexion results in forward movement of the jaw, protrusion of the chin and an anterior cross bite.

In micro-rhnic cases the concave profile often seen in Class III patients, is absent in rest position. It is often seen in centric occlusion, however, due to the forward movement of the condyles in the glenoid fossae required to avoid incisor interferences on closing.

2. Microtic dysplasia has more widespread effects on the growth of craniofacial components. The sphenoid and anterior parts of occipital bone

are reduced in size, the length of the anterior cranial base is reduced, as is the length of the clivus. The cranial base angle (N-S-Ba) is more acute and the distance from the glenoid fossa to the posterior aspect of the maxillary tuberosity is less.

Bimler stated that the essential feature is the "pathological" forward placement of the glenoid fossa. This observation was supported by Jacobson et al. (1974). The final morphological outcome of growth in these cases is closely linked to the height of the alveolar process with Class III malocclusion being associated with a short alveolar process and overclosure of the mandible, and open bite being associated with high alveolar process and pivotal rotation about the molars.

3. Leptoid dysplasia is associated with a "long face" syndrome where excessive vertical growth in the posterior alveolus and rapid mandibular growth exceed sagittal increase in the maxillary depth.

Bimler observed that these leptoid dysplastic types developed first an edge-to-edge incisor relationship which progressed to an open bite.

The primary aetiological events in these growth disturbances are unknown although Bilmer (1970) considers them to be "slight" forms of syndromes, such as Crouzon's and Apert's. In these syndromes, the cranial base is shortened and is associated with a midfacial deficiency. In almost all cases (of these two syndromes) craniosynostosis has been reported, usually in the sphenooccipital synchondrosis. In addition, it is reported that these are hereditary growth disorders transmitted by an autosomal dominant system (Stewart and Moore, 1982, p.78).

It would seem that early fusion of the synchondrosis rather than synostosis is more likely to be involved in Class III's with midfacial growth deficiencies. The formation of cranio-synostoses would result in cessation of growth in the area concerned (suture or synchondrosis) with the final extent of the deformity related to the area concerned, the age of fusion, and the extent of compensatory growth in other areas.

Premature fusion may be a more gradual process involving slow ossification of the cartilage of the synchondrosis, thus allowing some further "normal growth" to occur, although at a reduced rate. This, in combination with compensatory growth in other areas (which would differ in magnitude from person to person), may explain the great variability of Class III disorders and their progressive nature (Grabner, 1966).

The mode of inheritance postulated for Class III's (polygenic multifactorial) does, however, differ from that reported for these syndromes (autosomal dominant). This would though, provide an interesting area for further investigation.

Stark (1965) stated that various types of growth disturbance may occur as the result of oxygen deficiencies in early embryonic life, although proof of this is lacking.

Bryant (1981) related mandibular growth rotation to causation of Class III malocclusions. He posulated that the proportionality of growth in the condyle/glenoid fossa area to the maxillary sutures and alveolar process influenced the rotation of the mandible.

Equal growth rates in these areas resulted in linear translation of the maxilla and mandible. When growth in the condyle/glenoid fossa area exceeded that in the maxilla/alveolus, forward mandibular rotation resulted. Presumably this would also be associated with a forwards and upwards rotation of the maxilla.

Bryant (1981) listed the factors that influenced the height of the condyle/fossa area as:

- a. cranial base angle (N-S-Ba);
- b. length of posterior cranial base;
- c. height of the ramus;
- d. gonial angle.

Bryant did not speculate on what factors may be ultimately responsible for development of those morphological features listed above, as his study was primarily concerned with changes in morphology as the result of treatment.

The question of whether the aberrant growth patterns discussed here are due to primary pathological processes (in varying degrees) or the result of genomic expression is unanswered. Bimler (1970) referred to pathological forward positioning of the TMJ as being an associated morphological feature in certain types of Class III's.

None of these authors identified specific developmental occurrences as being primary aetiological events in the formation of the skeletal morphologies associated with Class III malocclusions.

Nelson (1969) observed that some syndromes such as Treacher-

Collins, Crouzons and Aperts are associated with Class III malocclusions.

Tessier (1971) examined the relationship of craniostenoses to the craniofacial dysostoses characteristic of these syndromes, and although these cases are extreme when compared to the majority of Class III malocclusions, enough similarities exist to justify a brief examination of his findings. In this way it may be possible to postulate how milder versions of these syndromes may occur.

These three syndromes are characterised by (amongst other things) an hypoplastic maxilla. Enlow (1983) asserted that the anterior cranial base formed the template for maxillary development and, if so, disturbances in cranial base growth (most likely involving the primary chondrocranium (Godfrey, 1986)) may result in corresponding disturbances in maxillary development.

Craniostenosis is premature closure of cranial sutures resulting from the formation of a synostosis across the suture concerned. The result is an interference in normal growth and aberrant cranial and facial development (Tessier, 1971).

The cause of the synostosis formation is unknown although it undoubtedly has a genetic basis. It is possible that normal (for these individuals) sutural growth is occurring and synostosis formation in these patients is equivalent to the ossification of sutures seen at maturity in normal individuals. This line of reasoning, whilst speculative, would infer a specific "trigger" for sutural ossification. The literature is, unfortunately, devoid of reference to this concept.

Compensatory mechanisms appear to come into play, although distinguishing the compensatory growth from the primary deformity is impossible (Tessier, 1971).

Depending on the location of the synostoses in craniostenotic patients, abnormal growth may be found in the sagittal, vertical or transverse dimensions of the cranial base, usually with associated facial (primarily maxillary) deformities.

Tessier quoted Delair (1961) as describing cases of faciostenosis which occurred in the absence of craniostenosis. Frequently in these cases the maxilla was found (surgically) to be retrusive rather than hypoplastic, and to be associated with normal mandibular development. Surgical investigation revealed in a number of cases a synostosis of the posterior aspect of the maxilla to the sphenoid bone.

It was not possible, however, Tessier stated, to identify in all cases whether maxillary retrusion or atresia was present. This was primarily due to the disturbance of a number of cephalometric landmarks (especially Frankfort horizontal) by the distorted growth.

Bearing in mind the dangers of relating pathological situations to what in the case of most Class III malocclusions may be "normal growth", several points worthy of consideration emerge.

Firstly, the occurrence of skeletal Class III's very early in life (deciduous and early mixed dentition) may be associated with the presence of mild forms of craniostenosis or faciostenosis. One indication is often "frontal bossing" or prominence of forehead.

The age at which the synostoses occur may influence the severity of the final outcome.

Secondly the presence of premature synostoses may dictate a skeletal development that is, in some cases, completely unaffected by either orthopaedic or orthodontic treatment.

Thirdly, premature synostosis may be a result of early ossification of the cranial base (primary chondrocranium).

The role of the cartilagenous nasal septum in growth of the maxillary complex is discussed in Chapter 3. Although I can find no reference in the literature it could be conjectured that premature ossification of the nasal septum is a potential aetiological factor in maxillary hypoplasia.

This discussion has concentrated primarily on abnormal growth of the cranial base/maxillary complex and mandibular morphology although a significant number of Class III's demonstrate excessive mandibular growth (Jacobson et al. 1974). To understand the potential aetiological role of mandibular growth, a detailed examination of the processes involved must be undertaken. This is presented in Chapter 3.

The role of skeletal factors in the development of Class III malocclusion has been discussed and, although a number of morphological features are regularly observed in the various forms of Class III malocclusion, the primary aetiological factors are unknown.

It is apparent, however, that certain types of Class III malocclusion are associated with specific morphological factors (such as gonial angle

variations) and others are due to growth disturbances where inadequate or excessive growth results in an overall interarch discrepancy. The primary cause of these growth disturbances, at a cellular or molecular level, are unknown.

2.3 Soft Tissue Components

A number of soft tissues and related functions have been implicated as possible aetiological factors in the development of Class III malocclusions. These include enlarged tonsils (Angle, 1907, Gold, 1949), difficult nose breathing (Angle, 1907), mouth breathing (Pac, 1970, Rakosi and Schilli, 1981), flat, anteriorly placed tongue (Rakosi and Schilli, 1981, Fränkel, 1970, Cozzani, 1981), hyperactive upper lip (Graber, 1970), hyperactive lower lip (Fränkel, 1972), habits (unspecified) (Rakosi and Schilli, 1981) and irregular masticatory movements (Korkhaus, 1970, Kreiburg, Jensen and Björk, 1978).

The question of whether these factors act in a primary role or are secondary, adaptive responses has elicited considerable controversy (O'Ryan, Gallagher, La Banc and Epker, 1982).

A number of authors have claimed that tongue posture may influence the morphology of the jaws.

Rakosi and Schilli (1981) stated that in patients with Class III malocclusion and mouthbreathing, the anteriorly displaced flat tongue causes a wide mandibular arch, narrow maxillary arch and high palate.

Cozzani (1981) claimed that low tongue posture may adversely influence the growth pattern.

Fränkel (1970) asserted that the tongue is an essential feature in the development of Class III conditions.

Little detailed explanation was presented by these authors regarding why tongue posture may be abnormal in these cases, or how it influenced the growth pattern.

It has been stated (Angle, 1907, Ballard, 1955, Moss, 1980) that normal occlusal development is associated with the maintenance of a balance between lip pressure and tongue pressure. It seems unlikely that the answer is as clear cut as this assertion implies. Mew (1981) claimed that experimental evidence (not cited, but presumably relates to work by Lear and Proffit) showed that tongue pressure greatly exceeded lip pressure, thus casting doubt on this assertion.

Assessment of normal tongue posture is exceedingly difficult (Proffit and Mason, 1975) as it is a highly mobile organ with a complex musculature. Likewise, description of tongue activity is difficult (Mew, 1981) and consequently relating tongue posture and function to the development of malocclusion is made more complex by these problems.

Moss (1968) describes growth and activity of the tongue as a component of the functional matrices regulating facial growth as a whole, and his hypotheses are described in detail in Chapter 4.

The development of a particular type of Class III (high gonial angle, anterior open bite) has been linked to low tongue posture and forward positioning of the mandible in some patients who respire orally (Subtelny, 1980). It was claimed that "oral respiration caused postural adaptations that

may have an effect on the positional relationship of the jaws and the developing occlusion".

This assertion has been challenged by O'Ryan, Gallagher, La Blanc and Epker (1982) who critically reviewed a number of key investigations into the possible relationship between oral respiration and dentofacial morphology. They concluded that the experimental evidence did not establish a definitive relationship between either mouth breathing or mandibular and tongue posturing and dentofacial morphology.

Subtelny (1980) studied 33 children with enlarged adenoids and found that (on the basis of radiographic examination) a number of features existed in these children.

1. there was a posterior downwards tipping of the palatal plane;
2. increased mandibular plane to SN angle with the mandible more divergent from gonion to symphysis;
3. antegonial notching and an increased curvature of the lower border;
4. some degree of maxillary retrusion.

His results indicated there was a relationship between altered morphology and adenoids but concluded that the associated oral respiration was probably a more precipitative or additive factor in cases where an hereditary propensity for vertical growth exists. Further he suggested that nasal respiration may be required to allow full development of the nasomaxillary complex.

Lip function has been included in the list of potential aetiological factors by Graber (1970). He stated that upper lip pressure may be a causal

factor by restricting anteroposterior growth of the upper jaw. He observed that the upper lip was frequently hyperactive in Class III cases although no evidence was presented to support this.

Fränkel (1972) considered that upper lip pressure may also be due to hyperactivity of the lower lip due to the upper lip being "embraced" by the lower lip in the lip seal position.

In contrast, Foster (1976) stated that lip form and function plays little part in variation of Class III's, but tend to vary with other factors.

With the exception of the information presented on tongue posture and mouth breathing, there is a virtual absence of data about soft-tissue-related variation in Class III malocclusion.

The contribution of soft tissues to the development of low gonial angle Class III's is uncertain, although muscle function (particularly the masseter) probably plays a role.

Kreiborg, Jensen and Björk (1978) stated that

1. Strong activity of the masseter muscles during maximal bite in the intercuspal position is associated with an anteriorly inclined prognathous mandible with a curved base and a tendency to deep overbite.
2. Weak masseter activity is associated with a posteriorly inclined mandible, retrognathia, flattened base and a tendency to anterior open bite.
3. Strong masseter activity during swallowing co-incides with prognathism and anterior indentation of both jaws.

Evidence to support muscle influence in craniofacial morphology was provided by examination of a case of craniofacial dystrophy of masticatory and facial muscles. Kreiborg, Jensen and Björk (1978) found a progressive skeletal dysplasia associated with the gradual degeneration of the muscles with the appearance of a severe anterior open bite.

They concluded that this was due to the effect of gravity being unreciprocated by the mandibular elevator muscles.

An attempt has been made here to restrict the discussion to soft tissue factors relating directly to Class III malocclusion. This has been brief due to the paucity of specific information in the literature.

It does appear that in some forms of Class III malocclusion, soft tissue form or function is an associated feature. The information available does not, however, allow a conclusive assessment of the primacy of the role of soft tissues in the aetiology of Class III malocclusions.

2.4 Dental Components

A number of features of the dentition have been stated to be causative or contributory in the development of Class III malocclusion.

Irregular eruption of the permanent incisors or loss of the deciduous incisors may result in an anterior crossbite of one or more teeth (Rubrecht, 1939).

Premature loss of the first permanent molars (Gold, 1949) or deciduous teeth, particularly mandibular deciduous molars (Rakosi and Schilli, 1981) may promote anterior sliding of the mandible.

Leighton (1969) quoted cases of postural mandibular protrusion due to "lingual occlusion of a permanent upper lateral incisor".

Single tooth crossbites in the anterior region are commonly found in Class III malocclusions (Rakosi and Schilli, 1981). They may be present in conjunction with a Class I skeletal relationships as in pseudo-class III (Foster, 1976) and in many cases they may be due to local occlusal irregularities (Kawata, Kishingami, Yamashita, Uyama, Toda and Uemura, 1982).

Deciduous canine tooth occlusal interferences have been implicated as a causative factor in early functional and true Class III malocclusion, in addition to labial crossbite (Godfrey, 1985). The problems of canine interference would be related to reduced intercanine width and, in turn, degrees of maxillary base deficiency. Whether the primary canine interferences are causative, aggravating or secondary is a matter for speculation. Many deciduous dentition, Class III patients have prominent primary canines associated with a forward postural shift of the mandible due solely to canine interferences, and this may support Godfrey's assertion.

The interference resulting from localised crossbites also results in a forward postural shift of the mandible (Kawata, Kishingami, Yamashita, Uyama, Toda and Uemura, 1982) and it has been claimed that in such cases this abnormal function may interfere with normal alveolar growth of the maxilla (Nakashima, Ichinose, Nakata and Takahama, 1982).

Schwarze (1970) warned that "the true mesial tendency may be masked by a crossbite of a single incisor". He did not state how this may occur, but one could imagine the possibility of a "scissor" bite associated with a single tooth crossbite forcing a retrusive mandibular closure

pattern, thus reducing the apparent severity of the skeletal discrepancy.

Litton, Ackerman, Isaacson and Shapiro (1970) stated that, in contrast to true prognathism, pseudoprognathism was manifested in centric occlusion (which was never co-incident with centric relation) due to incisor interferences. These authors claimed that co-incidence of centric relation and centric occlusion is a diagnostic feature of true prognathism, although admitting that secondary tooth guided mandibular shifts may occur - thus confusing the diagnosis.

If, as is apparently the case with these authors, diagnosis of pseudoprognathism is to be based on assessment of centric relation, then centric relation must be defined with reference to that assessment. The use of the most retruded position of the mandible as centric relation (a definition disputed by Kleinberg 1985) would result in the diagnosis of a less severe problem than actually exists.

In the posterior region, it has been claimed (Subtelny, 1980; Kawata et al., 1982) that continued eruption of molars in cases where a mouth breathing habit and an hereditary propensity to excessive vertical growth exist may contribute to deterioration of the developing facial skeleton. It is uncertain, however, whether this is a primary factor, or a secondary compensatory response to the existing abnormal growth pattern.

Posterior crossbites are frequently seen in skeletal Class III's due to a transverse discrepancy between the jaws. This has been discussed in 2.2.

Functional posterior crossbites in Class III malocclusions on skeletal

type I bases are most frequently associated with a functional shift of the mandible to avoid incisor (or canine) interferences (Chaconas and Caputo, 1982).

Bell and Le Compte (1981) claimed that this mandibular shift resulted in midline deviations and unilateral crossbite. Further, they stated that

subsequent dental, skeletal and neuromuscular adaptation results in a constricted maxillary arch of insufficient width to encompass the mandibular arch in normal closure.

Local factors such as persisting deciduous teeth have also been implicated in causing functional mandibular shifts associated with crossbite (Ryden and Magestrom, 1970).

Bilateral crossbites, particularly in the absence of mandibular posturing are most frequently associated with maxillary arch crowding and an underlying problem of transverse maxillary development. The skeletal aspect of posterior crossbites is discussed in section 2.2.

Björk and Skieller (1983) showed one case of late development of Class III but this is merely an unusual (unpredictable) aberration.

Leighton (1969) claimed that prenatal occlusion became apparent at about the time of eruption of the deciduous incisors and this may be seen as its primary manifestation.

Rakosi and Schilli (1981) stated that the step-by-step development of infantile (deciduous dentition) Class III's was as follows.

1. central incisors erupt into a neutral relationship with no overjet;

2. during the eruption of the lateral incisors into the same relationship, a crossbite of the central incisors develops;
3. several weeks later a crossbite of all incisors exists;
4. the tongue is flat and pressing against the mandibular incisors;
5. the child habitually moves the mandible into an anterior position.

These authors claimed that this sequence of events accounted for 10% of all early Class III malocclusions, although this scenario could be criticized on several points. No assessment of the degree of skeletal discrepancy at this early age was included. The great variability of Class III's (Chapter 1) would make this information essential.

Further, no reasons were presented for the eruption of the teeth into a neutral relationship, the development of the crossbite or the presence of an habitual anterior thrust of the mandible. The aetiological significance of this information is obvious.

Discrepancies in tooth size and number between the arches may also be a contributing factor in Class III malocclusions.

Bolton (1962) presented a case in the permanent dentition where an analysis according to his technique revealed an excess of tooth structure in the lower anterior region. He claimed that the discrepancy amounted to 4.5mm in this case. A Class III molar relationship existed and the incisors were edge-to-edge.

Hypodontia and oligodontia, particularly in the maxillary arch may also be associated with Class III malocclusion and this in conjunction with slight Class III growth tendencies may significantly worsen the prognosis.

CHAPTER THREE

CRANIOFACIAL GROWTH

The growth of the craniofacial complex has been the subject of extensive investigations by many authors over the years. This work has produced a number of theories and philosophical dissertations regarding the growth mechanisms, controls, sites and centres involved in craniofacial growth as well as numerous attempts at predictive growth assessments.

The clinical significance of growth studies has centred around three main areas:

1. attempts to identify areas where interceptive orthopaedic control of growth may be possible. These studies have been hampered by many factors, not the least of which is one of definition of such concepts as "growth centres", "growth sites" and "growth potential"
2. identification of areas where pathological, environmental or genetic factors may act to produce abnormal growth patterns
3. attempts to predict the final morphology of the craniofacial skeleton for individual patients.

Moss (1969) claimed that "growth" as a term must describe not only morphologic changes to a bone due to remodelling, sutural and cartilagenous growth, but include any spatial translation that takes place during that process.

Koski (1968) commented on the confusion that existed with regard to the definition of concepts. The terms "growth centres" and "growth sites"

appeared to be used frequently without definition and interchangeably. He defined "growth centres" as areas which possessed an inherent ability to produce a tissue separating force and thus induce a spatial translation of components. "Growth sites" were described as areas that grew in response to local environmental changes, but without an active separation of tissues. No conclusion was reached by Koski (at that stage) as to whether "growth centres" existed.

The concepts of "spatial translation" and "transformation" have also caused some confusion.

Moss's (1969) use of the term "spatial translation" was without definition and apparently intended to be self explanatory.

Björk and Skieller (1976) discussed the two concepts of "translational" and "transformational" growth.

"Translational growth" occurred when a component (of the facial skeleton) was moved, relative to other components, as the result of growth in another area, and without substantial alteration to its constituents.

"Transformational growth" resulted in a change in the shape, size and spatial position of a component by remodelling.

In the context of this treatise, these terms will be used as described.

Enlow (1983) stated that

the craniofacial complex is just that - a composite of many regional components, each of which relates to all the others. If there is an alteration in any region, its [sic] not likely to be merely a regional response, but there will be many other adjusting reactions elsewhere. (p.672)

Bearing this in mind, craniofacial growth will be here examined under three headings:

Control of facial growth

Mandibular growth

Nasomaxillary and cranial growth.

Emphasis will be placed on the relevance of growth to

1. development of Class III malocclusions
2. significance of growth studies to the treatment of class III malocclusions.

3.1 Control Mechanisms in Craniofacial Growth

Moss and Salentjin (1969) claimed that the volumetric growth of the oronasopharyngeal functioning spaces was the "primary morphogenetic event in facial skull growth", with the prime consideration being the maintenance of an airway.

This concept was the basis for the formulation of Moss's functional matrix hypothesis; a theory that has sparked much controversy over the years.

Enlow, Harvold, Latham, Moffett, Christianson and Hausch (1977) stated that

The functional matrix concept, (like Wolff's Law), is a useful concept in pointing out that growth interrelationships exist among tissues and that regulatory priorities are involved. This principle should not be misused, as Wolff's law has been misused, as a conceptual label substituting for explanations of the actual regulatory processes themselves.

There is little dispute regarding this statement. However, the mere existence of the theory that regulatory processes may be involved (which infers the presence of adaptive and compensatory growth mechanisms) has fuelled speculation that such processes may be controlled or influenced. This has special relevance to early Class III malocclusions, with the increasing use of chin cup, functional and maxillary protraction appliances.

Overall genetic control of growth has been expounded by many authors.

Cinasoni and Becks (1963) claimed that heredity was one of the principle elements determining the growth pattern. They supported this by referring to the experiments of Baker (1941) where foetal rat mandibles were transplanted into the leg muscle of a growing rat. The observation that in spite of surgical trauma and interruption to the blood supply, the mandible grew to half its normal size inferred some hereditary influence. This interpretation apparently ignored the possibility of hormonal influence on growth of the transplant - particularly by growth hormone.

Sarnat (1983) asserted that the blueprint of a bone is inherent, but conceded that

its final expression in the external form and internal architecture of a bone ... is dependent on the effects of prenatal and postnatal environmental influences.

Vig, Millicovsky and Johnston (1984) investigated, histologically, abnormal cranial development in rats and concluded that facial development is dependent on cells which arise from the cranial neural crest. Migration of these cells to their correct positions at the correct time is essential to normal development. Thus teratogenic, genetic or mechanical factors that interfere with these processes may result in facial deformity.

These authors qualified these observations by stating that there has been little work done that can relate directly to the human condition. The observations do, however, indicate that the growth pattern may be established well before birth and thus manifested very early in life.

In humans, this is a characteristic feature of Class III malocclusions (Leighton, 1969).

Logan (1968) discussed the remarkable recovery of a deformed dento-facial complex (resulting from Milwaukee Brace therapy for scoliosis) that occurred when the brace was removed. He observed a greatly accelerated rate of growth of the mandible and maxilla. Whilst his observations have important implications in orthopaedic therapy for Class III cases, they also appear to demonstrate that a predetermined pattern of growth was restrained by the brace and, when released, rapidly expressed itself. Presumably this pattern had a genetic basis, although a return to normal function undoubtedly played a role in the recovery.

Mills (1983) reviewed recent literature relating to facial growth and, although offering no substantial experimental evidence, concluded that genetics were of prime importance. Further, he stated that is no explanation of the mechanism by which genetic control asserted itself.

Sarnat (1983) reviewed normal and abnormal craniofacial growth in humans and animals and concluded that

There are many mechanisms (nervous, hormonal, metabolic, enzymatic) by which the environment directly induces adaptive changes. Environmental stresses can impact either directly, as with variations in temperature and oxygen levels, or indirectly through the genetic control of the enzyme forming system.

Further, Sarnat (1983) claimed that "the physiologic stability of bony components is the result of many interrelated factors, with normal functional use especially prominent."

The concept that normal function (as an "environmental factor") may influence bony morphology was extensively examined by Scott (1954) who presented work carried out on rabbits and rats where sectioning of muscles resulted in abnormal bony morphology. Specifically, muscles to the femur in rabbits were sectioned and, in growing rabbits only, the associated abnormal posture resulted in abnormal changes in femur morphology. Sectioning of the temporal muscle in rats resulted in a loss of the coronoid process after 3-5 months.

Scott concluded that three components existed that related muscle function to structure and form of the skull:

1. elements dependent on muscle growth and function but related to growth of other tissues;
2. elements related to muscle function through phylogenetic development and develop in a particular individual to a certain degree independently of the development or action of the muscles;
3. elements whose size and strength depend for their full development upon the degree of muscular function in the individual.

Thus the inference is that genetics and function are interrelated factors in growth but that individual response may vary the final outcome. As with many other studies it must be pointed out that the conclusions drawn relate mainly to animal studies and hence may not relate directly to humans.

Functional control of overall craniofacial growth has been expounded by Moss (1968, 1969, 1972, 1981) with his functional matrix hypothesis.

The hypothesis was developed in an attempt to construct a method of functional cranial analysis and is based on the assumption of a primary morphogenetic role of the functional matrix.

He described the head as a region carrying out a number of relatively independent functions viz. respiration, olfaction, vision, hearing, balance, chewing, digestion, swallowing, speech and neural integration (Moss 1968).

Moss (1968) went on to say

Each of these functions is carried out by a separate functional cranial component. Each component consists of all the tissues, organs, spaces and skeletal parts necessary to carry out a given function completely. The tissues, organs and functioning spaces taken as a whole comprise the functional matrix; while the skeletal tissues (osseous, as well as cartilagenous) related to this specific functional matrix are the skeletal unit.

Probably the most controversial aspect of this theory can be seen in the statement by Moss (1968):

There are no genes for bone. Bone structure and bone growth reflect extrinsic environmental factors rather than intrinsic genetic ones.

This concept is a radical departure from the long held theory of genetic control. It seems to be an oversimplification of what is a complex process, inferring that bone growth occurs by a process that is totally subservient to its (bone) local functioning environment.

Fränkel (1972) proposed a qualified interpretation of this theory. Bone growth was, he claimed, controlled by its functional matrix, but growth of the functional matrix occurred according to a genetic pattern, the genomic expression of which was influenced by epigenetic (environmental) factors.

The mechanism by which this process occurred was not clarified.

A balance between the growth rates and morphogenetic expression of the various functional components is, according to Moss's theory, essential for normal growth. If the proper relationships break down, the result will be deformity which may involve more than one functional region (Scott, 1969).

Whilst this theory provides a possible explanation for the inter-relationships existing between the functional components and their expression in the form of the skeleton, a satisfactory explanation of the strong hereditary pattern often found in craniofacial growth disorders (including Class III malocclusion) must await a more detailed understanding of growth control mechanisms at a molecular level.

Hereditary mandibular prognathism where bone growth appears to continue beyond normal functional limits seems difficult to explain purely in terms of environmental factors. Perhaps a more satisfactory explanation should include the concept of genetic determination of muscle design and the

particular functional influences these muscles would exert, in addition to influences of "growth disturbances" as previously discussed.

This rather broad "explanation" stresses the current incomplete understanding of craniofacial growth control processes which leave clinical assessment methods for the efficacy of interceptive orthopaedic therapy on a largely empirical base.

It also creates difficulties in attempting to explain and anticipate the variable morphogenetic expression of Class III's and thus to accurately formulate prognostic evaluations for treatment.

3.2 Growth of the Mandible

A study of 149 patients with Class III malocclusions and 112 patients with normal occlusions (Jacobson, Evans, Preston and Sadowski, 1974) revealed two main points.

1. the majority of the Class III patients owed their prognathism to mandibular characteristics rather than to a relative maxillary deficiency; and
2. the differences between normal and Class III mandibles are due more to morphology than size.

Consequently, a study of the growth processes that are responsible for the morphology of the mandible, may help to understand the complex morphological types, and degree of variation found in Class III malocclusions as well as to assess the prognosis for orthopaedic therapy.

The majority of studies on mandibular growth have been based on

cephalometric examinations of growing mandibles and have been of three types (Kerr, 1979).

1. Cross-sectional studies from which have been drawn longitudinal conclusions
2. combinations of cross-sectional and longitudinal studies also with longitudinally based conclusions
3. a limited number of longitudinal studies which have provided more accurate longitudinally based conclusions.

Longitudinal studies of the growth occurring in an individual over its growth period provide the most accurate information regarding the morphological changes that result in the final size and shape of the jaws.

Björk and Skieller (1983) presented a synthesis of longitudinal growth studies over a period of 25 years. These were based on the use of metallic implants and radiographic examination of positional changes of the implants occurring as the result of growth.

The findings resulted in a somewhat expanded version of Björk's (1969) concept of mandibular growth rotations.

These authors described three types of rotations that occur simultaneously during growth of the mandible. The overall morphology at the end of growth was described by the sum total of these rotations.

1. Total rotation

This is described as rotation of the mandibular corpus and is measured as a change in the inclination of a reference line or an implant line

in the mandibular corpus relative to the anterior cranial base. The total rotation does not have a fixed centre and its expression is dependent on the action of its two components, matrix and intramatrix rotation.

2. Matrix rotation

Centred at the condyles, matrix rotation is measured as a change in relationship of a tangential mandibular line (on the lower border) to the anterior cranial base. It is an expression of a rotation of the soft tissue matrix. Matrix rotation may occur in a forwards or backwards direction during growth in the same individual - in the manner of a pendulum with the condyles as the centre of rotation.

3. Intramatrix rotation

is described as the difference between total rotation and matrix rotation and is an expression of rotation of the mandibular corpus within the soft tissue matrix. Intramatrix rotation demonstrates remodelling of the lower border of the mandible and is defined as:

a change in the inclination of an implant or reference line in the mandibular corpus relative to the tangential mandibular line.

All three rotations are differently composed and show a changing interrelationship to each other during the entire growth period (Björk and Skieller, 1983). Further, they postulated that the explanation for this complex pattern of mandibular growth may be closely associated with growth at the condyles (both the direction and amount of growth) and that condyle growth may be part of a genetically determined growth pattern in the development of bone and muscle. On the basis of this theory, they hypothesised that intramatrix rotation exhibited a compensatory function to

allow normal occlusal development in cases of excessive total rotation.

Dibbets (1985) also discussed intramatrix rotation in terms of a compensatory mechanism for a genetically determined program of condylar growth. He related this type of rotation to condylar growth thus:

Assume two scenarios:

- a) circular condylar growth pattern which results in only intramatrix rotation and no mandibular enlargement
- b) linear condylar growth characterised by an absence of intramatrix rotation and significant mandibular enlargement.

Comparison of these two situations resulted in the postulate that intramatrix rotation may effectively neutralise condylar growth by inducing a curvilinear condylar growth direction and selective remodelling of the mandible to maintain morphology. Further, Dibbets (1985) stated that this rotational activity results in a selective enlargement of the mandible, apart and distinct from mechanisms that have been described in the literature.

How do these postulates tie in with other growth theories? Dibbets admitted that there was no answer to the question of genetic control of condylar growth, thus inferring that it is not known whether, with regard to his hypothesis, condylar growth causes intramatrix rotation or whether intramatrix rotation, under the influences of regional environmental factors (the local "functional matrix") controls condylar growth.

Overall rotations have been related causally to adaptive maintenance of an adequate pharyngeal airway (Moffet and Koskinen-Moffet, 1981). This observation is, however, based on studies of children with chronically

enlarged adenoids who exhibit a significant downwards and dorsal mandibular rotation (Koski and Lahdemaki, 1975) and thus may not be of significance in the development of Class III skeletal discrepancies.

How then does this information relate to Class III malocclusions? Whilst there is currently little evidence linking it directly with diagnostic and treatment planning decisions (Moffet and Koskinen-Moffet, 1981) it has potential relevance in several areas:

1. Growth prediction (discussed in greater detail in Chapter 3). The complex nature of the rotations, high degree of individual variation and unpredictable course of condylar growth (Björk and Skieller, 1983) complicate current methods of growth prediction. This is because they assume that either that growth will follow a standard course or that variations from the norm will be predictable (Petrovic, Stutzman and Grasson, 1981).

2. If growth at the condyle is genetically predetermined does there exist scope for extrinsic alteration of this growth?

3.3 Condylar Growth

For many years, the understanding of condylar growth was based on growth studies with epiphyseal cartilage (Durkin, 1972).

More recently, however, it has been found that important differences exist between the two. These findings influenced both the experimental approach to condylar growth studies, and the attitude toward orthopaedic treatment of mandibular growth disorders.

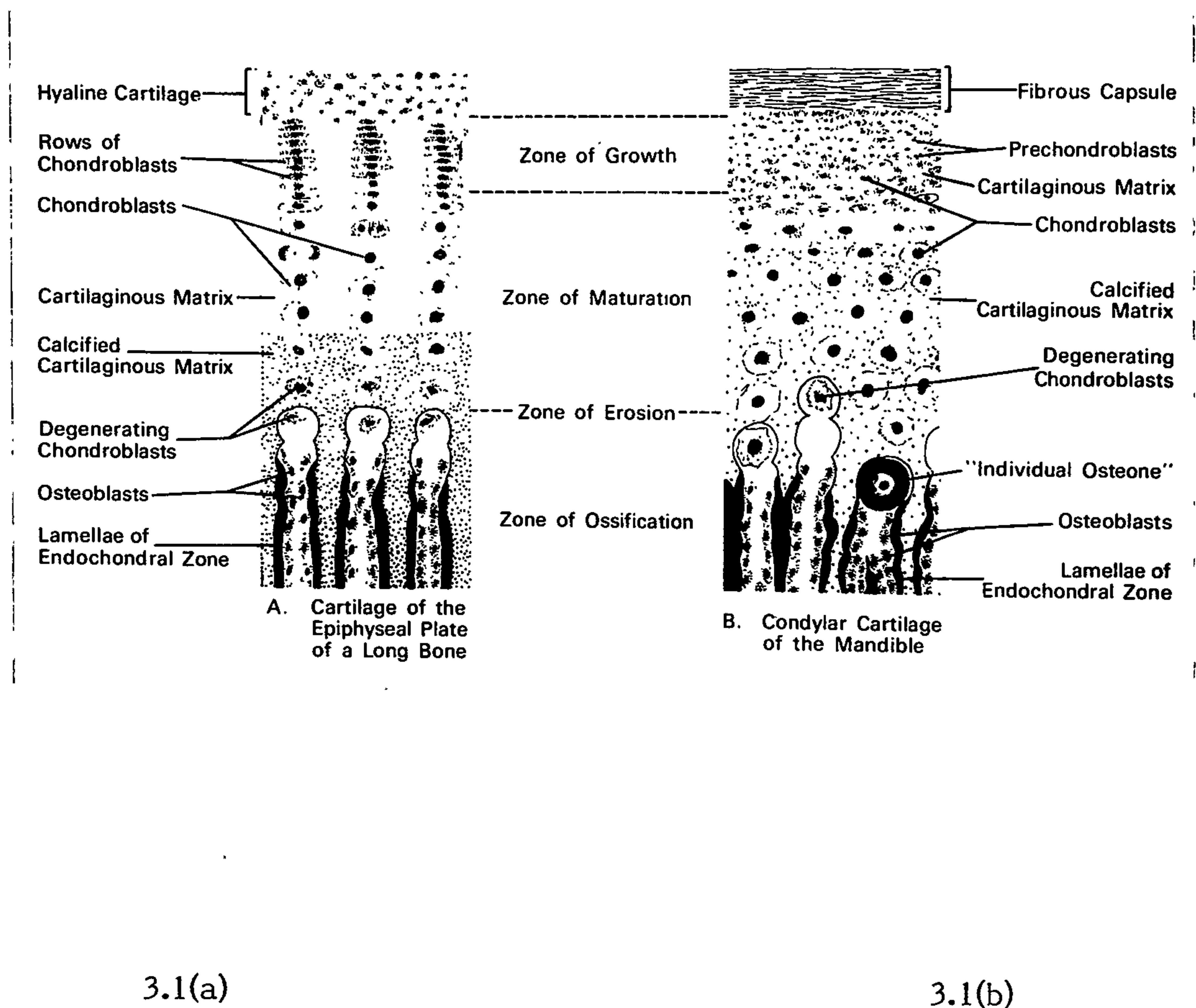


Fig. 3.1 Diagrammatic comparison of epiphyseal (3.1a) and condylar (3.1b) cartilage (from Petrovic, Stutzman and Oudet, 1975).

Condylar cartilage has been described as a secondary cartilage as an attempt to classify it with respect to its embryonic development (Durkin, 1972). Petrovic, Stutzmann and Oudet (1975) described secondary cartilages as those "that do not develop ontogenetically and phylogenetically from the primary cartilagenous skeleton". Mills (1983) stated that this type of cartilage forms in three areas:

- a) condyle;
- b) chin region;
- c) anterior border of the coronoid process.

Only the cartilage in the condyle persists and it does so as a thin cap lying beneath the articular surface of the TMJ and continuous with the periosteum covering the rest of the bone. Koski (1968), Durkin (1972, 1973), Rönning, Paumio and Koski (1979) are amongst a number of authors who point to the difference between condylar cartilage and epiphyseal plate cartilage.

Histologically it can be seen (Figs. 3.1a,b) that the arrangement of cells differs between the two. In epiphyseal cartilage the degenerating chondrocytes are arranged in parallel columns whereas in condylar cartilage this organisation is more haphazard (Petrovic, Stutzmann and Oudet, 1975). Durkin (1972), however, points out that this organisation of cells in epiphyseal cartilage is merely an intermediate phase in maturation of that cartilage and that in condylar cartilage the transformation of cells from the hypertrophic to the nonhypertrophic form occurs gradually and simultaneously across its entire width and that no such intermediate phase occurs (Fig. 3.1a, Durkin, 1972). Durkin points to histological evidence that the hypertrophic form of condylar cartilage possesses the embryonic characters described for both epiphyseal cartilage and articular cartilage, to support his assumption.

In spite of the apparent histological similarities it must be remembered, as Meikle (1973) points out, that "a detailed histological description of condylar cartilage, ... is not synonymous with an explanation of the growth processes of the cartilage".

Petrovic et al. (1975) stated also that in contrast to primary

cartilages (also termed embryonic cartilages by Durkin, 1972) condylar cartilage grows by division of prechondroblasts which are not yet able to synthesise typical cartilagenous matrix. In the case of epiphyseal cartilage the dividing cells are chondroblasts. Further they stated that the growth by division of prechondroblasts and differentiation to chondroblasts involved cells with a low response threshold to biomechanical incitation (either stimulatory or inhibitory). Chondroblasts, on the other hand, are cells adapted to division and growth in areas of pressure.

Different types of cells are thus responsible for growth in the two types of cartilage. The use of experimental data based on the response of epiphyseal cartilage to pressure may not be valid for understanding the likely response of condylar cartilage to pressure.

3.4 Experimental Studies of Condylar Growth

The extent to which condylar growth is responsible for spatial translation of the mandible has been the subject of considerable controversy.

Two schools of thought exist:

1. That the condyle is essential in overall length increase in the mandible. Proliferation and growth of the condylar cartilage propels the mandible forward, and this in conjunction with remodelling along the borders of the ramus regulate the anteroposterior position and length of the mandible (Sicher, 1949);

2. Condylar growth is secondary and compensatory to downwards and forwards movement of the mandible by growth of functioning soft tissues (Moss 1969). This is supported by experiments where condylectomy did not significantly change length and spatial position of the mandible (Gianelly and

Sarnat and Engie, 1953; Moorrees, 1965).

Condylectomy experiments have been criticised on several points. Pimanidis and Gianelly (1972) stated that the majority of condylectomy experiments were carried out in animals that had already completed a significant proportion of mandibular growth and consequently the experiments would demonstrate only what effect the absence of condyles had on the remaining growth and thus may not be an accurate representation of the true extent of effects. To test this these authors carried out condylectomy experiments in one day old rats. Their results were essentially the same as those previously reported, viz. a 15% reduction in mandibular length at maturity compared with controls and a normal incisor relationship. They concluded that their results supported the functional matrix hypothesis and that condylar growth is adaptive.

A second criticism was proposed by Meikle (1973). Removal of the condyle and observation of the effect on the overall growth of the mandible does not, he claimed, provide any information about the condylar growth mechanism. They indicate merely that growth is a complex mechanism and that the experimental animals are able to partially adapt to the loss of one growth site by compensatory growth in other growth sites. Consequently these experiments are capable of providing only a very rough indication of the quantitative contribution of the condyle to postnatal growth.

Brash (1956) termed condylectomy procedures as "mutilation experiments" and stated that the results only related to the particular experiment, although he produced no concrete evidence to support this statement.

Bernabei and Johnston (1978) observed that, even though the majority of these experiments yielded similar results, markedly different conclusions have been drawn:

1. Some authors (Gianelly and Moorrees, 1965, Pimenedis and Gianelly, 1972) have argued that as mandibles still grow in the absence of condyles, then condylar growth is secondary and serves to maintain the integrity of the temporomandibular joint.

2. Others (Sicher, 1965; Das, Meyer and Sicher, 1965; Skuble, Choucas and Toto, 1970) have emphasised the significant reduction in mandibular size and concluded that the condyles are primary growth centres.

It appears from the foregoing that condylectomy experiments fail to provide substantial information regarding the role of condylar growth or the mechanism by which it is regulated.

A variation on the condylectomy experiments has been carried out by Bernabei and Johnson (1978). This involved condylotomy (both unilateral and bilateral) and assessment of growth changes and alterations in spatial position of the components associated with growth hormone administration.

Their observations can be summarised:

1. in the anteroposterior direction little difference was observed between the growth hormone group and control group (both groups having had bilateral condylotomies), although the growth hormone group demonstrated a slight forwards displacement of the condylar fragments.

2. in a vertical direction the growth hormone group demonstrated a significant downwards displacement of the condylar implants. The ramus/body sector "collapsed" upwards and backwards in both groups but to a lesser extent in the growth hormone group.

The conclusions were:

1. the condyle is a "target organ" for growth hormone;
2. growth of the oropharyngeal space did not compensate for the loss of condylar support of the body/ramus and thus questioned the hypothesis that growth of functional spaces is responsible for translation of the mandible;
3. the condyle has some inherent growth potential but that it may be a rate-limiting factor in mandibular growth rather than a primary cause for displacement.

Here, too, it seems that we are in no better position to assess the level of control that the condyle exerts on mandibular growth and thus to ascertain whether that growth can be controlled to any great extent by external forces.

Condylar transplantation experiments have been carried out by many workers in an attempt to quantify the growth potential of the mandibular condyle in nonfunctional sites and thus assess its role in mandibular growth (Koski and Makinen, 1963, Koski and Mason, 1964, Meikle, 1973, Isotupa, Koski, Koskinen and Ronning, 1975, Engelsma, Jansen and Duterloo, 1980).

Again, both the results of these experiments and the conclusions drawn varied.

Koski and Makinen (1963) and Koski and Mason (1964) transplanted condylar cartilage with and without adjacent bone into subcutaneous tissue and brain and concluded that, as very little growth was seen, condylar cartilage had no significant inherent growth potential and that the results supported the theory of Moss.

Meikle (1973) conducted similar experiments using brain tissue as the supporting medium and observed chondrogenesis for a limited time followed by an increase in the rate of differentiation into osteogenic cells by cells of the proliferative zone. He concluded that condylar cartilage growth is ultimately under the control of the cellular genome and that, whilst the condyle did not control growth of the mandible, it was essential for enlargement of the ramus.

Engelsma, Jansen and Duterloo (1980) transplanted rat condylar head to the distal half of the 4th metacarpal and found that normal growth continued providing function in that site was not impaired. If this were so, growth was also impaired. This seemed to indicate some degree of intrinsic growth potential.

Mills (1983) on the basis of a review of the literature concluded that "growth in the length of the mandible, from condyle to chin, is produced by this cartilage and is not subject to environmental influences to any great extent". He supported this assumption by observing that in cases of bilateral cleft palate where maxillary hypoplasia was evident, mandibular growth was normal. In addition acromegalic patients exhibited excessive mandibular growth and a lack of both compensatory alveolar growth and matching maxillary growth.

This is in contrast with observations by Enlow, Harvold, Latham, Moffet, Christiaansen and Hausch (1977) of two TMJ disorders. In juvenile rheumatoid arthritis condylar growth is retarded. However, the mandible is still carried anteriorly as facial growth proceeds until the hypoplastic condyles disarticulate from the cranium. In fibrous ankylosis of the TMJs there is a distortion of the entire craniofacial complex as the condyles are unable to disarticulate from the glenoid fossa.

Tingey and Shapiro (1982) supported the observations of Enlow et al. (1977) with the results of papain-inhibited condylar growth experiments. Papain reduces the rate of endochondral growth by degrading the matrix protein and reducing the thickness of the maturation zone. It can be localised by injection of the active form of the enzyme and the effects are prolonged by steroids. They observed:

1. condylar growth inhibition;
2. compensatory growth of glenoid fossa resulting in a spatial translocation of the condyle;
3. reduction in ramus height;
4. little effect on overjet, overbite or midlines.

Further, they found that the effect of the induced condylar dysplasia depended on:

1. age;
2. duration of growth inhibition;
3. severity;
4. the capacity for compensatory growth of the individual.

They concluded that condylar growth is more important for vertical

ramus growth than for anteroposterior growth of the mandible.

Quantification of the role of the condyle in non-pathological mandibular growth is hampered by

1. varying experimental results;
2. varying interpretation of the results, particularly in comparing animal experiments with the human condition;
3. the difficulty of designing experiments that isolate condylar growth from the complex remodelling, compensatory and adaptive growth processes responsible for the final morphology of the mandible.

3.5 Maxillary Displacement

The processes whereby the maxilla is displaced anteriorly during growth has special relevance to a study of Class III malocclusions. Midfacial deficiency is a component of many Class III's (as discussed in Chapters 1 and 2) and aetiological considerations, as well as the possibility for antero-posterior modification of the developing maxillae dictate an examination of these processes.

The cartilage of the nasal septum has been the target of considerable debate regarding its role in downwards and forwards growth of the maxilla.

Scott (1953) claimed that the cartilagenous nasal septum played a role as a primary growth centre, resulting in spatial translocation of the maxilla.

The issue of the existence of primary growth centres is a contentious one (Koski 1968) and has been discussed on page 38 of this treatise.

Scott (1953) claimed that the septum slides downwards and forwards in a groove formed by the vomer.

Latham (1968, 1970) described a septomaxillary ligament which provided a firm attachment of the maxilla to the septum. This he claimed, added weight to the theory that the septum pulled the maxilla downwards and forwards.

Melsen (1977), however, asserted that the vomer was displaced downwards and forwards relative to the ethmoid bone during growth and claimed that this refuted the hypothesis presented by Scott (1953). Melsen based his assertions on studies with human autopsy material.

Melsen, Melsen and Moss (1981) claimed that the experiment used by Scott (1953) made it difficult to reconstruct a three dimensional model for septal growth in humans.

These authors carried out an assessment of growth changes in human septal cartilage using (they claimed) a more accurate histological, micro-radiographic and gross structural technique. Even so they were unable to arrive at any conclusions regarding the importance of the septum in maxillary displacement.

Moss (1968, 1969) constantly relegated growth of the nasal septum to a secondary, compensatory event in facial growth. The primary morphogenetic event being expansion of the functional spaces of the oronasopharynx and sinuses.

This view (of Moss) has been criticised by Johnston (1976) who felt that Moss's theory was merely a statement of a cause and effect with no evidence of the intervening mechanisms. He stated that, according to this theory,

facial growth is seen as a function of the function (or the need for the function) of functioning functional matrices.

and that an operational restatement of the hypothesis was required.

Animal studies have further provided a source for conjecture in this regard.

Moss and Bromberg (1968) excised the nasal septum in foetal rats and concluded that the resulting deformities represented merely a collapse of the roof of the nasal cavity.

Sarnat (1983) resected the septum in young growing rabbits and observed that severe lack of growth in the upper facial skeleton resulted. He pointed out that, whilst it was tempting to conclude that the nasal septum was a primary growth site, comparisons between rabbits and humans should not be made directly. Sarnat made no comment on the possible effect of surgical trauma in the production of these defects.

Babula, Smily and Dixon (1970) studied A/Jax mice with spontaneous bilateral cleft lip and palate. They observed that the nasal septum was shorter in these mice and concluded that this did not support the nasal septum thrust theory (which inferred that the septum in this case should have been at least normal length).

Koski (1981) referred to studies of the mitotic activity in rat nasal

septum tissue. The occurrence of phase differences in activity between the anterior, middle and posterior thirds of the septum (as determined by autoradiographic studies) he concluded, indicated the possibility of local control of growth of the septum. This he stated, could be hormonal in nature.

This conclusion was supported by Kvinnsland and Kvinnsland (1976) on the basis of the results of autotransplantation, transplantation to a culture medium and homotransplantation of the nasal septum in rats.

Koski (1981) observed that, in autotransplantation studies with septal tissue, a greater mitotic activity occurred in the transplants immediately following transplantation than in the controls. He postulated that this was due to local inhibitory factors in situ and thus supported the local control hypothesis. Koski concluded that, whilst the nasal septum was essential for normal growth, its role as a primary growth centre was not of "decisive magnitude and importance".

Other studies (Stutzman and Petrovic, 1976) have pointed to the importance of septal growth in the development of a snout in rats. Melsen and Melsen (1981) stated that this role was probably associated with the persistence of a premaxillo-maxillary suture in rats.

Mills (1983) asserted that primate studies (Sarnat, 1983) should be considered in the light of the fact that, unlike humans, early fusion of the premaxilla and maxilla does not occur in apes.

Evidence to support the hypothesis that the nasal septum lacks status as a primary growth centre is considerable in animal experiments. For obvious reasons, similar experiments in Man are lacking and most of the

information to date results from investigations into abnormal growth associated with trauma or congenital absence of tissues.

Latham and Burstone (1966) studied a case of a 9 month old baby suffering from congenital arrhinencephaly and observed deficient antero-posterior midfacial development. They concluded that the nasal septum is important in determining anteroposterior maxillary development but, in the light of near normal vertical development, is was not absolutely essential to growth in the latter dimension. Further, vertical growth of the maxilla appeared to be the result of surface opposition on the palate and alveolus (concomitant with tooth eruption) and corresponding enlargement of the nasal cavities and maxillary sinus.

Moss and Bromberg (1968) on the other hand, with similar material (two cases of 3 year old children with congenital arrhinencephaly) concluded that midfacial growth was independent of the nasal septum which merely played a passive role.

These two conclusions (based on similar material) underline the confusion that still exists.

The relative premaxillary protrusion and midfacial retrusion often seen in bilateral cleft lip and palate cases (Johnston, 1976) has been explained as being due to growth of the nasal septum in the absence of connections with the maxillae. This contrasts with claims by Babula, Smily and Dixon (1970) which have been mentioned, although phylogenetic differences between mice and human must qualify any comparison.

An area that has received little attention in the literature is the

restriction of midfacial growth that may result from premature ossification of the nasal septum. Further, the formation of abnormal synostoses with its bony neighbours may retard midfacial development. The effects of similar occurrences affecting the cartilagenous anterior cranial base in cases of congenital craniostenosis have been reviewed in Chapter 2.

Scott (1953) described four centres of ossification in the foetal nasal septum.

1. basioccipital plate;
2. basisphenoid centre;
3. presphenoid centre;
4. mesethmoid centre.

During growth, ossification in these areas proceeds until, in the adult, only the anterior third of the septum is unossified.

Fusion at the posterior extremities of the septum with the bones of the cranial base (sphenoid, basal, ethmoid) may provide a pathway by which abnormal cranial base growth may affect maxillary displacement - particularly in an anteroposterior direction (Tessier, 1971).

Surgical evidence of sphenomaxillary synostoses has been discussed by Enlow et al. (1977) who stated that

Anatomic research could shed light on whether maxillary hypoplasia is due to synostosis of the sutures or perhaps, to failure of normal differentiation in the anterior chondrocranial structures.

The formation of sutural synostoses may in turn, be merely a consequence of premature ossification of the anterior cranial base.

Björk and Skieller (1976) examined growth of the maxilla by means of implants. Changes in the relative positions of the implants were evaluated radiographically in 3 dimensions (sagittal, transverse and vertical) in a group of boys from 4 years of age to adulthood.

They concluded that maxillary displacement was associated with a complex combination of rotations of the two maxillae relative to each other in the transverse plane, forwards movement in the sagittal plane and a forward or backward rotation of the maxilla (as a whole) in the vertical plane.

They claimed that the three-dimensional displacement of the maxilla is associated with such a strongly differentiated growth pattern that the primary growth source is unlikely to be the nasal septum or the facial matrix. The inference is that a multi-factorial system of growth regulation is in operation.

Latham (1976) examined a number of foetuses and neonates with facial deformities. The cases examined were a 6 week old infant with bilateral cleft lip and palate, an arrhinencephalic child (from birth to 9 months of age) and two specimens of cyclopia.

Latham demonstrated that displacement growth of the maxilla occurred in these cases in the absence of:

1. normal septomaxillary connections;
2. the nasal septum;
3. the nasal capsule;
4. the nasal cavities.

He found further that osteogenesis was occurring on the posterosuperior surface of the maxilla.

On the basis of these observations Latham (1976) modified the nasal septum hypothesis (Scott, 1953) and stated that

the nasal septum appears to be an important maxillary pacemaker in prenatal but not post-natal life.

Further, he postulated that the primary mechanism of maxillary displacement was forward pressure applied by periosteal osteogenesis on fatty tissue encapsulated in a fibrous tissue matrix and lying adjacent to the posterosuperior surface of the maxilla.

This postulate appears to qualify the hypothesis of Enlow and Bang (1965) that the downwards and forwards movement of the maxilla is the result of growth that takes place in a posterior direction.

Growth and displacement of the maxilla is a complex process.

It is evident from the literature that, whilst there is some detailed understanding of the basic processes of bone growth (remodelling, sutural and cartilagenous growth), there is considerable diversity in opinion regarding the processes whereby these processes are regulated and more importantly, limited.

Enlow (1983) stated that

growth for any individual is normal, given the circumstances that exist for that individual. Growth is a process of striving toward a state of functional equilibrium - unless pathology is involved. Most structural displasias are in equilibrium.

The process of morphological expression of genetic information may never be fully understood and it is conceivable that the control processes are so complex and interdependent that their isolation and identification may well be impossible.

It does appear that a degree of adaptability to functional requirements and environment exists. The existence of adaptive growth may provide a possible explanation of differences of growth mechanisms between the "normal" and "abnormally" large or small maxilla/mandible. So-called abnormal growth patterns may be the result of variations in either control of adaptive growth or the extent of the adaptive response of the individual.

Determinations for methods by which these abnormal growth patterns may be influenced would require an understanding of the adaptive response at a cellular level. This is not currently forthcoming.

The majority of evidence to support the existence of adaptability in growth processes, however, relates to:

1. experiments with animals where phylogenetic differences may invalidate comparisons with the human condition, and
2. human material in which growth patterns in pathological states exists, or where there is congenital absence of tissues (for example congenital arrhinencephaly). Extrapolation of observations in these cases to individuals exhibiting "normal growth patterns" may also be invalid.

CHAPTER FOUR

DIAGNOSIS AND GROWTH PREDICTION

4.1 Diagnosis

The problems associated with classifying the various forms of Class III malocclusions into defined clinical entities have been discussed in Chapter 1. These problems also influence accurate diagnosis, treatment planning and prognosis.

In this chapter an examination of the literature will be made with respect to:

- a. methods of differentially diagnosing "true and pseudo Class III" malocclusions in the deciduous and mixed dentition;
- b. features of the malocclusion which may be used to relate the diagnosis to treatment regimes;
- c. features of the malocclusion which may be used to assess the prognosis for each case;
- d. the current status of growth prediction in relation to early Class III malocclusions and the use of such predictions in treatment planning and prognostic evaluations.

As can be seen from the foregoing, the variable and complex aetiology of Class III malocclusions raises problems with regard to accurate diagnosis. Class III malocclusions may be encountered which present features ranging from Class I skeletal with functional anterior shift to severe skeletal problems involving one or both jaws.

Obviously though, some attempt must be made to identify cases with

respect to suitable treatment regimes and prognostic evaluations.

Foster (1975) lists the main variables in Class III features as

1. the degree of Class III anteroposterior relationship of the dental arches;
2. the degree of lateral discrepancy of the maxilla and mandible;
3. the degree of incisor overjet and overbite;
4. the degree of crowding.

To this list could be added

1. the degree of anteroposterior skeletal discrepancy of the jaws;
2. the degree of dental compensation for skeletal discrepancies.

The initial clinical examination of facial profile, functional movements of the mandible, and the occlusion may provide valuable information.

Examination of the facial profile should include an assessment of the relative proportions of facial components - forehead, nose, lips and chin - with an evaluation of the overall balance of these components (Rakosi and Schilli, 1981). Total facial height and the size relationship of the upper, middle and lower thirds of the face should be evaluated visually. From this may be gained an approximate idea of the underlying skeletal pattern.

In addition, frontal assessment of the face may reveal the presence of any asymmetry in facial form.

Cozzani (1981) points to the importance of identifying forced anterior or lateral positioning of the mandible on closure into the maximal intercuspal position. He claims that this is generally due to abnormal tooth

tooth contact relationships which force the anterior shift to obtain a convenient occlusal position.

Table 4.1 shows the clinical significance of this functional anterior shift with regard to the anticipated degree of difficulty in treatment.

Further, Cozzani (1981) emphasised the importance of ensuring that the mandible is in its most retruded position for accurate diagnosis, even though in this position only a few teeth may be in contact.

It would be expected that this posterior position of the mandible would have the effect of increasing total and lower third face height (due to occlusal interferences) and decreasing the prominence of the chin in profile.

Type	Anterior position of mandible	Centric relation and centric occlusion	Significance
Pseudo Class III	Present	Not co-incident	Problem is less difficult than it appears (Class I)
True Class III	Present	Not co-incident	Problem is less difficult than it appears
True Class III	Absent	Coincident	Problem is as difficult as it appears

Table 4.1 The clinical significance of assessing mandibular movement from centric relation to centric occlusion from Cozzani, 1981.

Thus the profile assessment should involve the mandible in both full posterior occlusal position and most retruded position. Although Table 4.1 is

oversimplified and lacking in diagnostic criterion, it does illustrate that a "feeling" for the case can be gained at the initial examination.

The presence of anterior or posterior crossbites (or both), ectopic teeth (particularly incisors) and wear facets may be noted. Here again, an attempt should be made to assess whether the crossbites are due to postured positioning of the mandible and if so how this can be related to the presence of ectopic or malaligned teeth and wear facets. A crossbite of a single incisor may cause a pseudo-Class III malocclusion, or mask a true skeletal problem (Schwarze, 1970).

The presence of wear facets may help locate occlusal interferences. Likewise, the presence of wear facets on teeth which no longer contact may indicate recent rapid mandibular growth in skeletal Class III's.

It has been claimed (Rakosi and Schilli, 1981) that a family history of Class III features may be of importance in diagnosis and prediction of future growth. Whilst this may be so, information gained should be considered in the light of the variable expression of Class III malocclusions within families. This is vividly demonstrated by Schwarze (1970) in Fig. 4.1.

Cephalometric analysis is a widely used method of obtaining diagnostic and prognostic assessments of both dentally based and skeletally based malocclusions.

Maj and Luzi (1962) developed a technique for evaluating cephalograms based on an analysis of the correlations between specific features present in each patient.

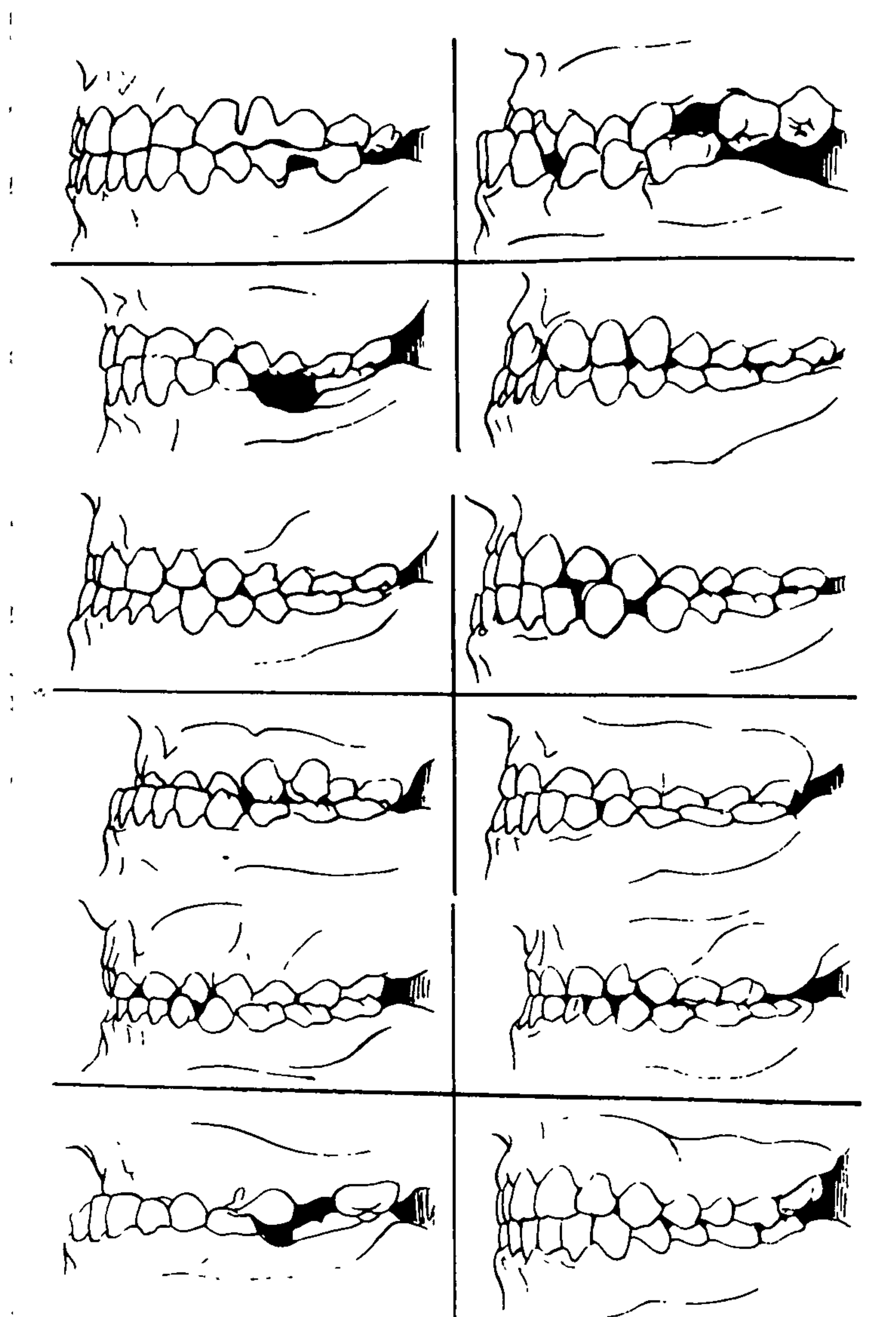


Fig. 4.1 Different phenotypes of Class III from the same family from Schwarze, 1970.

This was based on their theory that facial types were the result of "different combinations of the infinite variants in skeletal parts". They claimed that assessment of a particular measurement as a unit was less meaningful than assessments of the correlations between a number of measurements of factors that affect the degree of overjet.

A series of measurements (Fig. 4.2) are taken from which mandibular

size, shape and position, as well as the position of the lower and upper incisors relative to their supporting bones, are estimated.

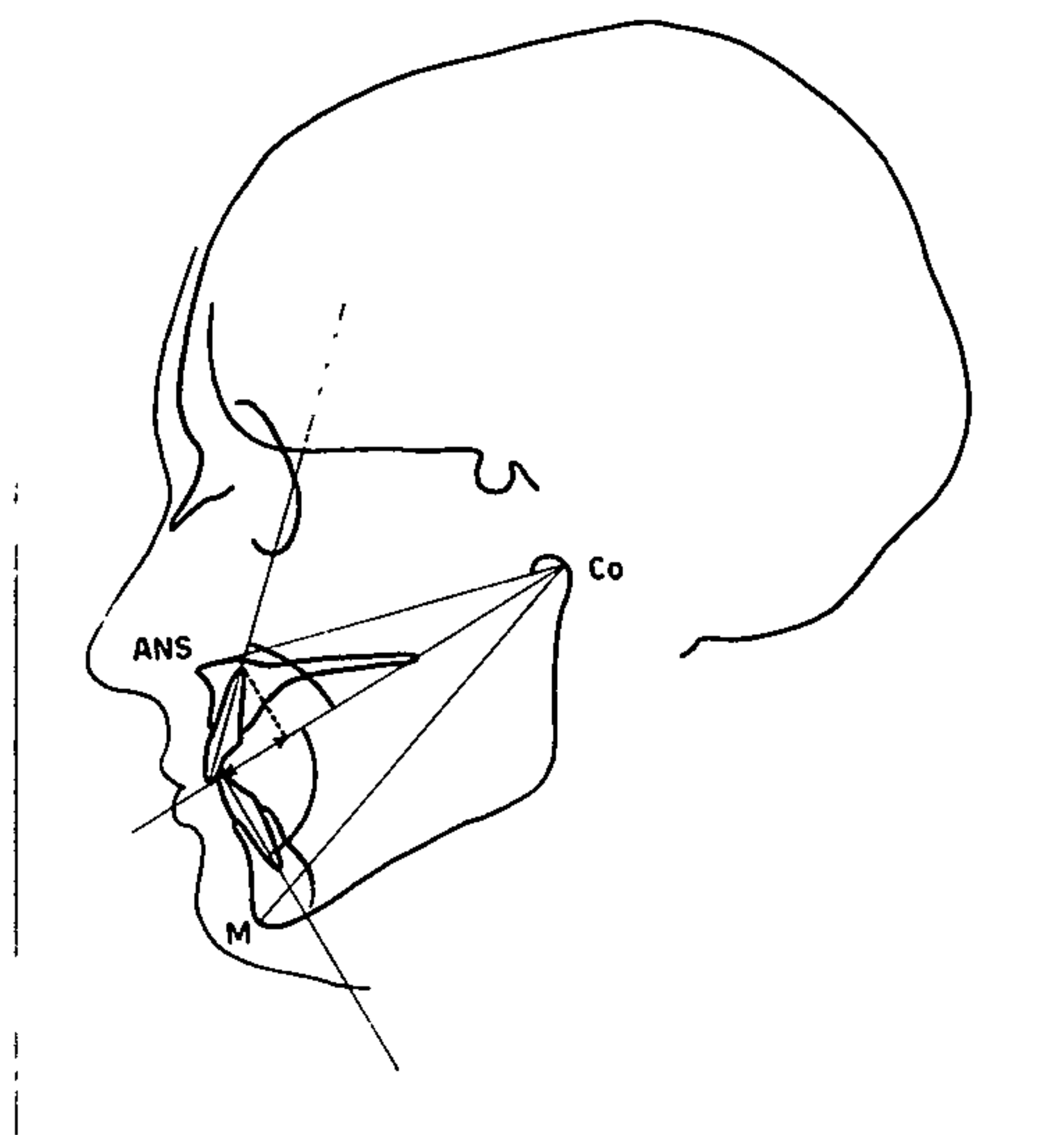


Fig. 4.2 Method of Analysis; skeletal and dental cephalometric criteria. Co-M = mandibular length; ANS-Co distance = anteroposterior maxillary position; Co-M and Co-ANS linear difference = length ratio of basal bones; interincisal axial inclination angle, as measured to the bisector of angle ANS-Co-M; distance between maxillary and mandibular incisor apices, as measured to the bisector of angle ANS-Co-M, from Maj and Luzi, 1962.

With this method these authors identified three variants in Class III malocclusions, which present widely different prognostic and therapeutic viewpoints.

1. The first is characterised by small negative values (up to -3) of the sum of the skeletal ratios. These cases it was claimed, are due to mesial movement of the mandibular dental arch on its bony base. These are primarily a dentally-based malocclusion, have a good prognosis and may be treated by conventional orthodontic methods.

2. The second variant is due to mesial mandibular displacement (pseudo Class III). The values are a slightly abnormal and negative sum of the skeletal ratios and with a normal mandibular length. It is further

characterised by a markedly horizontal position of the mandible and reduced facial height. The prognosis is stated to be favourable and responds well to activator therapy.

3. Thirdly, excessive size of the mandible gives values which are high and negative for the sum of the skeletal ratios, and high for total mandibular length (Co-M). When values for the skeletal ratios exceed -8 in these cases, the authors claim that orthodontic treatment is not advisable.

In addition, the prognosis is influenced by (Maj and Luzi, 1962, Bassani, 1970):

- a. the position of the mandibular condyles with respect to the cranial base;
- b. the gonial angle;
- c. the inclination (vertically) of the mandible.

With respect to these features the following are favourable (prognostically):

- 1. distal position of the condyles;
- 2. obtuse gonial angle;
- 3. downwards and backwards inclination of the mandible.

The method of Maj and Luzi (1962) is claimed by the authors to be suitable for all age groups, however the following points are noteworthy:

- 1. no specific mention is made of suitability for the deciduous dentition;
- 2. no assessment is made of lateral discrepancies between the jaws;
- 3. no allowance is made for retarded maxillary development (as regards maxillary size, shape or position;

4. no assessment of the length of the anterior cranial base is made.

All of these factors also play an important role in prognostic and therapeutic evaluations, as well as aesthetic considerations.

Rakosi (1970) compared mean values for 30 Class III cases with the average for normal cases (number of cases and definition of normal not specified) based on cephalometric tracings (Fig. 4.3) and found that the Class III's had (as expected) longer mandibles and shorter maxillas. However there were consistent differences in other measurements as well and Rakosi (1970) stated that "the basis of the anomaly is not localised in one of the jaws, but in the linear and angular correlations of the whole facial skeleton".

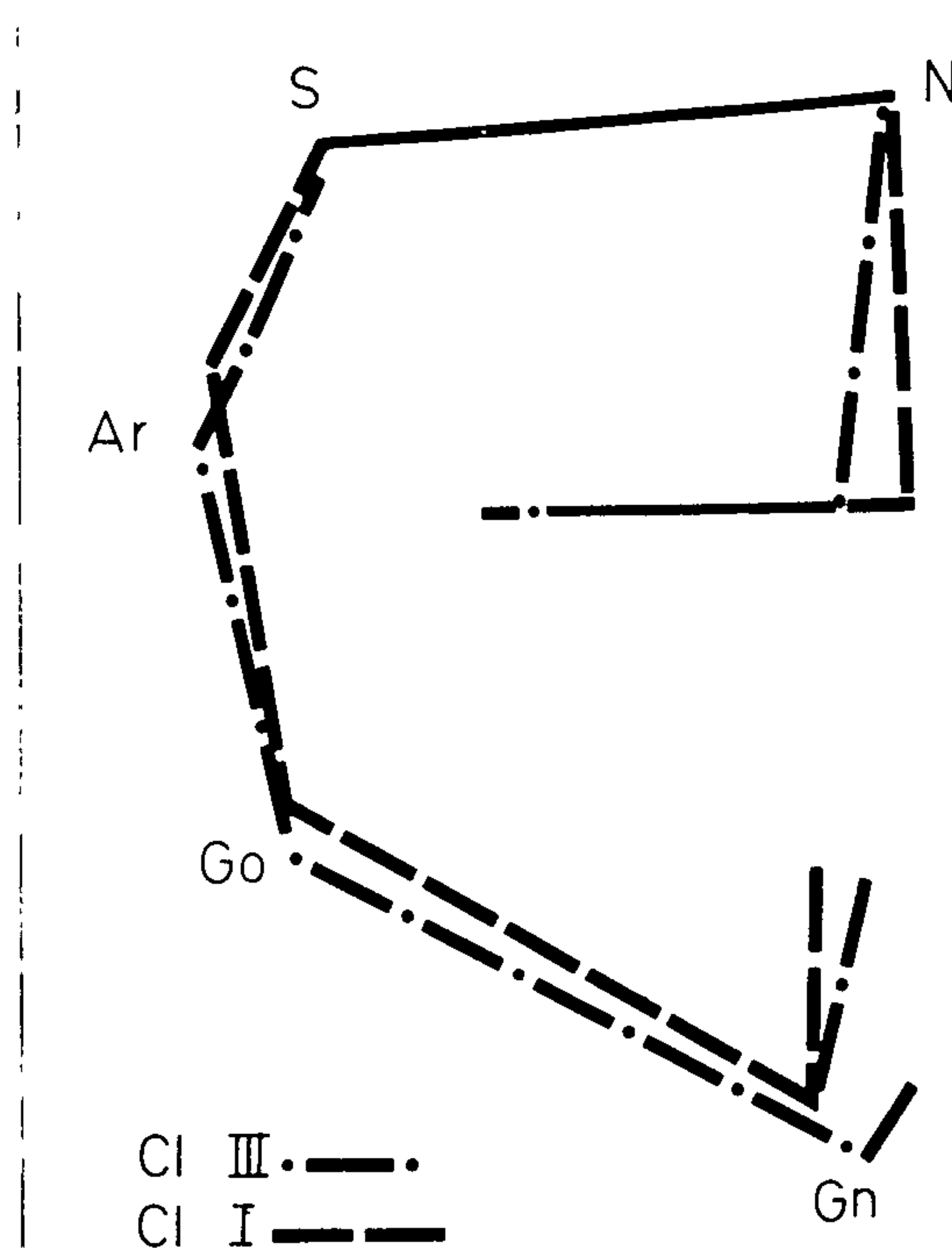


Fig. 4.3 Facial diagram comparing mean values of Class I and Class III from Rakosi, 1970.

The measurements used in this study are shown in Table 4.2. The significance of the values is in the difference between the Class III and Mean

(Class I) measurements, as the measurements themselves are valid only for the particular population (not stated) used in the study.

Measurement	Class II	Mean	Class III
Saddle angle (N-S-Ar)	129.2°	124.0°	122.2°
Angle of convexity (N-A-Pog)	+12.0°	1.24°	-12.5°
Joint angle (S-Ar-Go)	144.1°	143.0°	139.3°
Gonial angle (Ar-Go-mand.pl.)	122.0°	123.0°	132.0°
Go ₁ angle	-1.2°	9.0°	12.7°
Maxillary basal progn. (S-N-A)	82.7°	82.0°	81.5°
Mandibular basal progn. (S-N-Pog)	77.0°	81.0°	85.9°

Table 4.2 Comparison of Class II and Class III cephalometric measurements from Rakosi, 1970.

Ruhland (1975) examined 200 untreated Class III cases with mesio-occlusion and angular measurements are shown in Table 4.3.

Statistical analysis of these results (Students 't' test at 1% level) showed that the significant Class III features were:

- a. reduced SNA (permanent dentition only);
- b. increased SNB;
- c. increased ANB;
- d. reduced NS Ar (articulare);
- e. enlarged gonial angle;
- f. decreased maxillary length;
- g. increased mandibular length.

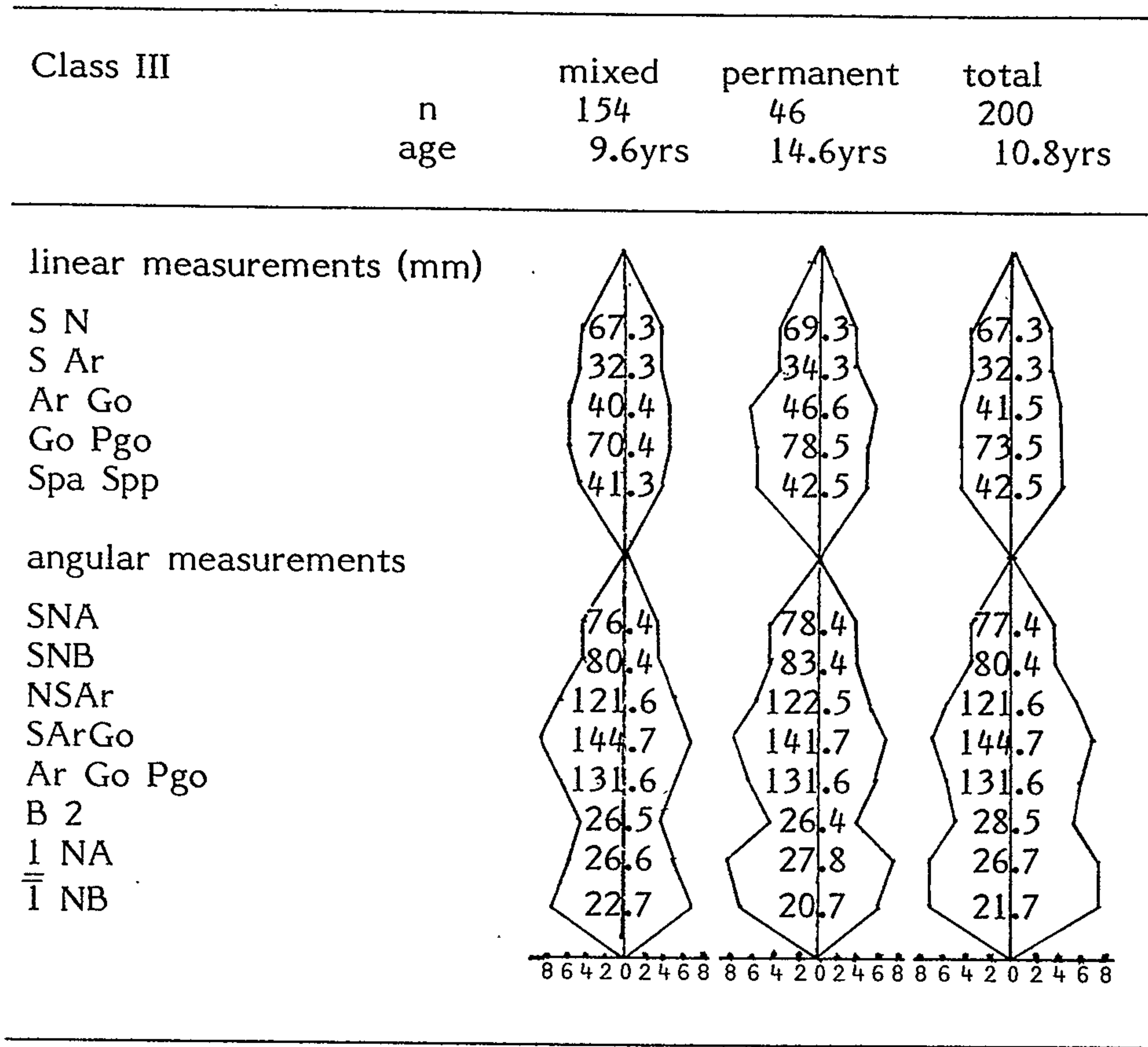


Fig. 4.3 Cephalometric measurements for 200 Class III patients in the mixed and permanent dentitions. The spread of mean values is shown by the mean standard deviation polygons, from Ruhland (1975).

Ruhland (1975) claimed that, whilst class III was a highly variable entity, the above measurements were consistently present and thus could constitute a factor analysis for diagnostic purposes.

This author also presented (Table 4.4) positive and negative prognostic factors although no post-treatment analyses were presented to support this.

Ruhland (1975) concluded that the results indicated a progressive development of Class III features and that this dictated that treatment is started early, with long retention to stabilize the result.

Class III factors	positive	negative
ANB angle	$\bar{+} 0^{\circ}$	< 0
$\underline{I}$ NA angle	retrusion	protrusion
$\bar{I}$ NB angle	protrusion	retrusion
Holdaway relation	2:1	1:2
B 2 angle max.-mandible planes	$< 20^{\circ}$	$> 30^{\circ}$
inclination of maxillary plane to the Frankfurt plane	+ $\bar{+}$	-
Go angle	$< 120^{\circ}$	$> 130^{\circ}$
Björk relation: $\frac{\text{length of cranial base}}{\text{length of mandible}}$	$x:(x+y)$ $y=5\text{mm}$	$x:(x+y)$ $y>5\text{mm}$
overbite	deep	flat, open
overjet	$\pm 0\text{mm}$	$< 0\text{mm}$

Table 4.4 Class III features associated with good (positive) and poor (negative) prognosis from Ruhland, 1975.

The material presented in this study would have provided a good opportunity to examine profile changes with growth, and to relate profile assessment radiographically to that observed clinically.

Moss (1976) assessed 18 children (mean age 14.7yrs) with skeletal Class III and 11 children (mean age 10.1yrs) with a postural Class III. His results are shown in Table 4.5.

Whilst the measured values in general conform with those previously presented for skeletal Class III's Moss's results indicate several marked differences in the postural Class III group.

Skull measurements for patients with Class III malocclusion				
Measurement	<u>Skeletal</u>		<u>Postural</u>	
	Means	s.d.	Means	s.d.
Overbite (mm)	-2.0	2.7	-4.1	3.3
Overjet (mm)	-4.0	3.2	-3.4	2.1
Maxillary/mandibular planes angle (°)	28.0	5.4	25.6	3.9
Angle SNA (°)	79.3	4.1	80.3	4.6
Angle SNB (°)	83.2	4.9	81.6	3.6
Angle upper incisors (°) to SN	112.8	10.9	111.1	11.3
Angle lower incisors to SN	83.0	10.4	87.5	6.1
Nasion-sella-articulare angle (°)	126.2	4.8	127.6	2.9
Gonial angle (°)	134.2	6.0	129.3	5.7
Angle of upper and lower incisors (°)	138.3	13.2	137.2	15.3
Nasion-menton distance (mm)	116.8	14.9	107.0	5.1
Maxillary/superior occlusal planes angle (°)	8.1	5.8	10.9	5.8
Maxillary/inferior occlusal planes angle (°)	4.9	5.3	3.6	5.1
Age (years)	14.7	6.4	10.1	1.7

Table 4.5 Dental and cephalometric measurements of 18 skeletal and 11 postural Class III children from Moss, 1976.

Facial height was greater in the skeletal group and this was associated with the more obtuse gonial angle and higher age in this group. The increased overbite in the postural group may be associated with some degree of overclosure and this may reduce facial height.

In addition, ramus height (Go-Co) and upper face height (ANS-N) may be contributing factors, although these were not assessed.

The ANB angle difference between the two groups is significant - particularly as the cephalograms of the postural group were taken with the mandible in the forward position and not edge-to-edge; a fact not stated by Moss (1976) but indicated by the deep overbite and negative overjet.

It has been claimed (Haralabakis and Spyropoulos, 1977; and Cozzani, 1981) that forward posturing of the mandible will potentiate both SNB and ANB measurements, and that recordings should be done with the mandible in its most retruded position.

This may more accurately reflect the anteroposterior relationship of the mandible to the maxilla, although it would tend to exaggerate total facial height and the mandibular plane to SN angle.

Gravely (1984) disputed the assumption that forward posturing occurred in postural Class III's when the posterior teeth were in occlusion. He used the term "posterior occlusion position" to indicate the position of the mandible when the upper and lower posterior teeth were in contact as compared with the edge-to-edge position where posterior occlusal contact was absent. He claimed that differences in SNB and ANB between the edge-to-edge and posterior occlusion position of the mandible were due to vertical movement of the mandible rather than posturing. Rotation of the mandible during closure would, he claimed move point B along an arc in a forwards and upwards direction. Tracings of the mandible and mandibular dentition were rotated about a pin placed at the most superior point of the condyle until the overbite was reduced to zero. Measurements of SNB, SNA and ANB were then taken and correlated to those taken with the incisors in the edge-to-edge position.

He claimed that, statistically, there was no significant difference between the two and concluded that, when the posterior teeth were in occlusion, no residual anterior displacement of the mandible existed. On the basis of this he further concluded that the skeletal pattern is accurately reflected in the posterior occlusion position and that there was no requirement for a cephalogram with the incisors edge-to-edge.

Several points warrant consideration.

1. Gravely (1984) assumed that the mandible rotates about a fixed point on the superior surface of the condyle.
2. SNB and ANB were significantly increased in the posterior occlusion position and this increase was related to the degree of overbite - the deeper the overbite, the greater the increase in SNB and ANB.

The concern here is that a shallow-overbite pseudo- or postural-Class III may well be misdiagnosed as a skeletal problem if the cephalometric assessment is made only in the fully closed position.

Campbell (1983) quoted Turpin (1981) as listing features of Class III malocclusions that indicated a favourable prognosis and the need to consider early treatment. These were:

- a. convergent facial type;
- b. anteroposterior functional shift;
- c. symmetrical condyle growth;
- d. young (growth remaining);
- e. ANB less than -2° ;
- f. good cooperation expected;
- g. no familial prognathism;
- h. good facial aesthetics.

The facial type in (a) could be better termed "hypo-divergent".

Incisor inclination has been omitted from this list, and no mention of it as a prognostic or diagnostic feature was included by Campbell (1983).

Several authors have discussed the significance of incisor inclination

(Ruhland, 1975, Rakosi and Schilli, 1981) in assessing the degree of dental compensation present for the skeletal discrepancy. Proclined maxillary incisors and/or retroclined mandibular incisors may restrict the amount of orthodontic compensation that may be achieved.

Carlotti and George (1981) list incisor inclination as one of a number of criteria for differentially diagnosing maxillary deficiency from mandibular prognathism (Table 4.6).

Whilst this list is primarily designed to assess adult patients pre-surgically it appears to have application in earlier age groups.

Tongue size, shape and position has been cited as important diagnostic and prognostic features.

Rakosi (1970) made seven measurements of the tongue on cephalograms taken in centric occlusion and rest position. He evaluated the root of the tongue, the back of the tongue in relation to the palatal vault and the position of the apex of the tongue relative to the lower incisors.

Rakosi (1970) found that among Class III patients the tongue was flat and anteriorly positioned, but not enlarged. In addition the tongue was more protrusive in rest position than in centric relation. The post-treatment assessment indicated little change in tongue position.

Bassani (1970) claimed that the tongue analysis was important as its size, behaviour and position may influence post-treatment stability although this author did not discuss the reasons why this may be so.

Maxillary deficiency	versus	Mandibular prognathism
I. Initial assessment - Frontal		
A. Tendency of upper lip to be thin (vermilion border less visible)		A. Normal upper lip form
B. Normal to deficient upper tooth-to-lip relation		B. Normal relation of upper tooth to lip
C. Shallow paranasal form (sallow)		C. Normal paranasal form
D. Narrow alar base width		D. Normal alar base width
E. Tendency to show sclera above lower eyelid		E. Normal sclera-to-lower-lid relation
F. More nearly normal chin projection		F. Prominent chin
G. Normal to decreased lower facial height (LFH)		G. Normal to increased lower facial height (LFH)
II. Facial Assessment - Profile		
A. Shallow paranasal form		A. Pseudoshallowness
B. Nasolabial line - Subnasale: subnasale-tip of nose usually not 1:1 ratio		B. Normal 1:1 ratio
C. Nasal tip down		C. Normal nasal tip
D. Obtuse nasolabial angle		D. Normal to acute nasolabial angle
E. Assuming proportionate nasal form, chin normally rotated to nasal tip		E. Chin anteriorly related to nasal tip
III. Smiling assessment		
A. Less incisor visible (teeth seem to be under the upper lip)		A. Good tooth/lip relationship
IV. Cephalometric assessment (Fig. 1)		
A. Normal to decreased total facial height		A. Increased total facial height
B. Maxillary molars distal to key ridge		B. Maxillary molars normal to key ridge
C. Decreased distance of pterygo-maxillary fissure to maxillary first molar		C. Normal distance of pterygo-maxillary first molar
D. Orbital plane anterior to maxillary canine apex		D. Orbital plane passing through maxillary canine apex
E. Facial convexity less than normal		E. Facial convexity normal
F. Normal ramus width		F. Narrow ramus width
G. Gonial angle normal		G. Gonial angle obtuse
H. IMPA normal		H. IMPA decreased
I. Lower incisor-NB line: NB line-pogonion, 1:1 ratio		I. Lower incisor-NB line: NB line-pogonion, not 1:1 ratio
V. Occlusal Assessment		
A. Class III malocclusion		A. Class III malocclusion
B. Tendency toward crowding and missing teeth		B. Normal maxillary arch
C. Transverse deficiencies noticeable in maxillary arch		C. Broad mandibular arch form
D. More nearly normal inclination of mandibular anterior teeth		D. Tendency toward linguoversion of lower anterior teeth

Table 4.6 Features used to distinguish skeletal Class III malocclusions due to maxillary deficiency from those due to mandibular excess from Carlotti and George, 1981.

Cozzani (1981) stated merely that low tongue posture may adversely influence the growth pattern. Again, the reasons for this were not discussed.

The role of the tongue in aetiology of Class III is discussed in Chapter 2.

Thus the importance of a complete and thorough examination is stressed. The variability of Class III malocclusions affects not only the diagnosis, but also the response to treatment and stability of the final result.

4.2 Growth Prediction

The ideal in growth prediction should "describe the sagittal and vertical intermaxillary relationships at the end of the growth period on the basis of the morphological features at any time during the growth period, and should also estimate the intensity and timing of growth, variations in direction and the ultimate size" (Lavergne, 1982).

This infers a truly individualised projection of growth based solely on the morphological features present in the patient being studied.

Growth is of course, a complex and multifactorial process, the outcome of which is determined by both the genetic code and local and general environmental factors (soft tissue function, habits, and nutritional status etc.). Consequently a method of growth prediction as described above would require a complete understanding of the processes involved and, as discussed in Chapter 3, it seems that this is unlikely to eventuate.

In the past, attempts have been made to correlate specific morphological features with growth direction and magnitude.

Hixon (1968) assessed the correlation of features present at particular ages with growth occurring through specific periods (Table 4.7).

Size Gain Relationships

Dimension	Correlated with r	Growth from
Stature at 6 yrs	.07	6 to 18 yrs
Body Weight at 9 yrs	.21	9 to 18
Bizygomatic Diam. at 5 yrs	.09	5 to 11
Nose Depth at 8 yrs	.29	8 to 14
Upper Lip Protrusion at 8 yrs	.16	8 to 14
Upper Face Depth (APOC ANS) at 5 yrs	.10	5 to 15
Mand. Length (APOC PO) at 5 yrs	.15	5 to 15
Mand. Length (Co Po) at 12 yrs	.09	12 to 20
Upper Face Ht. (N ANS) at 5 yrs	.11	5 to 15
Lower Face Ht. (ANS M) at 5 yrs	.15	5 to 15
Overbite at 12 yrs	.31	12 to 20
Max. Arch Width at 9 yrs	.15	9 to 15
Mand. Arch Width at 9 yrs.	.41	9 to 15

Table 4.7 Correlation of features present at particular ages with growth occurring through specific periods. Correlations above 0.4 are statistically significant academically although only those above 0.7 are clinically useful, from Hixon (1968)

At this point it should be stated that the value of r indicates the level of dependence of two events - a value of 0 indicating complete independence and a value of 1.0 complete dependence. Houston (1979) stated that whilst values above 0.4 were statistically significant in an academic sense, correlations above 0.7 were required before they may be used clinically with any degree of confidence.

As can be seen from Table 4.7, no clinically significant correlations were shown. Indeed, Hixon (1968) supported this - "the evidence is quite conclusive, there is nothing of clinical usefulness".

Hixon (1968) concluded that "the best estimate available of adult facial dimension for a given child is to use the dimension presented by the child and add to that the remaining average growth for the group".

This technique was used by Ricketts (1957) and statistically tested by Schulhof and Bagha (1975). Essentially, it involved assessment of the growth pattern (whether vertical or horizontal) from a cephalometric radiograph and the subsequent addition of the mean average increments for the group (appropriate to the patient) in a direction consistent with the originally assessed pattern.

Schulhof and Bagha (1975) claimed that "over the longer term, the majority of growth is consistent enough to be considered predictable (70%)".

It was not clear if the 70% quoted related to the percentage of growth that was predictable or the percentage of patients in which accurate prediction was made.

Shulhof, Nakamura and Williamson (1977) assessed the accuracy of computer predictions for Class III cases using a similar method. They modified the growth increments for the Class III pattern although the theoretical basis for the modifications was not clear (Houston, 1979). These modified increments were then added to the existing pattern.

Table 4.8 shows the comparison of the computer prediction with actual growth.

Patient	Effective yrs. growth	BA/NA			Corpus Axis		
		Actual growth	Computer prediction	Error	Actual growth	Predicted growth	Error
S.K.	3.7	4.0	5.5	1.5	6.5	10.67	+4.17
T. Tani	3.9	7.0	7.0	0.0	9.5	9.11	-0.39
K.I.	3.9	6.5	7.0	0.5	6.5	5.33	-1.17
M.E.	3.9	6.5	6.0	-0.5	8.0	7.11	-0.89
M.F.	4.0	5.3	5.5	+0.2	4.0	7.11	+3.11
T. Take	4.0	6.5	6.0	-0.5	6.5	4.89	-1.61
M.S.	3.9	6.0	6.5	0.5	6.0	6.67	+0.67
H.M.	3.5	7.5	5.5	-2.0	3.5	4.89	-1.39
J.U.	1.6	2.0	0.0	4.5	3.56	-0.94	
S.O.	3.7	4.0	4.5	0.5	8.0	9.78	+1.78
S.W.	3.8	6.0	6.0	0.0	11.0	8.89	-2.11
Y.Y.	3.9	4.5	6.0	1.5	4.0	6.04	+2.04
4.M.	3.9	5.0	7.0	2.0	7.5	6.22	-1.28
Y.T.	<u>3.9</u>	<u>7.0</u>	<u>6.0</u>	<u>-1.0</u>	<u>5.5</u>	<u>7.11</u>	<u>+1.61</u>
Mean	3.69	5.56	5.75	0.19	6.50	6.96	0.26
Standard deviation	0.62	1.52	1.28	1.05			1.97
RMS error				1.03			1.91
Per cent of growth				18			27

Table 4.8 Comparison of actual growth and computer predictions for cranial base (BA/NA) and mandibular corpus axis from Schulhof, Nakamura and Williamson, 1977.

These authors claimed that these results indicated an 82% accuracy for prediction of cranial base growth (BA/NA) and 73% for the mandible.

It can be seen from the table that whilst the accuracy is good for some patients, for others it is quite low. The authors offered no method of identifying which patients are likely to be unpredictable.

Marston (1979) claimed that, with respect of these findings, if the patients had grown by the average amount that the root mean square error is not appreciably greater than the computer prediction. In other words, the

Class III modification apparently had little influence on the increment amount.

Greenberg and Johnson (1975) compared the error with Ricketts' method (the difference between the actual growth and the computer forecast) with two alternatives:

1. an assumption of no further growth;
2. an assumption that each patient will change by an equal amount to the mean for a separate group of 80 subjects - they concluded that the method was superior to the first alternative but neutral with respect to the second and stated "less pretentious methods might prove equally satisfactory".

Johnston (1975) presented a forecast grid (Fig. 4.4) which, like the previous method involved the addition of average increments calculated for a group. At least two lateral cephalograms were required, and were superimposed on the anterior cranial base (SN) directly on the grid.

Johnston (1975) stated that inherent in any method of prediction using cephalograms were errors of accurate superimposition, point location and duplication.

This and other techniques using cephalometric landmarks have several disadvantages:

1. They assume no change in the position of point S, or the inclination of SN;
2. They rarely specify the various groups from which the "normal average increments" are taken;
3. They, by definition, do not allow for individual variation;

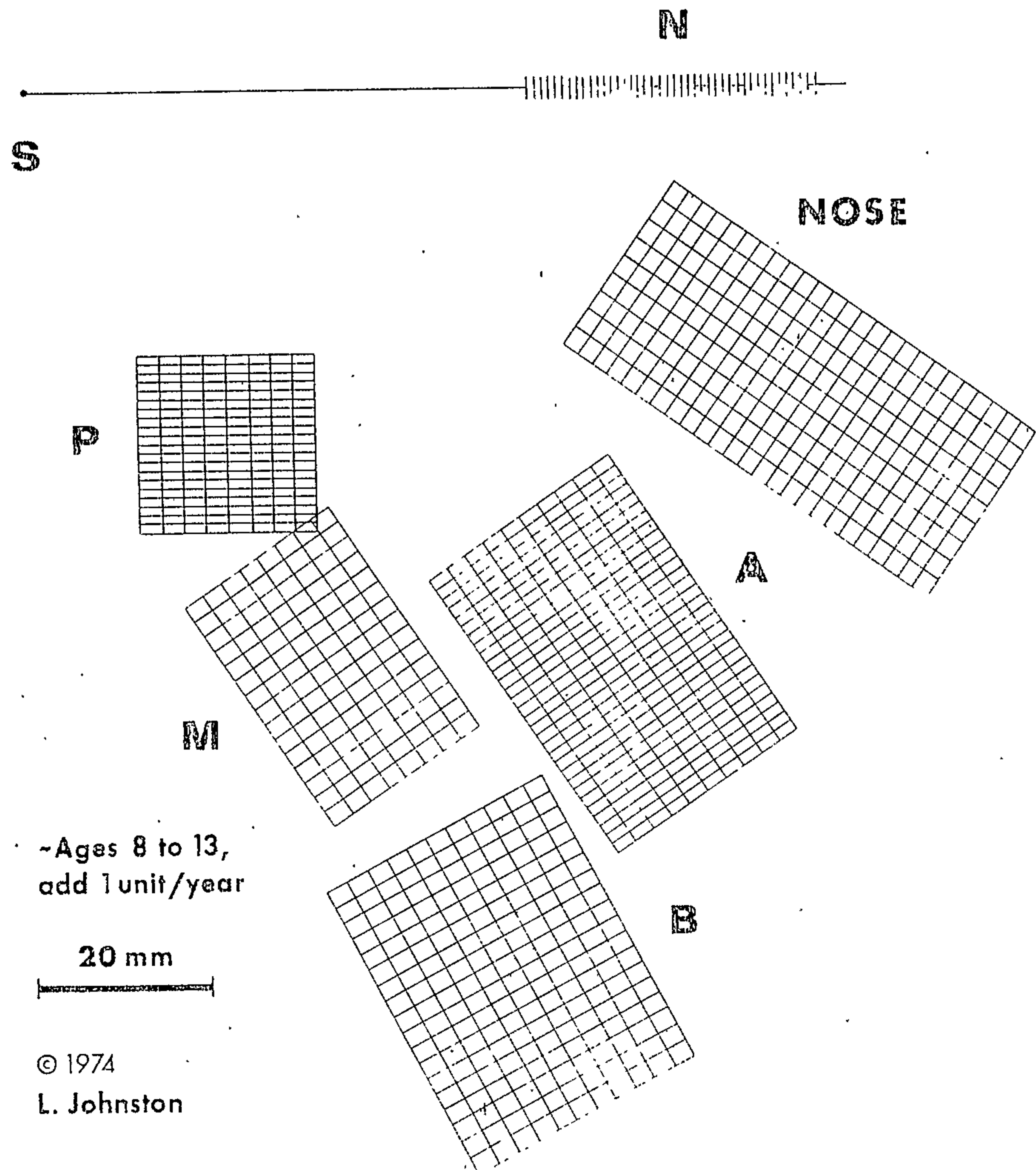


Fig. 4.4 Forecast grid. S, sella; N, nasion; P, posterior nasal spine; NOSE, tip of the nose; M, any point on the crown of the maxillary first permanent molars; A and B, subspinale and supramentale. A tracing of the landmarks is superimposed along S-N and registered at S. The points are then advanced downward and forward one unit per year; from Johnston, 1975.

4. They may indicate the direction of growth, but cannot indicate the magnitude;

5. The standard deviation for a group may in some cases be greater than the actual measurement. Björk and Palling (1955) showed that in Swedish boys the change in ANB from 12-20 years was 0.7° with a standard deviation of 1.3° .

Björk (1969) described three methods of growth prediction.

1. Longitudinal method based on annual cephalograms and the assessment of the growth pattern on the basis of previous growth. Björk claimed that "this method has the general limitation in the fact that the pattern of growth is not constant and the pattern recorded at a juvenile stage may well have changed by adolescence".

2. Metric method, where prediction of growth is based on facial morphology assessed on a single x-ray film. Björk found weak correlations only between the dimensions of the face at 12 years and residual growth and also the amount of vertical face growth.

3. Structural method. This was developed by Björk from his implant work and assessed the trends of mandibular growth as based on the identification of specific structural features associated with different types of rotation.

Several features were identified.

1. inclination of the condylar head;
2. curvature of the mandibular canal;
3. shape of lower border of the mandible;
4. inclination of the symphysis;
5. interincisal angle;
6. intermolar or interpremolar angles;
7. anterior lower face height.

Björk asserted that variations in the position of the mandible during growth were due to differences in the direction of condylar growth which was in turn related to these seven features.

Lavergne (1982) assessed Björk's structural method of growth

prediction using cephalograms taken four years apart, of a group of 42 children (mean age 11.4 years).

Mandibular rotation was assessed by observing changes in the cranial base inclination when the radiographs were superimposed on the natural mandibular reference structures defined by Björk (1969).

Lavergne (1982) concluded that

1. the method could demonstrate mandibular rotation;
2. there was no absolute correlation between structural growth prediction and the degree of mandibular rotation in those cases showing average rotation. There were correlations in the more severe cases of positive or negative mandibular rotation.

Other morphological features have been examined, particularly in the mandible, to ascertain correlations with growth outcomes.

Meredith (1959) examined the correlations between size at age 5 and change during the period from age 5 to 11, and dental arch width at age 4 and dental arch width change during the period 4-8 years. In both studies he concluded that no clinically useful information was obtained.

Maj, Luzi and Lucchese (1963) and Odegaard (1970) found correlations between mandibular growth and gonial angle.

In the former it was found that the greater the gonial angle, the greater the growth in mandibular length. In the latter, more acute gonial angles were associated with vertical and anterior mandibular growth.

Odegaard (1970) further stated that, on the basis of this one significant correlation, mandibular growth was related more to morphological features of the mandible than to features relating the mandible to the skull.

Ridley (1971) claimed (although did not quote the source) that the Y-axis was useful in growth prediction. The mean measurement was stated to be $60^\circ \pm 3^\circ$ and when the Y-axis was greater than this angle the growth was more vertical. The converse was also true, it was claimed. It seems difficult to accept that any valid long term prediction of growth directions for any individual could be made on the basis of a single measurement on a single radiograph and yet this is what people imply when they talk about "patterns". (Moyers and Bookstein, 1979).

McKeown (1975) asserted that craniofacial growth is potentially predictable as it is a process involving logarithmic expansion based upon the original form. McKeown claimed that the direction of growth of a chosen point may be expressed logarithmically as an expansive process taking place in straight lines and thus the direction of growth may be predicted.

This theory deserves comment.

1. It assumes a centre of allometry upon which superimpositions may be made - this centre being the point at which least growth (or no growth) occurs. It is a point which must be defined for predictions to be both possible and meaningful. Moss (1983) claims that the centre may exist, but that its location is not yet possible.

2. It assumes that the rate and direction of growth will remain constant.

3. It cannot predict the growth potential for an individual or for the anatomical features associated with particular landmarks.

Björk and Skieller (1983) studied the direction of condylar growth in studies over 20 years. These were expressed in a linear and logarithmic fashion, the significant features of which were the extreme variability between individuals, and the inconstancy in direction for individuals.

These authors considered that the prediction of condylar growth direction and magnitude was not currently possible, and was unlikely ever to be so.

This was a view shared with Ackerman and Proffitt (1970) who stated

precise prediction of amount and direction of skeletal growth is beyond our present ability.

It appears that currently proclaimed methods of growth prediction or forecasting are not yet of clinical usefulness. Many of the studies here discussed have examined only growth after 12 years of age, by which time the alternatives for interceptive or preventative orthopaedic therapies are severely limited.

Studies of infants have shown a consistent absence of significant correlations between features at this age and future growth.

As will be seen in ensuing chapters, the one reliable "rule-of-thumb" for Class III malocclusions with respect to their growth and development is that they very rarely improve.

CHAPTER 5

CHIN CUP THERAPY

As previously discussed, Class III cases may result from one or all of the following:

1. excessive mandibular development;
2. insufficient maxillary development;
3. both;
4. ideal maxillo/mandibular relationship with a dental Class III anterior and/or posterior relationship.

Emphasis has been placed on the variability of the Class III "syndrome", and the relationship between severity and prognosis, particularly in the last twenty years (Thilander, 1965, West, 1969, Kloeppel, 1972, Cleall, 1974, Campbell, 1983).

Orthopaedic influence on the growing mandible via an external appliance has been practised since the early part of the nineteenth century (Weinberger, 1926) and became widely used until the turn of the century (Weinberger, 1926). At this time Angle (1907) introduced multibanded appliance, and with the more widespread use of intermaxillary elastics. The chin cup apparently became somewhat outmoded (Armstrong, 1961, Graber, 1975).

The appliance has seen a resurgence in recent years however, and a great deal has been published on the efficacy of the chin cup, postulated modes of action (Graber, 1975) particularly in Japan and the United States,

and variability of patient response.

Armstrong (1961, 1963) effectively pioneered the resurgence of clinical testing of the appliance with a study assessing the effect of chin cup therapy alone on 100 children. Since then, there have been many published investigations of the chin cup appliance with varying levels of force, modification and results.

What then are the treatment objectives when deciding to apply chin cup therapy?

5.1 Aims of Treatment

The clinical success and postulated modes of action of the chin cup will be examined in detail in a separate section of this chapter.

At this stage, however, we must consider specifically the treatment objectives (for both the patient and clinician) with this appliance.

The potential psychological effects of the prognathism have been discussed in Chapter 1, but for most people this is only a potential problem in the more severe cases.

In situations where the malrelationship is due to overdevelopment of the mandible, or relative maxillary deficiency, one would hope to exert some control over the growing mandible and thus limit the extent of the disharmony. Whatever the mode of action, the basic requirements are to create a situation where growth of the midface is allowed to achieve its full potential, and growth of the mandible is restricted or redirected to maximise the functional and aesthetic harmony of the face, and minimise future treatment complexity (Irie and Nakamura, 1975).

5.2 Design of the Appliance

The chin cup appliance consists of strapping with or without a headcap or equivalent anchorage (such as circumcranial strapping), the orientation of which depends on the anchorage requirements. Elastics or springs are hooked or tied to the chin cup itself. The cup may be preformed plastic, custom-made acrylic or metal (Graber, 1969) and may include aeration holes to prevent excessive sweating under the cup.

Figs. 5.1 and 5.2 show some examples of the appliance.

The design is varied to suit the requirements of the individual case.

Sassouni (1972) described three sources of anchorage.

1. Cervical anchorage results in a line of force below the condyles and is used where the treatment objectives are a downward and backward rotation of the mandible - the aim being to reduce the protrusion of the chin cup by restricting horizontal growth.

2. Occipital anchorage provides a force through the condyles and is used when restriction of condylar growth is desired.

3. Frontal anchorage is used in the vertical chin cup. The treatment objectives in Class III cases with this appliance is frequently to counter undesirable side effects of rapid maxillary expansion, such as an increased mandibular plane to SN angle. This is discussed in Chapter 7.

Occipital anchorage is the form most frequently employed (Armstrong, 1961, Thilander, 1963, Irie and Nakamura, 1975, Vego, 1976,

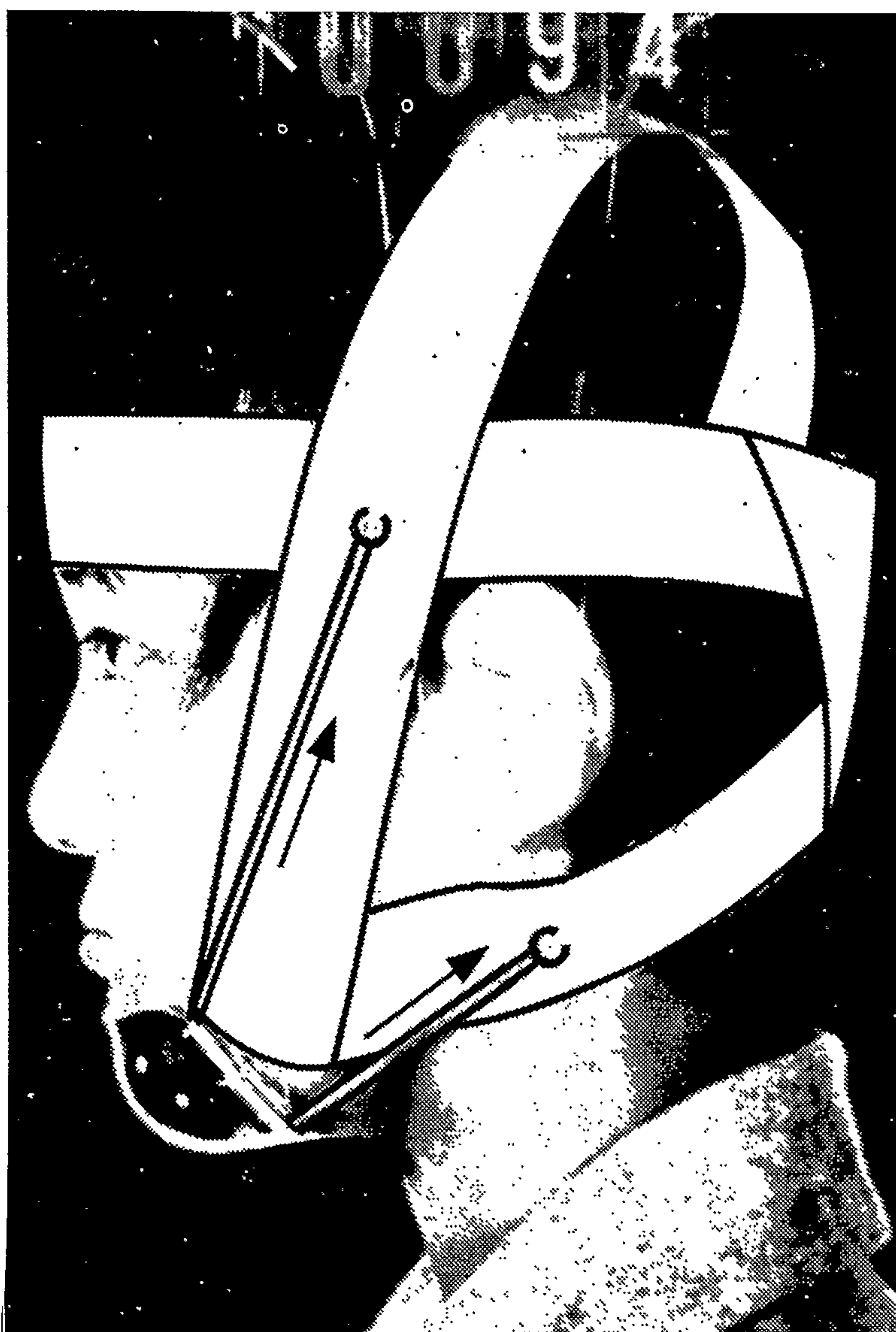


Fig. 5.1 Chin cup appliance (from Irie and Nakamura, 1975).



Fig. 5.2 Chin cup appliance in combination with maxillary protraction (from Cozzani, 1981)

Sakamoto, 1981, Mitani and Sakamoto, 1984) although the appliance design may vary.

Mitani and Sakamoto (1984) used an appliance with two straps from the chin, the first to the occiput with a force slightly anterior to the condyles. The second strap was cervically directed with the force vector approximately parallel to the lower border of the mandible. The resultant force vector, they claimed, passed through the condyles with a small degree of variation.

This was similar to the appliance used by Thilander (1963) although she used elastics attached to the straps.

Sakamoto (1981) directed the force through a point slightly forwards of the ears.

A number of other authors (Armstrong, 1961, Graber, 1963, Cleall, 1974, Campbell, 1983) stated merely that they apply "retrusive force" or a "force against the mandible".

West (1969) described the direction of force as occipital.

There exists little information in the literature relating particular designs to specific forms of Class III malocclusion, and this may be one reason for the variable treatment results seen with this appliance.

5.3 Distribution of Force

There is a paucity of published data on the stress distribution within the mandible and the teeth with this appliance.

Investigations by de Alba, Chaconas and Caputo (1976, 1979, 1982) using models of the craniofacial skeleton made with birefringent plastic, demonstrated the distribution of stress within the mandible using occipital headgear (chin cup).

Direct comparison of their models with the in vivo situation must be guarded. The models are constructed of a homogeneous material, with separate acrylic teeth and a polyurethane plastic "periodontal ligament". The human mandible consists of cancellous and cortical bone with foramina, vessels and tissues of varying densities. The cortical plate varies in thickness and these factors may modify translation of observed laboratory results to the postulated clinical situation. This fact is not mentioned in these authors' publications. The results are, however, interesting and bearing the foregoing in mind, warrant some discussion.

The distribution of stress was primarily restricted to the mandible although some areas of stress concentration were found at the posterior aspect of the glenoid fossa and the apices of the maxillary teeth (increasing posteriorly) (de Alba, Chaconas and Caputo, 1976). The authors pointed out that the lack of an articular disc may have modified the observed stress patterns in the glenoid fossa.

Overall, within the mandible, stresses were seen to follow the "trabeculae of the anatomic configuration" with concentrations in the areas of greatest bulk.

The results were summarised as follows:

- a. Stresses were noted in the area of the pterygoid plates at the sphenoid bone at the insertion of the simulated external pterygoid muscle.

- b. Because of direct contact of the condyle against the posterior surface of the glenoid fossa, forces were seen to be transmitted to this anatomic area.
- c. Stress trajectories followed the trabecular pattern of the anatomic configuration of the mandible.
- d. Beginning at the apices of the incisor teeth, the stresses emanated through the body, the angle and retromolar triangle of the mandible, radiating in a posterosuperior fashion, concentrating at the neck of the condyle.
- e. Selected sectioning of the mandibular model showed evidence of stress concentration at the lingual aspect of the external pterygoid muscle insertion.

Although these authors demonstrated the compressive effect on the condyle and an overall "bending" effect on the mandible, their observation of the effect on the simulated lateral pterygoid muscle are confusing. No explanation is offered about either the reasons for this effect, or its significance.

Interpretation of the results must be qualified by the fact that a static model does not relate to the dynamic system of the human condition. Variations in appliance effect that will result from a growth and treatment induced changes cannot be assessed by this method.

5.4 Clinical Supervision and Management

5.4.1 Magnitude of Force

Assessment of the magnitude of force required to "inhibit" mandibular (condylar) growth was, in the first half of this century, derived primarily from studies on growth inhibition in the epiphyseal plate of long bones (Armstrong, 1961).

Investigations by Haas (1948), Blount and Clark (1949) and Gelbke (1951) demonstrated that very high forces were needed.

Blount and Clark (1949) needed 6 staples $\frac{3}{32}$ " diameter to inhibit

epiphyseal growth in children requiring equalisation of limb size due to polio. Gelbke (1951) estimated, on the basis of research with growing dogs, that a force equivalent to one half the body weight was required to inhibit epiphyseal growth in the femur.

More recently it has been shown that there are considerable differences between the condyle and epiphyseal plate both histologically and in their response to force (see Chapter 3).

Selection of an appropriate force with this appliance, on the basis of published results is hampered by several factors. The first is the polymorphic nature of the Class III malocclusion, and thus the difficulty of relating clinical results with specific pretreatment features present in particular patients. A second is assessing the significance of the parameters or features by which authors assess the success or failure of the treatment. For example in mild discrepancy cases or patients with a more horizontal mandibular growth component (low gonial angle) simple correction of the anterior crossbite on completion of that phase of therapy may be assessed as successful. If this case then relapses during the course of further growth, was the treatment worthwhile? Should the level of applied force have been increased? In more severe cases where the end result is merely a lessening of the severity of the skeletal imbalance we could ask again does this justify treatment and appliance design?

These questions are inadequately answered in the literature and a survey revealed that few authors provided any basis for their selection of a particular force level. Several authors (Graber, 1970, Campbell, 1983, Mitani and Sakamoto, 1984) stated that they felt the results obtained by Thilander (1965) were inadequate due to the light forces (150-200 g) she applied.

Application of 500-600 g per side (total of 1000-1200 g on the chin) has been advocated by the majority of clinicians (Armstrong, 1961, Graber, 1970, Cleall, 1974, Sakamoto, 1981, Campbell, 1983, Mitani and Sakamoto, 1984). Graber (1975) mentions that "3-4 lbs per side" is required to inhibit mandibular growth. Thilander (1965) used lighter forces 150-200 g per side whilst Vego (1976) used slightly higher forces of 150-300 g per side. Kloeppel (1970) did not mention force at all when discussing the use of chin cup in the deciduous and mixed dentition.

There are other force-related factors affecting the treatment outcome that are scantily discussed in the literature.

High force levels (such as the 1300-2200 g per side used by Haas, 1970) are likely to cause problems of patient cooperation due to discomfort and the difficulty of placing the appliance. The effects on the skin over the chin area, and on the developing calvarial bones are further considerations.

5.4.2 Hours of wearing per day

Although there are a number of studies providing at least some scientific basis for the selection of force magnitude, there is very little (if any) similar basis for the selection of the duration (per day) of application of these forces.

The majority of clinical studies involve the patient wearing the appliance for at least 12 hours per day (Armstrong, 1961, Thilander, 1963, Sakamoto, 1981, Campbell, 1983, Mitani and Sakamoto, 1984). Several authors (Cleall, 1974, Graber, 1975) mention 12-16 hours per day whilst Mitani and

Sakamoto (1984), in the prepuberal growth spurt require patients to wear the appliance 24 hours per day, excepting meal times. Between periods of accelerating maxillary growth, and in combination with a maxillary protraction appliance the wearing was for as little as 9.2 hours per day.

Petrovic, Stutzman and Oudet (1975) stated that, with rats, more bodily growth occurred during periods of sleep than consciousness, and this apparently justifies night-time wear as advocated by most authors. Patient co-operation is probably a greater indicator for night-time wear than the claimed higher rate of growth nocturnally.

On the basis of published results, it appears that 12 hours per day is the minimum period of wear with this figure being increased if possible to a period commensurate with maximal patient co-operation. It is obvious from the literature that there is a requirement for substantial experimental evidence to more accurately and scientifically assess the effect of intermittent wear, or wearing for a period less than that stated above. I feel ethical problems and factors related to patient co-operation may limit the amount and accuracy of human studies. Comparison of controlled animal laboratory studies with human studies is limited by a number of factors. Control of appliance force, duration and intensity as well as "co-operation" is variable in the clinical situation (Graber, 1975) unlike the laboratory situation. In addition, the majority of animal studies convert an essentially normal occlusion into a malocclusion of varying degree (ibid.). Graber goes on to say that alteration of neuromuscular activity associated with an abnormal skeletal pattern may be more difficult than with a normal skeletal pattern.

5.4.3 Duration of treatment

Thilander (1963) stated that "it was not reasonable to expect a child to wear a chin cup throughout its growth period". This view is shared by a number of other authors (Kloepel, 1970, Sassouni, 1972) with some qualifications. Kloepel felt that orthopaedic therapy was "justified only in the deciduous dentition when it will overcome the problems of treating inferior prognathism as an entity or if only by an early start can the profile be changed". Sassouni (1972) felt that it was important to evaluate the biologic, mental, social and psychological needs of the patient before deciding on an orthopaedic treatment plan. Graber T. (1975) felt that 6 year treatments were not unreasonable and Graber L. (1974) recommended at least 3 years. Pascual (1975), Sakomoto (1981) and Mitani and Sakomoto (1984) used the appliance for approximately 2 years in their studies, although Sakomoto (1981) observed that even after this time in some cases the skeletal disharmony remained.

Strang and Thompson (1958) recommended chin cup therapy in the deciduous dentition and felt, as did Salzmann (1966) that it should be commenced as soon as the disharmony is diagnosed.

Creekmore (1978) stated that "in an excessively growing mandible, chin cup and protraction therapy would have to be continued over the entire growth period". Graber (1975) and Mitani and Sakomoto (1984) agreed and stated further that for redirection of growth short treatments (several years) were required and for growth inhibition long treatments were necessary - a view shared by Irie and Nakamura (1975).

Cleall (1974) related the long total period of treatment, as one of a number of factors, to inconsistencies with results, although Graber (1975) felt

that inadequate forces were the most significant factor.

Vego (1976) used chin cups on patients until the first permanent molar or second deciduous molar were in a flush terminal plane relationship, with a treatment duration of 2-9 months, but admitted that there is "no question but that these patients will require more chin cup therapy as pubescence is approached".

As mentioned previously, it appears that the requirements of the treatment will largely dictate the treatment time. Haas (1970) using vertical pull chin cups to offset the bite opening effects of RME and Class III inter-maxillary elastics found treatment times of 26 months with 9 months retention adequate - the effect of the chin cup being an alteration of growth direction. Pascual (1975) used the appliance 16 hours per day for two years - the objectives being alteration of mandibular morphology and, combined with exercises, modification of tongue activity.

Grossman (1970) continued until correction of the anterior crossbite was achieved, although he did not mention the time period required.

Thilander (1963) used the appliance for 1-4 years and felt that, on the basis of her results, routine use was unwarranted. This view was shared by Armstrong (1963) who, using the appliance for 18 months, did not find the observed changes justified widespread use.

Campbell (1983) selected patients for study on the basis of an anterior crossbite on edge-to-edge relationship, and found, with an age range of 7.1-11.7 years that the treatment time varied from 7 months to almost 2 years. Success was judged on correction of the incisal relationship to Class I

The appliance used was a reverse pull face crib and the only consistent finding was lingual tipping of the lower incisors.

Mitani and Sakomoto (1984) also used crossbite correction as an indicator of successful treatment and discontinued the appliance when this was achieved. In contrast to the previous study, the treatment times varied from several years to over six years. Recently Graber (1983) has advocated the use of chin cups for mandibular retraction in the 18 month to 48 month old infants. This is based on his assumption that there exists more potential for change in the rapidly growing organism.

Can the clinical recommendations of the foregoing, both positive and negative, be justified at a biological level? An examination of the changes observed in mandibular development in response to the application of force may provide an answer.

5.5 Mandibular Response to Force

The ability of growing bones to be morphologically modified by force or containment is well documented and has been practised for centuries in different cultures. Monson and Felts (1961) demonstrated permanent deformation of rat humeri by compression. The permanent changes in foot shape of some Chinese children and the cranial deformities induced in Columbian Indian societies, both the result of binding with boards, has been documented (Perez-Martinez, 1960). The direction of growth is altered, rather than the amount or rate.

On the basis of these observations, Sassouni (1972) stated

mechanical force can modify the direction of growth of bones ... and their shape and spatial position can be altered.

What basis is there to assume that permanent changes (favourable to Class III correction) can be induced in the growing mandible?

Graber (1975) stated that "whilst there is a wealth of material and research data available on mandibular hyperpropulsion and its ability to 'stimulate' mandibular growth, work on condylar growth restriction and redirection has been minimal".

Graber ascribes this to the low incidence of Class III in the population. The results of studies about mandibular growth have, however, been variable. Two sources have provided most of the information relating to force induced growth and morphological changes as well as the extent and permanence of these morphological changes observed as a result of application of force.

The sources are:

1. Animal studies;
2. Clinical human studies,

and these will be discussed, with reference to their limitations and the clinical significance of the results.

5.6 Animal Studies

5.6.1 Histological changes

The majority of reports have dealt with pressure-induced changes in the tissues comprising the head of the condyle, and those comprising the glenoid fossa. Observed changes in the prechondroblastic zone have been related to postulated effects on osteogenesis in the condyle, and macroscopic morphological changes in the mandible as a whole.

Charlier, Petrovic and Herman-Stutzman (1968) demonstrated a reduction in the width of the prechondroblastic zone in young rat condyles, as the result of retrusive force via a chin cup. The associated decrease in numbers of chondroblasts resulted in less degenerating chondroblasts and hence less endochondral bone formation. They pointed out that whilst prechondroblasts were sensitive to pressure, chondroblasts were not, thus, inferring that endochondral bone formation will continue, but at a reduced rate.

Petrovic, Stutzman and Oudet (1975) reported similar changes in rats and noted that there was an overall decrease in cellularity of the condylar head related to the experimental period.

Belhobeek (1975) also found similar changes in guinea pigs. In these experiments, the appliance design utilised the force provided by the growing mandible - no initial force being applied. This author found that when the force level reached 700 g, growth was inhibited.

No mention was made in these reports as to the occurrence of bone resorption in the area.

Joho (1973) did, however, report extensive bone resorption in experiments with Macaca mulatta. He observed that the distal aspect of the head of the condyles, roof of the glenoid fossa and the anterior portion of the post-glenoid surface all exhibited bone resorption. Resorptive changes of this nature were also observed by Breitner (1940) and Adams (1969) in rhesus monkey condyles.

In these experiments, Joho (1973) produced a Class III malocclusion from a Class I occlusion and noted also an initial upwards and backwards

movement of the maxilla, associated with the anterior positioning of the mandible.

Histologic changes in craniofacial structures with retrusive forces have been reported further by Janzen and Blucher (1965), Bare (1972) and Kulis (1972).

These changes have been interpreted by several authors (Graber, 1975, Mitani and Sakomoto, 1984) as retardation of condylar growth and related to changes observed in angular and linear measurements of the mandible.

5.6.2 Morphological changes

Petrovic, Stutzman and Oudet (1975) found that within 4 weeks changes in morphology of the mandible of rats had occurred. A decrease in the mandibular angle (corpus/ramus) of 2-3° was observed along with a slight decrease in the condyle to mental foramen distance.

Changes in the gonial angle have also been reported (Janzen and Blucher, 1965, Matsui, 1965, and Joho, 1973) in animal experiments.

In addition consistent findings of a lower rate of increase in mandibular length have been reported (Graber, 1975).

Although experimental animal studies examining the effect of orthopaedic force against the TMJ have demonstrated relatively consistent results (Mitani and Sakomoto, 1984) three factors must be considered when relating this to potential clinical situations. Firstly these animal studies generally involve converting a normal, skeletal relationship into an abnormal one and orthopaedic therapy aims to convert an abnormal relationship into a normal

one (Graber, 1983). The second is the fact that results with human investigations have been inconsistent (Mitani and Sakomoto, 1984). The reason for this inconsistency may be simply due to the difficulty in controlling variables (particularly duration of wear) in the way that they are controlled in animal experiments. Conversely it may be due to a different response of the human craniofacial complex to force than that observed in animals.

Histologic studies in humans are, for obvious reasons, extremely rare and thus assessment of the efficacy of the appliance must be made empirically on the basis of observed morphological changes and the assumption that, in these cases, variables have been maximally controlled.

The third factor is that specific differences exist in the TMJ anatomy between humans and animals. McNamara, Connelly and McBride (1974) noted in rhesus monkeys a smaller articular eminence, shallower glenoid fossa and larger postglenoid spine than exists in Man. McNamara et al. pointed out that consistent results have been observed in widely differing species, such as the rat and monkey, and state that on that basis it is not unreasonable to assume similar responses in Man.

5.7 Human Studies

The most consistent finding in clinical studies is an alteration in the incisor relationships with most authors reporting a retroclination of the lower incisors and proclination of the upper incisors (Armstrong, 1963, Thilander, 1965, Kloepel, 1970, Vego, 1976).

Cleall (1974) also observed retroclination of lower incisors and although he achieved correction of the dental relationship to Class I, was

uncertain whether this was due to forwards movement of the maxillary dentition, forward movement of the maxilla or growth inhibition in the mandible. Irie and Nakamura (1975) noted changes in incisor positions with a decrease in lower incisor to mandibular plane angle and an increase in upper incisor to SN angle.

It seems difficult to assess how this change in incisor relations occurs. Armstrong (1961) feels that it may be due to pressure on the lower incisors from the cup, transmitted via the lower lip. This view is shared by Thilander (1965) and Cleall (1974) although Thilander felt that retrusion of the alveolar process occurred. Graber (1977), in contrast, found lingual tipping of lower incisors and labial tipping of upper incisors in an untreated control group. The experimental group, treated with a chin cup showed a relative labial inclination of the lower incisors and thus concluded that orthopaedic correction of the crossbite occurred.

To date this has not been demonstrated elsewhere with chin cup studies. Logan (1968), however, did observe proclination of both mandibular and maxillary incisors in patients treated for scoliosis with the Milwaukee brace which he felt was due to intrusive effects on the posterior teeth and overclosure.

Armstrong (1963) noted that, even though lingual tipping of the lower incisors was observed in successfully treated cases, no such change was observed in cases that did not respond favourably to treatment. He postulated that changes in inclination of these teeth are secondary to some other effect.

Grossman (1970) postulated that retroclination of lower incisors was due to pressure from the chin cup and that whilst this effect was separate from observed orthopaedic changes, it was additive with respect to overall correction of the skeletal or dental disharmony.

Decreases in gonial angle (Suzuki, 1972, Graber, 1975, Irie and Nakamura, 1975, Sakomoto, 1979) and increases in the mandibular plane to SN angle (Graber, Chung and Aoba, 1968, Graber, 1975, Sakomoto, 1979, Nanda, 1980) have been regularly reported although Irie and Nakamura (1975) state that these changes do not always occur - particularly increased SN mandibular plane. They studied changes occurring with chip cup therapy in 10 boys and 19 girls with Class III anterior crossbites in the mixed dentition. On the basis of the results (post retention SN to mandibular plane) they divided their sample into 3 groups according to the pre and post treatment differences in the SN mandibular plane angle:

Group A comprised 59% of the sample and was considered a functional Class III. The SN mandibular plane angle did not change and they stated that crossbite correction occurred by backwards translation of the mandible, lingual tipping of mandibular incisors and "autonomous" labial maxillary incisor adjustment.

Group B (34%) demonstrated an increase in SN mandibular plane angle of 1.5° or more as well as similar incisor changes. In these cases correction occurred by "rocking open" of the mandible.

Group C (7%) was characterised by an initial down and backwards rotation, incisor changes as per group A and B, followed by an upwards and forwards mandibular rotation and a return to the pre-treatment incisor relationship. In this group the SN mandibular plane angle decreased overall.

These results, and others based on changes in angular measurements

must be considered in the light of work by Björk and Skieller (1983) on mandibular rotation occurring with normal growth - particularly with regard to small changes. Bjork and Skieller (1983) described total mandibular rotation as the result of the rotations of two independent rotary systems - the matrix rotation and intramatrix rotation. The matrix rotation is assessed by changes in a tangential mandibular line relative to SN, based on superimposition of implants. The centre of rotation is located at the condyles.

Intramatrix rotation is described as rotation of the mandibular corpus within the soft tissue matrix and is assessed by changes "in the inclination of an implant or reference line relative to the tangential mandibular line."

Björk goes on to say that there is usually considerable intramatrix rotation and moderate matrix rotation. The resultant of the two is the total mandibular growth rotation. To confuse the issue further, the matrix rotation may be in an opposite direction to the overall rotation.

The significance of this appears to be that matrix rotation during growth may affect the SN mandibular plane angle, whilst intramatrix rotation may affect the gonial angle - two measurements frequently used for assessing changes due to orthopaedic appliances on the mandible (Irie and Nakamura, 1975, Graber, 1977).

In addition to this, the reliability of landmarks used in the measurements has been questioned (Baumrind and Frantz, 1971) and whilst cephalometric analysis is satisfactory for assessing gross relationships, small angular or linear changes may be within or equal to the standard deviation and thus suspect.

It seems that there is little dispute that the shape and direction of bone growth can be altered. This has been clearly demonstrated in animal experiments. There is less certainty about the ability to inhibit mandibular growth and clinical studies have been inconclusive (Mitani and Sakomoto, 1984).

The wide variation found in clinical studies appears to result from a number of factors:

1. The difficulty in diagnosing the type of Class III in the deciduous or early mixed dentition and thus designing an appliance to control the specific growth patterns existing in each individual patient.
2. The frequent requirement for long treatments and the associated variations in patient co-operation.

Orthopaedic effects of the chin cup appliance in the maxilla and skull are rarely considered in the literature. It seems improbable that rotational effects on the mandible with these appliances does not have any effect on the maxilla and maxillary abutments. Forces must be transmitted across the occlusion, and thus at least some maxillary effect would be anticipated.

De Alba, Chaconas and Caputo (1976) observed stress lines radiating from the maxillary teeth - centred in the bicuspid area - in their birefringent plastic models. These authors did not, however, examine wider effects.

Occlusal contact may be significant with regard to maxillary effects. A tight interdigitating occlusion may actually act to restrict anterior maxillary development if retrusive forces on the mandible are effective.

Further, retroclination of incisors, together with any mandibular "bending" may well change tongue space with consequent effects on the maxilla and maxillary teeth.

Whilst the chin cup appears to be enjoying widespread usage - particularly in the United States and Japan - the evidence to support its effectiveness in humans is without a sound scientific base. In addition, the absence of any relationships between specific Class III features and appliance designs limits the clinician's ability to make accurate prognostic assessments for chin cup therapy.

CHAPTER SIX

MODIFICATION OF THE NASOMAXILLARY COMPLEX

The role of inadequate maxillary development in the aetiology of Class III malocclusions has been reviewed in Chapter 2, and it has been claimed (Bell, Proffitt and White, 1980) that this may be associated with 30-40% of Class III cases.

Consequently, anterior movement and expansion of the nasomaxillary complex and dentition is a desirable objective for many Class III treatments. A number of techniques have been published designed to achieve this end, although compared with that available for maxillary retrusion, the data relating to non surgical maxillary advancement is sparse.

Maxillary protraction has been advocated in Class III treatment by a number of authors, for a variety of reasons. It is essentially forwards movement of the maxilla or maxillary dental arch by anteriorly directed forces, usually applied with elastics to an extra oral crib. Specific designs are discussed in section 6.3.

The terms "deficiency", "hypoplasia" and "inadequate development" have been used, often synonymously, to describe maxillary involvement in Class III malocclusions.

In the context of this treatise, the terms used for maxillary size will be in accordance with Haas (1965) although the use of these terms will be qualified.

Haas (1965) asserted that Class III malocclusions may be associated with relative or real maxillary deficiency.

Relative maxillary deficiency exists when the maxilla is of expected size compared with the upper face and the cranium, but the mandible is too large when compared with these structures.

Real maxillary deficiency exists when the maxilla is of less than expected size when compared with these structures. The deficiency may be due to maxillary atresia or retrusion, in addition to lateral and vertical deficiencies (as discussed in Chapter 2) and variations in mandibular size may co-exist.

As mentioned, these terms must be qualified on several points.

1. If assessment of "expected" size is made visually, then subjective judgments may result in a wide variation of diagnosis;
2. If the assessment is determined cephalometrically, the "norms" used for a comparison should be standardised on a racial and population basis;
3. Positional variations of the mandible due to occlusal interferences may hamper accurate assessment of mandibular size by both clinical and cephalometric means.

6.1 Indications

Teuscher (1977) claimed that anteriorly directed force on the maxilla could "influence the course of events by accelerating maxillary translation and vertical development of the entire upper and lower anterior dentition". He goes on to qualify this statement by adding, "This means acceleration of normal growth components".

It has been conjectured (Chapter 2) that, in many Class III's, growth in the nasomaxillary complex may not be "normal" and this should be considered as a partial explanation for the variable responses reported with this form of therapy (Nanda, 1978, Campbell, 1983).

Nanda (1980) stated that maxillary protraction was only a part of a total treatment regime for Class III's. He suggested that cases most suitable are those where:

1. the maxilla is deficient and mandible normal;
2. less than 2 mm of negative overjet exists in centric relation;
3. the patient has not yet started the pubertal growth spurt.

Cozzani (1981) advocated early treatment with this technique (as young as four years of age) to take advantage of growth. The evidence relating to this statement is discussed in section 6.4. Further, it may be considered as an alternative in cases which, for reasons of profile preservation, are not amenable to extraction therapy. It may also, he claims, be used to avoid problems associated with conventional Class III orthodontic therapy viz. the extrusion of upper molars and lower incisors associated with Class III elastics and the resultant increase in vertical dimension and aggravation of an already compensated (by overeruption) position of the lower incisors.

Campbell (1983) selected patients for his study on the basis of an anterior crossbite, or at least an edge-to-edge relationship of the incisors. No assessment of incisor angulation was made, nor was there any mention of the presence of maxillary retrognathism. Had these been included, the results of his study would have had more significance with regard to the clinical application of these appliances.

6.2 Contraindications

There are few specific features mentioned in the literature as contraindications to maxillary protraction. This is probably due to the relatively limited usage of this form of therapy and thus the small amount of published data available.

Patient acceptance and operator scepticism are likely factors here and Nanda (1978) points to the difficulty in predicting the direction of maxillary movement (and associated maxillary and mandibular rotations) as a potential problem with clinical application.

This supported a warning by Teuscher (1977) who discussed the possibility that the maxilla and the maxillary dentition may react differently to one force vector, thus making the outcome of treatment difficult to predict.

Further, Teuscher (1977) claimed that the anterior rotation of the maxilla (counterclockwise) was contraindicated in cases of limited overbite as an open bite may result.

6.3 Appliance Design and Clinical Management

6.3.1 Animal studies

Experimental studies have formed the basis for the majority of information available on the tissue effects associated with, and the postulated mechanisms resulting in, anterior movement of the maxilla with protraction appliances.

In most of these studies cast metal splints have been bonded or ligated to the maxillary dentition of *Macaca* species.

Dillinger (1973) used elastics to a face crib and applied a force of 6 lbs. over 7 days.

Kambara (1977) constructed cast splints for the maxillary and mandibular dentition of Macaca irius and applied an elastic force on the maxillary splint from a head cap with an anterior bow. The force level applied was 300 g per side, 15 hours per day over a period of 90 days.

Nanda (1978) (with M. mulatta) applied a force of 500 g to a maxillary arch cast splint via a stainless steel spring attached to the splint in the midline. The extra-oral component consisted of a halo head frame secured to the skull by stainless steel screws and the appliance activation period ranged from 81 to 95 days.

Jackson, Kokich and Shapiro (1979), using a similar appliance to Nanda (1978) applied a force of 300 g per side in a direction parallel to the occlusal plane. The force application continued until there was an 11 mm overjet (achieved after 3-4 months of active treatment).

6.3.2 Human clinical studies

Nanda (1978) stated that there were few clinical studies examining methods of anterior displacement of retruded maxillas by extra-oral means, and that detailed analysis of the results of these studies were lacking. Since that time there appears to have been an increase in usage of this form of therapy.

Marx (1961) and Kiyomura, Niva and Hikino (1969) used an appliance (Fig. 6.1) employing occipito-mental anchorage to apply an anterior elastic force to gain forward maxillary movement during the late mixed dentition period.

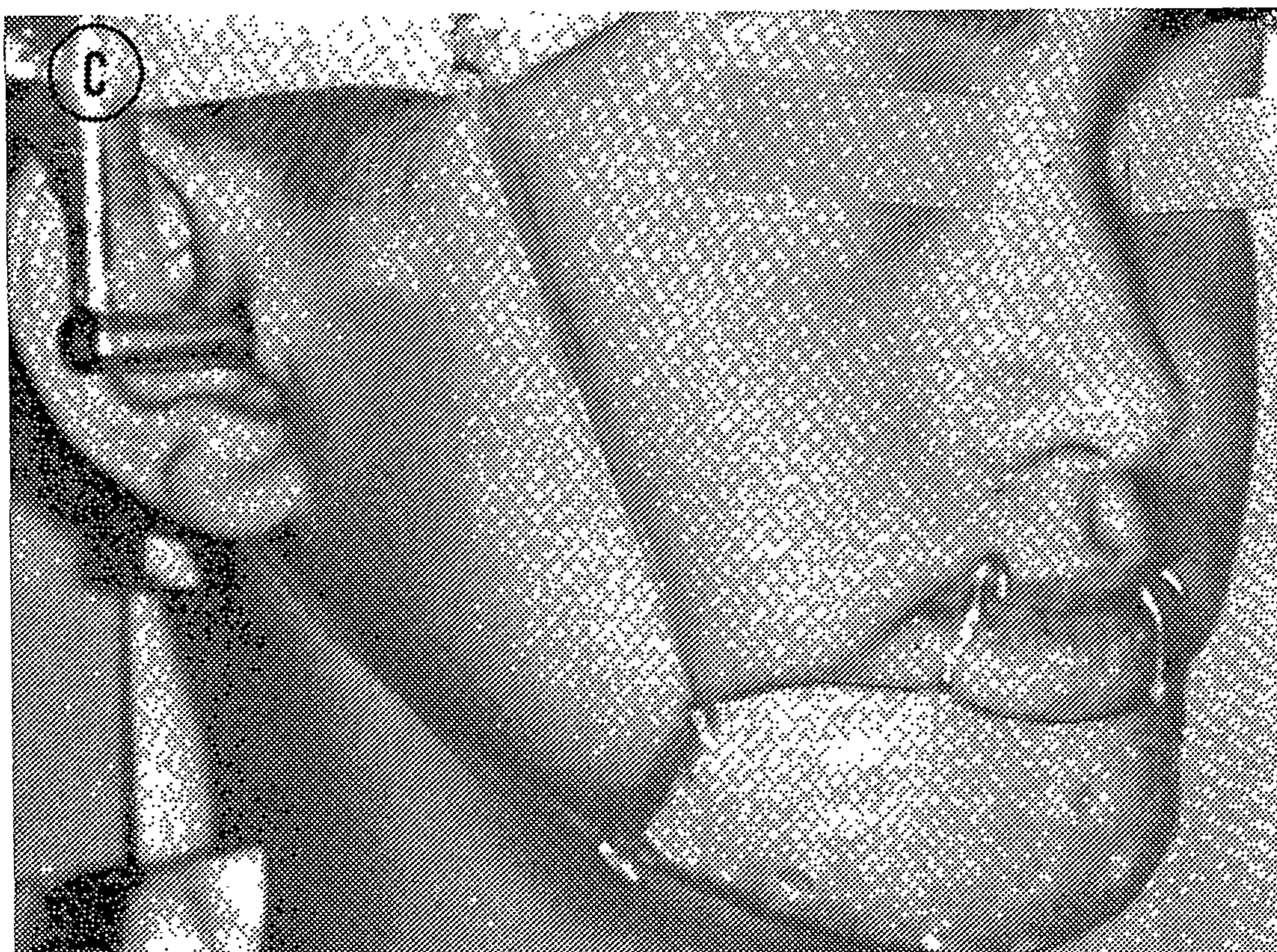


Fig. 6.1 Maxillary protraction appliance employing occipito-mental anchorage from Cozzani (1981).

Nelson (1968) used a football helmet type appliance with anchorage based on the forehead and sub-occipital region to move the maxillary dental arch forward.

Dillinger (1973) used a Hickam chin cup (similar to Fig. 6.1) in conjunction with rapid maxillary expansion on an eleven year old boy and an

eight year old girl. He applied a force of 6 lbs. for 14 days and this is the only instance I can locate of a force of this magnitude being used on humans for maxillary protraction. It is likely that patient compliance may be a problem with this approach. No other clinical details were discussed in this paper.

Irie and Nakamura (1975) used frontomental anchorage to support an appliance (Fig. 6.2) applying a 400 g anterior force to the maxilla. The point of application was the maxillary molars, which were supported by a palatal bar soldered to molar bands. In addition, a force of 800 g was applied between the chin cup and the occipital region of the skull. The patient's age at the beginning of the 10 months treatment was 10 years 6 months.



Fig. 6.2 Maxillary protraction appliance employing fronto-mental anchorage from Irie and Nakamura (1975).

Delaire, Verdon and Floor (1976, 1978) described an appliance that they claimed was used widely in Europe. The appliance (Fig. 6.3) consisted of a strap across the forehead, twin heavy gauge wires to a chin cup with a heavy metal bar supporting hooks to which were attached the elastics. Forces of 1000-2000 g were applied to the distal of the maxillary molars reinforced by a fully banded maxillary arch. Treatment times were generally 7-12 months.



Fig. 6.3 Delaire "face-mask" using fronto-mental anchorage.
(from Delaire, 1972)

Nanda (1980) claimed that the majority of the commonly used appliances (presumably in the U.S.A.) involved "some form of chin cup with a strap or support around the head and neck".

Elastics on hooks on the chin cup applied an anterior force to the upper molars which were supported by a fully banded arch with a heavy arch-wire. Nanda (1980) stated that this system caused extrusion and tipping of the first molars and suggested a modification using a headgear bow.

The intra-oral arms engaged the distal of the molar tubes and the elastics were applied to the arms of the outer bow. He claimed that the outer bow and the direction of pull could be designed:

according to the desired movement of the maxilla in relationship to the centre of resistance of the maxillary dentition, which was estimated to be in the apical area of the maxillary premolars.

With this appliance he applied a force of 500-600 g for an average of 17 hours per day. The treatment duration was related to patient co-operation such that for 20-22 hours wear per day, the treatment period was 4 months and for 15-18 hours per day, 7-8 months.

It should be pointed out that the maxillary protraction was only one phase of the overall treatment and was followed by a chin cup appliance worn until the full banding phase was completed - frequently a period of over 4 years total treatment time.

Cozzani (1981) presented cases treated with a Delaire type face mask, with the maxillary arch banded and additional support supplied by a heavy lingual wire ligated interdentally to the labial wire. He used a force of 500-1000 g per side for 3-4 months which was applied to hooks on the arch wire adjacent to the upper central incisors. He advocated starting very early

- as young as 4 years of age - to reduce the maxillary retrusion in the deciduous dentition, followed by a second phase of protraction just prior to exfoliation of the second deciduous molars.

In addition, Cozzani (1981) used an upper removable appliance with hooks adjacent to the lateral incisors which, provided retention of the appliance was adequate, was suitable for use in the deciduous dentition or the mixed dentition when exfoliated teeth would reduce the stability required by fixed appliances.

Campbell (1983) demonstrated the use of a reverse pull face crib and chin cup (similar to that described by Irie and Nakamura (1975) and, using $\frac{1}{4}$ " elastics, applied a force of 14-16 ozs per side.

The ages of the patients in this study ranged from 7 to 11 years and active protraction ceased when crossbite correction was achieved - a period of from 3.5 to 9 months.

This review of appliance design and management demonstrates several areas that are not satisfactorily stated by most authors:

1. the direction of the applied force;
2. factors to be considered in selection of this treatment mode.

6.4 Maxillary Response to Anteriorly Directed Force

Most experimental work in this area has been carried out on Macaca species, and the results of these studies have indicated that the direction of force is of paramount importance in determining the maxillary response to treatment (Nanda and Hickory, 1984).

A review of animal and clinical studies will be undertaken to form a basis for determining:

1. whether anterior maxillary movement is possible, and if so, stable;
2. how the resultant maxillary movement may be predicted;
3. the age range during which maxillary protraction may most readily be achieved.

6.4.1 Animal studies

These investigations have in the main been of two types, viz. assessment of skeletal changes determined by implants, and histological examination of sutural changes.

Dellinger (1973) reviewed the literature concerned with modification of maxillary sutures and found that, although there existed a wealth of information regarding expansion of the midpalatal suture, there was nothing concerning anterior movement of the maxilla, and the effects on maxillary sutures.

Dellinger (1973) applied a force of 6 lbs for 7 days to two young *Macaca speciosa* and analysed pre- and post-experimental lateral cephalograms by superimposing on implants placed at the occiput and above the supra-orbital ridges. He found that point A and a point on the labial surface of the incisors moved forwards 2.0 mm and 2.8 mm, measured parallel to the palatal plane. This, he claimed, indicated separation of the maxilla at the pterygomaxillary fissure although he admitted that visualisation of the fissure on a radiograph was difficult.

Although this study did not attempt to prove the specific location of the changes observed, it did indicate that under these experimental conditions, anterior movement of the chosen landmarks occurred. This could not be accounted for solely on the basis of growth or orthodontic movement as the treatment duration of 7 days is too short for either of these factors to play a significant role.

Kambara (1977) refined the experimental technique and, using tetracycline bone markers, examined skeletal changes during maxillary protraction by assessing bone growth in the circum-maxillary sutures. In addition, he used implants to aid cephalometric analysis of the changes.

In this study, a force of 300 g per side was applied, 15 hours per day, for a period of 90 days with the direction of force parallel to the occlusal plane.

Serial cephalograms were superimposed on the cribriform plate, clivus and sella turcica and changes assessed by measuring movement of four implants. These were placed at the left side of point A, the zygomatic process of the frontal bone, the middle region of the maxilla and near the apex of the mesial root of the first molar.

The results indicated that anterior movement of the maxilla and maxillary dentition occurred with a counter clockwise rotation of the maxilla - presumably about a centre of resistance. This centre was not identified or discussed. The observed changes were much greater in those monkeys in the mixed dentition, than in adults.

All the sutures examined (zygomatico-maxillary, transverse palatine, zygomatico-temporal, zygomatico-frontal, frontomaxillary, sphenozygomatic and pterygopalatine) exhibited changes in structure. The greatest separation (and subsequent osteoblastic activity) was seen in the zygomaticotemporal and transverse palatine sutures.

The author claimed that, on the basis of his results, "The sutures in the craniofacial skeleton were remodelled as a secondary growth centre" (Kambara, 1977).

Kambara (1977) then concluded that

1. anterior movement of the maxilla was possible (in monkeys);
2. stretching of the sutural connective tissue stimulated bone formation and thus sutural growth; and
3. the response was greater in young monkeys.

No assessment was made in this study of the resultant maxillary movement or sutural reactions to varying force directions and point of application. This would have had valuable clinical application with regard to prediction of maxillary movement - both in amount and direction.

The study did, however, illustrate the adaptive ability of facial sutures (Koski, 1968) and thus formed a basis for examining human responses to anterior force on the maxilla.

Nanda (1978) using *Macaca mulatta* assessed specifically variations in maxillary movement and suture response resulting from alterations in the line of force with a standard point of application.

The histological analysis of the sutures was in agreement with the results of Kambara (1977).

Spatial changes in the maxilla were related to the location of the centre of rotation of the maxilla. This was determined by joining the images of the implants on the pre- and post-treatment cephalograms and drawing a line perpendicular to these implant lines. The point of intersection was claimed to be the centre of rotation of the maxilla (COR).

It was unclear in this study whether the centre of rotation was

synonymous with the centre of resistance, and if not, what the relationship was between the two.

The point of application was varied by movement of the spring up or down the extra-oral bar of the splint. No attempt was made to explain how varying the point of application in this way would relate to the use of different points intra-orally.

Nanda (1978) concluded that the location of the COR varied with the point of application and the line of force (relative to the original occlusal plane) such that:

1. with a high line of force to occlusal plane angle, the COR was low and within the maxilla, sutures above this point were compressed and maxillary movement was predominantly vertical;
2. as the line of force became lower the COR moved superiorly and translation of the maxilla became more horizontal;
3. within each suture, different moments of force were generated due to sutural morphology. This changed the degree of bony displacement in a way that was difficult to predict.

Further, this study showed that the effects were not limited to the maxilla but that all the articulating bones (of the maxilla) exhibited some changes in orientation. Effects beyond these bones were not discussed or assessed.

Nanda (1978) concluded that, whilst anterior movement of the maxilla was possible, prediction of the resultant direction and amount of movement required an understanding of the location of the centre of rotation, centre of resistance and the sutural morphology of the maxillary

articulation. Thus, direct clinical application of the technique may be difficult.

Nanda and Hickory (1984) examined specifically changes in the zygomaxillary suture resulting from anteriorly directed forces on the maxilla. The results were in agreement with the study by Kambara (1977) and Nanda (1978) and showed further, that due to the tortuous path of the suture margin, areas of compression and tension were present throughout the suture. These areas formed points of resistance and their location varied with the direction of force and point of application on the maxillary denture, and was related to the COR.

A significant finding in this study was that new bone formed in the sutures was stable when examined six months after termination of force. This supported a study by Jackson, Kokich and Shapiro (1979) which examined the post-treatment response in monkeys to maxillary protraction.

At the end of active therapy, these authors found that the sutural response was proportional to its distance from and orientation to the force applied. The closest sutures, or those whose orientation to the direction of force allowed greater antero-posterior movement showed the greatest response.

This study also demonstrated remodelling on the bony surfaces of facial bones away from the sutures, and it was claimed that this was due to a direct effect of the "periosteal envelope" which was placed under tension by the extra-oral force.

No consideration was given to the possible altered muscle function (particularly as it relates to muscle orientation) that may result from spatial

alterations to the maxilla, and thus to the insertion points of facial muscles attached to the maxilla. It is conceivable that these changes (although small) may also exert some tension on the periosteum and may contribute to relapse.

These authors found that the degree of relapse was directly proportional to the length of stabilisation and, as all animals exhibited some relapse an assessment of the likely reasons for the relapse (i.e. sutural connective tissue tension, periosteal or muscular effects) would have been valuable. It was found that dental relapse was four times greater than skeletal relapse and, although the authors likened the facial sutures to the periodontal ligament with regards to their similar response to force, no explanation for the difference in relapse was offered.

The review of animal studies has shown that, in monkeys:

1. the maxillary complex can be protracted using anteriorly directed forces;
2. sutural response and maxillary displacement vary with the direction of force and point of application, and are related to the location of the centre of rotation of the maxilla;
3. the effects are not limited to the maxilla but that most facial bones articulating with the maxilla are affected. Some changes in the cranial base angle were also reported (Jackson, Kokich and Shapiro, 1979);
4. stability of achieved changes requires a lengthy period of retention.

6.4.2 Human clinical studies

Dellinger (1978), in relation to the paucity of published studies concerning maxillary protraction in humans, commented:

... it is ironic that we have limited ourselves in maxillary orthopaedics to the lateral components.

He stated that anterior movement of point A with midpalatal suture splitting appliances was well documented and claimed that this was due to separation in the pterygomaxillary fissure, although no evidence to support this was presented.

Dellinger (1973) presented two cases of maxillary protraction using a Hickam chin cup in conjunction with a non-activated palatal expansion appliance. A 6 lb force applied to the maxillary appliance in an 8 year old girl and an 11 year old boy produced an anterior movement of point A of 2.0 mm and 2.3 mm respectively.

Whilst no discussion of the location, direction or stability of these changes was offered, it seems unlikely that growth or remodelling at point A as a result of anterior movement of the dentition could account for this change during the 14 days of active treatment.

Irie and Nakamura (1975) briefly discussed maxillary protracting appliances as an alternative to surgery in cases where both an under-developed maxilla, and an overdeveloped mandible exist. They claimed that correction of the mandible to a deficient maxilla was not satisfactory and that "forward growth of the maxilla at an early age was more desirable"

These authors presented cases treated with a maxillary protracting appliance/chin cup combination and demonstrated (on cephalometric tracings) anterior movement of the incisors, maxillary molars and incisive fossa.

No assessment was made of the movement of other maxillary landmarks, the relative skeletal and dental changes or post-treatment stability. Considerable alterations to the mandibular plane angle were demonstrated, with a downwards and backwards rotation of the mandible.

As the point of attachment of the protraction elastics was the first maxillary molars, and a downwards and forwards movement of the maxillary dentition was demonstrated, there seems to be an indication that significant extrusion of the maxillary dentition occurred. In the absence of a detailed analysis of the movement of other parts of the maxilla, no valid conclusions can be drawn regarding either the location of the changes or the stability of the results in this study.

Nanda (1980) used a modified chin cup-protraction appliance as part of the total treatment regime for 20 patients. He demonstrated anterior displacement of the maxilla of 1-3 mm and maxillary dentition of 1-4 mm.

These changes were assessed cephalometrically by superimposing on sella and measuring to point A and ANS. He claimed that they were due to

1. normal growth and development;
2. protraction headgear;
3. orthodontic tooth movement.

Remodelling at point A associated with anterior movement of the incisors may have also been contributory, although this was not mentioned by Nanda (1980).

Further, he asserted that the cephalometric analysis (by superimposition) indicated the changes were due to:

1. anterior displacement of the maxilla;
2. protraction of the maxillary dentition;
3. flaring of the maxillary incisors;

although no assessment of the relative contribution of each factor was made.

Cozzani (1981) treated eight patients ranging in age from 5 years 2 months to 19 years 10 months with a Delaire face mask and assessed the treatment result on the basis of changes in SNA, SNB and ANB angles. He found that the greatest change occurred in the SNA angle and the change was largest in younger patients. The treatment time varied from 7 months to 36 months and there was no correlation between treatment duration and the magnitude of the change.

As with several other authors, no assessment of the relative contribution of skeletal and dental changes to the overall result was made, nor was the stability of the improvement discussed.

Campbell (1983) treated 11 patients under 12 years of age with a reverse-pull face crib. Four of these patients also had rapid maxillary expansion and one had maxillary expansion in addition to orthodontic appliances.

He assessed the changes in the same way as Nanda (1980) - point A/ANS to sella, and incisor position by superimposition on S.

He found that seven of these patients demonstrated forward movement and/or growth of the maxilla. All had lingual tipping of the lower incisors and the majority (90%) had labial tipping of the maxillary incisors and mesial movement of the maxillary molars. In addition 30% had an increase of mandibular plane to SN angle.

Campbell (1983) asserted that, despite the claims of maxillary advancement by previous authors, proof of this was questionable and described the term relative maxillary advancement to describe the net changes with maxillary protraction treatment. He defined this as

the effective therapeutic change in the anteroposterior relationships among the maxilla, cranial base and mandible.

He claimed that relative maxillary advancement was achieved in growing patients by:

1. Stimulation of anterior growth of the maxilla at its sutural articulations;
2. Bone remodelling at both points A and B;
3. Labial tipping of the maxillary incisors;
4. Lingual tipping of the mandibular incisors;
5. Posterior positioning of the mandible if functional forward positioning is present;
6. Mandibular rotation;
7. Changes in growth vectors resulting from the normalised functional environment.

The evidence gained from animal studies indicates that anteriorly directed forces on the maxilla will result in:

1. increased activity and altered width of maxillary sutures;
2. anterior movement of the maxilla and the maxillary dentition with the direction of movement related to the line of force;
3. a greater degree of movement in young animals;
4. a degree of relapse inversely proportional to the period of stabilisation.

For obvious reasons, histological studies in humans are lacking, and clinical results have shown a range of variation in response to this form of treatment.

Definite proof of maxillary mobilisation and movement is lacking. No accurate method of predicting the precise changes has been determined and long term follow up assessments of stability are unavailable.

It appears that some skeletal modification does occur, but how much of this is due to normal growth is unknown.

It is likely that dental changes, with associated remodelling of point A are a major component in the overall changes.

The evidence from both animal and clinical studies indicates that the greatest response occurs in young patients, particularly during the mixed dentition period.

Although it would be anticipated that most protraction appliances would have a "chin cup" like effect on the mandible, this possibility is rarely discussed.

Campbell (1983) referred to changes in lower incisor inclination and mandibular rotation associated with use of his appliance, but speculation on possible condylar growth effects was absent.

CHAPTER SEVEN

MAXILLARY EXPANSION

Introduction

In the context of Class III treatment, maxillary expansion may be required to correct maxillary arch deficiencies which may be of two types (Haas, 1965):

1. "Real", where the maxilla is deficient in the lateral, antero-posterior or vertical direction, or a combination of all three.
2. "Relative", where the associated crossbite is due to excessive mandibular arch size or to anterior positioning of the mandible to avoid occlusal interferences. These interferences are generally considered to be due to incisor position, although Godfrey (1985) claims that the primary canine teeth may be principally involved in the development of anterior positioning movements of the mandible in many early Class III cases. These interferences may be involved in both "real" and "relative" maxillary deficiencies.

A distinction should be made, at this stage, between maxillary and mandibular bases and their respective dentitions. There are some cases of Class III in which no dental arch changes are required, only basal relocation.

These types are generally associated with major skeletal discrepancies and require surgical treatment and as such are beyond the scope of this treatise.

7.1 Indications

Maxillary expansion may be indicated to orthopaedically alter the

maxilla such that separation of the midpalatal suture occurs.

The widespread effects of this form of therapy have been discussed in Chapter 6 with regard to the circum- and inter-maxillary sutures and protraction. In addition, other effects may be required, such as when midpalatal suture separation results in an increase in nasal volume and thus nasal airflow in cases where oral respiration is considered either a likely aetiological factor in Class III development, or due to a nasomaxillary deficiency.

Selected maxillary arch length problems where extractions are undesirable due to profile considerations and the arch length deficiency is small have also been treated by maxillary expansion (Haas, 1970, Berlocher, Mueller and Tinanoff, 1980). The stability of the resultant corrections have however, been questioned (Cleall, 1974, Timms, 1981).

7.2 Clinical Effects of Rapid Maxillary Expansion

Of particular interest in early Class III treatments are the antero-posterior and vertical effects that may be associated with maxillary expansion appliances using orthopaedic force levels.

Appliance designs such as those in Figs. 7.1 have been shown to produce forces of up to 10 lbs. when expansion of the screw thread (which does not equal bony expansion) is occurring at the rate of 0.4-0.5 mm per day (Isaacson, Wood and Ingram, 1964). This study also demonstrated that the level of accumulated force is related to the age of the patient, with the force level at each activation of the appliance generally being below this level in 8 to 10 year olds.

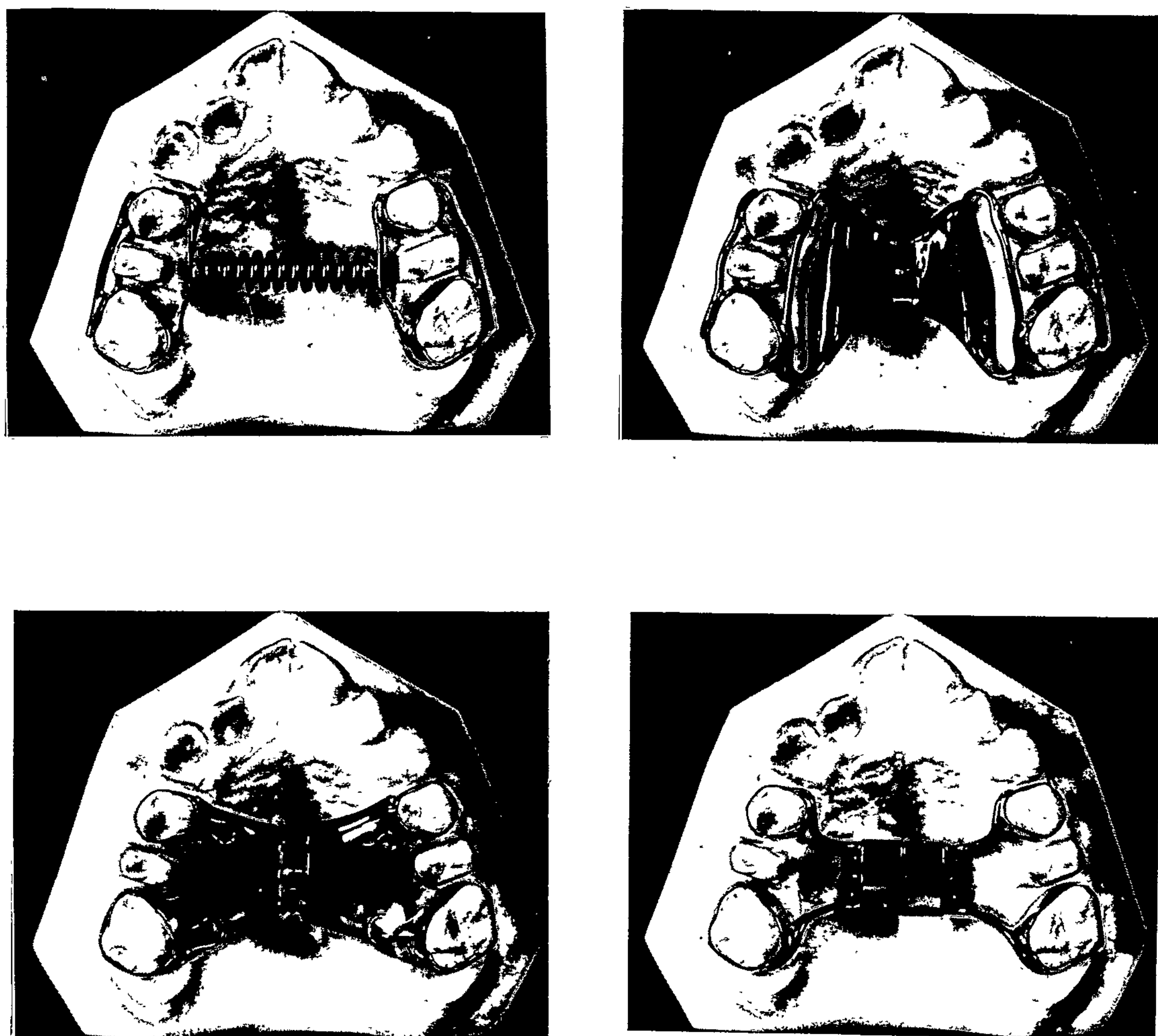


Fig. 7.1 Variations in design of appliances used for rapid maxillary expansion, from Timms (1981).

Separation of the midpalatal suture and an increase in intermolar width is routinely observed in young patients (Haas, 1970, 1980, Chaconas and Caputo, 1982) during active treatment. In addition, downwards and occasionally forwards maxillary movement has been reported (Haas, 1961, 1970, 1973, 1980, Wertz, 1970, Bell, 1982).

Grossman (1970) claimed that forwards and downwards movement of the maxilla was routinely observed with rapid maxillary expansion. Further he stated that molar extrusion occurred, and that this, in conjunction with downwards maxillary movement resulted in a downwards and backwards mandibular rotation and a desirable increase in vertical face height. The anterior movement of the maxilla may also correct the anterior crossbite.

No indication was given as to how the extent of these changes was assessed (whether by skeletal landmarks or movement of the dentition) nor how the extent may be predicted.

Extrusion of the anchor teeth, together with opening of the mandibular plane to SN angle, have also been observed by other authors (Henrikson and Jensen, 1977, Greenbaum and Zacchrisson, 1982) although here too, there is a lack of long term evidence relating to the permanence of these changes.

Dellinger (1973) claimed that there were well documented studies showing anterior movement of point A, and stated that this was due to an increase in the pterygomaxillary fissure distance. No indication was given as to the primary cause of this movement, nor was there offered any explanation as to how a laterally directed force resulted in an anteriorly directed effect.

This effect has been noted by others (Wertz, 1970, Bell, 1982). However, the reason for it has not been explained.

Wertz (1970) observed that, in a study of 60 cases treated by palatal expansion, movement of point A was unpredictable and irregular.

Further, extrusion of the teeth as well as "dropping downward of the maxilla", was routinely observed, with an associated rotational effect on the mandible, point B and the ANB angle, all of which were favourable in deep bite Class III cases.

Like others, this author did not discuss the reasons for the movement of point A or downwards maxillary movement, although he claimed that these changes appeared to be more stable in Class III cases than Class II cases and postulated that this was due to occlusal adaptation.

Sicher (1949) stated that the sutures between the maxilla and its neighbouring bones are oriented in such a way that growth at the sutures would result in a downwards and forwards movement of the maxilla. Whilst the primary role of sutures in facial growth is in doubt, the orientation as described by Sicher (1949) may be a partial explanation for their anterior and downwards maxillary movement sometimes seen with rapid maxillary expansion.

Haas (1961) presented 10 cases treated with rapid maxillary expansion appliances. He found that, at the end of active expansion, point A had moved forward in all cases and downward in 5 cases. During the four month period following treatment point A moved posteriorly 2 mm and SNA decreased 1°. He claimed that

this would seem to indicate that, as the maxillae move laterally, marked activity and adjustment must be occurring at the sutures of the cranial and facial bones with which the maxillae articulate. As sutures open and bones slide, the maxilla is displaced forward and sometimes downward. During the retention period this activity continues toward re-establishing the former proximity of the bones.

There is, however, a degree of discordance of opinions regarding the

frequency with which this is observed. As mentioned, Wertz (1970), Cleall (1974) and Bell (1982) have discussed the irregularity and unpredictability of this change. On the other hand, Haas (1965) states that forward movement of the maxilla is "a phenomenon that always seems to occur in palate expansion".

Detailed analysis of this is absent in the literature as most attention appears to be focused on lateral changes.

7.3 Rapid Maxillary Expansion with Class III Elastics

Anterior movement of the maxillary dentition and/or maxilla has been regularly demonstrated when rapid maxillary expansion is supplemented with vigorous Class III elastics to a fully banded lower arch.

Haas (1970) demonstrated significant change in the position of point A and pogonion subsequent to rapid maxillary expansion and heavy Class III elastics applied to the maxillary molars. Haas (1970) claimed that this caused an anterior movement of the "more mobile maxilla" as well as a downwards tipping posteriorly. In addition, some extrusion of the maxillary molars was observed. There was a resultant downwards and backwards pivotal rotation of the mandible (about the molars) with a decrease in overbite, increase in vertical face height, net distal movement of point B and correction of the anterior crossbite or edge-to-edge incisor relationship. These effects were favourable in deep bite Class III cases, but, he asserted, definitely contra-indicated in open bite Class III's. The latter should, he claimed, be controlled with a vertical-pull chin cup.

This author also demonstrated radiographically, opening of the

pterygomaxillary fissure, which he claimed definitely proved anterior movement of the maxilla.

Grossman (1970) stated that Class III elastics may be used with rapid maxillary expansion for Class III correction, but no other details were given.

Teuscher (1977) claimed that rapid maxillary expansion with intra-oral (and other) forces may cause anterior and downward displacement of the whole (maxillary) complex. This author also claimed that this technique was contra-indicated where no overbite existed.

Adams, Meikle, Norwich and Turpin (1972) demonstrated anterior maxillary movement with Class III elastics to maxillary splints (non-active). They claimed that this was due to a combination of sutural, periodontal and temporomandibular joint remodelling.

Haas (1980) demonstrated similar results to his of 1970, and claimed that, in one case, vertical and anteroposterior corrections were stable after 16 years.

7.4 Clinical Application of Rapid Maxillary Expansion in Class III Malocclusions

Whilst it appears from this review that anteroposterior corrections of Class III malocclusions can be made using rapid maxillary expansion and auxiliaries there is very little specific information concerning

1. the relative contribution of dental and/or skeletal movement;
2. long term stability of skeletal alterations - particularly anterior and vertical;

3. the effect on the mandibular dentitions with respect to
 - a. anteroposterior movement of the dentition as a whole
 - b. extrusive effects as the result of Class III elastics.

In addition, diagnostic problems (discussed in Chapter 4) have to some extent restricted the clinical application of some of these appliances. The lack of consistent diagnostic criteria in the literature used to determine the most appropriate treatment regime for each particular case is probably a significant factor in the relatively limited usage of orthopaedic techniques such as those previously described.

7.5 The Fränkel Appliance

Modification of the maxilla and its dentition has been claimed by Fränkel (1970, 1972) using the function corrector - FRIII.

Fränkel (1970) examined the use of the FRIII and FRII (for Class II cases) in two age groups - to to 7 years and 11 years of age.

He found that the reference points nasospinale, A point, posterior nasal spine and the upper first molar changed their position anteriorly more in the Class III cases than they did in a posterior direction with the Class II cases. In addition slightly greater changes were observed in the older age group than the younger age group. He concluded that the results demonstrated that anterior repositioning of the maxilla was possible with this appliance. He postulated that this may be associated with forward traction on the maxilla by the perioral musculature, displaced anteriorly by the appliance and, via the periosteal attachment to the bone, stimulating forward maxillary growth. The stability of the axial inclination of the upper incisors in the majority of cases, he claimed, supported this view.

In this study, changes were assessed radiographically by transferring the marked landmarks on transparent foil (superimposed on the "occipital base - the anterior edge of the occipital condyles") to subsequent lateral head radiographs. The limits of error with this technique were claimed to be less than 1.0 mm. As the measured changes over the ten month period were, in most cases less than 2 mm the percentage error of the total measured change may have been considerable. This may cast some doubt on the clinical significance of the results.

Further, there was no distinction between functional and true Class III's so accurate assessment of mandibular effects is difficult.

Correction of the anterior crossbite was the criterion of successful treatment and, whilst this is a reasonable objective, it does not necessarily relate to skeletal effects of the appliance. On this latter point, Fränkel (1970) stated that the investigation demonstrated that aggravation of antero-posterior discrepancies may be prevented and did not claim correction of skeletal dyscrasias was necessarily possible. Fränkel (1980) claimed that lateral expansion of the maxillary arch has been frequently observed with this appliance, apparently as the result of tension on the periosteum due to buccal displacement of the soft tissues of the cheek by the vestibular shield. The extent of basal involvement in this expansion and the post treatment stability of the expanded arch were not examined by that author.

CHAPTER EIGHT

DISCUSSION

It is apparent that Class III malocclusions present difficulties, not the least of which is their treatment.

8.1 Classification

The absence of a satisfactory method of classification in widespread use creates difficulties in assessing the significance of treatment responses to various treatment regimes, and identifying the most suitable treatment for individual cases (based on the advocacies of various authors).

8.2 Aetiology

Considerable confusion exists regarding the aetiology of Class III malocclusion. This stems partly from its immense variability although other factors such as the multifactorial nature of the skeletal abnormalities compounds this problem.

It does appear that the maxilla is a contributory factor in the development of Class III malocclusion in more cases than is generally thought (Bimler, 1970). Reduction in the size of the maxilla in the anteroposterior direction or a retardation of the anterior displacement of the maxilla during growth (or a combination of both) may be involved.

Reduced size of the maxilla has been linked to abnormal muscle function (Graber, 1966, Frankel, 1972), size or position although the cause and effect relationship is not understood.

Retarded anterior displacement of the maxilla may be associated with growth disturbances such as premature ossification of the nasal septum and anterior cranial base cartilage or the formation of synostoses in the maxillary sutures. These may in fact all be interrelated and it is thought that they may develop as mild forms of syndromes (such as Crouzon's or Apert's) which exhibit more severe midfacial deficiency (Bimler, 1970, Stewart and Moore, 1982).

The underlying cause of the growth disturbance is unknown although it seems likely to have a genetic basis which could predispose to the early onset of osseous change in these specialized cartilagenous tissues. No work has been done to examine the possibility of associated effects occurring in epiphyseal cartilage.

A genetic basis would explain the early appearance of Class III features which is routinely observed (at the time of eruption of the deciduous incisors or earlier) (Leighton, 1969).

Mandibular prognathism is similarly, a confusing entity.

Research on condylar growth has recently attributed to it a compensatory role rather than a primary one (Gianelly and Moorrees, 1965; Moss, 1969; Enlow et al., 1977). This is generally supported, particularly by proponents of Moss's functional matrix theory, although a small group consider that the condyle possesses a genetically predetermined growth potential (Mills, 1983), the expression of which may be influenced by environmental factors (Fränkel, 1969). In addition it has been recently demonstrated that "Class III mandibles" differ from normal more in morphology than size, although there is frequently a small size difference (Jacobson et al., 1974).

The occasional case where the mandible grows beyond the apparent functional constraints of the facial "matrix" are inexplicable on the basis of compensatory mandibular or condylar growth. There appears to be a primary driving force in some area or areas that is programmed or stimulated to "abnormal growth". In the absence of pathology, this abnormal growth may in fact be normal for these individuals and this concept (of "normal" growth) may have important ramifications with regard to treatment.

An important factor in treatment planning with Class III's is estimating the final outcome of growth, based on the presenting features - particularly of the young patient. In the ideal situation, growth prediction could be used to justify early treatment and to accurately assess treatment-induced and growth changes. Currently used methods of growth prediction are at best approximations, with the expected error increasing in younger patients (Ackerman and Proffit, 1970; Björk and Skieller, 1983).

8.3 Chin Cup Appliance

Clinical studies of chin cup appliances indicate that it is possible to achieve changes, although the most consistent and reproduceable of these, lower incisor retroclination (Armstrong, 1963; Thilander, 1965; Kloeppel, 1970; Vego, 1976) is unlikely to be adequate to correct many skeletal disharmonies.

Skeletal changes have been regularly reported (Suzuki, 1972; Graber, 1975; Irie and Nakamura, 1975; Sakomoto, 1979) although the significance of these is in doubt for a number of reasons.

1. The majority of animal experiments involved creating Class II malocclusions in normally growing animals. Comparison with abnormal situations may not be valid especially considering the phylogenetic

differences involved.

2. Few clinical studies standardised patients with regard to specific morphological features, thus extrapolation of results to Class III's in general may not be accurate.

3. Treatments were never randomly assigned and thus operator preference and experience must be considered a factor in the treatment result. This is rarely considered in the literature.

Chin cups are probably best indicated in cases which exhibit the following features.

1. Class I or very mild Class III (ANB 0° or greater);
2. anterior functional mandibular shift;
3. lower incisors not retroclined and upper incisors not proclined;
4. minimal lower incisor crowding, preferably spacing;
5. minimal negative overjet;
6. good co-operation.

As previously mentioned, some skeletal changes have been observed with this appliance. In general these were associated with very long treatment periods (up to 7 years, Graber, 1975; Sakomoto, 1979) with maximal daily periods of use up to 24 hours per day in some cases.

The very possibility of achieving skeletal changes poses a dilemma: does withholding chin cup therapy sentence patients to surgery at a later date?

It has been claimed that true Class III malocclusion is progressive in nature and thus could be reasonably expected to deteriorate with growth.

The high demands on patient co-operation over long periods and the difficulty of predicting treatment effects must be weighed against the possible psychological and social effects of prognathism.

In reality only the most severe cases request surgery, a number are talked into surgery whilst many more elect no surgery. Further, it is not yet possible to predict whether chin cup therapy even when commenced at an early age will eliminate the need for surgery. It frequently acts as a pacifier with the appearance of "something being done" easing the minds of both parent and clinician.

In summary, there does not appear to be a sound scientific basis to justify the use of chin cups in true skeletal Class III malocclusions. With the high demands on patient co-operation, the appliance should be used in cases where success can be reasonably assured - that is, where correction is primarily required by dento-alveolar changes.

8.4 Maxillary Protraction

Maxillary protraction appliances have only recently been examined under formal experimental conditions. Consequently data relating to their effectiveness is sparse and the results inconclusive.

Animal studies have regularly demonstrated skeletal alterations, primarily centred in the maxillary sutures (Kambara, 1977; Nanda, 1978; Nanda and Hickory, 1984). The nett changes were found to relate to:

1. the point of application;
2. the direction of the force vector;
3. force magnitude;
4. period of retention.

One study showed that dental changes were initially greater than skeletal changes, although dental relapse was four times greater than skeletal relapse (Jackson, Kokich and Shapiro, 1979).

Human studies have demonstrated anterior movement of both the maxilla and the maxillary dentition, although the extent of maxillary movement and the location of skeletal changes are uncertain and inconsistent (Dellinger, 1978; Nanda, 1980).

Reverse pull appliances must also have a chin-cup-like effect although scant mention is made of this in the literature.

Again, Campbell (1983) mentioned alterations in lower incisor inclination and mandibular plane angle (with mandibular rotation). No reference is made to possible effects on condylar growth or long term changes in mandibular morphology or rotation.

In summary, it is apparent that:

1. maxillary sutural changes can be induced with these appliances, but the extent, geometry and stability are either difficult to predict or unknown;
2. greater sutural effects were observed in young animals and patients;
3. dental effects will be more stable if anterior crossbite correction is achieved and a good interlocking occlusion is present;
4. the overall treatment results are likely to be due to a combination of effects - maxillary sutures, dentition, mandibular positional retrusion and "normalised" growth environment.

The criteria for selection of this mode of treatment may be listed as

1. a mild real maxillary deficiency with a negative overjet of less than 2 mm;
2. minimal dental compensation existing;
3. forward postural positioning of the mandible exists;
4. where sufficient anchorage exists in the maxillary arch.

Maxillary protraction must be considered one phase of the overall treatment plan and treatment periods must be short (7-8 months) so as not to jeopardise co-operation in further treatment.

8.5 Maxillary Expansion

Rapid maxillary expansion has been shown to produce skeletal changes beneficial to many Class III's (Haas, 1961; Wertz, 1970; Bell, 1982). These effects appear to be potentiated by heavy Class III elastics.

Associated effects such as molar and lower incisor extrusion, downward mandibular rotation and root resorption of anchor teeth should be considered before using this appliance (Henrikson and Jensen, 1977; Greenbaum and Zacchrisson, 1982).

Where the overbite is shallow, the appliance is contra-indicated unless the bite opening effect is controlled by the use of a vertical pull chin cup (Haas, 1970; Sassouni, 1972). Obviously this increases the complexity of the treatment, with the attendant effects on patient co-operation.

8.6 Fränkel Appliance

There is little in the literature to support the use of Fränkel appliances in Class III cases. It appears that this is not because no changes can be achieved but rather, the same changes can be attained using other

appliances that are less complex to construct and less susceptible to breakage.

8.7 Overview

The discussion on the latter two appliances has been brief as it is apparent that selection criteria for all "Class III type" appliances have similarities.

Of paramount importance is patient co-operation. This applies not only to the success or failure of early treatment, but to the likelihood that in most Class III malocclusions some permanent dentition treatment will be required. Hence treatment in the deciduous or mixed dentition should be short (less than 12 months), atraumatic and be judged to have a high chance of success.

Skeletal discrepancies must be mild as a review of the literature reveals that corrections occur primarily as the result of dental changes unless the appliance is used for the entire growth period.

The appliances all have some untoward "side-effects" and careful consideration should be given to these. In addition careful monitoring during treatment is essential to ensure that improvement rather than deterioration occurs.

Correction of the anterior crossbite and elimination of abnormal anterior mandibular movements are the prime requirements for early Class III treatments. In addition, reduction of the severity of skeletal dysplasias is desirable.

There is evidence to support the view that the former is possible with the appliances herewith reviewed and if the guidelines for selection of treatments are adhered to, a high level of success may be expected.

Reduction of the skeletal discrepancy, if mild, may not be required to achieve dental corrections. If severe, it seems unlikely that a difference to growth outcome or surgical difficulty will occur that is of a magnitude to justify the effects on patient co-operation of very long treatment periods and to balance the uncertainty of treatment outcome.

The final, but by no means least, problem in the area of early orthopaedic treatment is that of judging "successful" treatment.

The most widely used criterion is that of anterior crossbite correction, and, when present, elimination of forward postural positioning of the mandible (Irie and Nakamura, 1975; Nanda, 1980; Campbell, 1983).

Whilst these are reasonable treatment objectives, successful treatment should have broader implications - particularly with respect to long term effects.

The acute problems associated with primary canine and incisor interferences on the teeth themselves and postulated problems of abnormal dentoalveolar development, aberrant muscle activity and temporomandibular joint pathology may be enough to "justify" early treatment in those cases where correction seems assured.

The objective of permanent alterations to bone morphology and growth rate, which are widely proclaimed as the "reason" for this form of

treatment, are well documented but inadequately supported. In the few cases where permanent changes have been observed, treatment throughout the entire growth period was required.

So, in this well-worked area of early treatment, confusion reigns supreme.

Uncertainty exists about the classification of Class III's, aetiology, selection criteria for the various appliances, assessment of treatment effects and the adequacy of the term "successful treatment".

In the overview, however, it does seem that the appliances herein reviewed have a place in the orthodontic armamentarium.

Selection of orthopaedic techniques should be carefully balanced against the potentially deleterious effects on co-operation and, when used, be considered as only one phase of the overall orthodontic therapy for the child's problem.

CHAPTER NINE

SUMMARY AND CONCLUSIONS

In order to evaluate the hypothesis the literature was reviewed with respect to classification, aetiology and diagnosis of Class III malocclusions in the deciduous and mixed dentitions.

It is apparent that considerable confusion exists in all areas, in particular:

1. The great variation in skeletal morphological features and growth patterns make accurate classification of Class III types difficult.
2. This variability makes identification of specific aetiological factors impossible in the majority of cases.
3. In consequence, the allocation of specific forms of therapy and prognostic evaluations to specific clinical diagnoses is difficult, and an area neglected in the literature.

The aetiology of Class III malocclusions is complex, involving effects on skeletal, dental and soft tissue components by both genetic and functional/environmental influences. It is evident that a strong genetic component exists in many Class III malocclusions, although the mode of expression and transmission, is unclear.

Currently proposed theories of condylar growth and maxillary displacement mechanisms are of no great value in identification of aetiological factors or assessment of treatment effects for several reasons:

1. The use of animal models, human autopsy material and human patients in which pathological states or congenital absence of tissues exist is of dubious value in assessing the mechanisms of "normal" growth (in the absence of pathology).

2. Whilst several popular growth models have been proposed, sufficient refuting evidence exists to deny the model's total support. The use of a hypothetical situation as the basis for proving a mechanism or process is not in accordance with scientific protocol.

Chin cup appliances were examined and it was found that:

1. The majority of operators use force levels of 500-600 g per side with the line of force directed through or just superior to the condyle, applied for periods ranging from a minimum of 12 to a maximum of 22 hours per day.

2. The appliance is most frequently used as a prelude to fixed orthodontic therapy, either intermittently or full time throughout the growth period.

3. The only consistently reported effect was lower incisor retroclination, although decreases in gonial angle were frequently observed. Some more extensive morphological changes have been reported in cases where the appliance is worn 22 hours per day.

4. Many studies on the effectiveness of the chin cup demonstrated a lack of control or identification of variables such as skeletal type and co-operation. Further, selection criteria for use of the appliance were rarely presented.

Formal investigations into the use of maxillary protraction appliances have to date been based largely on animal experiments and little clinical research has been done.

In animals, skeletal alterations have been achieved and the extent and direction of these changes are influenced by

1. the point of application of force;
2. the direction of the force;
3. the magnitude of the force;
4. the period of retention.

It appears that the greatest effects are observed in the anterior movement of the dentition, although considerable relapse occurred in both dental and skeletal changes.

Clinically, the appliances were most effective in the correction of anterior crossbites in cases where mild skeletal and dental discrepancies existed.

The major problems with use of the appliances were related to prediction of the direction of movement of both the dentition and maxilla, and the form and duration of post-treatment retention.

Consistent dental and orthopaedic changes have been reported with rapid maxillary expansion and the dental effects seem to be potentiated by the use of heavy Class III elastics.

Molar extrusion and downwards movement of the maxilla result in a downwards and backwards rotation of the mandible with an associated reduction in overbite, or the development of anterior open bite.

The use of the function regulator of Fränkel, the FRIII, has been advocated for the improvement of the muscular environments of the jaws, and some dental and skeletal changes have been demonstrated with its use.

The principal disadvantages of the appliance are

1. susceptibility to damage;
2. heavy dependence on operator experience.

Conclusions

The deciduous and mixed dentition Class III malocclusion presents a difficult clinical problem for the orthodontist.

The complex aetiology and great variability make categorisation of types difficult, and this has ramifications with regard to appliance selection.

There is a lack of solid scientific evidence to support the contention that mandibular growth can be regularly inhibited or significantly redirected by methods that are of a force magnitude and duration which will not jeopardise the long term patient co-operation and are consistent with the social well being of the patient.

Dental changes have, however, been regularly reported and the appliances herein reviewed may be of benefit in certain situations.

In general, these appliances are indicated when correction by dento-alveolar changes are desired and may be especially effective in postural Class III malocclusions.

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